Advances in Neurosurgery 8



Surgery of Cervical Myelopathy

Infantile Hydrocephalus: Long-Term Results

Edited by W. Grote M. Brock H.-E. Clar M. Klinger H.-E. Nau

With 178 Figures in 215 Separate Illustrations and 138 Tables

Springer-Verlag Berlin Heidelberg New York 1980 Proceedings of the 30th Annual Meeting of the Deutsche Gesellschaft für Neurochirurgie Essen, September 16-19, 1979

ISBN-13: 978-3-540-09949-9

e-ISBN-13: 978-3-642-67605-5

DOI: 10.1007/978-3-642-67605-5

Library of Congress Cataloging in Publication Data. Main entry under title: Surgery of cervical myelopathy - infantile hydrocephalus: long-term results. (Advances in neurosurgery; v. 8) "Proceedings of the 30th annual meeting of the Deutsche Gesellschaft für Neurochirurgie, Essen, September 16-19, 1979." Bibliography: p. 1. Spinal cord-Surgery-Congresses. 2. Vertebrae, Cervical-Surgery-Congresses. 3. Hydrocephalus in children-Surgery-Congresses. 4. Cerebrospinal fluid shunts-Congresses. 5. Nervous system-Surgery-Congresses. I. Grote, Wilhelm. II. Deutsche Gesellschaft für Neurochirurgie. III. Series. [DNLM: 1. Spinal cord diseases-Surgery-Congresses. 2. Hydrocephalus-Surgery-Congresses. 3. Hydrocephalus-In Infancy and childhood-Congresses. W1 AD684N v.8 / WL400 D486s 1979] RD594.3.S93 617'.482 80-13685.

This work is subject to copyright. All rights are reserved, whether the whole or part of the material is concerned, specifically those of translation, reprinting, re-use of illustrations, broadcasting, reproduction by photocopying machine or similar means, and storage in data banks.

Under § 54 of the German Copyright Law, where copies are made for other than private use, a fee is payable to the publisher, the amount of the fee to be determined by agreement with the publisher.

© by Springer-Verlag Berlin Heidelberg 1980

The use of registered names, trademarks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

2122/3140-543210

Welcoming Address W. GROTE

Ladies and Gentlemen, dear colleagues, we wish you a happy welcome, particularly to our Italian colleagues and their wives under the leadership of the President of the Società Italiana di Neurochirurgia, Professor Papo, who accepted our invitation for this friendly get-together and who were not averse to making the long trip into the heart of the Ruhr region. I am delighted that you make it possible, in this way, for us, to return the compliment of our visit to Taormina in 1972 and the generous hospitality associated with it. I am particularly pleased that during the coming days I can try to offer you a scientifically interesting and also a happy and eventful stay in a warm and friendly atmosphere. My great handicap in this regard is clear to me from the outset, for, who here can offer anything comparable to the landscape of Taormina or to the charm of southern countries? Even so, I will not allow myself to be discouraged und will attempt to convey to you something of the magic of our landscape, and try to convince you that in the meantime Germanic hospitality has undergone a maturation process. We are all delighted that you will be spending some time here with us and we earnestly hope that you will feel at home in our community. Of course, no one should be forced into a friendship, but rather a willingness to enter such a relationship must be present in order that it may flourish at all. We should open our hearts to one another and it should not prove difficult to experience friendship over and above the mutual respect we feel for each other, especially since we are all bonded together by our love to our difficult and often very burdensome profession.

This is naturally true not only in regard to our behaviour towards our Italian colleagues but applies to all of us, for how can we prevail in our profession, in the long run, without the knowledge that we have many friends around who will support and help us in time of need? And what neurosurgeon does not need the advice and assistance of a friend much of the time? In order to ensure that personal encounters, personal conversations and simple personal contacts can take place, we have consciously arranged this congress so that, when the morning's scientific programme is over, the rest of the day will be at the disposal of the participants for cultivation of the personal sphere. Ladies and gentlemen, you naturally have the right to relax and rest after the morning's scientific work and I trust that the so-called Additional Programme will find favour with all of you.

The scientific sessions in the mornings will be concerned with two important disease patterns. On Monday *Diagnosis and Therapy of Cervical Myelopathy* will be reported and discussed, and on Wednesday the *Long Term Results* attained in the *Operative Treatment of Hydrocephalus in Children*. Tuesday will be devoted to open discussion and it is expressly hoped that younger colleagues will avail themselves of the opportunity to

present scientific work to the assembly. In this context, no upper limit will be set upon the age of youth, though I know that some really young colleagues will take the floor.

I extend my best wishes to all departmental heads in their desire to improve our knowledge and increase our understanding. I expect from them, however, the courtesy of keeping within the imposed speaking time limits though this appeal for fairness towards colleagues who appear later on in the programme seems to me to be unnecessary in view of the exemplary team of speakers we have here.

This meeting, which I now declare open for those who need such an official statement, has two special features. One is that it involves a friendly get-together with our neurosurgical colleagues from Italy and the other is that it represents the 30th Annual Meeting of the German Society for Neurosurgery. I have said something about the friendly get-together at the outset. Now let me make some observations on the 30th Annual Meeting which also falls at this time. This still youthful but certainly marriageable age arises from the fact that the Founding Assembly, originally planned from 6th to 7th October 1939 in Würzburg, could not take place because of the outbreak of war. Only subsequent, post-war attempts led to the official foundation of the *German Society for Neurosurgery* in Bonn on September 13th, 1950. In the account of that first gathering it is reported that seven members were present or represented. This little clan has since developed itself single-mindedly and has multiplied itself today into a troop of over 200 ordinary and over 100 extraordinary members, to which 87 corresponding members may be added.

This explosive development of our Society more or less corresponds with the development and grandiose extension of the discipline of Neurosurgery throughout the world. Our general knowledge of neurosurgical ailments and the possibilities for treatment which have resulted therefrom have, in recent decades, witnessed substantial and sometimes decisive progress. This is so even if new knowledge sometimes leads to new questions which will have to be answered in the future. Neurosurgery has certainly experienced a maturation although it is very far from being complete. The problems still to be solved appear infinite in number, and although we are urged by our zeal, our ambition and our medical duty oblige us to try to throw some light on still outstanding problems. the keenness of our younger collaborators, to work together on problems as yet unsolved, is admirable and their sense of mission is so strong that they are prepared to renounce personal things. The fruits of their labours are then hawked around at congresses such as this, and found to be edible, especially delicious or even worm-eaten and rotten. I very much hope – indeed I am certain – that during the coming days we shall be offered aromatic and tasty fruit.

Normally, a special symbol is chosen for a congress and we too have endeavoured to find something suitable for this gathering. We believe that KOSMAS and DAMIAN, the patron saints of this city, who were doctors and apothecaries, are particularly apropriate in this role. There are numerous pictures of these two saints and, at our suggestion, a draughtsman in our clinic has created a further representation which will perhaps meet with your approval. Here the two patrons appear in mediaeval-modern garb combined together with neurosurgical emblems such as that of trepan as a symbol of operative activity on the nervous system. This is supposed to indicate the nerve cells. So now you know what the two gentlemen are up to as they hold before them the annointing urn and snake. The connection of the city patrons of Essen with neurosurgery should also exemplify the intimate connection of the city with the Neurosurgical Clinic of its University. Actually, the city of Essen is largely responsible for the fact that, some 11 years ago, neurosurgery could come into existence at all. This, respected Senior Mayor, implies, at the same time, a hearty "thank you" to you and your city.

Now I have arrived at the thanking stage. I should like to thank you, Madam Minister Huber, for making the time to come to us today and for wanting to greet our guests. I thank you, Rector and Dean, for tendering the greetings of the University and the Medical Faculty, and I should like to thank you, Madam Mayor Möller-Dostali, for welcoming us in the name of the city of Essen and, additionally, for your willingness to support our meeting in manifold different ways and for your invitation to a reception given by the city of Essen this evening.

Thanks to this we shall be able to go directly to the reception at the end of the offical welcoming addresses.

Before this happens, however, may I express my special thanks to the director of the Folkwang Museum, Professor Vogt, for making available to us this evening all of the rooms in his museum, and for permitting us to use these for subsequent receptions and viewing. The bandmaster, Mr. Scheytt, and the members of the Collegium Musicum – our medical colleagues, reinforced today by members of the Folkwang School, should also be heartly thanked for their willingness to make this opening ceremony more festive and beautiful with their music.

I wish you all an enjoyable evening and hope that you all will have many enriching experiences during the course of our scientific meeting and friendly get-together, as well as happy hours in a friendly atmosphere, and that you will also come by much new knowledge.

Contents

Cervical Myelopathy

H. KUHLENDAHL: Cervical Myelopathy	3
H. VOGELSANG and R. C. SCHMIDT: Indications, Results, and Adverse Effects of Cervical Myelography with Amipaque (Study on 300 Examinations)	8
K. SARTOR, DK. BÖKER, N. FRECKMANN, and R. SCHÄFER: Computed Tomography in Spondylogenic Narrowing of the Cervical Spinal Canal	13
HE. NAU, J. LIESEGANG, P. HIEDL, and K. ROOSEN: Electromyographic (EMG) Investigations in Cervical Myelopathy	22
J. SCHRAMM: Clinical Experience with the Objective Localization of the Lesion in Cervical Myelopathy	26
HD. HERRMANN and H. JASCHKE: Measurement of Spinal Elastance in Patients with Spondylogenic Narrowing of the Spinal Canal	33
H. ALTENBURG, G. SITZER, W. WALTER, and G. BRUNE: Indication for Decompressive Laminectomy in Cervical Myelopathy	38
B. KUGELGEN, K. LIEBIG, and W. HUK: Neurological Approach to Differential	
Diagnosis and Indication for Surgery in Chronic Cervical Myelopathy A. FANTIS: Spinal Symptoms Accompanying Cervical Root Compression	41
A. FRANCO and B. MATRICALI: Anterior Disc Surgery in Cases of Cervical	47
Myelopathy	50
the Cervical Vertebral Body	53
Approach in Chronic Cervical Myelopathies?	59
C. ARIENTA, G. DE BENEDITTIS, L. INFUSO, and R. VILLANI: Operative Results in the Treatment of Cervical Spondylotic Myelopathy	64
K. ROOSEN and W. GROTE: Late Results of Operative Treatment of Cervical Myelopathy	69
G. DALLE ORE and C. VIVENZA: Cervical Spondylotic Myelopathies: Long-Term Results of Surgical Treatment	78
M. SUNDER-PLASSMANN and F. ZAUNBAUER: Long-Term Follow-Up After Surgery for Chronic Spondylogenous Myelopathy	83
F. REALE, D. GAMBACORTA, G. B. SCARFO, and G. P. CANTORE: Cervical Myelopathy Due to Spondylosis and Disc Protrusion: Operative Results in 70	
Patients	86

HU. THAL, H. MILTZ, W. J. BOCK, and H. KUHLENDAHL: Long-Term Results	
After Operative Treatment of Cervical Myelopathy by Laminectomy	90
J. HAMER and M. KAHL: Long-Term Results After Decompressive Laminectomy	
in Cases of Multisegmental Cervical Spinal Stenosis	95
K. LIEBIG, B. KUGELGEN, D. HOHMANN, and W. HUK: Results of the Treatment	
of Patients Affected by Chronic Cervical Myelopathy by Surgical Decom-	
pression and Ventral Fusion According to Cloward	100
B. GUIDETTI, A. FORTUNA, C. ZAMPONI, and P. P. LUNARDI: Cervical	
Spondylosis Myelopathy	104
D. FISCHER, H. D. HERRMANN, and F. LOEW: Chronic Spondylogenic	
Myelopathy: Analysis of Data of 62 Patients Operated on by the Anterior	
Approach	112
E. HAMEL, R. A. FROWEIN, and A. KARIMI-NEJAD: Classification and Prognosis	
of Cervical Myelopathy	115
T. DEMIREL, M. MAKSOUD, and W. BRAUN: Therapeutic Results Following	
Laminectomy and Dural Enlargement in Cases of Cervical Myelopathy	120
B. WILLIAMS: Surgery for Vertebrobasilar Ischaemia in Cervical Spondylosis	122
P. GRUSS, W. GRUNINGER, and FR. ENGELHARDT: Differential Therapy of	
Cervical Radiculopathy and Myelopathy in Degenerative Changes of the	
Cervical Vertebral Column	130
I. SCHOTER and J. WAPPENSCHMIDT: The Value of Computed Tomography for	
the Diagnosis of Spinal Lesions	138
P. C. POTTHOFF: Anterior Cervical Steel Plate Spondylodesis in Mobile or	
Unreducible Traumatic and Osteolytic Cervical Dislocations	144

Hydrocephalus in Childhood

R. HEMMER: Long-Term Results in the Operative Treatment of Hydrocephalus in	
Children	155
R. SCHÖNMAYR, J. ZIERSKI, and A. L. AGNOLI: CT Follow-Up of Hydrocepha-	
lus in Children	164
C. SPRUNG and TH. GRUMME: CT Images of Periventricular Lucency (PVL) in	
Various Forms of Hydrocephalus	172
H. COLLMANN, W. MAUERSBERGER, and G. MOHR: Clinical Observations and	
CSF Absorption Studies in the Slit Ventricle Syndrome	183
A. Ambrosio, L. Benvenuti, E. Bianchi, S. Briani, G. Cagnoni,	
A. Carteri, M. Colangelo, M. Fontana, S. M. Gaini, M. Gerosa, R.	
Giuffre, G. Liguori, I. L. Longatti, G. Luccarelli, C. Mazza,	
F. Migliavacca, A. Moise, E. Occhipinti, L. Palma, A. Pasqualin,	
G. PEZZOTTA, G. TOMEI, and R. VILLANI: Cooperative Study: Long-Term	
Results of the Operative Treatment of Hydrocephalus in Children	187
F. ZAUNBAUER, K. GLONING, E. HIFT, W. KOOS, and M. SUNDER-PLASSMANN:	
Investigations of Factors Influencing the Development of Hydrocephalic	
Children	212
M. KLINGER, G. GROHMANN, W. HAUBNER, S. KUNZE, and H. ELM:	
Long-Term Results of Shunt Operations over a Period of 10 Years	217

J. LIESEGANG, E. W. STRAHL, and H. R. STREICHER: Complications Following Shunt Operations in Children	222
B. RAMA and O. SPOERRI: Surgical Treatment and Long-Term Results in Children with Hydrocephalus	227
K. TORNOW, W. PIOTROWSKI, and H. G. LENARD: Behavior of Hydrocephalus after Shunt Procedures	231
P. C. POTTHOFF: Early and Late Mortality Following Shunt Procedures in Early Infancy	235
E. W. STRAHL, M. DUCHTING, H. C. NAHSER, and H. E. NAU: Long-Term Follow-Up Studies in Hydrocephalus Patients with Spina Bifida or Encepha-	
locele	247
Ventriculo-Cardiac Shunt Material	252
Insertion	255
H. ARNOLD, F. BLAKER, and B. HILLENKAMP: Immune Complex Disease Associated with Chronic Infection of Ventriculoatrial Shunts	263
V. REINHARDT and HE. NAU: Histological Investigations and Clinical Considerations on Shunt Dysfunctions	267

Free Topics

H. W. PIA and R. LORENZ: Transoral-Transpalatine-Transclival Approach to Aneurysms of the Vertebral and Basilar Artery	275
B. WILLIAMS: Investigations on the Pathogenesis of Syringomyelia	277
J. GILSBACH and H. R. EGGERT: Technique and Results of the Cervical Discectomy	284
H. BAUMANN, M. SAMII, and K. VON WILD: Contribution to Ventral Microsurgi-	
cal Foraminotomy in Case of Cervical Nerve Root Compression	287
W. GRUNINGER and P. GRUSS: The Influence of the Cloward Fusion Operation	
on the Motility of the Cervical Spine	291
P. DISTELMAIER, I. VLAJIC, and J. WAPPENSCHMIDT: Discitis After Discography.	297
S. GIOMBINI and C. L. SOLERO: Considerations on 100 Anterior Cervical	
Discectomies Without Fusion	302
F. COLOMBO, A. ALEXANDRE, D. CURRI, and A. BENEDETTI: Posterior Selective	
Spinal Thermorhizotomy for Control of Intractable Pain Syndroms	308
B. GUIDETTI, S. MERCURI, and R. VAGNOZZI: Intramedullary Ependymomas:	
Long-Term Results of Surgical Treatment	313
J. SZANTO: Diagnostic Problems of Neurosurgical Interest in Tick-Borne	
Encephalitis	318
T. WALLENFANG, J. BOHL, K. KRETZSCHMAR, and M. MAYER: Experimental	
Investigation on the Development of Brain Abscess in Cats	321
A. SCUCCIMARRA, V. PENSABENE, N. PANDOLFO, and F. DE BLASI: Study of	
Various Types of Experimental Brain Edema Using the Electrical Impedance	
Technique and the Electron Microscope	332
M. GAAB, H. A. TROST, I. HAUBITZ, K. W. PFLUGHAUPT, and E. HALVES:	
Osmoregulation, Brain Damage and Prognosis	337
	551

M. BORTOLUZZI and G. MARINI: Brain Ischemic Disorders: Role of the Perfusional Brain Scanning in the Neurosurgical Diagnosis	347
I. PAPO, G. CARUSELLI, M. SCARPELLI, and A. LUONGO: Intracranial Pressure	
Time Course in Massive Ischemic Brain Infarction	352
Anastomoses	356
Brain Before Surgical Treatment of Internal Carotid Artery Lesions W. HUK: Treatment of Vascular Malformations of the Head with Detachable	363
Balloon Catheters	366
of Computer-Tomography: Report of Eight Cases	368
Cognitive Defects	374
Cerebro-Spinal Fluid After Osmotherapy with Sorbitol in Neurosurgical Patients	381
K. E. RICHARD and R. A. FROWEIN: Significance of VF-Lactate Analysis in Lesions with Increased Intracranial Pressure in Respect to the Prognosis	386
M. A. GEROSA, A. OLIVI, P. L. LONGATTI, L. MIETTO, S. TEOLATO, and A. CARTERI: Longitudinal Investigation on CSF Levels of Cyclic Nucleotides and Adrenergic Metabolites in Non-Neoplastic Hydrocephalus	394
DK. BOKER: Immunoelectrophoretic Studies on Human Intracranial Tumors G. PENDL and W. KOOS: Microsurgery of Brainstem Tumors in Childhood and	400
Adolescence: A Review of Past Experience	403
Tumours	409
Thermal Rhizotomy Without General Anaesthesia	414
Device	419
Pathophysiology of Chronic Subdural Haematoma	421
After Experimental Denervation and Microsurgical Secondary Suture of the Peroneal Nerve in the Rabbit	431
D. STOLKE, BU. SEIDEL, and H. MULLER: Results After Anterior Transposition of the Ulnar Nerve for Tardy Ulnar Palsy.	440
K. HUSE: Hemodynamic Changes During Controlled Hypotension with Sodium- nitrousprusside in Thiopental-Anesthesia	444
Subject Index	450

List of Editors and Senior Authors

ALEXANDRE, A.: Divisione di Neurochirurgia, Ospedale Civile, Via Muscheria 19, I-36100 Vicenza

ALTENBURG, H.: Neurochirurgische Universitäts-Klinik, Jungeblodtplatz 1, D-4400 Münster

Амвrosio, A.: Divisione di Neurochirurgia dell'Ospedali Riuniti per Bambini, I-80100 Napoli

ARIENTA, C.: Divisione di Neurochirurgia, Università di Milano, I-20100 Milano

ARMENISE, B.: Divisione di Neurochirurgia, Ospedale Generale Regionale "V. Fazzi", I-73100 Lecce

ARNOLD, H.: Abteilung für Neurochirurgie, Neurologische Universitätsklinik Hamburg, Martinistrasse 52, D-2000 Hamburg 20

BAUMANN, H.: Neurochirurgische Klinik der Städtischen Kliniken, Krankenhaus Nordstadt, Haltenhoffstrasse 41, D-3000 Hannover

BOKER, D.-K.: Institut für Neuropathologie der Universität, Sigmund-Freud-Strasse 25, D-5300 Bonn-Venusberg

BORTOLUZZI, M.: Divisione di Neurochirurgia, Spedali Civili, Università di Brescia, Piazza Spedali Civili, I-25100 Brescia

BROCK, M.: Universitätsklinikum Steglitz, Neurochirurgische Klinik und Poliklinik, Freie Universität Berlin, Hindenburgdamm 30, D-1000 Berlin 45

CLAR, H.-E.: Neurochirurgische Klinik im Universitäts-Klinikum Essen, Hufelandstrasse 55, D-4300 Essen 1

COLLMANN, H.: Neurochirurgische Klinik im Klinikum Charlottenburg der Freien Universität Berlin, Spandauer Damm 130, D-1000 Berlin 19

Соlомво, F.: Divisione di Neurochirurgia, Ospedale Civile, Via Muscheria 19, I-36 100 Vicenza DALLE ORE, G.: Divisione di Neurochirurgia, I-37100 Verona

DEMIREL, T.: Neurochirurgische Abteilung, Bethesda-Krankenhaus, Hainstrasse 35, D-5600 Wuppertal 1

DISTELMAIER, P.: Neuroradiologische Abteilung der Neurochirurgischen Universitätsklinik, Sigmund-Freud-Strasse 25, D-5300 Bonn-Venusberg

FANTIS, A.: Neurochirurgie, Versorgungskrankenhaus, Maulbeerallee, D-3280 Bad Pyrmont

FISCHER, D.: Neurochirurgische Universitätsklinik, Medizinische Fakultät der Universität des Saarlandes, D-6650 Homburg/Schwarzenbach

FRANCO, A.: Divisione di Neurochirurgia, Ospedale dei Pellegrini , Via Scipione Capece 3, I-80122 Napoli

GAAB, M.: Neurochirurgische Universitäts-Klinik, Kopfklinikum, Josef-Schneider-Strasse 11, D-8700 Würzburg

GEROSA, M. A.: Istituto di Neurochirurgia dell'Università di Padova, Via Giustiniani 5, I-35100 Padova

GILSBACH, J.: Neurochirurgische Klinik, Albert-Ludwig-Universität Freiburg, Hugstetter Strasse 55, D-7800 Freiburg

GIOMBINI, S.: Divisione di Neurochirurgia, Istituto Neurologico "C. Besta", Via Celoria 11, I-20133 Milano

GRAZIUSSI, G.: Ospedale S. Gennaro, Divisione di Neurochirurgia, Via F. Crispi 27, I-80121 Napoli

GROTE, W.: Neurochirurgische Klinik im Universitäts-Klinikum Essen, Hufelandstrasse 55, D-4300 Essen 1

GRUNINGER, W.: Rehabilitationsklinik für Querschnittsgelähmte im Krankenhaus, Hohe Warte 8, D-8580 Bayreuth

GRUSS, P.: Neurochirurgische Universitäts-Klinik, Kopfklinikum, Josef-Schneider-Strasse 11, D-8700 Würzburg

GUIDETTI, B.: Istituto di Neurochirurgia, Università di Roma, Viale dell'Università 30, I-00 100 Roma

HAMEL, E.: Neurochirurgische Universitätsklinik, Joseph-Stelzmann-Strasse 9, D-5000 Köln 41

HAMER, J.: Neurochirurgische Abteilung des Chirurgischen Zentrums der Universität Heidelberg, Im Neuenheimer Feld 110, D-6900 Heidelberg

XIV

НЕММЕR, R.: Neurochirurgische Universitätsklinik, Hugstetter Strasse 55, D-7800 Freiburg

HERRMANN, H. D.: Neurochirurgische Klinik, Universität des Saarlandes, D-6650 Homburg/Saar

HUK, W.: Neurochirurgische Klinik der Universität Erlangen-Nürnberg, Schwabachanlage 6, (Kopfklinikum) D-8520 Erlangen

HUSE, K.: Institut für Anaesthesiologie und Neurochirurgische Klinik der Universität Düsseldorf, Moorenstrasse 5, D-4000 Düsseldorf

KLINGER, M.: Neurochirurgische Klinik der Universität Erlangen-Nürnberg, Schwabachanlage 6, D-8520 Erlangen

KUGELGEN, B.: Neurologische Klinik der Universität Erlangen-Nürnberg, Schwabachanlage 6, D-8520 Erlangen

KUHLENDAHL, H.: Hubbelrather Weg 14, D-4006 Erkrath

LIEBIG, K.: Orthopädische Universitätsklinik, Rathsbergerstrasse 57, D-8520 Erlangen

LIESEGANG, J.: Neurochirurgische Klinik im Universitäts-Klinikum Essen, Hufelandstrasse 55, D-4300 Essen 1

LINS, E.: Neuroradiologische Abteilung der Neurochirurgischen Universitätsklinik, Sigmund-Freud-Strasse 25, D-5300 Bonn-Venusberg

MEINIG, G.: Neurochirurgische Klinik der Universität Mainz, Langenbeckstrasse 1, D-6500 Mainz

NAU, H.-E.: Neurochirurgische Klinik im Universitäts-Klinikum Essen, Hufelandstrasse 55, D-4300 Essen 1

PAPO, I.: Divisione di Neurochirurgia, Ospedale Generale Regionale, Via Panoramica 34, I-60100 Ancona

PENDL, G.: Neurochirurgische Universitätsklinik Wien, Alserstrasse 9, A-1090 Wien

PIA, H. W.: Zentrum für Neurochirurgie, Klinikstrasse 29, D-6300 Giessen

POTTHOFF, P. C.: Neurochirurgische Klinik der Universität Ulm, Reisensburgerstrasse 2, D-8870 Günzburg

RAMA, B.: Neurochirurgische Universitätsklinik, Robert-Koch-Strasse 40, D-3400 Göttingen

REALE, F.: Istituto di Neurochirurgia dell'Università di Siena, Via Valdimontone 6, I-53100 Siena

REINHARDT, V.: Institut für Neuropathologie im Universitäts-Klinikum, Hufelandstrasse 55, D-4300 Essen 1

RICHARD, K. E.: Neurochirurgische Universitätsklinik, Joseph-Stelzmann-Strasse 9, D-5000 Köln 41

RICHTER, H.-P.: Neurochirurgische Abteilung des Bezirkskrankenhauses, Reisensburger Strasse 2, D-8870 Günzburg

ROOSEN, K.: Neurochirurgische Klinik im Universitäts-Klinikum Essen, Hufelandstrasse 55, D-4300 Essen 1

SARTOR, K.: Bereich Neuroradiologie, Strahlendiagnostische Abteilung, Allgemeines Krankenhaus Altona, Paul-Ehrlich-Strasse 1, D-2000 Hamburg 50

SCHONMAYR, R.: Zentrum für Neurochirurgie, Klinikstrasse 29, D-6300 Giessen

SCHOTER, I.: Neurochirurgische Universitätsklinik, Sigmund-Freud-Strasse 25, D-5300 Bonn-Venusberg

SCHRAMM, J.: Universitätsklinikum Steglitz, Neurochirurgische Klinik und Poliklinik, Freie Universität Berlin, Hindenburgdamm 30, D-1000 Berlin 45

SCUCCIMARRA, A.: Divisione di Neurochirurgia, Ospedali Riuniti, I-89100 Reggio Calabria

SPRING, A.: Neurochirurgische Klinik der Medizinischen Hochschule Hannover, Karl-Wiechert-Allee 9, D-3000 Hannover 61

SPRUNG, C.: Neurochirurgische/Neurologische Klinik und Poliklinik, Freie Universität Berlin, Hindenburgdamm 30, D-1000 Berlin 45

STEUDEL, W. I.: Abteilung für Allgemeine Neurochirurgie, Zentrum der Neurologie und Neurochirurgie, Klinikum der Johann Wolfgang Goethe-Universität, Schleusenweg 2–16, D-6000 Frankfurt/Main 71

STOLKE, D.: Neurochirurgische Klinik der Medizinischen Hochschule Hannover, Karl-Wiechert-Allee 9, D-3000 Hannover 61

STRAHL, E. W.: Neurochirurgische Klinik im Universitäts-Klinikum Essen, Hufelandstrasse 55, D-4300 Essen 1

SUNDER-PLASSMANN, M.: Neurochirurgische Universitätsklinik Wien, Allgemeines Krankenhaus, Alserstrasse 4, A-1090 Wien

SZANTO, J.: Department of Neurology and Psychiatry, County Hospital, Arany Janos u. 61 Zalaegerszeg, Hungary THAL, H.-U.: Neurochirurgische Klinik der Universität, Moorenstrasse 5, D-4000 Düsseldorf

THOMALSKE, G.: Abteilung für Funktionelle Neurochirurgie im Zentrum für Neurologie und Neurochirurgie, Klinikum der Johann Wolfgang von Goethe-Universität, Schleusenweg 2–16, D-6000 Frankfurt 71

TORNOW, K.: Institut für Neuroradiologie, Klinikum Mannheim, Universität Heidelberg, Theodor-Kutzer-Ufer, D-6800 Mannheim 1

VOGELSANG, H.: Abteilung für Neuroradiologie, Zentrum Radiologie der Medizinischen Hochschule Hannover, Karl-Wiechert-Allee 9, D-3000 Hannover 61

WALLENFANG, T.: Neurochirurgische Klinik der Universität Mainz, Langenbeckstrasse 1, D-6500 Mainz

WASSMANN, H.: Neurochirurgische Universitätsklinik, Sigmund-Freud-Strasse 25, D-5300 Bonn-Venusberg

WEIGEL, K.: Neurochirurgische Universitätsklinik, Hugstetter Strasse 55, D-7800 Freiburg i. Br.

WILLIAMS, B.: Midland Center for Neurosurgery and Neurology, Holly Lane Waley, GB-Birmingham 67

ZANDER, E.: Service de Neurochirurgie, Centre Hospitalier, Universitaire Vaudois, Rue du Bugnon 15, CH-1011 Lausanne

ZAUNBAUER, F.: Neurochirurgische Universitätsklinik Wien, Allgemeines Krankenhaus, Alserstrasse 4, A-1090 Wien

Cervical Myelopathy

Cervical Myelopathy

H. KUHLENDAHL

"This is one more condition which has been taken out of the scrap basket into which we used to put multiple sclerosis or spinal degenerative disease". (GILBERT HORRAX, 1954)

A brief review of the discovery and development of our present-day concept of the complex "cervical myelopathy" would seem justified, since, among other things, it throws some light on the background of the unaltered current interest of the discussions about the actual pathological mechanisms of this disease of the spinal cord. The birth of this "new" disease of cervical myelopathy at the beginning of the nineteen-fifties, was not free from labour pains. In the initial period, neurology did not find it easy to come to terms with the concept of a bio-mechanical pathogenesis for classical neurological clinical pictures, and many a neurologist accepted this new knowledge only after a great deal of hesitation. After all, the discovery of the aetiological-pathogenetic principle of spondylogenic myelopathy represented a somewhat revolutionary break with the formerly apparently unshakable concept of an inflammatory or primarily degenerative pathogenesis of this spinal-neurological syndrome, which was now to be replaced by the concept of a mechanical causal complex.

This fact must be borne in mind when, today, we unquestioningly accept that spondylogenic cervical myelopathy is the most frequent disease affecting the spinal cord in the middle-aged and the elderly, beyond the age of 45. In order to be able to say this with such certainty, however, the research and clinical experience of more than two decades was necessary from the first demonstration of the clinical and pathogenetic overall concept of the causality between spondylotic changes and neurological pathological processes by the neurologist Sir RUSSEL BRAIN (in cooperation with the neuro-surgeon, D. NORTHFIELD and the pathologist MARCIA WILKINSON) in the year 1952.

The clinical entity, cervical myelopathy, belongs within the larger framework of biomechanical processes as a cause of disease in the field of neurology, whose recognition was introduced as early as in 1934, by MIXTER and BARR, with their clarification of the pathogenesis of ischalgia. (In passing, we might recall that the idea of an "inflammatory" aetiology for ischialgia did not finally disappear until the discovery was made that the identical causal relationships obtained both in the case of cervical radicular brachialgias, and cervical myelopathy).

First of all, however, a systematic differentiation must be made between the spinal cord lesion due to a *prolapse of the intervertebral disc* (which usually manifests as a subacute clinical picture), and the primary chronic *spondylotic form of myelopathy*. Occasionally, however, it is difficult to classify a particular case in the one or the other group. After all, the spondylotic, stenosing pathological-anatomical process takes its origin in the cartilage of the intervertebral disc, so that the osseous protrusions (spurs) developing reactively from the margin of the vertebral bodies, also usually have a fibrous-cartilaginous core. We go along with ROBINSON in employing the term "chondro-osseous protrusion" to describe this phenomenon.

But direct spinal-cord damage occasioned by a soft prolapse (in Anglo-Saxon usage, the so-called "soft disc"), which occurs predominantly in the fifth decade already, also belongs to the sector of cervical myelopathy. Here, however, there are scarcely any problems, neither with respect to the pathological mechanism of spinal-cord damage, nor with respect to the surgical therapy that is indicated.

By contrast, all the more problems are encountered with the entity *chronic spondylotic myelopathy*. In the following passage, this *spondylotic* form of myelopathy is discussed. Ever since cervical myelopathy has existed as an aetiological *clinical entity*, there has been a continuing lively discussion as to the mechanisms of its pathology, and the manner in which this spinal-cord lesion arises.

In any case, we are dealing here with a *complex pathogenic process*, which involves the interaction of a number of different factors. But is the triggering mechanism the direct mechanical effect of pressure upon the substance of the cord, (cord compression), or does the damage arise via the mechanisms of a vaso-circulatory ischaemia? And, further, does the latter arise as a consequence of the compression of the anterior spinal artery, or of radicular arteries, or of the epimedullary vasocorona? Is the causative factor simply static pressure occasioned by the protrusion, or do *dynamic* factors also play a part, and, if so, what is their role? The complex *bio-mechanical chain of causalities* must be investigated in more detail, not only on account of its fundamental significance for our understanding of the pathogenesis of the pathological process and of the neurological symptomatology, but, in particular, with reference to the *problems of surgical therapy*.

In this connection, dynamic factors play a decisive role, namely, both exogenous and internal (physiological) kinetic (dynamic) forces, the interplay of which is responsible for the neurological multiformity and determine the variable course of the disease. This pathological process can occur only in a mobile and actually moving cervical spine! The individual factors, however, vary both in their significance and their effect (Table 1).

- 1. Among the morphological factors, the relatively or absolutely narrow cervical vertebral canal as measured in the sagittal diameter is of predominant importance. The restricted freedom to move of the spinal cord is a conditio sine qua non for the development of myelo-pathy. No spondylogenic myelopathy ever develops in a wide cervical vertebral canal! The borderline between wide and narrow is at about 14 mm with respect ot the bony canal. The enveloping tissues account for some 2-3 mm. The diameter of the cervical spinal cord, however, is 9.5-10 mm. Numerous investigations into these measured values have been carried out.
- 2. The second and without a doubt, most important factor is the spondylotic *protrusion* (singular or multiple) at the ventral wall of the cervical vertebral canal, which exercises an additional stenosing effect. It can reduce the sagittal diameter to 10-8 mm,

Table 1. Synopsis of causal factors of cervical myelopathy

A. (Patho-)morphogenetic factors	B. Dynamic factors
$\stackrel{\lambda}{\vec{r}}$ 1. Narrowness of the canal!	1. Motion of head and neck!
2. Erect or kyphotic cerv. spine	Bulging of lig. flav.
2. Erect or kyphotic cerv. spine 3. Theca and arachnoid restraint A of the cord	3. Active spontaneous motion! of cord:
4. Chondro-osseous protrusions!	a) with respirationb) rhythmically transvers.
5. Cuff-shaped adhesive fibrosis > of the pia-arachnoid	with blood pulsation c) rhytmic changes of cord
of the pla-arachnoid G 6. Intima-fibrosis of the small g intramedullary arteries (Jellinger) 7. Diminished plasticity of the	volume due to blood pulse d) rhytmic pressure changes
$\stackrel{5}{0}$ 7. Diminished plasticity of the ω cord	
Biomechanic effects:	
Compression, constriction, tension,	(over-)stretching, friction,

vasocirculatory impairment

and lead to an absolute stenosis. In a wide canal of 17, 18 or 19 mm, an identical protrusion leads to no harmful effects.

- 3. A further important factor is the lack of lordosis, the erectness or, frequently, kyphotic, form of the cervical vertebral column. (This is, by the way, not a "compulsory posture" imposed by the disease (reflectorily, as it were), but is always part of a constitutional, more or less imperfect variant of the overall construction of the vertebral column). As compared with the lordotic posture, however, in erect of kyphotic cervical spinal columns, the vertebral canal is longer by 2-4 cm, with the result that, during flexing movements, a not inconsiderable traction effect is exerted upon the spinal cord (BREIG).
- 4. The restriction in the mobility of the cord "hung" within the vertebral canal, in particular due to the relative fixation of the epidural space and of the nerve roots and to the dentate ligaments, and other arachnoidal structures, is enhanced in the overstretched or kyphotic cervical spinal column.

In this unfavourable anatomical situation, the permanent dynamic forces which lead to a continuous back-and-forth movement of the cervical spinal cord and which tug at the cord in the narrowed canal giving rise to friction and chafing, develop a potentiating effect and lead to continuous *microtraumatization*. (The various dynamic forces can be seen in the right-hand side of the Table 1). With every beat of the pulse, the spinal cord moves up and down through a distance of several millimetres in the axial (longitudinal) direction. In the same rhythm, the volume of the cord also fluctuates, finding expression as a movement in the transverse diameter of the vertebral canal. Superimposed on these movements is a slower motion due to the movements of respiration.

Particular attention msut be given to the role of the *ligamentum flavum*. In the narrowed spinal canal, the ligamenta flava, which, during extension, bulge inwards, impinge upon the posterior tracts and posterior roots. This is particularly true when lordosis of the cervical spine presents. Vis-à-vis a protrusion, a pincer-like mechanism comes into effect. In consequence of this, we often find the typical paraesthetic conditions that occur in conjunction with certain lesions of the head, in the hands. Frequently disturbances in sensation begin or predominate in the upper extremities.

Friction and chafing effects in the narrowed canal lead to more or less extensive, leptomeningeal adhesions, in particular on the ventral surface of the spinal cord. We have confirmed this regularly when opening the dura while performing a laminectomy. Of course, the direct effects of pressure (compression) upon the cord, which sits in the now too-narrow canal like a plug, also occure, as do circumscribed impressions in the cord and, naturally, impairment of circulation, involving both the venous vessels and also the arteries. The question as to an arterial vascular blockade remains a debatable point.

In my opinion, too little is known about the extent of the dynamic processes that take place within the vertebral canal. For this reason, a short film was shown.

On the *neurological symptomatology*, I shall merely make only few general remarks. On the one hand we find the more or less symmetrical, more or less marked symptomatology of transverse lesions of the cord and, on the other the *imitation* of all systemic diseases of the spinal cord, including multiple sclerosis, of course.

Naturally the neurological symptomatology is also in part determined by the level involved, as well as by the nature, form and extent of the protrusions which can manifest as paramedian, semi-lateral bulgings, or as uniform transverse thickenings, either single or multiple, and sometimes as a chain of hard nodules. Furthermore the symptomatology is partly determined by the lordotic or kyphotic shape of the cervical spinal column.

In the subacute cases of "soft disc", a more or less marked Brown-Séquard's syndrome is frequently seen, since the prolapse is usually expressed in the paramedian area. In the case of a markedly chronic course with stenosing due to osseous bar-shaped protrusions and in view of the frequent localization in the lower part of the cervical spine, the syndrome of amyotrophic lateral sclerosis with the typical nuclear atrophies of the small muscles of the hand, is remarkably frequently found.

In addition, we also observe both purely motor tetra-spastic or paraspastic syndromes, and also cases with marked sensory transverse-lesion symptomatology. However, the sensory symptomatology, too, manifests quite unsystematically. We have insufficient time to deal at any length with the differential diagnosis, here. Since there is no uniform neurological clinical picture, the differential diagnostic problems involved are often not of a minor nature. Here, too, the fact that the diagnosis established in a more or less large number of cases is *wrong*, should not be lost sight of. From the state of our knowledge today, however, I would no longer agree with C. ADAMS when (in the Handbook of Clinical Neurology, 1976) he states that the diagnosis can be established only on the basis of excluding all the other possible diagnoses. In particular, the subtle radiological diagnostic work-up employing water-soluble contrast media, which is possible today, permits an adequately reliable positive diagnosis in many cases. The complex bio-mechanical pathogenetic constellation, however, also makes the *prognostic* assessment difficult. The big question mark is the *spontaneous* course which, in the case of chronic spondylotic myelopathy rarely progresses in a linear manner to complete paraplegia certainly only rather rarely. In a large number of cases, the process apparently "stagnates" in a more or less high-grade stage of deficitor would very probably often remain stagnant even without any surgical treatment. In consequence, the question as to whether an unchanged post-operative state or finding can be chalked up as a therapeutic success (prevention of further progression?) is an open one. What experience certainly does teach us, however, is that, in individual cases, after years of quiescence, a progressive deterioration again sets in. In any case, there is a need for more comprehensive postoperative long-term follow-up observations.

I should now like to briefly characterize the particular problems associated with *surgical therapy* on the basis of a few short headings.

- a) The too narrow canal necessitates the relief afforded by laminectomy, where required with plastic enlargement of the epidural space.
- b) The compression by the hypomochlion of the protrusion at the anterior wall requires the removal of the latter via the anterior approach.
- c) The injurious effect of *exogenous* dynamic fordes suggests that immobilization by fusion of the vertebral bodies is necessary. The range of the therapeutic concepts, therefore, lie between temporary immobilization with the aid of a supportive device on the one hand, and, at the other end of the range, a two-stage surgical procedure with or without fusion. The complexity of the pathological mechanisms should induce us to analyse the situation in each individual case, and to maintain appropriate flexibility with respect to the indication and the choice of the most suitable surgical procedure. Without a doubt, the ideal objective can only be the establishment of a spinal cord with adequate space allowing freedom of movement.

This paper was introduced with a remark about the success achieved by the scientific search for knowledge. I should like to end by giving expression to a certain degree of resignation. This resignation is occasioned by the fact that the illumination of the aetiopathogenetic relationships, which have led to *diagnostic success*, have not led to any *comparable therapeutic gains*. For, in my opinion, at any rate, the successes of surgical treatment certainly do not provide us with any good reason for singing and dancing - they are not even particularly satisfactory - neither those of my own direct experience, nor those described in the international literature, which, it might be said in passing, provides much food for controversy. After all, in our specialty, we all too often have to be satisfied with a more or less limited success of surgery.

Despite this, we must continue to operate - and we must also continue with our research work, in order to obtain better results at some time or other.

Indications, Results and Adverse Effects of Cervical Myelography with Amipaque (Study on 300 Examinations)

H. VOGELSANG and R. C. SCHMIDT

Myelography with the water-soluble, non-ionic contrast medium Amipaque has given rise to decisive advances in the diagnosis of cervical myelopathy. The possibility of discerning excellent detail of anatomical structures in the spinal canal (this applies even for vessels) permits exact visualization of pathological changes. Since the amount and concentration of the contrast medium have a critical influence on side effects, they should be kept as low as possible. This is achieved via the lateral approach at C 1/2 by fractionated administration, and ideal positioning of the patient without tilting. As can be seen from Table 1, the high frequency of examinations in the meantime shows that the diagnostic value of this procedure has been recognized by clinicians.

Table 1. Myelography with amipaque (n = 900)

Lumbal	4138		
Thoracic	155%		
Cervical	3328		

Degenerative conditions originating in the vertebrae with effects on the central and peripheral nervous system take first place among the indications (Table 2). Cervical myelography with Amipaque permits farreaching differentiation of the causes of pain syndromes and neurological disorders (Table 3).

Table 2. Diagnosis in cervical myelography (n = 332)

Degenerative disease	59%	
Tumor	88	
Traumatic lesion	5%	
Miscellaneous	5%	
Normal	23%	

Table 3.	Degenerative	cervical	spine	disease	(n =	193)

Disc herniation	30%
Spondylosis and Narrow spinal canal	38%
Osteochondrosis with nerve root compression	32%

1. Herniated Disc

Detachment of the anterior wall of the dural sac with consequent narrowing of the ventral subarachnoid space, with or without touching of the spinal cord, can be seen in the lateral view. In the frontal view, the herniation leads to a thinning of the contrast medium column and to a broadening of the lateral subarachnoid space, similar to the situation observed in the presence of an intramedullary tumor, but limited to one intervertebral space.

2. Retrospondylosis with Primary Stenotic or Constricted Spinal Canal

The anterior wall of the dural sac is detached and the ventral subarachnoid space is constricted by dorsal retrospondylosis, the extent of which (including touching of the spinal cord) can be shown impressively. In many cases, a constriction in the sense of the pincer mechanism is also found from dorsal upon retroflection of the head. The reduction in size of the spinal cord for several segments can be measured and is in some cases substantial. X-ray pictures in the frontal view resemble those seen in disc herniation, only that in many cases several levels and the nerve roots are also affected.

3. Osteochondrosis with Nerve Root Compression

These are the most frequent cause of shoulder-arm-syndrome with and without neurological deficits, mostly resulting from arthrosis of the uncovertebral joint, retrospondylosis and stenosis of one or several intervertebral foramina. Myelography also reveals typical findings here, especially in the frontal view: the nerve roots or root sleeves are lacking at one or several levels, and in some cases they are displaced. In their place, a filling defect is found oriented medially. On the other hand, in the lateral projection findings are negative or a slight identation of the ventral subarachnoid space is found laterally (tomogram).

Such findings, not obtained with negative or oily contrast media, prove the value of the method. However, the value of an investigation is also determined by its adverse affects. The general observation must be made here that no neuroradiological examination and no contrast medium can be regarded as free of adverse effects (i.e. indifferent). This also applies to Amipaque. Two possible complications encountered with other aqueous contrast media can be neglected, however: the occurrence of epileptic seizures and manifestations of spinal irritation. These are incontrovertible advantages of the new generation of contrast media. Manifestations of spinal irritation are actually to be feared only when severe kyphoscolioses prevent or delay the contrast medium from draining away in the caudal direction. The autonomic symptoms such as headaches, nausea and vertigo remain. These are, in some cases, caused by a "hypobaric" syndrome, but in other cases they have to be considered a direct effect of the contrast medium. The dependence on the amount and concentration of the contrast medium as well as the region of examination can be regarded as demonstrated (Tables 4 and 5).

Psychopathological syndromes are observed as a new phenomenon in Amipaque myelographies. They may occur especially after thoracic, but also after cervical examinations. Here the dependence of the amount and concentration, as well as of the age of the patient, or of a brain with prior damage, play the decisive role. For this reason, elderly

<u>-</u>	Punction	Volume (ml)	Concentration (mg 1/ml)
Lumbal	L 3/4	8-10	170
Thoracic	L 1/2	10-14	220-240
Cervical	C 1/2 lat.	6-10	170

Table 4. Myelography with Amipaque

Table 5. Adverse effects

	Lumbal myelography (%)	Thoracic myelography (%)	Cervical myelography (%)
Headache	31	36	23
Nausea	3	6	4
Vomiting	8	32	11
Dizziness	7	8	4
	n = 120	n = 50	n = 105

patients should continue to be given oil myelography to demonstrate the thoracic region when the Queckenstedt-test is negative. The usually mild (but sometimes severe) psychopathological symptoms we observed, subsided within 24 hours (at the latest after 48 hours) and did not lead to any secondary effects.

To summarize, in 332 cervical myelographies performed via the lateral approach at C 1/2, a high diagnostic accuracy was achieved which had not been attained so far. This applies in particular to cervical myelopathy. Touching of the spinal cord can be recognized unequivocally, and measurements performed. The adverse effects of the contrast medium Amipaque (autonomic symptoms, brief psychopathological symptoms in elderly patients) are therefore justifiable.

References

- AHLGREN, P.: Myelography with Metrizamide in the cervical region. In: Metrizamide-amipaque. LINDGREN, E. (ed.), p. 85. Acta radiol. Suppl. 355 (1977)
- AMUNDSEN, P.: Metrizamide in cervical myelography. In: Metrizamideamipaque. LINDGREN, E. (ed.), p. 85. Acta radiol. Suppl. 355 (1977)
- AMUNDSEN, P., SKALPE, I.O.: Cervical myelography with a water soluble contrast medium (Metrizamide). Neuroradiology <u>8</u>, 209 (1975)
- PEETERS, F.L.M.: Myelography with metrizamide. Radiol. clin. <u>46</u>, 203-213 (1977)
- SCHMIDT, R.C.: Die zervikale und thorakale Myelographie mit dem neuen wasserlöslichen Kontrastmittel Metrizamid. Akt. Neurol. <u>5</u>, 97 (1978)
- SCHMIDT, R.C.: Probleme der Myelographie mit Metrizamid (Amipaque) bei älteren Patienten. In: Amipaque Workshop Berlin 1978. FROMMHOLD/ HACKER/VOGELSANG/SCHMITT (Hrsg.), S. 20. Amsterdam: Excerpta medica 1978
- SKALPE, I.O., AMUNDSEN, P.: Thoracic and cervical myelography with Metrizamide. Radiology <u>116</u>, 101 (1975)

- SKALPE, I.O., SORTLAND, O.: Myelography lumbar-thoracic-cervical with water soluble contrast medium. Oslo: Tanum-Norli 1978
- SORTLAND, O., SKALPE, I.O.: Cervical myelography by lateral cervical and lumbar injection of Metrizamide. In: Metrizamide-amipaque. LINDGREN, E. (ed.), p. 154. Acta radiol. Suppl. 355 (1977)
- VALK, J.: Myelography with metrizamide (Amipaque). Medicamundi 21, 164 (1977)
- VOGELSANG, H., BUSSE, O., SCHMIDT, R.C.: Die zervikale Myelographie mit wasserlöslichem Kontrastmittel (Amipaque). Fortschr. Röntgenstr. 125, 225 (1976)
- VOGELSANG, H., SCHMIDT, R.C., BUSSE, O., DANGEL, U.: Myelographie mit Metrizamid (Amipaque). Acta neurochir. (Wien) <u>40</u>, 157 (1978)





Fig. 1 (left). Medial herniated disc at C 5/6 with norrowing of the ventral subarachnoid space and touching of the spinal cord Fig. 2 (right) Retrospondylosis at C 5/6 with narrowing of the ventral subarachnoid space, without touching of the spinal cord



Fig. 3. Typical myelography in a case of cervical myelopathy by retrospondylosis showing marked compression of the spinal cord. Note also the pince mechanism from dorsal by retrofection of the head



Fig. 4. Filling defect of the nerve sleeve at C 5/6 on the right by uncovertebral arthrosis

Computed Tomography in Spondylogenic Narrowing of the Cervical Spinal Canal

K. SARTOR, D.-K. BÖKER, N. FRECKMANN, and R. SCHÄFER

Introduction

Since development of general purpose scanners, computed tomography (CT) of the entire body has become possible $(\underline{6})$. Spatial resolution of modern equipment is already rather good with regard to dense structures such as bone. As a method of axial radiography, CT appears, therefore, well suited for the investigation of the bony spinal canal, for reasons of anatomy and ease of positioning, particularly in the cervical region.

Material and Approach

We performed CT scans on 70 patients with spondylogenic narrowing of the cervical spinal canal. The etiology of bony stenoses varied. Stenosis secondary to degenerative disease of the spine predominated, however. A certain number of patients were examined for other reasons, and stenosis of the cervical spinal canal of some kind, usually localized and spondylogenic in origin, was an accidental finding.

A total body scanner (EMI 5005/12) was used for all examinations. In order to reduce radiation and improve image quality, the smaller scan field (24 cm) was selected. All studies were performed with the 320 matrix. The slow scanning mode was preferred. Slices were 13 mm thick. Overlapping was 3 - 5 mm, depending on the nature of regional pathology. For visual and statistical evaluation of the data an independent diagnostic unit was available.

About 50% of the examinations were performed without any or with intravenous contrast enhancement, the remaining after intrathecal administration of metrizamide (intrathecal contrast enhancement). For level localization purposes horizontal beam lateral radiographs of the cervical region with the spine straightened as much as possible were obtained before each CT study in almost all cases. For the scanning procedure the patient was put in the same position with the cervical spine again straightened and placed parallel to the couch table top. Depending on the situation the duration of the entire examination varied between 20 and 90 minutes.

Results

Bony structures were viewed best at wide window settings. Non-invasive demonstration of intraspinal soft tissue structures was best at high contrast window settings. The cord could be seen most frequently in the high cervical region, particularly after intravenous contrast enhancement. The clearest differentiation of spinal cord, subarachnoid space and epidural space was possible, however, after intrathecal contrast enhancement (Fig. 1, normal cases for comparison).

In the narrow cervical spinal canal syndrome CT was very helpful since it demonstrated the *overall* width and shape of the spinal canal on the different levels, as well as rotational changes of the cervical spine as a whole. Particularly after intrathecal enhancement, the morphological aspects of the spinal cord could be evaluated very well (Fig. 2). Constitutional changes of width and shape at the craniospinal transition were far better appreciated on the CT scans than on conventional radiographic studies (Fig. 3 a-c) (Slight narrowing of the foramen magnum due to a tumor of almost bony density was similarly diagnosed, Fig. 3 d).

In several trauma cases, fractures of the neural arch, at best suspected on conventional radiographs, were clearly demonstrated by CT. Again, overall width and shape of the spinal canal at the level of the fracture could be evaluated most easily (Fig. 3 e-f). Also, intraspinal displacement of isolated bone fragments could be recognized or excluded.

In one patient with an osteogenic giant cell tumor of C 2 and C 3 the (marked) degree of narrowing of the spinal canal secondary to osteoblastic changes involving the left half of the vertebral bodies and neural arches was only seen on the appropriate CT scans (Fig. 3 g-h).

In degenerative disease of the cervical spine CT helped assessing the possible involvement of the cord due to osteophytes. Because of the axial visualization of the spinal canal, exact location (bilateral, unilateral, midline) and shape of bony spurs including their relative stenosing effect was very well shown. Compression and deformation effects on the spinal cord could be appreciated best after metrizamideenhancement of the subarachnoid space (Fig. 4 a-d). In a few cases with subtotal spinal block on ascending metrizamide myelography CT permitted the differential diagnosis (Fig. 4 e-h).

Discussion

CT is a method of axial radiography. It is therefore particularly well suited for the investigation of the spine. Non-invasive visualization of the soft tissue content of the spinal canal is still a challenge for research. Presently, with commercially available scanners metrizamide-enhancement (of the subarachnoid space) appears to be the best way of demonstrating the spinal cord and its relationship to ajacent bony structures (Fig. 1 e-f; 2; 4 c-d, g-g'). The spine itself, including surrounding soft tissue, can be well demonstrated non-invasively (Fig. 1 a-d; 3 c, e, f, h; 4 a-b, f). With regard to demonstration of the bony spinal canal CT is superior to the usually applied conventional radiographic methods as it provides the most valuable plane, namely the axial. Only in this plane can spondylogenic narrowing of the canal be really appreciated. In the cervical region, including transitions, this holds true for any kind of bony stenosis be it of developmental (Fig. 2; 3 b-c), traumatic (Fig. 3 e-f), neoplastic (Fig. 3 h), degenerative (Fig. 4 a-d, f) or of other origin (1, 2, 3, 4, 5, 7, 8). Optimal positioning of the cervical spine during the scanning procedure is of utmost importance, however, particularly when CT slices are relatively thick. Otherwise the partial volume effect will affect the evaluation of the real width of the cervical spinal canal.

Conclusion

Computed tomography is a valuable method of investigation in suspected spondylogenic narrowing of the cervical spinal canal of any cause.

References

- BALÉRIAUX-WAHA, D., MORTELMANS, L.L., DUPONT, M.G., JENAMART, L.: Computed tomography for lesions of the craniovertebral region. Neuroradiology <u>13</u>, 59-61 (1977)
- COIN, C.G., PENNINK, M., AHMAD, W.D., KERANEN, V.J.: Diving-type injury of the cervical spine: Contribution of computed tomography to management. J. Comput. Assist. Tomogr. <u>3</u>, 362-372 (1979)
- 3. HAMMERSCHLAG, S.B., WOLPERT, S.M., CARTER, B.L.: Computed tomography of the spinal canal. Radiology <u>121</u>, 361-367 (1976)
- 4. HYMAN, R.A., MERTEN, C.W., LIEBESKIND, A.L., NAIDICH, J.A., STEIN, H.L.: Computed tomography in ossification of the posterior longitudinal spinal ligament. Neuroradiology 13, 227-228
- HELLER, M., RINGE, J.D., BÜCHELER, E., KUHLENCORDT, F.: Morbus Paget - Manifestationen an der Wirbelsäule. Computertomographische Untersuchungen. Fortschrt. Röntgenstr. <u>130</u>, 652-658 (1979)
- LEDLEY, R.S., DI CHIRO, G., LUESSENHOP, A.J. et al.: Computerized transaxial X-ray tomography of the human body. Science <u>186</u>, 207-212 (1974)
- POST, D.M.J., GARGANO, F.P., VINING, D.Q., ROSOMOFF, H.L.: A comparison of radiographic methods of diagnosing constrictive lesions of the spinal canal. Toshiba unit vs. CT scanner. J. Neurosurg. 48, 360-368 (1978)
- TADMOR, R., DAVIS, K.R., ROBERSON, G.H., NEW, P.F.J., TAVERAS, J.M.: Computed tomographic evaluation of traumatic spinal injuries. Radiology 127, 825-827 (1978)

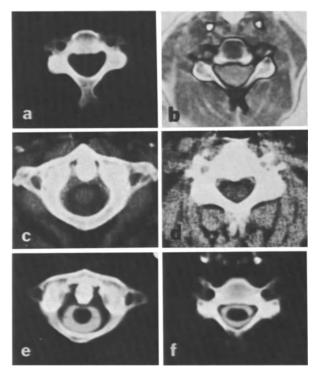


Fig. 1 a-f. Normal CT-scans of cervical spine and spinal cord. a Scan at level of C 3 (no enhancement). b Scan at level of C 5 (no enhancement). c Scan at level of atlas showing epidural tissue, subarachnoid space and spinal cord (no enhancement; high contrast window setting). d Scan at level of C 5; spinal cord can be differentiated (i.v. enhancement; high contract window setting). e Scan at level of atlas (metrizamide-enhancement). f Scan at level of C 6 (metrizamide-enhancement)

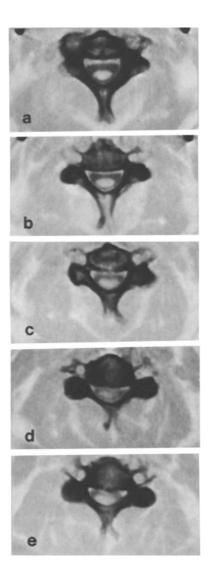


Fig. 2 a-e. Congenitally narrow cervical spinal canal (metrizamideenhancement / CT-myelogram). <u>a-c</u> Scans at level of C 2, C 2/3 and C 3 showing relatively wide subarachnoid space and thin cord. <u>d</u> Scan at level of C 4/5 interspace showing extreme anterior-posterior flattening of spinal cord and very narrow subarachnoid space secondary to constitutional narrowness of spinal canal plus posterior spondylosis. <u>e</u> Scan at level of C 5

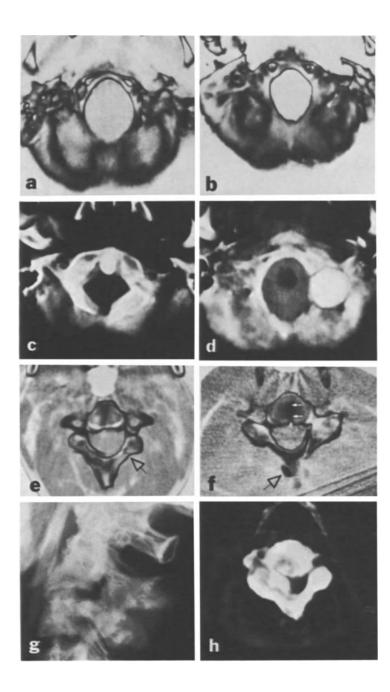


Fig. 3 a-h. Narrowing of foramen magnum and cervical spinal canal of various etiologies. a Normal foramen magnum for comparison. b Narrow foramen magnum in a patient with congenitally narrow spinal canal. c Narrow neural canal in basilar impression with assimilation of atlas. d Slight narrowing of foramen magnum of non-spondylogenic origin (highly calcified tumor at craniospinal junction). e Bilateral fracture of neural arch of C 2 with slight narrowing of spinal canal; arrow points to fracture on right. f Probably bilateral fracture of neural arch of C 6, narrowing of spinal canal more pronounced. White arrows point to vertical fracture of vertebral body, single arrow points to unusual extension of spinous process of C 5 (no fracture!). g Osteogenic giant cell tumor at C 2 and C 3 (lateral radiograph of upper cervical spine). h Corresponding CT-scan at level of C 3 shows marked bony stenosis of spinal canal

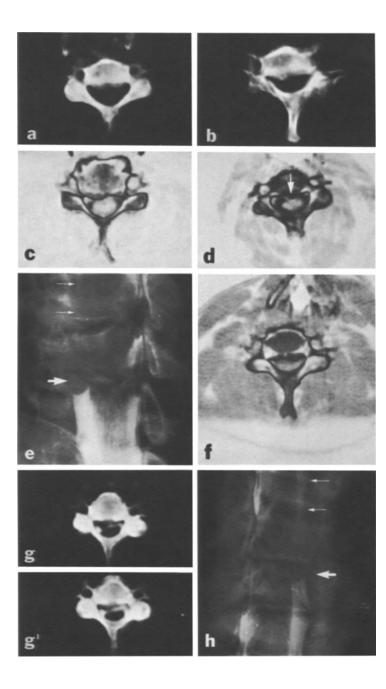


Fig. 4 a-h. Narrowing of cervical spinal canal in degenerative disease of the spine. a Transverse posterior osteophyte at C 5/6 interspace with moderate narrowing. <u>b</u> More laterally (left) located posterior osteophyte at C 6/7 interspace with marked narrowing. Also narrowing of neural foramen on both sides, and of transverse foramen on right secondary to spondylarthrosis and degenerative changes of LUSCHKA's joints. c Compression of subarachnoid space, probably also already of spinal cord due to unilateral (left) posterior osteophyte at level of C 5/6 (metrizamide-enhancement / CT-myelogram). d Midline compression of subarachnoid space and spinal cord by prominent osteophyte (arrow) at level of C 4. e-h Differential diagnosis by means of CT: Subtotal block to flow of contrast medium at ascending metrizamide-myelography at level of C 5/6 interspace (fat arrows) in two patients; myelographic appearance quite similar, small arrows point to little contrast medium above obstruction. Disk herniation? (e and h). CT scans show different causes of obstruction: Prominent unilateral osteophyte with relatively narrow spinal canal (\underline{f} , corresponding with e) and protruding intervertebral disk plus some spondylosis (g + g', corresponding with h). Operative confirmation

Electromyographic (EMG) Investigations in Cervical Myelopathy H.-E. NAU, J. LIESEGANG, P. HIEDL, and K. ROOSEN

Introduction

This study dealing with EMG alterations in cases of cervical myelopathy was motivated by the rare publications (1-6) analysing only a small number of patients. We report on the pathological EMG changes in a larger number of patients who have been operated on.

Patients and Method (Table 1)

Twenty-two patients (15 males and 7 females with an average age of 55.6 years) suffering from cervical myelopathy came to admission for operative management last year. The diagnoses on admission were different: protruded disc in 13 cases, spinal space occupying lesion and narrowing of the spinal canal in 3 cases each, unknown spinal diseases in the rest. All underwent clinical and neuroradiological examinations (cervical myelography in 21 cases, additional discography in 17 patients, and discography only in 1 case).

Electromyographic investigations were performed in all patients by a three channel DISA electromyograph (type 14 A 30) and concentric needle electrodes. The program was standardised in derivations of the deltoid, biceps, and triceps, brachioradialis, adductor pollicis brevis, abductor digiti V, quadriceps femoris, tibialis anterior, gastrocenmius, and the segmental nuchal muscles, at rest as well as under various voluntary innervations.

Clinical data were compared with the neuroradiological and electrophysiological findings.

All patients were operated on. In 10 patients excision of one cervical disc was performed, followed by interbody fusion. In 4 cases two neighbouring discs were operated on in this way. Five patients underwent laminectomy, 3 were treated by fusion followed by laminectomy. Eighteen patients were followed up for a long time. All showed a good improvement.

<u>Results</u> (Table 1)

The clinical courses (according to history) were acute in 4 cases, subacute in 6, and chronic in 12. Thirteen patients had a spastic tetraparesis, 6 a paraparesis, and 1 a medullary hemiparesis. Twelve of them had segmental motor deficit and/or radicular disturbances of sensitivity in one arm or both arms. All but three patients had disturbances of gait. Three could not walk at all.

Patients	22 } 15 males Average age 55.6 years 7 females
History	Acute 4 Subacute 6 Chronic 12
Neurological examination	Tetraparesis 13 Paraparesis 6 Hemiparesis 1 Segmental deficits 12
Electromyography	Spontaneous activity7Rarefaction of intention pattern21Increased amplitudes18Polyphasic potentials13Monosegmental alteration7Polysegmental alteration14Agreement with neurological findings9Discrepancy to neuroradiology11
Neuroradiology	Plain X-ray22Degenerative signs17Myelography21Protruded disc(s)9Stop (functional, complete)10Discography19Pathological findings14
Operation	Fusion 14 Laminectomy 5 Combined surgery 3

Table 1. Findings in patients suffering from cervical myelopathy

Plain X-ray of the cervical spine revealed degenerative signs in 17 patients. In 1 case each a subluxation and a false posture was found. Only three cases seemed to have a normal spine.

Myelography was performed in 21 patients. In 9 a ventral space occupying process was found due to one or more protruded discs. Two cases showed a complete, resp. incomplete stop, 8 a functional contrast stop.

Discography was performed in 19 patients. In 9 one disc only was found to be abnormal, in 4 two discs simultaneously, and in only 1 case three discs. Five discographies did not reveal any disc protrusion. The most frequent disc involved was that between the 5th and 6th vertebral bodies (36% of all cases).

Electromyographic investigations were performed in all patients. In 7 cases spontaneous activity consisting of fibrillations or - less frequently - of positive sharp waves was found, in one chronic case fasciculation potentials. The clinical course of those patients with fibrillation potentials and positive sharp waves was acute or subacute in 5 cases. No spontaneous activity could be seen in the lower limbs.

While normal voluntary activity was found in the upper myotomes, the muscles innervated by the compromised cervical nerve roots revealed a normal interference pattern in 1 patient only. All others showed non-maximal recruitment with signs of peripheral neurogenic disorder of these muscles. The severest deficit in recruitment was found in cases of acute or subacute clinical course. In 6 of the 12 chronic cases only a slight deficit of recruitment could be seen.

The heights of amplitudes varied very much. In 17 patients augmented amplitudes from 2 to 7 millivolts were seen, in one additional case amplitudes up to 15 millivolts were recorded. The more chronic the clinical course was, the higher the amplitudes found.

In the same way, the most severe increase in number of polyphasic waves was found in the chronic patients. In 9 of them nearly no normal action potentials could be derived.

In the arm muscles these EMG signs of peripheral neurogenic disorders were segmental in 20 patients. In 7 cases the disturbances showed a monosegmental pattern, in all others at least 2 segments seemed to be involved. In these cases the upper segment was used for the neurophysiological determination of the site of the spinal lesion. The distribution of electromyographically and neuroradiologically determined sites is shown in Table 1. In 9 cases EMG location was in accordance with the neuroradiological findings. In 11 cases EMG localisation was one or two segments below the neuroradiological. EMG examination of the muscles of the leg showed a more or less decrease of voluntary activity (slight decrease in 11 patients, moderate in 3), sometimes accompanied by a slight increase of polyphasic potentials, in accordance with the severity of the paresis. This accordance berween EMG alterations and clinical findings was noted both preoperatively and postoperatively.

Discussion

Up to now, in the publications (1-6) concerning EMG alterations in cervical myelopathy, pathological EMG findings of only a few patients have been described in a very global way. That is why we routinely performed EMG examinations in all our patients with cervical myelopathy. In doing so we found the same kind of pathological alterations but with different degrees and distribution patterns.

Spontaneous activity was found in 8 patients, 7 of them showing fibrillation potentials accompanied by positive sharp waves. In 1 patient only fasciculation potentials could be derived. With the exception of 2 patients with a chronic course cervical myelopathy, this spontaneous activity was found in those patients with an acute or subacute history. These results correspond to the findings in cases of peripheral nerve or nerve root compression where spontaneous activity can be found mainly in the more acute stages.

In the upper limbs voluntary activity of all but one patients showed a more or less pronounced rarefaction, increase in the number of polyphasic potentials, and an increase in the amplitude, signs of a chronic peripheral neurogenic disturbance. These disorders had a monosegmental or, more frequently, a bisegmental distribution pattern, seldom a polysegmental one. In contrast to these observations in the muscles of the arms, we found a slight or moderate deficit of recruitment in the muscles of the legs in 3/4 of our patients. All these EMG alterations of the arm muscles reveal a disorder of the peripheral neuron. But it is not possible to differ whether the noxic factor acts at the anterior horn cells, the nerve root fibres, or the spinal nerve roots. These findings, in combination with the signs of central paresis in the muscles of the legs, can be a clue to the diagnosis of cervical myelopathy. In this way, it seems to be possible to differentiate this disease from polyneuropathy, amyotrophic lateral sclerosis. In comparison with the neuroradiological findings, it is difficult to evaluate the exact site of the disturbance, because in most of the cases we found polysegmental disorders. Even in those cases where only one disc was the cause of cervical myelopathy, EMG alterations were found in the myotomes lower than expected by neuroradiological and intraoperative findings.

Summary

22 patients suffering from cervical myelopathy underwent EMG investigations before and after operation. In these cases we found an extremely high incidence of polysegmental chronic denervation (increased number of polyphasic potentials, increased duration of motor unit potentials, as well as rarefaction of interference patterns in maximal voluntary movement) which depended on the severity of clinical symptoms. Activity at rest was only found in some cases.

EMG investigations can provide good diagnostic clues, and offer the possibility for follow-up investigations in cases of cervical myelopathy with mixed segmental and medullary symptoms.

References

- HOEFER, P.F.A., GUTTMAN, S.A.: Electromyography as a method for determination of level of lesions in the spinal cord. Arch. Neurol. Psychiat. <u>51</u>, 415-422 (1944)
- JÖRG, J.: Die cervicale Myelopathie als differentialdiagnostische Erwägung bei Gehstörungen im mittleren und höheren Alter. Nervenarzt <u>45</u>, 341-353 (1974)
- KAPLAN, P.E.: Cervical spondylitic myelopathy (Letter). Arch. Neurol. <u>25</u>, 772 (1978)
- KAPLAN, P.E.: Cervical bilateral polyradiculopathy as a manifestation of cervical transverse myelopathy. Electromyograph. Clin. Neurophysiol. <u>18</u>, 159-164 (1978)
- 5. LIESEGANG, J.: Neurophysiologische Untersuchungen bei zervikalen Wurzelkompressionen. Habilitationsschrift Essen, 1975
- 6. LUDIN, H.-P.: Praktische Elektromyographie. S. 75. Stuttgart: F. Enke 1976

Clinical Experience with the Objective Localization of the Lesion in Cervical Myelopathy

J. SCHRAMM

The diagnosis and the differential diagnosis of cervical myelopathy (CM) remains problematic (1, 2, 6, 10). It is not only problematic to differentiate vascular myelopathy from spondylogenic CM but also from the spinal type of multiple sclerosis and other spastic clinical pictures. Degenerative cervical spine alterations are quite common in older age and quite often multisegmental. Myelography often confirms the multisegmental vertebral changes without, however, clarifying which of the lesions is affecting the spinal cord. Utmost localizing accuracy as to the segment and the extent of spinal cord involvement is desirable when planning the operative procedure in such cases. The aim of this study is to evaluate the usefulness of somatosensory evoked potential recordings (SEP) in cases of cervical spondylotic diseases with and without myelopathy.

Material and Methods

Two groups of patients are included in this study: 18 cases of cervical myelopathy and 22 cases of cervical radiculopathy, either due to cervical spondylosis and/or to cervical disc protrusions (Table 1). Twelve patients from the first group and 21 from the second group were operated on by the ventral approach (ROBINSON-SMITH) with a modified stabilization procedure using methacrylate (9). All patients were subjected to an SEP examination using the segmental stimulation technique. In 21 cases the SEP examination was repeated up to 3 times and used as a control. Usually the second recording was made during the first postoperative week; the third recording was repeated much later after periods ranging up to 24 months. The mean duration of symptoms in the radiculopathy group was 7.6 months (minimum 1 week, maximum 2 years) and in the cervical myelopathy group 3.5 years (minimum 2 months, maximum 10 years).

	No. of cases	Operations	Myelograms		ked poten- l studies
				1	2 or more
Cervical myelopathy	18	11	12	9	9
Cervical radiculopathy	22	21	22	10	12
Total	40	33	34	19	21

Table 1

Evoked potentials were recorded with scalp electrodes and a standard EEG amplifier and averaged in a signal averager (NICOLET 1072). One hundred and twenty-eight responses were summated at a frequency of 1/sec and with an analysis time of 100 ms. The stimulating current intensity was adjusted at 5 times the voltage of the sensory threshold in segmental stimulation and above the motor threshold in peripheral nerve stimulation (<u>11</u>). Usually, SEPs were bilaterally recorded, at least from 3 or 4 cervical segments and from the peroneal nerve and/ or the S₁ segment as a control of the long fiber tracts, regardless of the clinical sensory findings. In the myelopathy cases, multisegmental studies wer done. The nomenclature has been described priorly (<u>3</u>, <u>11</u>, <u>12</u>). SEP alterations were classified according to the following patterns: generalized, regional, polysegmental, monosegmental, uni-

Results

<u>Cervical Myelopathy</u>: Clinically, normal sensory findings were seen in 3 patients. In 8 patients a sensory transectional level or unilateral sensory disturbance was found. Eleven patients showed long fiber tract signs such as spasticity, paraparesis and extensor plantar response.

In the CM group, a high incidence of pathological SEP findings was seen (15/18). Mildly abnormal findings were just as common as more severe findings (Table 2). All anatomical patterns of SEP alterations were found. The two most common patterns were the transectional and the polysegmental pattern (Table 3). In the former, it was possible to find normal recordings several segments below the lesion; in the latter, more than one segment in the *same* region showed SEP alterations.

		Evoked]	potentia	al findings	
	No.	Normal	Change	es	
			Mild	Moderate	Severe
Cervical myelopathy	18	3	8	4	3
Cervical radiculopathy	22	11	10	1	0

Table 2. Degree of SEP alterations

Table 3. Pattern of SEP change

	No.	Nor- mal	Mono- seg- mental	Poly- seg- mental	Re- gion- al	Uni- lat- eral	Trans- ectional	General- ized
Cervical myelo- pathy	18	3	3	5	2	2	6	3
Cervical radiculo- pathy	22	11	7	3	1	0	0	

The correlation between clinical sensory findings and SEP data was good in 4/12 cases. SEP alterations exceeding what would be expected from clinical testing were seen in 8 cases. Alterations less pronounced than clinically expected were seen in 6 cases. Thus, in 12 of the 18 patients with CM, SEP alterations were as marked or more extensive than clinical examination. Three additional points deserve mention regarding the correlation in the CM group:

- 1. In one case of unilateral clinical sensory deficit, the SEP pattern was transectional.
- 2. Of the three clinically normal patients, one had a transverse SEP pattern below $\rm T_{10},$ one showed a monosegmental lesion at C_7 and the third had a normal SEP.
- 3. In two patients with normal SEP findings, there was a hypesthesia involving 2 segments of the affected extremities.

The overall incidence of false negative results is, therefore, 2 out of 18 patients. On the other hand, SEP alterations could be detected in two cases otherwise considered normal.

Control recordings were performed in 9 patients. In two of these, postoperative improvement was seen. No change occurred in 3 and further impairment in 4 (Table 4).

Table 4

		Follow-up SEE	^o recordings	
	No.	Improvement	Impairment	No change
Cervical myelopathy	9	2	4	3
Cervical radiculopathy	12	4	5	3

<u>Cervical Radiculopathy</u>: The majority of these patients showed clinical sensory impairment in a circumscribed area confined to one or more segments. There were no hemisyndromes, no transectional sensory findings, and no long fiber tract signs. In 2 cases sensory testing was normal.

In half the cases with cervical radiculopathy, SEP findings were normal (Table 2). The other 11 showed only mild SEP changes with one exception, in which moderate SEP alterations restricted to the legs were due to a polyneuropathy. The SEP alterations were only of the monosegmental and polysegmental pattern (except in the polyneuropathy patient). The correlation between electrophysiological and clinical (sensory) findings was precise in 9 cases, poor in 8, while in 5 cases SEP alteration was more extensive than the clinical findings.

SEP recordings were controlled in 12 patients, usually during the first postoperative week. No change was seen in 3 cases, impairment in 5 and improvement in 4.

In summary, these findings appear to indicate that, in the radiculopathy group, evoked potentials reveal only focal neurological involvement, either in one or in a number of neighboring segments, indicating the radicular or focal spinal involvement, while, in the CM group, SEP alterations are more common, more severe and of a more varied nature, reflecting the more extensive involvement of spinal cord pathways.

Discussion

The diagnosis of a cervical myelopathy is often difficult. It may be impossible to differentiate between a craniocervical tumor and a case of spinal multiple sclerosis (2, 6, 10). The problem is not made easier by the fact that there are cases of \overline{CM} with and without pronounced osteochondrosis, not to forget that in the age group above 40 years nearly all patients show radiographic signs of osteochondrosis (2). In spondylotic cervical myelopathy, operative measures can be $ta\overline{k}en$ to interrupt the progress of the disease $(\underline{2}, \underline{4}, \underline{9})$. There have been controversial opinions as to which is the best operative procedure. The dorsal approach has its limitations in the remaining ventral spurs, and the ventral approach is limited to two (rarely three) levels. In any case, it is mandatory to obtain as much information as possible in order to pose the proper surgical indication. MATSUKA et al. used the evoked spinal electrogram in a clinical trial, but this appears to be a very cumbersome procedure (8). In cases with pyramidal signs but without sensory deficit, pathological SEPs reveal definite spinal cord involvement, thus supporting the decision to operate. The usefulness of this criterium becomes apparent through the comparison of our two groups, a fact also underlined by JÖRG (6). By using the SEP technique, it is possible to differentiate between purely radicular involvement, polyradicular involvement or transverse spinal cord involvement. This will be helpful for the assessment of the extent and the necessity of an operation as well as of prognosis.

As demonstrated in Fig. 1, a level of spinal cord involvement may be sharply demarcated, indicating the level of the ventral approach. Even in those cases where SEPs show alterations only in one or two segments, despite more severe clinical findings, the maximum damage to the sensory pathways can be pinpointed.

Difficult to explain are the bisegmental SEPs quite common in the radiculopathy group, even if the sensory deficit is in good agreement with one of the affected segments. If, for example, C_5 and C_6 on the same side are altered in the SEP examination, one cannot differentiate between a focal spinal or a biradicular cause for this change. It is impossible so far to differentiate between the root or the cord itself as being the site of the lesion. Myelography or electromyographic examinations have to provide additional information ($\underline{6}$). If there is only one myelographic level and two-level EP involvement, a fairly large disc prolapse may be assumed, causing focal spinal alterations. If there are two myelographic levels and two SEP levels, no differentiation is possible between a focal spinal involvement or two compressed roots.

The evoked potential method does not allow differentiation between purely spondylotic forms of CM and vascular forms of CM. In the spondylotic form of CM, not only mechanic or dynamic-mechanic causes are discussed (2, 5, 6, 7, 9), but a vascular factor is also taken into consideration by some authors (6, 13). Once vascular alterations are present, the probability of a transverse or unilateral SEP alteration pattern is much larger. The combination of mechanical and vascular factors in the pathogenesis of cervical spondylotic myelopathy may be used to explain the uncertain prognosis in postoperative evolution and the postoperative combination of improvement and impairment of the SEP patterns in CM patients.

Conclusions

The clinical application of *segmental* evoked potential studies in cases of cervical myelopathy yields a high percentage of abnormal recordings.

It is possible to differentiate between purely radicular or polyradicular involvement and transverse spinal cord involvement.

Normal evoked potentials exclude a cervical myelopathy with a high degree of probability. Segmental evoked potential studies may be a significant aid in the differential diagnosis between CM and other spinal cord disease.

SEP findings in cervical radiculopathy differ sufficiently from findings in DM as to allow the confirmation or exclusion of long fiber tract involvement.

References

- BAUST, W., ILSEN, H.W., JÖRG, J., WAMBACH, G.: Höhenlokalisation von Rückenmarksquerschnittssyndromen mittels corticaler Reizantwortpotentiale. Nervenarzt 43, 292-304 (1972)
- BRAAKMANN, R.: Cervical spondylotic myelopathy. Advances and technical standards in neurosurgery. Vol. 6, pp. 137-169. Wien, New York: Springer 1979
- FUKUSHIMA, T., MAYANAGI, Y.: Neurophysiological examination (SEP) for the objective diagnosis of spinal lesions. In: Advances in neurosrugery, Vol. 2. KLUG, W., BROCK, M., KLINGER, M., SPOERRI, O. (eds.), pp. 158-168. Berlin, Heidelberg, New York: Springer 1975
- GROTE, W., BETTAG, W., WÜLLENWEBER, R.: Technik und Ergebnisse zervikaler Fusionen. Acta neurochir. <u>22</u>, 1-27 (1970)
- HOFF, J.T., WILSON, C.B.: The pathophysiology of cervical spondylotic radiculopathy and myelopathy. Clin. Neurosurg. <u>24</u>, 474-487 (1977)
- JÖRG, J.: Die cervikale Myelopathie als differentialdiagnostische Erwägung bei Gehstörungen im mittleren und höheren Lebensalter. Nervenarzt <u>45</u>, 341-353 (1975)
- KUHLENDAHL, H., HIRSCHBIEGEL, H., BOCKEM, K.F.: Die klinischneurologische Symptomatik der chronischen cervicalen Myelopathie vertebraler Genese. In: Wirbelsäule und Nervensystem. TROSTDORF, E., STEUDER, H. (eds.), pp. 108-112. Stuttgart: Thieme 1970
- MATSUKA, D. Y., YOSHIDA, M., GOYA, T., SHIMOJI, K.: Classification of cervical spondylosis or disc protrusion by preoperative evoked spinal electrogram. J. Neurosurg. 44, 435-441 (1976)
- 9. ROBINSON, R.A., AFEICHE, N., DUNN, E.J., NORTHING, B.E.: Cervical spondylotic myelopathy: Etiology and treatment concepts. Spine <u>2</u>, 89-98 (1977)
- RICHERT, S., BERNHARDT, W.: Ungeklärte Tetraspastik der Erwachsenen. Fortschr. Neurol. Psychiat. <u>45</u>, 206-222 (1977)
- 11. SCHRAMM, J., HASHIZUME, K.: Somatosensory evoked potentials (SEP) in patients with peripheral, spinal and supraspinal lesions of the sensory system. In: Advances in neurosurgery, Vol. 4. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 250-256. Berlin, Heidelberg, New York: Springer 1977

- 12. SCHRAMM, J., OETTLE, G.J., PICHERT, T.: Clinical application of segmental somatosensory evoked potentials (SEP) - experience in patients with non-space occupying spinal lesions. Proceedings of the Nottingham International Evoked Potentials Symposium, Lancaster, MTP-Press (in print)
- 13. TAYLOR, A.R.: Vascular factors in the myelopathy associated with cervical spondylosis. Neurology (Minneap.) 14, 62-68 (1968)



Fig. 1. a Air myelogram of a 70 year old female with cervical myelopathy. Clinical findings: tetraparesis with tetraspasticity. Sensory level below T₄ for all modalities and isolated hypesthesia in C₆ bilaterally. Small arrows designate normal ventral subarachnoid space. Arrowheads on spurs from C₄ to C₇

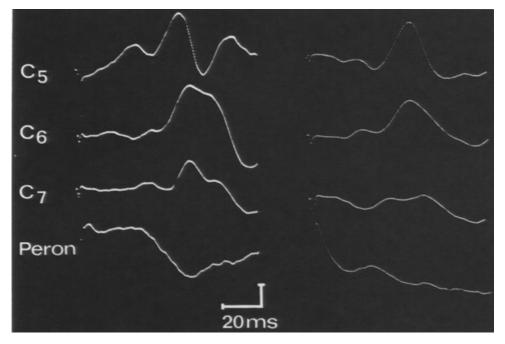


Fig. 1. b SEP from left side in the same patient as in Fig. 1a; left column preoperative recordings, right column 4 months postoperative. Normal C5 tracings in both recordings, latency delay in C6 and more severe change in other recordings show transectional lesion (right-side recordings showed a similar change). The C6 latency is normalized postoperatively, while the other alterations persisted

Measurement of Spinal Elastance in Patients with Spondylogenic Narrowing of the Spinal Canal

H. D. HERRMANN and H. JAKSCHE

To substantiate the obstruction of the spinal subarachnoid space in degenerative deformations of the spine causing a spondylogenic myelopathy, we measured spinal elastance $(\underline{3}, \underline{6})$.

Method

Lumbar puncture is performed in lateral recumbent position. Through a 3-way stop-cock, a syringe (to inject different amounts of normal saline) and a Statham-transducer were connected to the needle. A volume change - ΔV - of 1, 2, 5 and 10 ml was induced. The resultant pressure ΔP and pressure decrease was recorded. The measurements were performed in ante- and retroflexion of the head (4).

Two groups of patients were examined:

- Group 1: Patients with lumbar disc disease but normal cervical spine (control group N = 5).
- Group 2: Patients with cervical spondylosis narrowing of the cervical spinal canal, cervical spondylogenic myelopathy (N = 10).

Results

Elastance curves in the control group: Each patient examined had a higher elastance in retroflexion of the head than in anteflexion (Fig. 1), what proves the physiological narrowing of the subarachnoid space observed in cadavers (1, 2).

The elastance curves of the group with degenerative narrowing of the cervical spinal canal at $\Delta V = 5$ and 10 ml was significantly higher (< 0.01) than in the control group, particularly in retroflexion (Fig. 1), what substantiates the obliteration of the subarachnoid space possibly by a pincers mechanism (5).

The time course of pressure decrease with unobstructed spinal canal showed a slow, strictly monoexponential slope with a mean time constant of 100 sec (Fig. 2).

Sometimes, in retroflexion and at $\Delta V = 10$ ml, a slight secondary pressure increase could be observed, followed by the slow slope (Fig. 3).

Patients with partial obstruction of the spinal canal always showed a pronounced secondary pressure increase in retroflexion, sometimes also in anteflexion (Fig. 4). In cases of severe obstruction a plateau remained. The secondary pressure increase was followed by a biexponential pressure decrease with a fast slope with a mean time constant of 38 sec and the slow slope with the mean time constant again of 100 sec.

We have no proven explanation for the secondary pressure increase.

The slow slope seems to be a physiological parameter and possibly represents the resorption time. The fast slope could represent the pressure drop at a point of stenosis, since it can be influenced by operation.

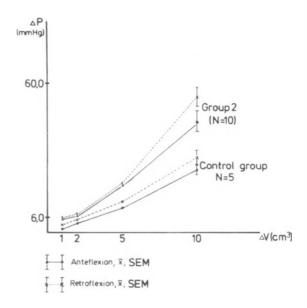
The fast slopes at $\Delta V = 10$ ml of a patient with spondylogenic myelopathy drawn on semilogarithmic scale in Fig. 5 shows that the preoperatively retarded fast slope in anteflexion was markedly accelerated after removal of the spondylotic spurs from the anterior approach. The time constants in retroflexion were unchanged.

Conclusions

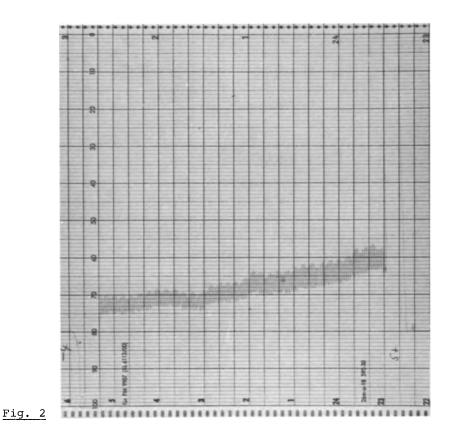
All measurements were well tolerated, without complaints of any discomfort, except for the lumbar puncture. Our results prove the obstruction of the subarachnoid space in ante- or retroflexion of the head. This may allow differentiation between predominantly ventral or dorsal impingement on the subarachnoid space as aid in the decision for ventral or dorsal operative approach in the treatment of spondylogenic myelopathy. Further, this method allows postoperative quantification of the achieved patency of the subarachnoid space.

References

- ADAMS, C.B.T., LOQUE, V.: Studies in cervical spondylotic myelopathy. Brain 94, 557 (1971)
- BRAIG, A.: Biomechanic of the central nervous system. Stockholm: Almqvist and Widsell 1960
- 3. GILLAND, O.: CSF dynamic diagnosis of spinal block: II The spinal CSF pressure-volume curve. Acta neurol. scand. 41, 487 (1965)
- 4. KAPLAN, L., KENNEDY, F.: Effect of head posture on manometrics of cerebrospinal fluid in cervical lesions: New diagnostic test. Brain 73, 337 (1950)
- KUHLENDAHL, H.: Pathogenese der sog. zervikalen Myelopathie, biomechanische und vasizirkulatorische Faktoren. Münch. med. Wschr. 111, 1137 (1969)
- LÖFGREN, J., ZWETNOW, N.N.: Cranial and spinal components of the cerebrospinal fluid pressure-volume curve. Acta Neurol. scand. 49, 575 (1973)







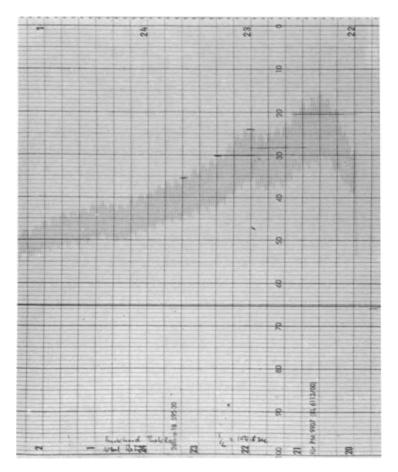


Fig. 3

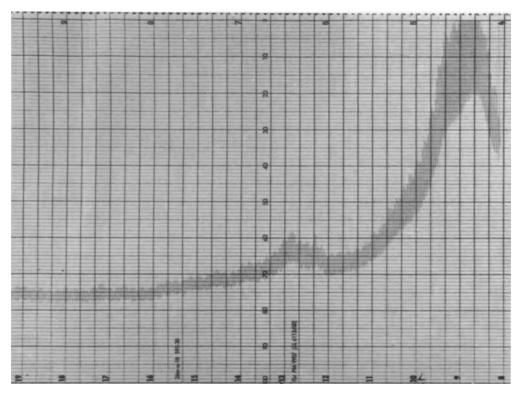
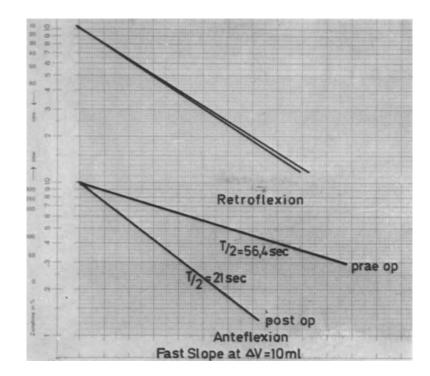


Fig. 4



<u>Fig. 5</u>

37

Indication for Decompressive Laminectomy in Cervical Myelopathy H. ALTENBURG, G. SITZER, W. WALTER, and G. BRUNE

The discussion about surgical indication and best operative approach for decompression in cases of *chronic* cervical myelopathy (CCM) is still in progress (4, 6, 9, 10). In our opinion, neurosurgical reflections on this problem have become considerably more differentiated in the last years $(\underline{8})$.

Cervical myelography with positive contrast media is the method of choice for the differential diagnosis of CCM (5, 7). Clinical and myelographic features of CCM represent the most decisive hints as to indication and best operative procedure.

If a disc protrusion or a spondylosis is limited to two spaces, the treatment of choice is the anterior approach according to CLOWARD. The indication for decompressive laminectomy in CCM is justified if there are multiple ventral or dorsal deformations of the dye column at myelography (Fig. 1) as well as an extensive multisegmental narrowing of the cervical spinal canal, with a sagittal diameter below 12-14 mm.

There is no question that intensive conservative treatment should be performed as a trial before indicating decompressive laminectomy, except for those cases with pain or frank neurological deficits resistant to therapy, which we are forced to operate on as early as possible $(\underline{2})$.

Our operative procedure in the above-mentioned cases consists in a laminectomy, extended to one or two segments above and below the damaged discs associated to a posterior foraminotomy for cervical root decompression, if required. The operation is always performed in the sitting position. Incision of the dura in the midline and cutting of the dentate ligaments follow. Many cases need a dural graft with lyophilized dura in order to widen the intradural space. The main value of extended decompressive laminectomy in the above mentioned form of CCM is, at least, to stop the progression of the disease.

Some patients recover well from myelopathy in the postoperative course. In literature improvement is described in 20-60% of cases. In our experience with 20 cases of CCM, the follow-up results were objectively satisfactory in one third of the patients operated on with the described surgical technique.

As to prognosis and operative results, preoperative duration of the disease, the type of operation, and especially the preoperative neurological condition are the most meaningful parameters (1, 3, 6). The aim of neurosurgical treatment is the earliest differentiated use of the possible operative procedures.

In conclusion, dorsal decompression by a wide laminectomy is indicated in those progressive cases of CCM showing diffuse cervical spondylosis and osteophytes at multiple levels, and multisegmental sagittal stenosis of the cervical spinal canal at myelography and lateral roentgenograms of the cervical column, and showing resistance to conservative treatment.

References

- ALSHARIF, H. et al.: The results of surgical treatment of spondylotic radiculomyelopathy with complete cervical laminectomy and posterior foramen magnum decompression. Acta Neurochir. <u>48</u>, 83-100 (1979)
- BRONISCH, F.W.: Erkrankungen im Bereich der oberen Wirbelsäule aus klinisch-neurologischer Sicht. Med. Welt 25, 1741-1748 (1974)
- FAGER, Ch.A.: Results of adequate posterior decompression in the relief of spondylotic cervical myelopathy. J. Neurosurg. <u>38</u>, 684-692 (1978)
- HAMEL, E. et al.: Cervical myelopathy. Neurosurg. Rev. <u>1</u>, 101-110 (1978)
- HILLEMACHER, A., KÜGELGEN, B.: Zum Wert der seitlichen HWS-Aufnahme bei der Diagnose der chronischen zervikalen Myelopathie. Fortschr. Röntgenstr. 129, 44-46 (1978)
- JEFFREYS, R.V.: The surgical treatment of cervical spondylotic myelopathy. Acta Neurochir. <u>47</u>, 293-305 (1979)
- JÖRG, J.: Die zervikale Myelopathie als differentialdiagnostische Erwägung bei Gehstörungen im mittleren und höheren Alter. Nervenarzt <u>45</u>, 341-353 (1974)
- KAZNER, E., KOLLMANNSBERGER, A.: Differenzierte neurochirurgische Behandlung des zervikalen Bandscheibenvorfalls. In: Neurologische und psychiatrische Therapie. FLüGEL, K.A. (ed.), pp. 102-107. Erlangen: Dr. med. D. STRAUBE 1978
- MURAKAMI, N. et al.: Cervical myelopathy due to ossification of the posterior longitudinal ligament. Arch. Neurol. <u>35</u>, 33-36 (1978)
- PHILLIPS, D.G.: Surgical treatment of myelopathy with cervical spondylosis. J. Neurol., Neurosurg., Psychiatr. <u>36</u>, 879-884 (1973)

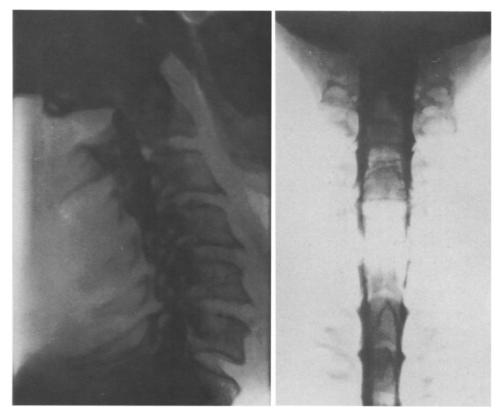


Fig. 1. Myelography with positive contrast medium in multisegmental chronic cervical myelopathy

Neurological Approach to Differential Diagnosis and Indication for Surgery in Chronic Cervical Myelopathy

B. KÜGELGEN, K. LIEBIG, and W. HUK

The pathogenesis of chronic cervical myelopathy still leaves some questions to be elucidated. These are associated with problems encountered in diagnosis, differential diagnosis, therapy and in the prognosis of this disease.

Since 1975, in cooperation with neurosurgeons and orthopaedists, we have been examining patients who were referred to us with the request to determine whether a chronic cervical myelopathy (CCM) was present. We have been able to observe some of these patients for a period of four years. We have tried to clarify the question as to which symptoms and difficulties are typical of a chronic cervical myelopathy, what significance should be attributed to them in establishing a differential diagnosis, when surgery is advisable, and what prospects can be held out for patients who are not operated on.

We have included 56 of our patients in this investigation. - Chronic cervical myelopathy is a disease which affects persons of middle or advanced age (Fig. 1), men being more frequently afflicted than women. It follows an insidious course (Table 1). Among the difficulties encountered there are: gait disturbances, weakness in the legs, diffuse pains and sensations of discomfort (Table 2), whereas radicular disorders are more rare. Spasticity predominates among the findings but disorders may be completely absent (Table 3). It was interesting to note that none of our patients provided reliable clues to an insufficientia vertebrae.

Acute cervical myelopathy, which is predominantly due to by a median disk prolapse or a ruptured intervertebral disk, must be distinguished from the chronic variety. The patients in question are usually younger, the lesion develops considerably faster - immediately or within a few days. Neurologically, the picture is similar to that of a transverse lesion of the cord. X-rays of the cervical spine can provide conclusive information as to the absence of degenerative changes or when a ruptured intervertebral disk can be demonstrated.

It may be difficult to establish differential diagnosis from amyotrophic lateral sclerosis if it starts with spasticity (10% of the cases). Even in CCM, sensory disorders are absent in 27% of the cases, uncharacteristic sensations of discomfort may also accompany amyotrophic lateral sclerosis (AL). The clinical pictures of both follow a similar progressive pattern. Distribution of pareses, electromyographic evidence that the second neuron to the legs is affected, or cranial nerve disturbances, can rule out the diagnosis of CCM. Spinal vascular processes, myelitis, funicular spinal diseases, and disseminated encephalomyelitis can be confirmed by follow-up, clinical examinations and laboratory analyses. Cerebral processes and changes at the cervico-occipital junction can be ruled out by radiography

<u>Table 1</u> . Breakdown by sex $(n = 56)$,	duration of anamnesis			
Men 45				
Women 11				
Duration of anamnesis up to the time	of diagnosis (n = 56)			
2 years and 2 months on average				
<u>Table 2</u> . Complaints $(n = 56)$				
Disturbed micturition	27%			
Gait disturbance 77%				
Weakness in the legs	77%			
Weakness in the arms	46%			

Diffuse pains, sensations of discomfort68%Radicular arm pains39%Lhermitte's sign5%

<u>Table 3</u>. Neurological findings (n = 56)

Paraspasticity	218
Tetraspasticity	66%
Pareses of the upper motor neuron	
Arms only Legs only Arms and legs	9% 21% 18%
Radicular pareses of arms	188
Disturbance of sensory system	
Non-characteristic Postural sensibility Radicular Similar to transverse cord lesion Dissociated Absent	39% 9% 23% 16% 4% 27%

and CT examinations, as also can other space-occupying processes in the region of the cervical column (Table 4).

<u>Indication for Surgery.</u> We have been able to observe 10 patients over periods of at least 6 months, some of them even over years without resorting to surgery, although a number of them was operated on later. Among the patients not operated on, there was only one whose condition deteriorated, 9 had no clearly progressive course or increase in neurological deficit. The longest period of time with constant symptomatology was 4 years (Table 5). (These figures are not representative, but only prove that CCM is not necessarily progressive). LIEBIG et al. will report on the surgical results. All patients have been operated on according to the method of CLOWARD. With the exception of LHERMITTE's sign and disturbed micturition, none showed a substantial improvement after surgery. Most patients reported that their difficulties had diminished. Radicular disorders and deficits, in particular, were found

42

	Course	Clinic	Special examinations
Acute cervical myelopathy	+ +	+	Myelogram? X-ray of cervical spine
Space-occupying spinal process	(+)	+	Myelogram
Anterior spinal artery syndrome	+++++	++++	
Myelitis	++++	+	Liquor
Disseminated encephalomyelitis	+	+	Liquor electrophoresis
Funicular spinal disease	+	+	B12 examinations (Schilling test)
Amyotrophic lateral sclerosis	(+)	+	EMG
Cerebral process, lesion of the cervico-occipital transition	+	+	X-ray, CT

Table 4. Differential diagnoses

Table 5. Progressive course of non-operated-on patients (not representative)

Total Constant Worse	10 patients 9 patients 1 patient	
Period of	observation	1 year 8 months on average at least 6 months 4 years at most

likely to improve. However, patients not submitted to surgery also make varying statements regarding their complaints, some reporting spontaneous improvement.

There is no symptom pathognomonic of CCM. The value of a lateral X-rays of the cervical column is limited, since CCM may also occur with a spinal canal of 14 mm. The myelogram can be typical, but by no means conclusive. In no instance have we noted a complete obstruction to the flow of contrast medium. We have seen three patients with amyotrophic lateral sclerosis who were referred to us as being affected by CCM on the grounds of a typical myelogram.

A diagnosis of CCM must be based on the 4 criteria shown in Table 6. Needless to say, the indication for surgery is ultimately left to the surgeon. If deficits are clearly progressive, we recommend surgery to prevent further deterioration. In the case of radicular disturbances and disorders of micturition, the condition is even expected to improve and the complaints usually diminish. Surgery should not be performed if the medullary deficits, regardless of their extent, are not progressive. Admittedly, it is not easy to appraise spasticity over a period of years, especially when different examiners have been involved and when surgery is accompanied by additional intensive physiotherapy and chemotherapy.

Table 6. Four criteria for the diagnosis of chronic cervical myelopathy

- 1. Typical i.e. slow development
- 2. Neurological finding pointing to the cervical region of the spinal cord
- 3. Positive clues in the myelogram
- 4. Exclusion of another disease

In the case of acute cervical myelopathy, the extent of the deficits and persisting spinal compression will decisively influence the indication for surgery. Here, the results are much better and considerable medullary deficits also improve well. But even among our patients with milder deficits and no longer afflicted by spinal compression, who were not treated surgically, we have noted very good regression. Deteriorations of the neurological findings immediately after surgery of chronic cervical myelopathy are by no means rare and can be compared to the acute cervical myelopathies. They, too, showed good regression tendency without exception. The same applies to cervical radiculopathies.

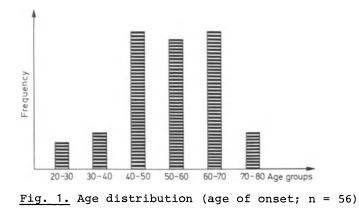
Summary

The most important differential diagnosis of CCM is acute cervical myelopathy or amyotrophic lateral sclerosis if it commences with spasticity. In both cases, differentiation can be very difficult. If the progress of spasticity is evident, we recommend operation for chronic cervical myelopathy as well as for cases with disturbed micturition. Surgery is indicated in cases of positive LHERMITTE's sign and when patients suffer from severe and continuous complaints. As in the case of acute cervical myelopathy, the degree of deficit and persisting root and spinal cord compression are decisive for surgical indication.

Duration of disturbances prior to surgery seems to be of secondary importance only, while the susceptibility of the different neurological functions is a weighty factor. Whether these functions will recover after surgery, will depend on the deficits to which the systems involved are subjected.

References

- GRÜNINGER, W.: Die chronische Myelopathie im höheren Lebensalter. Habilitationsschrift 1977, Würzburg
- HILLEMACHER, A., KÜGELGEN, B.: Zum Wert der seitlichen HWS-Aufnahme bei der Diagnose der chronisch zervikalen Myelopathie. Fortschr. Röntgenstr. <u>129</u>, <u>1</u>, 44-46 (1978)
- KAZNER, E., KOLLMANNSBERGER, A.: Differenzierte neurochirurgische Behandlung des zervikalen Bandscheibenvorfalls. In: Neurologische und psychiatrische Therapie. FLüGEL, K.A. (Hrsg.). Erlangen: perimed Verlag 1978
- KESSLER, J.T.: Congenital narrowing of the cervical spinal canal. J. Neurology, Neurosurgery, Psychiatry 38, 1218-1224 (1975)
- KUHLENDAHL, H., FELTEN, H.: Die chronische Rückenmarksschädigung spinalen Ursprungs. Langenbecks Arch. und Dtsch. Z. Chir. Bd. <u>283</u>, 96-128 (1956)
- MARTIN, G.: Cervical spondylotic myelopathy in wide canals. New Zealand Med. J. 85, 475-476 (1977)
- PESERICO, L., UIHLEIN, A., BAKER, G.S.: Surgical treatment of cervical myelopathy associated with cervical spondylosis. Acta Neurochir. <u>10</u>, 214-275 (1962)
- RITTER, G., HOPF, H.C.: Die zervikale Myelopathie. Akt. neurologie <u>3</u>, 79-89 (1967)
- THOMALSKE, G., WILD, K.v., LAMMERT, E.: Zur chirurgischen Behandlung der zervikalen Myelopathie. Nervenarzt 43, 520-524 (1972)



Spinal Symptoms Accompanying Cervical Root Compression Syndromes

A. FANTIS

The diagnosis of cervical myelopathy and the indication for surgery are mainly based on the evidence of a spinal cord compression by spondylotic spurs. During our 15 years experience in the neurosurgical treatment of cervicobracchialgias, we have observed the occurence of medullary and vertebrobasilar symptoms in cases of common cervical spinal root compression. Although clinically they evoked suspicion of cervical myelopathy, a spinal cord compression could not be prooved by myelography. There we - re pyramidal signs on the legs, spinal hemiparesis, often combined with sensory disturbances such as haemihypaesthaesia, segmentary "tricot-like" hypaesthaesia on the trunk, patchy hypaesthaesia ("quadrant syndrome"), and trigeminal irritation. Moreover, vertebrobasilar signs with vestibular disturbances and syncopes occured. These symptoms we characterised as "concomitant spinal syndrome" of cervical spinal root compression.

- In the present paper we pose the following questions:
- 1. How often did we meet this "concomitant spinal syndrome" in our patients.
- 2. Which pathophysiological mechanism could be responsible for the mentioned symptoms.
- 3. How far could these signs and symptoms be treated surgically.

Material and Methods

A group of 300 personal cases of cervical root compression has been evaluated statistically. All patients have been operated on. In the majority we performed a decompressive foraminotomy, by posterior approach, either of one or more intervertebral canals. The indication for foraminotomy was based on the neurological and electromyographic examinations as well as on the correlation with X-ray findings. On suspicion of myelopathy Pantopaque or Amipaque myelograph was performed and, sometimes, vertebral angiography. The technique of decompressive posterior foraminotomy was a standard one (1). During the last five years we have refined the method and the foraminotomy is performed exclusively by microsurgical technique. The skin incision is about 3 cm, the spinous process is marked praeoperatively by means of methylen blue-contrast dye mixture percutaneously injected under radioscopic control. The decompression is made with a high speed surgical air drill, and the osteophytes are removed. The perineural sheats are kept intact and a resection of the whole uncinate process is not performed. The most frequent radicular syndromes were at C6 and C7. We operated in sitting position under general endotracheal anaesthesia.

Results

In our study of 300 foraminotomized patients, there were 116 females and 184 males. The average age of the patients was 50.5 years. We observed the "concomitant spinal syndrome" in 78 patients (26,0%) without myelographic proof of spinal cord compression. Following cervical root decompression a significant improvement of subjective complaints and objective findings was observed in 55 patients (70,5%). In two cases severe praeoperative syncopal attacs ceased after the simple cervical root decompression by means of foraminotomy (Table 1).

Table 1, Clinical material and results

Cervical root compression

300 patients (194 males, 116 females)

Concomitant spinal syndrome

78 patients (26,0%)

Improvement of spinal signs following decompressive foraminotomy

55 patients (70,5%)

Discussion

Cervical spinal root decompression, according to our experience, was capable to alleviate the "concomitant spinal syndrome" in a significant number of cases (Fig. 1). Now the result of this decompression can be understood as a model test with variation of only one condition: decompression in the intervertebral canal. With other operative techniques, e.g. CLOWARD's operation it is not a selective procedure. In our opinion, the origin of the pathological findings is to be seen in the simultaneous compression of the spinal root and the radicular vessels within the narrowed intervertebral canal, followed by a localized circulatory disturbance of the respective spinal region.

The important contribution of the radicular arteries to the spinal cord circulation has been demonstrated experimentally in the monkey (3) and by retrograde angiography $(\underline{2})$. The anterior and posterior cervical spinal arteries are supplied, along their course, by up to 6 radicular arteries from both sides. They originate from the verte-bral artery and cervical vessels. When an important radicular artery becomes functionally excluded (e.g. compressed) and the arteries above and below in the spinal cord are insufficient to compensate the circulatory deficit, an area supplied by this artery becomes "a distant field beyond the reach of the irrigation system" (6). Intermittent circulatory disturbances and spinal cord ischaemia may result from a compression radicular as well as of intramedullary vessels (4). The quick remission of signs and symptoms following foraminotomy, in correspondence with the improvement of the radicular compression syndrome speak in favor of the vascular genesis of the above mentioned syndrome. We suppose that the compression of a radicular branch in the intervertebral canal is not without vasomotor effect on the vertebral artery itself. The influence of simple decompression in the intervertebral canal upon vertebrobasillary disturbances in some cases makes this probable.

Conclusion

On the basis of our experience in 300 cases we should like to point out that the simultaneous chronic compression of the cervical spinal root and of the radicular artery in the narrowed cervical canal can lead to direct as well as remote reversible circulatory disturbances in the cervical spinal cord and cause the development of the "concomitant spinal syndrome" in ordinary cervical root compression. This syndrome may simulate the cervical myelopathy or other form of cervical spinal cord compression. By means of a simple decompressive foraminotomy, without major operative procedures, a significant and quick improvement can be reached with remarkable remission tendency of signs and symptoms.

References

- FANTIS, A.: Contribution to the surgical treatment of cervicobrachial syndrome. Cs. Neurol. <u>28</u>, 397-402 (1965)
- FORTUNA, A., La TORRE, E., OCCIPINTI, E.: The direction of the blood flow in the cervical cord. Eur. Neurol. <u>5</u>, 335-342 (1971)
- FRIED, L.C., DOPPMAN, J.L., Di CHIRO, G.: Direction of the blood flow in the primate cervical spinal cord. J. Neurosurg. <u>33</u>, 325-330 (1970)
- 4. JELLINGER, K.: Pathomorphological aspects of the biomechanics of the C.N.S. Proc. German Soc. Neurosurg. <u>3</u>, 27-35 (1973)
- 5. TURNBULL, I.M., BREIG, A., HASSLER, O.: Blood supply of cervical spinal cord in man. J. Neurosurg. 24, 951-965 (1966)
- TURNBULL, I.L.: Blood supply of the spinal cord: Normal and pathological consideration. Clin. Neurosurg. <u>20</u>, 56-84 (1973)

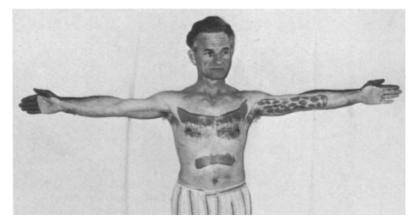


Fig. 1. A case of bilateral compression of the spinal root C6. "Concomitant spinal syndrome" with "tricot like" hypaesthesia of the trunk. Radicular hypaesthesia C6. Hyperpathy on the forearm (*dotted area*) Anterior Disc Surgery in Cases of Cervical Myelopathy A. FRANCO and B. MATRICALI

With the use of Amipaque better myelographic diagnosis in the region of the cervical spinal cord has become possible. Leaving aside patients with radicular pain because of a cervical disc herniation, we want to consider those who show signs and symptoms of spinal cord compression. During the last two years we have operated upon 16 patients with clear signs of medullary compression produced by a cervical slipped disc. The first three were first submitted to decompression laminectomy. Because of the impossibility of eliminating the cause of the compression by a posterior approach, all patients underwent a new myelographic study with Amipaque, in which a clear view of the pathological disc was obtained. At the second operation the disc was removed by an anterior approach.

The first patient, a 71 year old man (A.Z. v.d.L. NCH 19470/78), was admitted to our clinic with progressive quadriplegia. Lumbar puncture revealed a block with the neck in retroflexion. Myelography showed a stop of the contrast medium at C 4-5. Decompression was performed by posterior laminectomy. The neurological condition improved but not sufficiently. A control myelography was performed. Here a clear disc was observed, and the patient operated upon by an anterior discectomy. He recovered well from his quadriplegia. The second and third patients are identical cases and, therefore, we will consider only the myelographic studies pre- and post-laminectomy. In all patients the radiograms were made in prone position with moderate hyperextension of the neck.

Because we disliked this double procedure, a more extensive myelographic study was made before surgery. Today, if we find a myleographic block, we continue the study even with anteflexion of the neck. In this way we can visualize one or more discal impressions. In the case of a normal result, however, the examination must be repeated. If the cervical myelopathy is caused by a disc herniation, this must be demonstrated. The patient to be discussed next is a 38 year-old man (A.Z. W. NCH/79/ with progressive quadriplegia caused by a perforated disc at C 5-6 confirmed at surgery. Myelography demonstrated this block. However, at anteflexion the contrast medium passed without showing the responsible disc, even in a stratigraphic examination. A second cervical myelography was performed and in one tomogram the perforated disc was visible. The patient was operated upon by the anterior approach, and the slipped disc removed. The postoperative course was uneventful with a clear neurological improvement.

Anterior cervical disc removal is an easy and safe procedure. The operation is performed with the aid of a loupes (2X) for a slight magnification, and with the help of a cold headlight. This gives a maximal view of the intervertebral space and the posterior longitudinal ligament, and free movement of the surgeon's head. Entire

procedure must be performed under fluoroscopic control. No fusion is necessary if only one disc is removed, since the stability of the cervical spine is untouched. Postoperative bed rest is not necessary and a quick mobilization and dismissal from hospital are possible.

In conclusion, we advocate the necessity of a good preoperative myelography. If the signs and symptoms are those of a slipped disc, but myelographic findings are normal, this examination has to be repeated with a stratigraphic study. Fortunately the use of Amipaque gives us an opportunity of doing this without contaminating the patient with iodine oil solution.

References

- ARONSON, N., BAGAN, N., FILTZER, D.L.: Result of using the Smith-Robinson approach for herniated and extruded cervical disc. J. Neurosurg. <u>32</u>, 721-722 (1970)
- ARONSON, N.I.: The management of soft cervical disc protrusions using the Smith-Robinson approach. Clin. Neurosurg. <u>20</u>, 253-258 (1973)
- BOHLMAN, H.H.: Cervical spondylosis with moderate to severe myelopathy: treatment of anterior cervical discectomy and fusion. Presented at Cervical Spine Research Society, Philadelphia, Pennsylvaina, November 20, 1976
- 4. CONNOLLY, E. S., SEYMOUR, R.J., ADAMS, J.E.: Clinical evaluation of anterior cervical fusion for degenerative cervical disc disease. J. Neurosurg. <u>23</u>, 431-438 (1965)
- EPSTEIN, CARRAS, LAVINE, EPSTEIN: The importance of removing osteophites as part of the surgical treatment of myeloradiculopathy in cervical spondylosis. J. Neurosurg. 30, 219-226 (1969)
- GALERA, R.G., TOVI, D.: Anterior disc excision with interbody fusion in cervical spondylotic myelopathy and rhizopathy. J. Neurosurg. 28, 305-311 (1968)
- 7. HANKINSON, H.L., WILSON, C.B.: Use of the operating microscope in anterior cervical discectomy without fusion. J. Neurosurg. <u>43</u>, 452-456 (1975)
- HIRSCH, C.: Cervical disc rupture: diagnosis and therapy. Acta Orthop. Scand. <u>30</u>, 172-186 (1960)
- 9. JOMIN, BOUSQUET, DELANDSHEER, LAINE: Traitement des complications radiculo-médullaires de la cervicarthrose par la méthode de Cloward résultats à propos de 370 malades opérés. Neuro-chirurgie <u>21</u>, 21-28 (1975)
- MAYFIELD, F.H.: Cervical spondylosis: a comparison of the anterior and posterior approaches. Clin. Neurosurg. <u>13</u>, 181-188 (1965)
- 11. ROBERTSON, J.T.: Anterior cervical disc removal with and without fusion. Presented at the 33rd Annual Meeting of the American Academy of Neurological Surgery, Lake Tahoe, Nevada, September 29, 1971
- VERBIEST, H.: A lateral approach to the cervical spine: technique and indications. J. Neurosurg. 28, 191-203 (1968)
- WILSON, D.H., CAMPBELL, D.D.: Anterior cervical discectomy without bone graft. J. Neurosurg. 47, 551-555 (1977)

- 14. HENRY, A.K.: Extensile exposure. 2nd ed., pp. 58-72. Baltimore: Williams & Wilkins Co. 1957
- 15. SCHMIDEK, H.H.: Operative Neurosurgery, pp. 303-321. G&S. 1977

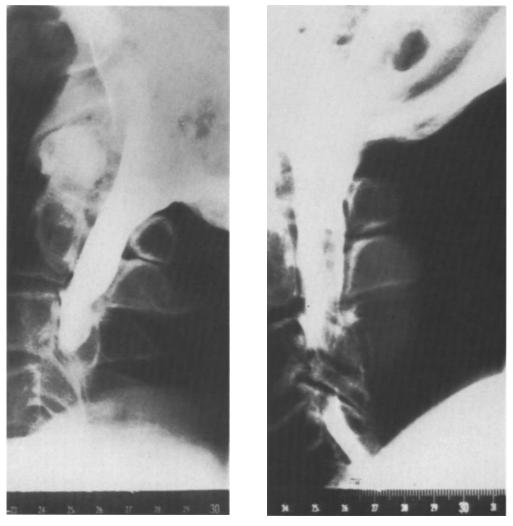


Fig. 1 (left). Radiograms showing a complete block at c 3-4 with the head in slight hyperextension

Fig. 2 (right).Following laminectomy, Amipaque passed along the decompressed area while the partial block caused by a disc protrusion is clearly visible

The Ventral Resection of the Cervical Vertebral Body B. ARMENISE, M. CANCELLIERE, and A. MONTINARO

Introduction

For the treatment of certain pathological and traumatic fractures, we have employed subtotal ventral resection (VSR) of the cervical vertebral body, replacing it with bone graft or acrylic. This was deemed necessary in order to obtain a definite stability of the cervical spine; to relieve any impingement or potential impingement on the neural structures and, finally, to favour a rapid mobilization of the patient. This method has been employed individually or along with the well known ventral and/or dorsal surgical methods. We have been able to observe that the surgical procedure will depend on a considerable number of variable anatomical and clinical situations. The theoretical surgical method may be subject, case by case, to intraoperative variations and adjustments, due to specific aspects of the lesion which may arise during surgery and which were not obvious in the preoperative X-rays.

Acrylic is already commonly used as an alternative to the bone graft in the ventral approach of cervical intervertebral disc (2) or of neoplasia affecting the cervical vertebral body (1). We are convinced that because of its adaptability and texture, acrylic has many advantages if compared to bone graft, namely faster application, sparing of the iliac crest or the tibial bone, satisfactory long-term stability, and proper alignment of the vertebral segments. All these advantages will permit a rapid mobilization of the patient.

We felt the need for a VSR of the cervical vertebral body after the *first case* we treated in June 1975. It was a 21-year-old boy, L.R. (Case 1) with a C5 fracture-dislocation due to diving in shallow water. Two days after the posterior fusion from C4 to C7 by means of wire and acrylic we observed a progressive worsening of the tetraparesis. X-rays showed posterior dislocation of the right hemivertebral body of C5. At the ventral intervention we carried out a VSR of the vertebral body, which was fractured in several fragments (not visible in the X-ray film), as well as the complete removal of the intervertebral disc and filling of the empty space with acrylic. The postoperative course was normal, with regression of neurological signs, except for a bilateral hyperreflexia. After 4 years there still is a good stability of the cervical spine.

Case Reports

<u>Case 2:</u> C.E., age 21. Fracture-dislocation of C-6 due to diving in shallow water. The neurological examination showed a complete cord syndrome. The patient was operated upon on 28 July, 1975 by means of a VSR of the C6 body, and replacement with acrylic. Following another

dislocation, due to spontaneous rapid mobilization, a posterior wire fixation was performed. Neurological situation unchanged.

<u>Case 3:</u> M.O.R., age 52. Fracture of the body and posterior arch of C5 due to a fall from a tree. Neurological examination showed paresis of the upper limbs. On October 8, 1975, he was operated upon. We performed a VSR of the C5 body and replacement with acrylic and, subsequently, a posterior fixation with wire. After 3 1/2 years a left C7-C8 hypoaesthesia is still evident.

<u>Case 4:</u> B.R., age 21. Fracture of the C5 body due to diving in shallow water. Neurological examination showed an incomplete spinal cord syndrome. The patient was operated upon on June 28, 1977: VSR of the C5 body and replacement with acrylic. A year after the intervention the patient showed an incomplete Brown-Séquard syndrome from D3 downwards.

<u>Case 5:</u> R.F., age 38. Pathological fracture of the C5 body due to myeloma. Left C5-C6 motor radicular syndrome. Operated upon on February 21, 1978, by means of VSR of C5 body and replacement with bone graft taken from the iliac crest. Metal plate between C4 and C6, with wiring between the plate and the bone prosthesis. During a checkup, a year and half later, the neurological examination was normal and the patient had a very good stabilization of the spine (Fig. 1).

<u>Case 6:</u> J.S., age 53. Fracture-dislocation of C5 due to a fall from a tree. The neurological examination showed paresis of the upper limbs and spasticity of the legs. Operated upon on April 11, 1978: VSR of the C5 body and replacement with acrylic. After 8 days, due to a spontaneous rapid mobilization, another C5-C6 dislocation appeared. The patient was reoperated ventrally with application of a metal plate. After 1 year there were no neurological signs.

<u>Case 7:</u> C.C., age 40. Fracture-dislocation of C6 due to a car accident. Neurological examination showed paresis of the upper limbs. Operated upon on July 27, 1978: VSR of the C6 body and replacement with acrylic. Neurological improvement after the operation. After 90 days, flaccid tetraplegia, with no alteration of sensitivity. Myelography revealed a diffuse arachnoiditis also extended to the lumbar segments. Exitus occurred due to sepsis (Fig. 2).

<u>Case 8:</u> N.V. age 20. Fracture of C6 due to a car accident. Neurological examination showed an upper limbs paresis, more evident on the right. Operated upon on September 19, 1978 by means of a VSR of the C6 body and replacement with acrylic. Neurological normalization throughout the 7-month check.

The next two cases concern patients suffering from myelopathy due to cervical spondylosis affecting two contiguous discs with deformation of the intermediate vertebral body. They were operated upon by the Cloward procedure. Yet, there was a thin residual central bone portion of the vertebral body, unable to provide support to the bone graft or acrylic. Therefore, a VSR of the vertebral body and replacement of the remaining cavities with bone graft (case 9) or acrylic were carried out (Case 10).

<u>Case 9:</u> C.A., age 74. Myelopathy due to cervical spondylosis. Spastic tetraparesis and hyperreflexia. Operated upon on May 18, 1978 by the above mentioned method. Neurological normalization; 1 year later only exaggerated deep tendon reflexes.

<u>Case 10:</u> C.M.C., age 57. Myelopathy due to cervical spondylosis. Spastic tetraparesis. The patient was operated upon on January 27, 1979 as mentioned above. Five months later there was improvement of the tetraparesis and spasticity.

Discussion

All operations were carried out with the patients under skeletal traction by means of Crutchfield tongs. Skin incision was along the anterior margin of the right sternomastoid muscle. Skeletal traction was maintained until the acrylic had polymerized or the bone graft inserted. VSR of the vertebral body was carried out by means of a microdrill. The upper and lower intervertebral discs were removed. The spinal dura mater was protected with Surgicel® and Spongostan® before introducing the acrylic. We have been particularly careful with the fixation of the acrylic or bone graft between the remaining side fragments of the vertebral body and between the upper and lower vertebral body. We have used the following methods, depending on the anatomical and pathological conditions (Figs. 3, 4):

- Metal plate ventrally attached to the upper and lower vertebral bodies, and wire between the plate and the acrylic or bone prosthesis (cases 5 - 6, Fig. 3B).
- 2. Small microdrill excavation of the upper and lower vertebral displacement (Fig. 3A). A wire is introduced between the excavations. It is then surrounded by the acrylic so as to make the position of the prosthesis visible (cases 1, 2, 3, 4, 8; Fig. 3A).
- 3. The acrylic is fixed by means of wiring between the upper and lower vertebral bodies (case 7, Fig. 4A). A variant of this method was adopted in case 9, in which the wiring included the remaining posterior portion of the vertebral body (case 9, Fig. 4B).

The cases here described are due to various causes such as trauma (7 cases), spondylotic myelopathy (2 cases) and neoplasia (1 case). In two cases autologous bone graft was employed (cases 5 and 9), whereas in the remaining 8 VSR of the vertebral body and acrylic filling were carried out. The follow up period varied from 4 years to 5 months (until June 1979). This follow-up period appears to be sufficiently long so as to consider the validity of our procedure. Control neurological examination was normal in 3 patients, unchanged in 1, whereas in 5 patients it improved considerably, showing a slight deficit which did not affect the working capacity. One patient died from diffuse arachnoiditis and sepsis. In all cases cervical alignment was stable, with no pathological angulations, as shown in the X-ray controls.

<u>References</u>

- CROSS, G.O., WHITE, H.L., WHITE, L.P.: Acrylic prosthesis of the fifth cervical vertebra in multiple myeloma. J. Neurosurg. <u>35</u>, 112-114 (1971)
- ROOSEN, K., GROTE, W., BETTOG, W.: Komplikationen zervikaler ventraler Fusionoperation. Neurochirurgie 18, 1-11 (1975)

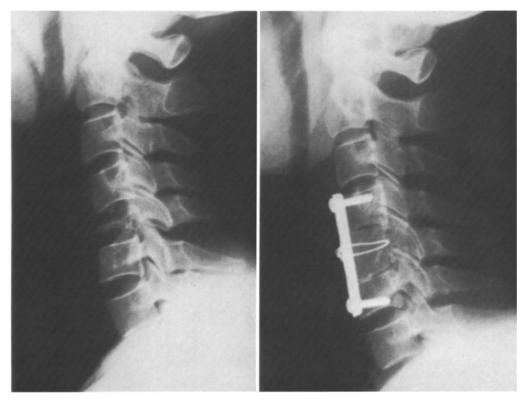


Fig. 1. Preoperative (*left*) and postoperative (*right*) lateral X-ray films showing the pathological fracture of the C5 body due to myeloma, and the metal plate between C4 and C6, with wiring between the plate and the iliac bone graft

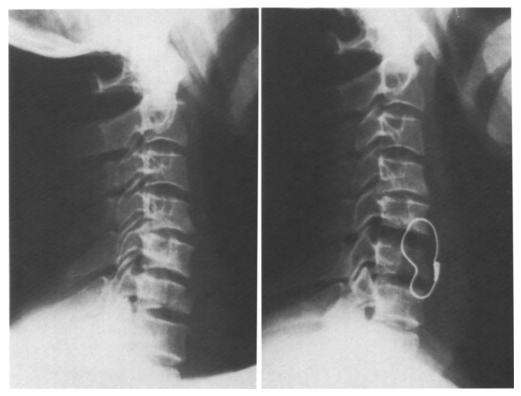


Fig. 2. Preoperative (*left*) and postoperative (*right*) lateral X-ray films showing the fracture of the C6 body and the wire fixing the acrylic between the upper and lower vertebral disc

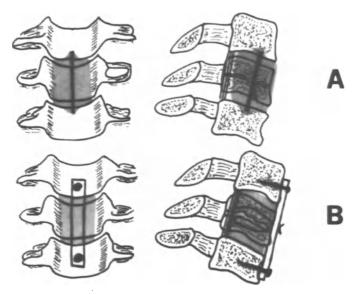


Fig. 3. Drawing shows two procedures adopted: <u>A</u> Small excavations are made by means of the microdrill in the upper and lower vertebral bodies, a wire is introduced between the excavations and thereafter surrounded by the acrylic. <u>B</u> Metal plate with wire between the plate and the acrylic or bone prosthesis

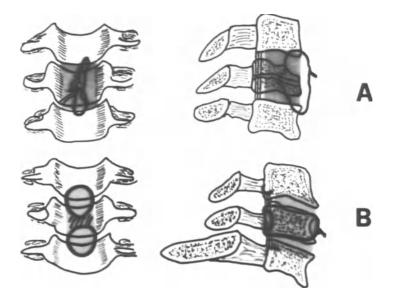


Fig. 4. Drawing shows the other procedures adopted: <u>A</u> The acrylic is fixed by means of wiring between the upper and lower vertebral bodies. <u>B</u> The wiring includes the posterior portion of the vertebral body

Ventral or Dorsal Surgical Approach in Chronic Cervical Myelopathies? G. THOMALSKE, W. BRAUNSDORF, and W. GALOW

Introduction

The discussion about the best surgical approach for decompression in cases of cervical myelopathy still persists. A comparison of operative results is difficult, due to the conflicting views of different surgeons. Therefore, we thought it interesting to compare our own cases operated upon by dorsal or ventral approach.

Case Material and Method

From 1954 to 1978 132 cases of cervical cord or root compression (excluding traumatic and tumor cases) were operated upon in our clinic (Table 1).

Approach	Cervical disc lesion	Cervical myelo- pathy	- Total	
Dorsal	4	77	81	
Ventral (CLOWARD)	36	15	51	
Total	40	92	132	

Table 1. Operations on the cervical column (excluding traumatic cases) - 1954-1978 (N = 132)

Forty operations were performed for cervical disc herniation alone, 92 for cervical myelopathy. In the last-mentioned group 23 patients presented an additional intervertebral disc prolapse. Eighty-one patients were operated upon by dorsal and 51 by ventral approach. The dorsal procedure comprised a laminectomy, including at least one segment above or one below the radiological findings. The dura was opened and the denticulate ligaments were dissected. A decompressing duraplasty (fascia lata or lyophilisied dura) was performed to enlarge the intradural space. The ventral operations were performed in typical way by CLOWARD's method. Curettage of the intervertebral space without fusion was performed only in exceptional cases. As to the extent of decompression in our 92 patients with cervical myelopathy see Table 2.

Maximal decompression comprised 6 segments. Catamnestic data were obtained from 110 of 132 patients operated upon (Table 3). <u>Table 2</u>. Number of segments operated upon in cases of cervical myelopathy (N = 92)

Approach	1 segment	2 segments	3 segments	4 segments	5 segments	6 segments
Dorsal		12	40	17	7	1
Ventral	9	6				

Table 3. Follow-up data (questionnaire and clinical checkup) (N = 110)

Approach	Cervical disc prolapse	Cervical myelopathy	Total	No information
Dorsal	4	62	66	15
Ventral	29	15	44	7
Total	33	77	110	22

These data are from 66 patients with dorsal and 44 patients with ventral approach. Thirty-three had a prolapsed cervical disc and 77 a cervical myelopathy. Nearly 1/3 of the patients followed up were female and 2/3 were male. Nine of the patients operated upon by dorsal route for cervical myelopathy had an additional foraminotomy. The average age was about 46 years (14 to 74 years at surgery). The single case operated upon at the age of 14 had a compression of the spinal cord by a congenital malformation at the cranio-cervical level in presence of a MORQUIO-Syndrome.

Surgical results were compared with the aid of a neurological examination and an interview by questionnaire. Special attention was attributed to the self-assessment of postoperative improvement by the patient himself (% of improvement in comparison to the preoperative condition). The questionnaire comprised questions about the pre- and postoperative evolution and clinical condition. A part of it, dealing with the pre- and postoperative clinical examination was filled in by the medical staff.

The present report includes only 77 patients with cervical myelopathy, 62 of whom were operated upon by dorsal and 15 by ventral approach.

Results

One should take into account that such a comparison may not be completely correct since nearly all cases with cervical spondylosis and osteophytes at multiple levels were chosen for a dorsal approach. Such patients often showed a considerably more marked clinical picture. We have chosen the ventral approach mainly in cases with compression at one or two levels only. The chances of improvement seem to be better in these patients. On the other hand, the small number of patients operated upon by ventral approach permits only a limited statistical assessment.

Concerning the dorsal approach, we found no complete relief by the operation in our 62 cases (Table 4). In 24 cases (more than 1/3)

Table 4. Postoperative clinical checkup in cervical myelopathy (N = 77)

Approach	Relief	Improved	No progression	Worse	N = 77
Dorsal	ø	24	20	18	62
Ventral	3	9	3	Ø	15

there was an *improvement*, in 20 cases (about 1/3) *no progression*, and in 18 cases (less than 1/3) *deterioration* of the preoperative condition. In the 15 cases with ventral approach, we observed *complete relief* in 3, *improvement* in 9 and *no progression* in 3. This means that the rate of improvement (relief and improvement) for the dorsal approach was about 2/5 smaller than for the ventral route.

Keeping in mind the lack of statistical significance by reason of the small number of cases, we found an improvement in about 38,7% operated upon by the dorsal approach and in 80% operated upon by ventral approach. If the postoperative *standstill* of this nearly always progressive illness is also considered as a good result, an acceptable result can be observed in 71% of the cases operated upon by in 71% and in nearly all cases operated upon by ventral approach.

Deterioration was observed only following the dorsal approach. But this procedure, as lined out above, was more or less preferred in cases with more pronounced clinical findings.

As to *lethality*, we found immediate postoperative deaths only in dorsally operated patients (Table 5).

Table 5. Lethality in operated patients with cervical myelopathy (N = 21)

				_
Approach	Immediately postoperative	Late	Total	_
Dorsal	5	12	17	
Ventral	Ø	4	4	

Immediate lethality of dorsal operation is about 1/3 of all deaths following the dorsal operation. This corresponds to 8% of all patients with cervical myelopathy operated upon by the dorsal route. Following the ventral approach we observed only late lethality from unrelated causes.

Discussion

Concerning preoperative *clinical symptomatology* we should like to stress the following cases:

- One case with osteophytic protrusion from C4 to C7 had cough syncopes, which also existed postoperatively, but were less frequent.
- Two cases with LHERMITTE-syndrome were operated upon at the segments C 3/4 respectively C4 to C7 by dorsal approach. Postoperatively this sign was still present.

- One case, having had a laminectomy from C4 to C7 suffered from generalized vascular disease, which required bilateral aortoiliac bypass for bilateral stenosis of the iliac artery.
- Of sixteen cases with tetraspasticity none improved.
- In another 16 cases with paraparesis, 3 were flaccid and 13 spastic. Flaccid paraparesis showed better postoperative improvement than the spastic ones.
- Two BROWN-SEQUARD-syndromes were observed. Both still presented this syndrome following operation.
- Twenty-two patients had marked gait disturbances due to paraparesis, hemilesions, spasticity or ataxia.
- Five cases suffered from preoperative vertebral vertigo, which remained unchanged.
- Seven patients had disturbed micturition and defecation, which improved in 2 cases.
- Four cases suffered from typical "migraine cervicale", which improved by surgery in 2.

Pathologic-anatomical particularities were found in the following patients operated upon by dorsal route:

- In 1 patient with myelopathy due to an osteophytic protrusion at C5/6, a meningioma was found to be the cause of a stop myelographic at the C1/C2 leve. This patient had been operated upon by ventral approach in another hospital, but her condition deteriorated and she was admitted to our clinic. Certainly these two pathogenetic components together were responsible for the clinical symptoms.
- One case with an osteophytic protrusion at C7/Th1 had an epidural cyst at the same level.
- In 2 cases a pathological thickening of the arachnoid was found, once of unknown origin, once probably due to a lues cerebrospinalis.

The following *complications* were observed:

- a) Intraoperatively, due to anesthesia: a herniated cuff of the intratracheal tube in once case.
- b) Additional postoperative symptoms:
 - 1. Tetraparesis due to a transverse lesion on one patient.
 - 2. Impotentia coeundi in 5 cases following a dorsal approach, and in 3 patients following ventral approach.
 - 3. Tetraspasticity in one case.
 - 4. BROWN-SÉQUARD-syndrome in one case.
 - 5. Gait disturbances in four cases.
 - 6. Disturbances of micturition in 3 cases.

Following dorsal decompression there never was the necessity to operate again by ventral approach, but in 3 cases we had to decompress by dorsal approach after a CLOWARD operation.

An obvious advantage of the ventral operation in cervical myelopathy seems to be indicated by the above tables. For the above mentioned reasons, this advantage is only relative because the group operated upon by dorsal route includes more severe cases. Fifteen of them were suspected to have a medullary vascular process from their preoperative clinical symptoms. Moreover, the group operated upon by dorsal route, the number of patients with distinct gait disturbances was approximately the double of that in the CLOWARD group.

On the other hand, the average age of the group treated by dorsal approach was markedly higher (50 : 41 years). With respect to the biomechanical pathogenetic factors of cervical myelopathy, the indication for ventral or dorsal approach should be related to the extent and the severity of the lesions in the future too.

Patients with plurisegmental lesions, especially in presence of a narrow spinal canal, should be operated upon by the dorsal approach. Patients with lesions in one or two segments only, particularly when combined with disc herniation and with a more wide spinal canal, should be preferred for ventral approach.

Only a prospective study comprising patients with multiple extensive lesions leading to chronic cervical myelopathy, also operated upon by anterior approach on more than two levels, would permit more significant conclusions as to the advantage of the one or the other method.

Operative Results in the Treatment of Cervical Spondylotic Myelopathy

C. ARIENTA, G. DE BENEDITTIS, L. INFUSO, and R. VILLANI

Introduction

Progressive myelopathy due to cervical spondylosis is a recognized clinical entity. For many years the accepted alternative to conservative treatment has been surgery. The standard cervical approach has been via the posterior route, performing decompressive laminectomy, foraminotomy or dentate attachments' division in varying degree and combination. The anterior approach has been devised more recently as an alternative procedure whenever the pathologic condition in cervical spondylosis lies anterior to the spinal canal and in single-level lesions. Indications and limits of these two major surgical procedures have been extensively discussed in literature, whereas follow-up of patients operated upon have seldom been reported in detail (13, 7, 9, 1, 5, 11, 10, 6).

Material and Method

This report considers immediate and long-term operative results of 75 consecutive patients in the period 1967 - 1978. A higher incidence was found among men - 55 (73%) - as in series reported in the literature. The age of the patients ranged from 37 to 74 years: 72% were in the Vth and VIth decade. The duration of symptoms varied from 1 month to 7 years (mean duration: 3 years).

Pre-operative clinical features related to cervical syndromes are shown in Table 1: progressive myelopathy was present in 33 cases (44%), radiculopathy in 13 patients (17%), whereas evidence of combined impairment of spinal cord and roots was found in 29 instances (39%).

	No. of cases	Severe	Moderate	Mild
Radiculopathy	13	-	7	6
Myelopathy	33	18	15	-
Combined syndrome	29	11	18	-
Total	75	29 (39%)	40 (53%)	6 (8%)

Table 1. Pre-operative clinical features related to cervical syndromes

Varying degrees of impaired function were seen, and patients were grouped into 3 categories, according to neurological findings, subjective complaints and disability (5):

- 29 patients (39%) had "severe" spondylotic cervical myelopathy (i.e., spastic tetraparaparesis, transverse sensory level, etc.).
- 40 patients (53%) had "moderate" sensory-motor symptoms and signs (i.e., spastic weakness in legs, pain, etc.).
- 3. Only 6 patients (11%) showed "mild" impairment of spinal cord function (reflex changes, numbness, etc.).

The clinical picture was inconstant: the initial symptom, in order of frequency, included pain (particularly in radiculopathic spondylosis), motor or sensory disturbances, alone or in combination.

The fully developed syndrome included sensory, motor and algetic phenomena, in which segmental root symptoms were often combined with signs due to spinal cord compression.

Radiological Features. Plain radiograms of cervical spine and preoperative myelography were performed in all cases. Two common types of abnormalities related to the spinal cord encroachment were found:

- a) shell-like defect, caused by ventral midline ridges at two or more levels: 44 cases;
- b) central bar, cue to complete or partial block to passage of contrast medium: 31 cases.

Stenosis of cervical canal was disclosed in 13 patients (17%). Foraminal osteophytes were present in 17 cases (23%), whereas evidence of associated lumbar spondylosis was found in 24 instances (32%).

Operative Treatment. Two types of surgical procedures were conducted:

1. Posterior approach in 51 patients (68%).

2. Anterior approach in 25 patients (32%) (Table 2).

Table 2. Surgical procedures

No. of cases: 75

Posterior approach: 51 (67%)	Operative techniques	
	Laminectomy Laminectomy - dura closed Laminectomy - dura open (dural graft) Dentate ligament's division Foraminotomy	All cases 11 40 20 24 17
Anterior approach: 25 (33%)		
	Anterior interbody fusion Non fusion op.	20 5

 The standard posterior approach included extensive decompressive laminectomy, one or more segments cranially and caudally beyond the compression area demonstrated radiologically. Usually the removal of 4 to 6 laminae was adequate: almost 2/3 of cases (67%) had at least 5 laminae removed. Laminectomy was extended as far laterally as possible without jeopardizing stability of the cervical spine. In 40 patients (78%) the dura was opened throughout the full length of the exposure: direct reapproximation of the dura was performed in 11 cases, whereas a dural graft was employed to enlarge the dural tube in 20 patients. Twenty-four patients had section of dentate ligaments in order to provide additional biomechanical release (2, 4). Foraminotomy was carried out in 17 patients. With radicular syndrome due to narrowing of the foramen intervertebrale by exostosis, this surgical tactic seems to ensure satisfactory mobilization of compressed nerve roots, and may enhance blood supply to the spinal cord by decompression of radicular vessels (13).

2. The anterior approach: combined cervical disc excision, removal of posterior spurs or ridges with interbody fusion (3) or without fusion (8, 12). All patients had only 1 interspace exposed. Interbody fusion was performed in 20 cases by homograft with iliac bone disc, whereas "non-fusion operation" (discectomy) was carried out in 5 cases. The C5 - C6 disc space accounted for the majority of the disc spaces operated upon (72%). In patients with multilevel lesions, opeation aimed at relieving the prominent compression area. In 1 patient a combined anterior-posterior approach was required in order to remove anterior and posterior spinal compression. There were no serious complications in our series. Minor complications of the anterior cervical approach included ventral displacement of the graft in 2 cases which, nonetheless, received a fair end-result rating.

Results

<u>Immediate Results</u>. "Improved" results were assigned to 58 (77%) patients without complaints and with improved physical signs. Sixteen (27%) patients were "unchanged", whereas 1 was made "worse" by surgery.

Late Results. Follow-up has been obtained for 57 patients (76%). The observation period ranged from 1 to 10 years (mean period: 3,5 years).

In evaluating late surgical results, patients were assessed not only on the basis of the neurological examination, but also on the basis of physical and social disability. Results related to preoperative clinical syndromes are shown in Table 3: improvement was maintained in 15 patients (28%), their neurological deficit and functional disability having disappeared completely or partially. All returned to work or to their former activities when over retirement age. Thirty patients (57%) were rated as stabilized since they showed persistence of neurological deficit and physical disability. Eight cases (15%), classified as worse, developed signs of extension of the spinal cord and involvement or increase of pre-existing symptoms.

Clinical syndrome	No. of cases	Improved	Stabilized	Worse
Severe	19	2	10	4
Moderate	33	11	18	3
Mild	5	2	2	1
Total	57	15 (28%)	30 (57%)	8 (15%)

Table 3. Late results compared with pre-operative disability

/. 4 deaths from unrelated causes.

Immediate vs. late results are shown in Table 4: The early postoperative course can be considered as a fairly reliable predictor of the effectiveness of surgery in the long run. Clinical improvement is likely to be maintained following removal of the primary compressive cause, whereas "unchanged conditions" suggest that the disease process is not effected by surgery.

Table 4.	Immediate	versus	late	results

		Cured	Stabilized	Worse
Improved	49	15	26	4
Unchanged	8	0	4	4

/. 4 deaths from unrelated causes.

A tentative surgical prognosis for cervical spondylotic myelopathy as related to preoperative clinical conditions is shown in Table 5.

	Pre-op. conditions	Immediat results	Immediate results		Late results		
Severe	00000 00000 00000 00000	00000 00000 0000	00000	00	00000 00000	0000	
Moderate	+++++ +++++ +++++ +++++ +++++ +++++ ++++	+++++ +++++ +++++ +++++ +++++ +++++	+++	+++++ +++++ +	+++++ +++++ +++++ +++	+++	
Mild	-					-	
		Improved unchanged worse		Improved stabilized worse			

Table 5. Surgical prognosis in cervical spondylotic myelopathy

/. 4 deaths from unrelated causes.

Age, sex, duration of symptoms, and surgical approach did not influence significantly the outcome of the patients (Table 6).

Table 6. Late results related to the type of surgery

	No. of cases	Improved	Arrested	Worse
Posterior approach	40	9 (24%)	20 (54%)	8 (22%)
Anterior approach	18	6 (35%)	10 (59%)	-

/. 4 deaths from unrelated causes.

Conclusions

Myelopathy associated with cervical spondylosis can be treated with lasting benefits by surgery, whenever conservative measures prove to fail. In choosing any particular approach, the aim remains adequate elimination of severe pathogenic factors. Chronicity seems to play the major role in limiting reversal of neurological findings. Disability and little success can be expected if unalterable conditions have been created by a predominantly vascular-degenerative process.

Improvement or arrest of the disease process as a unit of favorable results has been achieved in up to 85%. However, we are unable to identify specific prognostic predictors for operation. Unfortunately our follow-up series is unadequate to evaluate pros and cons of the posterior and the anterior approach in the long run.

References

- BISHARA, S.M.: Posterior operation in treatment of cervical spondylosis with myelopathy: long-term follow-up study. J. Neurol. Neurosurg. & Psychiat. <u>34</u>, 393-398 (1971)
- 2. BREIG, A.: Biochemanics of the central nervous system. ALMQUIST and WIKSELL (eds.), pp. 1-183. Stockholm 1960
- CLOWARD, R.B.: The anterior approach for removal of ruptured cervical disc. J. Neurosurg. <u>15</u>, 602-614 (1958)
- CUSICK, J.F., ACKMANN, J.J., LARSON, S.J.: Mechanical and physiological effects of dentatotomy. J. Neurosurg. 46, 767-778 (1977)
- FAGER, C.A.: Results of adequate posterior decompression in the relief of spondylotic cervical myelopathy. J. Neurosurg. <u>38</u>, 684-692 (1973)
- GREGORIUS, K., ESTRIN, T., CRANDALL, P.: Cervical spondylotic radiculopathy and myelopathy: long-term follow-up study. Arch. Neurol. <u>33</u>, 618-625 (1976)
- 7. GUIDETTI, B., FORTUNA, A.: Trattamento conservativo e chirurgico delle mielopatie da discoartrosi. Proceedings of the XXVI Italian Congress of Neurology, Rome Oct. 23-26, 1967, pp. 184-238. Roma: Pensiero Scientifico Editore, 1967
- HIRSCH, C., WICKBOM, I., LIDSTRÖM, A., et al.: Cervical disc resection: a follow-up of myelographic and surgical procedure. J. Bone Joint Surg. <u>46A</u>, 1811-1821 (1964)
- 9. JACOBS, B., KRUEGER, E.G., LEIVY, D.M.: Cervical spondylosis with radiculopathy. Results of anterior diskectomy and interbody fusion. J.A.M.A. <u>211</u>, 2135-2139 (1970)
- 10. JOMIN, M., BOUSQUET, C., DELANDSHEER, J.M., LAINE, E.: Traitement des complications radiculo-medullaires de la cervicoarthrose par la méthode de CLOWARD. Resultats à propos de 370 malades opérés. Neuro-chirurgie <u>21</u>, 21-28 (1975)
- 11. PHILLIPS, D.G.: Surgical results of myelopathy with cervical spondylosis. J. Neurol. Neurosurg. & Psychiat. <u>36</u>, 879-884 (1973)
- ROBERTSON, J.T.: Anterior removal of cervical disc without fusion. Clin. Neurosurg. <u>20</u>, 259-261 (1973)
- STOOP, W.L., KING, R.B.: Chronic myelopathy associated with cervical spondylosis: its response to laminectomy and foraminotomy. J.A.M.A. <u>192</u>, 281-284 (1965)

Late Results of Operative Treatment of Cervical Myelopathy K. ROOSEN and W. GROTE

Various experiences with the ventral $(\frac{4}{2}, \frac{5}{2}, \frac{10}{2}, \frac{13-17}{2}, \frac{21}{2}, \frac{24}{2}, \frac{30}{2}, \frac{34}{27}, \frac{29}{2}, \frac{31}{23}, \frac{35}{2})$ in surgery of cervical spondylotic myelopathy (c.m.) have been reported. Therefore, we evaluated 75 patients operated upon from 1969 - 1977. During this time, 90 patients underwent surgery. Eight patients could not be traced, and 4 were living abroad; they answered a questionnaire, reporting good results. For lack of clinical and radiological data these patients were not included. Three patients died in the meantime, from diseases unrelated to c.m.

Age and sex distribution of the 75 patients were in agreement with other series ($\underline{6}$, $\underline{19}$, $\underline{20}$, $\underline{25}$, $\underline{32}$). Age of the 12 female and 63 male patients ranged from $\underline{27}$ - $\underline{84}$ years with a mean of 58 years. Follow-up evaluation inclused detailed pre- and postoperative case history, neurological investigation, X-rays of the cervical spine in 4 projections and in functional positions.

Time interval between operation and re-examination was longer than 1 1/2 years in 86%, and longer than 3 years in 50%. In 23 cases, postoperative course was 4 - 7 years. For evaluation of the clinical results a scale (Table 1) was used, taking into account neurological findings as well as subjective complaints. In a modification of NURICK's classification (28), the degree of disability was graded from A to E based on gait disturbance (Table 2).

Grade	Definition			
I	Symptom-free No neurological deficit ("excellent")			
II	Subjective complaints markedly improved, Mild neurological disorders, Well compensated ("good")			
III	Complaints unchanged, Preoperative neurological status improved ("fair")			
IV	No change in symptoms and neurological findings ("unchanged")			
V	Deterioration in patient's condition ("worse")			
I/II +	Postoperative grade I or II for at least 12 months;			
IV/V	then newly developed signs of c.m.			

Table 1. Clinical categories for evaluation of late results

Table 2. Disturbances Gait (modified after NURICK, 1972)

Grade	Definition
A	None
В	Mild
С	Severe - without help for walking
D	Severe - walking help needed
Е	Disability - chairbound, bedridden

Because of the prognostic importance, three types of c.m. were differentiated, depending on the duration of anamnesis. Preoperative course up to 4 months was defined as "acute type" (15 patients = 20%); the subacute form (29 patients = 39%) lasted from 4 to 12 months; anamnesis longer than 12 months characterized the chronic c.m. (31 patients = 41%).

Eleven (= 73%) of 15 patients suffering from acute c.m. recovered or were markedly improved; the percentage of recovery was only 56% in the subacute type and 22% in the chronic form (Fig. 1). The poorest late results were seen after treatment of chronic c.m.; deterioration was arrested by the operative decompression in 18 cases; in 6 patients the condition worsened.

The different age-groups are equally represented in all categories (Fig. 1). Course and long-time results do not depend on age. The youngest patient, a 27 year old coalmine worker suffering from high-grade chronic spondylotic myelopathy did not improve after ventral operation. On the other hand, the oldest patient, a 84 year old female, who acutely developed an incomplete spastic tetraparesis, had become normal after ventral fusion at C4/5 when examined 1 1/2 years later.

We selected the anterior approach with cervical disc excision and subsequent interbody fusion in 64 cases. In all cases bone cement (polymethylmethacrylate) was used. In 7 patients, we performed a laminectomy as the first step prior to the ventral fusion. In 5 cases we had suspected a spinal tumor, which proved to be a herniated disc with extra- or intradural sequesters; in 2 patients there was a narrow spinal canal.

Two cases deteriorated neurologically and 2 cases did not improve after ventral fusion. Therefore we performed a consecutive posterior decompression by wide laminectomy without enlargement of the dural sac or resection of the dentate ligaments. Late results (2 grade IV patients and 2 grade V) were unsatisfactory. Probably the vascular factor was predominant in pathogenesis of c.m. (2, <u>18</u>, <u>23</u>, <u>25</u>, <u>26</u>, <u>32</u>, <u>37</u>).

There is a significant relation between the eventual clinical condition, whether a lesion of the spina lcord is primarily mono- or multifocal, and what is found intraoperatively (Figs. 2, a + b). Monosegmental processes with an acute or subacute course have the best prognostic recovery chances. When a vascular disturbance is not the main cause for the clinical picture, acute onset of symptoms is probably caused by soft herniated discs (Fig. 2 b); chronic diseases are rather caused by hard disc protrusion or dorsal osteophytic spurs ($\underline{9}$) Prognosis in these cases is significantly worse. This correlation is also proven by the unfavorable late results in cases with marked osteochondrosis and spondylarthrosis.

Analysis of pre- and postoperative symptoms (Fig. 3 a) demonstrates a total improvement rate of 30%, with the exception of sphincter disturbances, which seem to have a better prognosis irrespectively of the c.m. type. On the basis of the clinical course, normalization or improvement is definitely higher in the acute or subacute type of c.m. Recovery of gait (Fig. 3 a, b), which is psychologically the most important sign for the patient, also depends on the duration of preoperative disturbance. Seventy-two out of 75 patients complained of disturbed walking. Operative treatment shortly after the onset of symptoms (acute c.m.) caused favorable effects in 66%. Even in the subacute phase, 56% of the patients improved definitely. Poor results were only seen in cases of chronic course.

The influence of intensive postoperative physical therapy is demonstrated by Fig. 4. Excellent rehabilitation prevents clinical deterioration and generally improves the effects of surgery. All patients graded I + II returned to work. Only those graded III, IV and V were disabled and retired, an interesting socio-psychological finding.

Our indications for surgery are based on the following results (Table 3):

Anterior approach	1.	Mono	segmental	Prolapse Protrusion	
	2.	Bi		Spondylosis	
Laminectomy					
+ anterior fusion	1.	. Multisegmental protrusions with or without dorsal osteophytic spurs			
	2.	DD: Tu	mor - herniate	d disc	
Laminectomy		Narrow spinal canal			
	2.	Deteri	oration after	cervical fusion	
"The sooner, the bette	r"				

Table 3. Indications for different operative techniques

Excellent and good late results in 45 (5%); prophylaxis of clinical deterioration by operative therapy in 44\%, and bad clinical courses in 10,5%.

An amelioration of late results can be expected, if:

- diagnosis and, consequently, indication for surgical treatment are made earlier,
- the duration of thedisease, the degree of degenerative changes of the cervical spine as well as the general vascular condition are more seriously considered, and
- 3. an adequate psycho-physical rehabilitation is ensured.

References

- 1. ABOULKER, J., DAVID, M., ENGEL, P., BALLIVET, J.: Les myelopathies cervicales d'origine rachidienne. Neurochir. <u>11</u>, 89-198 (1965)
- BARTSCH, W.: Klinik der spinalen Durchblutungsstörungen. Acta Neurochir. 7, 255-260 (1961)
- BEKS, J.W.F., PENNING, L.: Ergebnisse neurochirurgischer Behandlung von ausgewählten Fällen spondylotischer Myelopathie. Neurochirurgia <u>7</u>, 77-86 (1964)
- 4. CANTORE, G., FORTUNA, A.: Intersomatic fusion with calf bone "Kiel Bone Splint" in the anterior surgical approach for the treatment of myelopathy in cervical spondylosis. Acta Neurochir. 20, 59-62 (1969)
- 5. CRANDALL, P.H., BATZDORF, U.: Cervical spondylotic myelopathy. J. Neurosurg. 25, 57-66 (1966)
- 6. DIECKMANN, H.: Cervikale Myelopathie. Internist 7, 94-105 (1966)
- FAGER, C.A.: Results of adequate posterior decompression in the relief of spondylotic cervical myelopathy. J. Neurosurg. <u>38</u>, 684-692 (1973)
- FELTEN, H.: Die chirurgischen Aspekte der chronischen Myelopathie (Primäre Rückenmarkserkrankung oder sekundäre Degeneration?). Zbl. Neurochir. <u>16</u>, 142-149 (1956)
- FRYKHOLM, R.: Die cervicalen Bandscheibenschäden. In: Handbuch der Neurochirurgie, Vol. VII, Part 1. OLIVECRONA, H., TÖNNIS, W. (Hrsg.), S. 73-163, Berlin, Heidelberg, New York: Springer 1969
- 10. GALERA, R.G., TOVI, D.: Anterior disc excision with interbody fusion in cervical spondylotic myelopathy and rhizopathy. J. Neurosurg. 28, 305-310 (1968)
- 11. GONZALEZ-FERIA, L., PERAITA-PERAITA, P.: Cervical spondylotic myelopathy: a cooperative study. Clin. Neurol. Neurosurg. <u>78</u>, 19-33 (1975)
- 12. GORTER, K.: Influence of laminectomy on the course of cervical myelopathy. Acta Neurochir. <u>33</u>, 265-281 (1976)
- GREGORIUS, F.K., ESTRIN, T., CRANDALL, P.H.: Cervical spondylotic radiculopathy and myelopathy. A long-term follow-up study. Arch. Neurol. 33, 618-625 (1976)
- 14. GROTE, W., RÖTTGEN, P.: Die ventrale Fusion bei der zervikalen Osteochondrose und ihre Behandlungsergebnisse. Acta Neurochir. 16, 218-240 (1967)
- 15. GROTE, W., BETTAG, W., WÜLLENWEBER, R.: Indikation, Technik und Ergebnisse zervikaler Fusionen. Acta Neurochir. <u>22</u>, 1-27 (1970)
- 16. GUIDETTI, B., FORTUNA, A.: Long-term results of surgical treatment of myelopathy due to cervical spondylosis. J. Neurosurg. <u>30</u>, 714-721 (1969)
- 17. HERZBERGER, E.E., KINDSCHI, L.G., BEAR, N.E., CHANDLER, A.: Treatment of cervical disc disease and cervical spine injury by anterior interbody fusion. A report on early results in 72 cases. Zbl. Neurochir. 23, 214-227 (1963)
- 18. JELLINGER, K.: Morphologie und Pathogenese der spinalen Mangeldurchblutung in Abhängigkeit von der Wirbelsäule. In: Wirbelsäule und Nervensystem. TROSTDORF, H., STENDER, H.St. (Hrsg.), S. 75-89, Stuttgart: Thieme 1970

- 19. JÖRG, J.: Die cervikale Myelopathie als differential-diagnostische Erwägung bei Gehstörungen im mittleren Alter (unter besonderer Berücksichtigung neuropathophysiologischer Grundlagen). Nervenarzt 45, 341-353 (1974)
- 20. KAESER, H.E.: Zur Klinik der cervicalen Diskushernien. Nervenarzt 27, 257-262 (1956)
- 21. KRAYENBÜHL, H.: Die ventrale Operationsmethode von CLOWARD zur Exstirpation zervikaler Diskushernien. Therap. Umsch. <u>20</u>, 444-447 (1963)
- 22. KUHLENDAHL, H.: Die Grundlagen und die Indikationsstellung zur operativen Behandlung in der Wirbelsäulentherapie. In: Die Wirbelsäule in Forschung und Praxis. JUNGHANNS, H. (Hrsg.), S. 29-50, Stuttgart: Hippokrates 1956
- 23. KUHLENDAHL, H., FELTEN, H.: Die chronische Rückenmarkschädigung spinalen Ursprungs. Langenbecks Arch. klin. Chir. <u>283</u>, 96-128 (1956)
- 24. MAYFIELD, F.H.: Cervical spondylosis: A comparison of the anterior and posterior approaches. Clin. Neurosurg. <u>13</u>, 181-188 (1965)
- 25. MUMENTHALER, M.: Cervicale Spondylose und cervicale Discushernien. Acta Neurochir. 5, 553-604 (1957)
- 26. NEUMAYER, E.: Über Diagnose und Differentialdiagnose der chronischen vaskulären Myelopathie. In: Wirbelsäule und Nervensystem. TROSTDORF, E., STENDER, H.St. (Hrsg.), S. 105-108, Stuttgart: Thieme 1970
- 27. NITTNER, K.: Raumbeengende Prozesse im Spinalkanal. In: Handbuch der Neurochirurgie, Vol. VII, Part II. OLIVECRONA, H., TÖNNIS, W. (Hrsg.), S. 1-606, Berlin, Heidelberg, New York: Springer 1972
- NURICK, S.: The pathogenesis of the spinal cord disorder associated with cervical spondylosis. Brain <u>95</u>, 87-100 (1972)
- 29. NURICK, S.: The natural history and the results of surgical treatment of the spinal cord disorder associated with cervical spondylosis. Brain 95, 101-108 (1972)
- PHILLIPS, D.G.: Surgical treatment of myelopathy with cervical spondylosis. J. Neurol. Neurosurg. Psychiat. <u>36</u>, 879-884 (1973)
- 31. SCOVILLE, W.B.: Cervical spondylosis treated by bilateral facetectomy and laminectomy. J. Neurosurg. <u>18</u>, 423-428 (1961)
- 32. SEITZ, D.: Spinale Störungen bei der Osteochondrose der Halswirbelsäule. Dtsch. Z. Nervenheilk. <u>176</u>, 457-468 (1957)
- 33. STOOPS, W.L., KING, R.B.: Chronic myelopathy associated with cervical spondylosis. JAMA <u>192</u>, 281-284 (1965)
- 34. STRACHAN, W.E.: Cervical myelopathy treated by anterior decompression and fusion. J. Neurol. Neurosurg. Psychiat. <u>38</u>, 823-829 (1975)
- 35. THOMALSKE, G., v. WILD, K., LAMMERT, E.: Zur chirurgischen Behandlung der cervikalen Myelopathie. Nervenarzt <u>43</u>, 520-524 (1972)
- 36. VERBIEST, H., PAZ Y GEUSE, H.D.: Anterolateral surgery for cervical spondylosis in cases of myelopathy or nerve root compression. J. Neurosurg. 25, 611-622 (1966)
- 37. ZÜLCH, K.J.: Mangeldurchblutung an der Grenzzone zweier Gefäßgebiete als Ursache bisher ungeklärter Rückenmarksschädigungen. Dtsch. Z. Nervenheilk. <u>172</u>, 81-101 (1954)

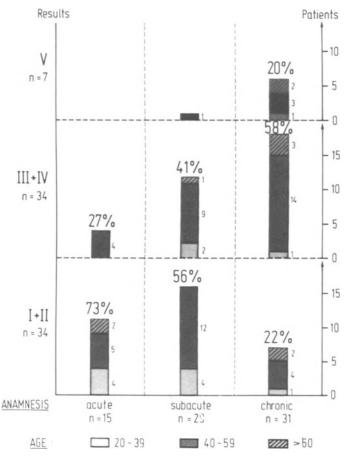
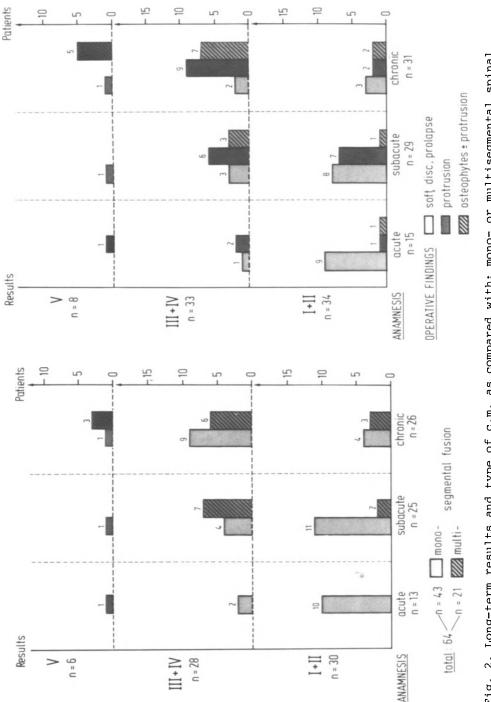


Fig. 1. Correlation between anamnesis, age and late results





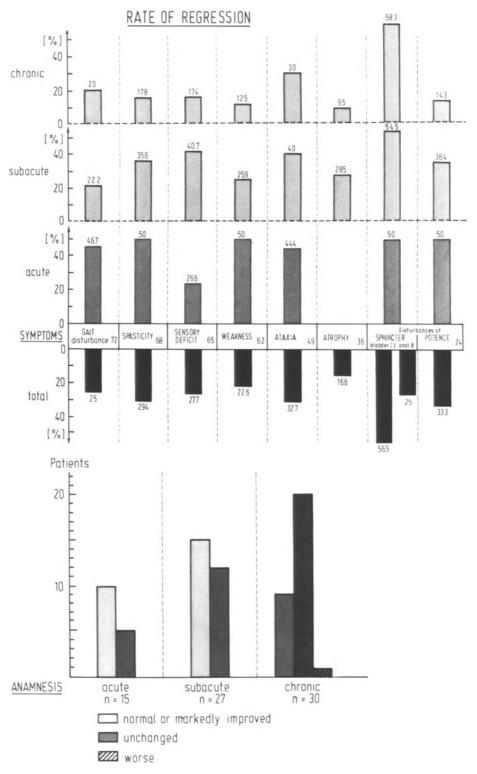


Fig. 3. Improvement of symptoms. *Above:* Clinical signs depending on the preoperative course of c.m.; *below:* gait disturbances

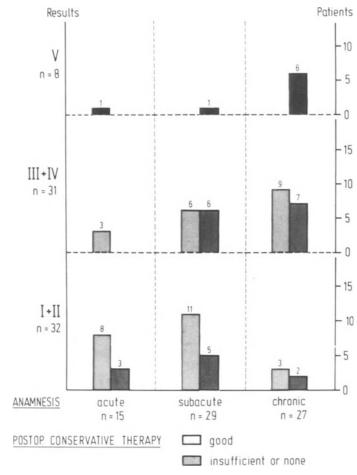


Fig. 4. Effects of postoperative physical rehabilitation

Cervical Spondylotic Myelopathies – Long-Term Results of Surgical Treatment G. DALLE ORE and C. VIVENZA

Cervical spondylotic changes are very frequent and benign lesions. When associated with congenital or acquired stenosis of the cervical canal, cervical trauma, or other factors, they can originate a slowly pregressive medullary compression syndrome.

The surgical techniques presently available permit a stable and complete decompression of the cervical nervous structures. Thus, as happens in the surgery of benign spinal neoplasms, one would expect that decompressive surgery for spondylosis would, in time, give a complete cure. Long-term follow-ups, however, show that incomplete improvement, albeit stabilized in time, are more frequent than the rare complete cures. For this reason, even in long-term follow-up, the results are mostly reported as "improvements" in literature.

Long-term follow-up seems to indicate that in cervical spondylotic myelopathy medullary compression is not the only pathogenetic factor involved, as occurs in benign neoplastic lesions.

The present series of 186 patients with myelopathy due to cervical spondylosis, including 147 cases operated on by CLOWARD's anterior route, 34 cases by laminectomy and 5 by both anterior route and laminectomy, confirms the above stated.

Clinical and neuroradiological data of our patients support the fact that stable postoperative improvements are obtained when some clinical and neuroradiological features, typical of cervical myelopathy, are present. Other features will predict an unfavourable result.

The clinical picture of cervical spondylotic myelopathy shows a wide range of severity and evolution of the medullary signs. In some cases a differential diagnosis from "medical myelopathy" is difficult to make, even in the presence of cervical brachialgia, and slowly progressive ataxic paraparesis.

We observed that many cases with stable postoperative improvement had shown one or more of the following clinical signs, not observed in cases without or with only transitory improvement.

- LHERMITTE's sign.
- Mild or severe BROWN-SEQUARD syndrome.
- Predominantly unilateral motor deficit.
- Immediate transitory worsening after cervical trauma.
- Improvement after wearing a firm collar.
- Early appearance of tonus alterations.

- Myotonic phenomenon of the hand.
- Mild bladder disturbances.

Mild and short-lasting symptoms are followed by the best results, but severe and long-lasting symptoms may also give stable improvements.

On cervical X-rays we noted:

- The degree of disc degeneration, shown by progressive disc flattening. In some cases there can be a fusion of the adjacent bodies.
- Alterations of the vertebral bodies: with time they assume a trapezoidal shape, when only one disc is involved, or a rectangular shape when two adjacent discs are involved.
- The degree and shape of posterior osteophytes.
- Narrowing of the spinal canal, as evaluated by the distance between the laminae in the lateral view.

In the patients with stable long-term improvement the most significant finding was the concomitant presence of posterior spondylosis and stenosis of the cervical canal. The best results were obtained in patients with stenosis and spondylosis at only one level. A large spinal canal frequently gave none or only transitory improvement.

In our patients ascending myelography was usually performed with 9 cc of Myodil[®] in prone position. In this position, the spontaneous extension of the head narrows the spinal canal. The following flexion of the head enlarges the spinal canal and allows a dynamic study.

Complete blocks were rare. The most frequent pictures were:

- Anterior defects in AP and lateral views at one or more levels, with disappearance of radicular pouches and free flow of the Myodil through the spinal canal.
- Anterior defects with partial block at the level of spondylosis. When the head is extended, Myodil stops at the level of spondylosis, when the head is flexed it flows again, although with some difficulty. In these cases a dorsal filling defect caused by the yellow ligaments is frequently shown at one or more levels.

Cases with this picture of partial block have more long-term stable improvements and seems to represent the typical picture of myelopathy due to cervical spondylosis. A free flow of Myodil in a large cervical canal often is associated to no or to only transitory long-term improvement, even if anterior spondylotic defects are present.

Conclusions

In cases of spondylotic cervical myelopathy surgical treatment provides less favourable long-term results than expected, due to the small number of complete recoveries (Table 1). Instead, it has proven very useful in obtaining a great number of stable improvements.

Complete cures are mostly obtained in a small number of patients with one level spondylosis, stenotic canal, unilateral syndromes, worsening after cervical trauma, mild and shortlasting symptomatology, LHERMITTE's sign and BROWN-SEQUARD syndrome.

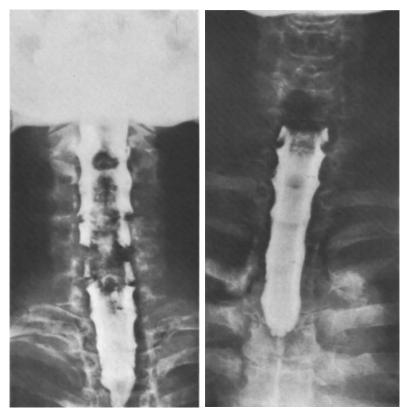
Table 1. Cervical spondylotic myelopathy

	Cases
CLOWARD	147
Laminectomy	34
CLOWARD and laminectomy	5
	86

Stable improvements are more frequent and occur in patients who present one or more of the following symptoms: LHERMITTE's sign, BROWN-SÉQUARD syndrome, almost unilateral motor deficit, worsening after minor cervical trauma, improvement after wearing firm collar, early appearance of tonus alterations, myotonic phenomenon of the hand, mild bladder difficulties.

The radiological finding of cervical spondylosis associated with stenosis of the cervical canal and the myelographic picture of partial block have the most favourable prognostic value. Spondylosis with large spinal canal rarely is followed by stable improvement after operation.

The anterior approach has practically no mortality or morbidity. For this reason the operation can be proposed also when a good or stable improvement can not be foretold.



<u>Fig. 1</u>. Myelography, AP views: extending the neck, the contrast medium flows over the "partial block"



Fig. 2. Lateral views: narrow canal with anterior myelographic defects. C3 body with a trapezoidal shape. C4 and C5 bodies with rectangular shapes

Long-Term Follow-Up After Surgery for Chronic Spondylogenous Myelopathy M. SUNDER-PLASSMANN and F. ZAUNBAUER

Introduction

Both in published series (1-8) and in our own material the surgical management of cervical spondylogenous myelopathies has been found to be rather disappointing, since it has no effect other than halting the progression of neurologic deficits in most of the cases. This prompted us to conduct a follow-up study, the purpose being to shed light on those factors which might improve postoperative results.

Material

Our study included 66 patients with chronic spondylogenous myelopathy who had undergone surgery between 1964 and 1976 and were, accordingly, followed up for periods from 2 to 12 years. Patients with radicular symptoms and discogenic myelopathies were excluded. There was a definite male preponderance, the ratio males: females being 49 : 17. Patient age ranged from 37-77 years. The peak-incidence coincided with the age of 55-65 years (mean: 57.5 years).

The mean duration of symptoms was 11 months, with a range of 2 months to 10 years. Both the onset of the disease and its clinical course were extremely variable. Consequently, the rate of misdiagnoses was fairly high, degenerative central nervous disorders and primary vascular myelopathies predominating.

At the time of surgery quadriparesis and paraparesis were present in 42 and 24 patients, respectively. Twenty-seven patients walked unsupported, while 31 definitely needed support. Eight were unable to walk. Bladder and rectal disorders were observed in 10 cases. In 27 patients sensory function was intact, 15 had dissociated sensory losses.

Spondylogenous myelopathy was diagnosed by plain X-rays of the cervical spine, which consistently showed osteochondrotic reduction of spinal canal width, and by myelography. This revealed complete occlusion in 25 cases and functional occlusion in 22. In 19 patients there was substantial impairment of radiopaque flow.

Surgery consisted in KAHN's laminectomy in 62 cases. Four patients with monosegmental reduction of spinal canal width underwent ventral fusion. In the immediate postoperative period neurologic symptoms were found to deteriorate in 3 cases. Two patients who had been able to walk prior to surgery developed complete postoperative paraplegia, which was irreversible in one.

<u>Results</u>

Fifty patients were followed for periods of 2 to 12 years after surgery. At the time the study was undertaken 10 patients were no longer alive and 6 were lost for follow-up. Of the 50 patients available, no more than 3 were in complete remission; 12 were improved neurologically so that they were able to return to work or, if retired, take care of themselves without any aid. The remaining 35 patients were more or less dependent on help. Neurologic symptoms were found to be improved in 23 of the 35 cases and unchanged in 9. In 3 patients initial improvement was followed by deterioration of the neurologic symptoms within 7 months to 6 years after surgery. Plain X-ray films showed increased osteochondrotic changes in the non-laminectomized region of the cervical spine. Re-operation was refused by these patients, who were invariably older than 65 years.

Overall, 15 patients were thus rehabilitated, while 35 depended on assistance.

Discussion

The rather discouraging outcome of our follow-up study prompted us to look for possible causes. An analysis of our material in terms of pre-operative duration of symptoms, clinical course, and pre-operative neurologic as well as radiologic findings, showed some correlation with the postoperative results. All patients who returned to work after surgery had a pre-operative history of less than 1 year and, with one exception, had been able to walk unsupported before operation. On myelography completely occluded passage was only observed in the patient unable to walk. In all others there was a functional stop or an imparied radiopaque flow. As regards the frequent misdiagnoses during the course of the disease, and its therapeutically poor prognosis, we should like to quote KUHLENDAHL (3): A diagnosis of primary vascular cervical myelopathy and, we would add, of atypical degenerative central nervous disorders, should not be made unless vertebral stenosis and intraspinal space-occupying processes have been ruled out by plain X-rays and myelography. Since almost our entire material was treated by KAHN's laminectomy, we are unable to assess the merits and demerits of other procedures on the basis of our own experience. But insufficiently extensive laminectomy, no doubt, was the factor underlying the deterioration of symptoms in 3 of our cases.

Our results clearly document the need for early diagnosis and early surgery. While conservative treatment may temporarily improve neurologic symptoms, it fails to halt the progression of the osteochondrotic process. Progressive reduction of spinal canal width, and compression of the spinal cord increase the risks of surgery and reduce the regenerating potential of the spinal cord.

Conclusion

Postoperative follow-up studies in patients with chronic spondylogenous myelopathy showed the chances for rehabilitation to be highest if the pre-operative history is less than 1 year, if patients were able to walk unsupported before surgery, and if myelography revealed less-thancomplete occlusion. In all other cases surgery will generally have no effect other than halting the progression of the disease.

References

- BISHARA, S.N.: The posterior operation in treatment of cervical spondylosis with myelopathy: a longterm follow-up sutdy. J. Neurol. Neurosurg. Psychiat. <u>34</u>, 393-398 (1971)
- GUIDETTI, B., FORTUNA, A.: Long-term results of surgical treatment of myelopathy due to cervical spondylosis. J. Neurosurg. <u>30</u>, 714-721 (1969)
- KUHLENDAHL, H.: Pathogenese der sogenannten zervikalen Myelopathie. Biomechanische und vasozirkulatorische Faktoren. Münch. med. Wschr. <u>111</u>, 1137-1140 (1969)
- 4. MAYFIELD, F.H.: Cervical spondylosis: a comparison of the anterior and posterior approaches. Clin. Neurosurg. <u>13</u>, 181-188 (1965)
- 5. NORTHFIELD, D.W.C.: Diagnosis and treatment of myelopathy due to cervical spondylosis. Brit. Med. J. 2, 1474-1477 (1955)
- PHILLIPS, D.G.: Surgical treatment of myelopathy with cervical spondylosis. J. Neurol. Neurosurg. Psychiat. 36, 879-884 (1973)
- 7. SCOVILLE, W.B., DOHRMANN, G.J., CORKILL, G.: Late results of cervical disc surgery. J. Neurosurg. 45, 203-210 (1976)
- VERBIEST, H.: The management of cervical spondyloses. Clin. Neurosurg. <u>20</u>, 262-294 (1973)

Cervical Myelopathy Due to Spondylosis and Disc Protrusion: Operative Results in 70 Patients

F. REALE, D. GAMBACORTA, G. B. SCARFÒ, and G. P. CANTORE

The aim of this paper is to assess the results of surgical treatment in spondylosis and cervical disc protrusion, and to indicate some operative parameters which have an influence on the outcome. Particular attention is devoted to the operative techniques used - that is the posterior laminectomy and the anterior approach according to CLOWARD - and to the indication for their choice.

The series includes 70 cases operated on betwedn 1974 and 1978 in the Department of Neurosurgery of University of Siena: 58 operated on for spondylosis and 12 for soft herniation or disc protrusion. The average follow up is 27 months.

As to the evaluation of the results, 5 categories have been considered: "Excellent" when symptoms disappeard and neurological deficit improved; "Good" when symptoms disappeared almost totally and neurological deficit either improved or remained unchanged; "Poor" when symptoms partly improved or remained unchanged, together with neurological deficit; "Worse" when an exacerbation either of the subjective or the objective symptomatology was found; "Unknown" for those patients who have not undergone a post-operative check-up. Table 1 shows the results according to the above mentioned categories: a satisfactory result (namely excellent + good) can be seen in 62% of the patients, while the condition worsened in 8.5% of the cases. In the same table the results are also divided from the standpoint of pathology: the difference is fairly strong: in 75% of patients operated on for herniation the results are satisfactory, while only 59% of the operated on for spondylosis have the same outcome.

	Excellent	Good	Poor	Worse	Unknown	Total
Herniations	5 (42%)	4 (33%)	2 (17%)	-	1 (8%)	12 (100%)
Spondylosis	20	14	13	6	5	58
	(35%)	(24%)	(22%)	(10%)	(9%)	(100%)
Total	25	18	15	6	6	70
	(36%)	(26%)	(21%)	(8.5%)	(8.5%)	(100%)

Table 1. Operative results - January 1974 / December 1978

Although the criterium of classification is based both on the subjective judgement of the patient and on the objective one of the examiner, we have also taken into consideration the working disability (4, 7, 8),

a strictly objective parameter on the basis of which 4 classes of patients have been classified: "Absent" - no hindrance and possibility of full-time work; "Mild" - possibility of full-time work even if with slight handicaps; "Moderate" - impossibility of full-time work but no handicaps in every-day life; "Severe" - inability to look after oneself and one's elementary needs. Figure 1 shows the postoperative course of working capacity, which has improved in 47% of the patients remained unchanged in 48% and worsened in 3 cases, corresponding 5%. The patients operated on for herniations have shown a course substantially similar to those treated for spondylosis, even if among the latter the 3 cases of worsening are included.

The multiformity of symptomatology of cervical spondylosis makes the identification of preoperative parameters which are unquestionably significant for long-term results particularly difficult. From the data of this series it has been possible to point out that the shorter the clinical history and the milder the neurological deficit the better the outcome.

Age, considered as important by some authors (3, 6), has not, in our opinion, a decisive role, since the results do not show a significantly different course in the various age classes (Table 2). Only in patients over 70 we find, besides unexpected very good results, a greater number of poor results: a careful evaluation of the general condition and neurological deficits allows us to choose those aged patients who presumably will gain benefit from surgical treatment.

In average, patients suffering from herniation have been found to have a shorter clinical history and less severe neurological findings as compared to those affected by spondylosis. This explains the better operative results in the former.

	Excellent	Good	Poor	Worse	Total
- 41 years	3 (42%)	2 (29%)	2 (29%)	-	7 (100%)
41/50 years	8	3	3	2	16
	(50%)	(19%)	(19%)	(12%)	(100%)
51/60 years	7	11	4	2	24
	(30%)	(46%)	(16%)	(8%)	(100%)
61/70 years	5	2	2	2	11
	(46%)	(18%)	(18%)	(18%)	(100%)
+ 70 years	2 (33%)	-	4 (67%)	-	6 (100%)

Table 2. Comparison of results by age

Proper choice of the operative technique is certainly important: following the indications of GUIDETTI and FORTUNA (5), for a diffused spondylosis posterior approach is preferred, namely an extended laminectomy with foraminotomy; in disc protrusions and spondylosis limited to not more than two spaces, anterior approach is chosen according to CLOWARD's technique (2), using, in the majority of cases, a graft of calf bone purified in hydrogen peroxide and sterilized by gamma rays (1). CLOWARD's technique is preferred - more recently also with the aid of the operative microscope - since it allows the removal of the osteophytic spurs with a careful toilette of the foramina, of essential importance also in operations by anterior approach in the presence of radicular symptoms.

The results by type of operation are shown in Table 3. In 4 patients operated on by anterior approach, after an initial improvement, the symptomatology has reappeared - in one after four years. These patients have been submitted to posterior laminectomy with a new recovery in all cases. These cases are too few to justify a special category, and their results are scheduled as if the two operations had been performed on two different patients. From the data of the table we can see that better results are obtained by the anterior approach. We must say, however, that the preoperative neurological status of the patients operated on by anterior approach was generally better than that of the patients operated on by posterior approach and, if we consider the results divided into every category of neurological status, we can state that the course is substantially the same for both types of operation.

	Anter	ior approach	Posterior approach		
Excellent	14 (4	5%)	13	(34%)	
Good	9 (2	98)	10	(26%)	
Poor	5 (1	6%)	11	(29%)	
Worse	3 (1	0%)	4	(118)	
Total	31 (1	00%)	38	(100%)	

Table 3. Results by type of operation

There has been a case of death owing to sudden intraoperative hypotension in a patient operated on in sitting position.

Finally, we can state that surgery of spondylosis and cervical herniations on the whole gives good results. However, in the case of a long clinical history and severe neurological deficit, the possibilities of a useful therapy are reduced. Nevertheless, even in these cases, considering the overall results and the low risk, it is necessary to try and stop the evolution of the disease. Not even old age is, in itself, a contra-indication.

The increasing importance given to vascular impairment due to compression of the anterior spinal artery in the pathogenesis of the "cervical syndrome", has induced us to prefer the anterior approach in some cases of diffuse spondylosis. This technique, especially if accompanied by foraminotomy, gives very good results, not only in cases of myelopathy but also of radicular symptoms, allowing subsequent operation by posterior approach when the result is not good enough.

References

 CANTORE, G.P., FORTUNA, A.: Intersomatic fusion with calf bone ("Kiel bone splint") in the anterior surgical approach for the treatment of myelopathies from cervical spondylosis. Acta Neurochir. (1969)

- CLOWARD, R.: The anterior approach for removal of ruptured cervical disks. J. Neurosurg. <u>15</u>, 602-617 (1958)
- GORTER, K.: Influence of laminectomy on the course of cervical myelopathy. Acta Neurochir. <u>33</u>, 265-281 (1976)
- 4. GREGORIUS, F.K., ESTRIN, T., CRANDALL, P.H.: Cervical spondylotic radiculopathy and myelopathy. Arch. Neurol. <u>33</u>, 618-625 (1976)
- GUIDETTI, B., FORTUNA, A.: Trattamento conservativo e chirurgico delle mielopatie da discoartrosi. Atti del XVI Congresso Nazionale di Neurologia. Vol. I, pp. 181-238. Rom: Pensioro Scientifico (1967)
- 6. GUIDETTI, B., FORTUNA, A.: Long-term results of surgical treatment of Myelopathy due to cervical spondylosis. J. Neurosurg. <u>30</u>, 714-721 (1969)
- 7. LEES, F., TURNER, J.W.A.: Natural history and prognosis of cervical spondylosis. Br. med. J. 2, 1607-1610 (1963)
- SYMON, L., LAVENDER, P.: The surgical treatment of cervical spondylotic myelopathy. Neurology <u>17</u>, 117-127 (1967)

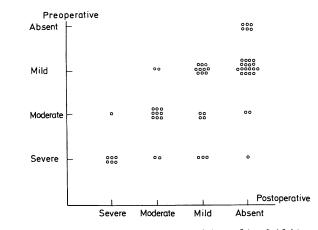


Fig. 1. Pre- and postoperative working disability

Long-Term Results After Operative Treatment of Cervical Myelopathy by Laminectomy

H. U. THAL, H. MILTZ, W. J. BOCK, and H. KUHLENDAHL

We report on the late results in patients suffering from cervical myelopathy operated on by laminectomy in the fifties and sixties. Surgical treatment consisted of a more or less extensive laminectomy; in each case the dura mater was opened and the dentate ligaments cut. Early results were published by KUHLENDAHL and FELTEN (9). Patients operated on by other methods during the last decade are not considered in this report.

Considering the various factors contributing to the pathogenesis of cervical myelopathy, there can be no single method for operative treatment. Laminectomy and cutting of the dentate ligaments was initially the method of choice (3, 4, 5, 8, 10). In 1960 REID (11) demonstrated that cutting of the dentate ligaments to mobilize the spinal cord appeared superfluous. Consecutively, larger series of cases with extensive laminectomy without opening of the dura mater were reported (1, 2, 12). After the introduction of the ventral approach by DEREYMAEKER 1956 and by CLOWARD 1958, this technique has often been used in the operative treatment of cervical myelopathy (6, 7, 10).

The majority of the 96 patients of our series suffered from severe neurological deficits. Seventy cases had a spastic tetraparesis, 13 of which also had nuclear atrophies of the muscles innervated by the lower cervical segment. Nine had spastic paraparesis, and 17 showed lesions of the BROWN-SEQUARD type. Our cases were subdivided into patients with prolapsed cervical discs and with chronical spondylotic myelopathy.

Thirty-nine patients, 33 men and 6 women, had prolapsed discs, mainly in the $C_{V/VI}$ segment (16 cases), 12 in $C_{IV/V}$ and the rest in $C_{III/IV}$ and $C_{VI/VII}$.

Fifty-seven patients, 50 men and 7 women, had spondylotic myelopathy, more than half of them several bars.

More than 50% of the patients of the prolapsed disc group were younger than 50 years of age. On the other hand, spondylotic myelopathy patients predominated in the age group above 50 (Figs. 1a, 1b).

Forty-five patients have died in the meantime. Twenty of these (out of 96) had prolapsed discs, 25 spondylotic bars.

One case of acute spinal cord compression by a prolapsed disc at $C_{III/IV}$ died two months after the operation with tetraplegia; he had had tetraplegia and respiratory troubles, however, on admission to the hospital. Six further patients died, 2 of pulmonary artery embolism following the operation, 1 of acute gastrointestinal hemorrhage, 1 of pneumonia, endocarditis and diabetes mellitus, 1 of septicaemia, 1 of diabetic coma.

Thirty-eight patients died many years after the operation because of other diseases or old age.

More than half of the patients with prolapsed discs had a history of less than 6 months, the rest up to a maximum of 2 years. This is different in patients with spondylotic bars: in this group almost half of the patients had first symptoms dating between 10 months and 3 years before the operation; 22 patients had a history of less than 10 months, 6 of 3 to 10 years (Fig. 2).

Operation in the patients of our series dates back as far as 27 years; 16 patients of the prolapsed disc group were followed up for a period of 6 to 15 years, 21 patients of the spondylotic myelopathy group for a period of 3 to 10 years, another 8 for 10-27 years (Fig. 3). Higher age at the onset of symptoms in patients with spondylotic myelopathy is responsible for shorter follow-up periods until these patients died.

Follow-up data were obtained partly by questionnaires, in the majority of cases by neurological examination. We distinguished the late results into improved, unchanged and worsened as compared to the preoperative situation. Excluding 32 patients whose addresses could not be found out, we considered 29 patients with prolapsed discs and 35 patients with spondylotic myelopathy.

Results

Of 29 patients with prolapsed discs non worsened, 19 were improved and 10 remained unchanged, i.e. with stationary neurological deficits. Special reference is made to the fact that in some cases complete recovery could be obtained.

In the spondylotic myelopathy group we found 14 patients improved, 12 unchanged and 9 with progressive signs of the underlying disease (Table 1).

	Cervical discs	Spondylotic myelopathy		
Improved	19	14		
Unchanged	10	12		
Worsened	-	9		

Table 1. Long-term results after operative treatment

These results correspond to the findings of other authors $(\underline{3}, \underline{4}, \underline{5}, \underline{7}, \underline{10}, \underline{13})$.

To discuss the question of how far the laminectomy should be extended, we have split up our cases according to the number of laminae removed. Our findings suggest that better results can be achieved by extensive laminectomy. Data are listed in Table 2. GORTER, however, did not confirm this in his findings.

From our data we conclude:

Laminectomy limited to two laminae as performed in the fifties appears to be insufficient as revealed by long-term follow-up in-vestigations. We think that at least four laminae comprising the C_{III}vertebral arch cranially in any case should be removed.

Laminae	Improved	Unchanged	Worsened	Total
2	4	4	6	14
3	7	6	2	15
4	2	2	1	5
5	1	-	-	1
Total	14	12	9	35

Table 2. Late result after operative treatment of spondylotic myelopathy in correlation to the extent of the laminectomy

2. Should there be neuroradiological evidence of monosegmental spinal compression the ventral approach is recommended.

 Some cases - particularly when a very narrow spinal canal (10-12 mm) is present - require combined operative procedures.

References

- 1. ABOULKER, J., DAVID, M., ENGEL, P., BALLIVET, J.: Les myélopathies cervicales d'origine rachidienne. Neurochirurgie 11, 87-198 (1965)
- BRAAKMAN, R.: Cervical spondylotic myelopathy. Advances and Technical Standards in Neurosurg. <u>6</u>, 136-169 (1979)
- FAGER, Ch.A.: Results of adequate posterior decompression in the relief of spondylotic cervical myelopathy. J. Neurosurg. <u>38</u>, 488-507 (1977)
- 4. FAGER, Ch.A.: Management of cervical disc lesions and spondylosis by posterior approaches. Clin. Neurosurg. 24, 488-507 (1977)
- GORTER, K.: Influence of laminectomy on the course of cervicyl myelopathy. Acta Neurochir. <u>33</u>, 265-281 (1976)
- GROTE, W., BETTAG, W., WÜLLENWEBER, R.: Technik und Ergebnisse zervikaler Fusionen. Acta Neurochir. 22, 1-27 (1970)
- GUIDETTI, B., FORTUNA, A.: Long-term results of surgical treatment of myelopathy due to cervical spondylosis. J. Neurosurg. <u>30</u>, 714-721 (1969)
- KAHN, E.A.: The role of the dentate ligaments in spinal cord compression and the syndrom of lateral sclerosis. J. Neurosurg. <u>4</u>, 191-199 (1947)
- KUHLENDAHL, H., FELTEN, H.: Die chronische Rückenmarksschädigung spinalen Ursprungs. Langenbecks Arch. Klin. Chir. <u>283</u>, 96-128 (1956)
- 10. MAYFIELD, F.H.: Cervical spondylosis: A comparison of the anterior and posterior approaches. Clin. Neurosurg. 13, 181-188 (1966)
- 11. REID, J.D.: Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. J. Neurol. Neurosurg. Psych. <u>23</u>, 214-221 (1960)
- 12. STOOPS, W.L., KING, R.B.: Neural complications of cervical spondylosis: their response to laminectomy and foraminotomy. J. Neurosurg. 19, 986-999 (1962)
- 13. STOOPS, W.L., KING, R.B.: Chronic myelopathy associated with cervical spondylosis - its response to laminectomy and foraminotomy. J.A.M.A. <u>192</u>, 281-284 (1965)

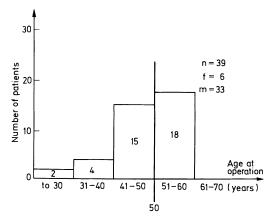


Fig.1. a Age distribution of patients with prolapsed cervical discs

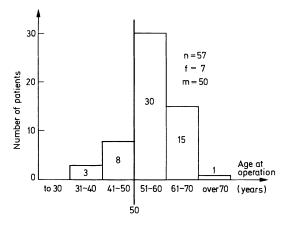
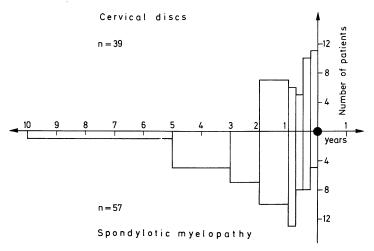
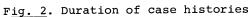


Fig 1. b Age distribution of patients with spondylotic myelopathy





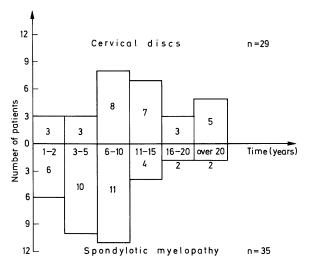


Fig. 3. Duration of follow-up after operative treatment

Long-Term Results After Decompressive Laminectomy in Cases of Multisegmental Cervical Spinal Stenosis

J. HAMER and M. KAHL

Introduction

The primary cause of spondylotic cervical myelopathy is always a more or less *chronic* compression of the spinal cord. Besides one-sided cervical disc prolapse with monosegmental compression of the cord, multiple protrusions with vertebral exostosis in the presence of a congenitally narrow spinal canal are the most important factors for severe biomechanical irritation. In cases with primary spinal stenosis and multisegmental narrowing of the cervical canal, the surgical treatment of choice is generous decompressive laminectomy with the aim of enlarging the spinal canal. Since cervical myelopathy is generally the consequence of a chronic lesion of the cord, the immediate postoperative results often seem not to be very convincing. The present follow-up study was carried out in order to estimate the long-term influence of decompressive laminectomy on spondylotic myelopathy.

Selection of Patients and Methods

This clinical investigation includes 20 patients (5 females, 15 males), who could be followed-up after a sufficiently long period of time since operation, the average being 3 years. All patients had primary spinal stenosis with the sagittal diameter of the middle cervical spine less than 14 mm (which is the lower limit of normal cervical width) and additional narrowing by multiple protrusions and osteophytic bars (see Fig. 1). In all cases, air myelotomography (3) of the cervical spine was performed, which allowed exact measurement of the subarachnoid spinal space. The age of the patients ranged from 34-72 years, the mean being 56. Laminectomy from C2-C7 with enlargement of the spinal sac by dural plasty was performed in all patients. In some cases, bilateral resection of the dentate ligaments was carried out. According to the most prominent clinical symptom, i.e. spastic paraparesis of the legs with subsequent gait disturbance, the results of surgery were graded into 4 groups:

- 1. Patients free of spinal symptoms and having no more complaints (grade I).
- 2. Spastic paraparesis markedly improved, with only minor residual neurological symptoms, such as increased tendon reflexes, positive BABINSKI reflex etc. (grade II).
- 3. Neither improvement nor deterioration (grade III).
- 4. Marked deterioration, either immediately after operation or in the later course of the disease; patients are bedridden (grade IV).

Results

Among the 20 patients, 5 were free of symptoms (grade I), 7 markedly improved (grade II), 5 unchanged (grade III), and 3 worse (grade IV), two of which deteriorated immediately after operation. Whereas in 12 patients (60%), decompressive laminectomy showed a very favourable effect on cervical myelopathy, in particular on the spastic paraparesis of the legs, the influence on the associated spinal and radicular disturbances was less convincing: 8 patients diffuse, non-segmental sensory disturbance and painful dysaesthesia in the upper limbs additionally to the spastic paraparesis. Laminectomy improved these symptoms in only 3 cases. Twelve patients had associated radicular paresis and segmental sensory loss in the arms and hands. Here, the effect of operation was still more unfavourable: only 3 patients improved after decompressive laminectomy. Tables 1 and 2 clearly show which factors influence most the surgical outcome: the main factor is the duration of symptoms, secondly the advanced age of the patient.

Patients older than 60 years and with symptoms lasting more than one year had a more or less unfavourable operative prognosis.

Table 1. Duration of neurological symptoms, and not patient age, primarily influences the result of surgery

	Grade III - IV	Grade I - II
Total	n = 8	n = 12
Over 60 years	4	4
Duration of symptoms > 1 year	7	2

<u>Table 2</u>. Both duration and age play the decisive roles for either very favourable or very bad operative result

	Grade IV	Grade I
Total	n = 3	n = 5
Over 60 years	2	1
Duration of symptoms > 1 year	3	-

Discussion

In spite of the limited number of patients, the present clinical investigation shows a clear trend, and permits evaluation of the value of decompressive laminectomy in cases of spondylotic myelopathy due to mulitple spinal stenosis. In about 60% of the patients, laminectomy favourably influenced the leading clinical symptoms (i.e. gait disturbance due to spasticity and weakness of the legs). As shown in Table 3, this success rate is fairly consistent with operative results from larger clinical studies (1, 2, 4, 5, 6, 7, 8, 9). While motor disturbances of the long spinal tracts are best influenced by operation, diffuse anaesthesia or burning dysaesthesia are less frequently improved. Associated radicular symptoms are even more unfavourable. This may be because decompressive laminectomy alone does not remove the causal ventral compression due to disc protrusion and foraminal osteophytes. In those cases where myelopathic and radicular symptoms are equally prominent, laminectomy should be completed by segmental

Author	Improved	Unchanged	Worse
PESERICO, 1962 n = 47	18	26	3
ABOULKER, 1965 n = 52	36	8	8
GUIDETTI, 1969 n = 86	63	9	14
BISHARA, 1971 n = 59	36	23	-
THOMALSKE, 1972 n = 41	21	8	12
FAGER, 1973 n = 35	24	9	2
GORTER, 1976 n = 71	42	13	16
PIEPGRAS, 1977 n = 80	35	42	3
Total (n = 471)	275 (58%)	138 (30%)	58 (12%)

Table 3. Results of decompressive laminectomy in cases of cervical myelopathy

dorsolateral foraminotomy ($\underline{6}$). The most important factor which determines the surgical prognosis is the duration of symptoms. In our 5 patients with grade I duration of neurological signs was much less than one year (2-4 months). The eminent significance of symptom duration has also been stressed by PESERICO et al. (7) and, in particular, by GUIDETTI ($\underline{6}$), who surveyed the largest patient group with spondylotic myelopathy. He states: "the 51,8% success rate obtained in duration under 6 months falls steadily to 16% for duration exceeding 12 months".

Our follow-up study showed, in agreement with GUIDETTI's results, that age may be another important prognostic factor. It was surprising, however, to see that the severity of neurological deficit per se did not necessarily influence the result of surgery in a negative way: We found very good recovery of spastic paraparesis in 4 patients with marked spinal disturbances, but with short case history (duration of symptoms less than three months!). The same has been observed by GUIDETTI (6).

Finally, a comparison between the early postoperative neurological condition and the follow-up results clearly demonstrated that, in the majority of patients, the beneficial effect of decompressive laminectomy on cervical myelopathy can not be seen immediately after operation. This observation emphasizes the chronic nature of spondylotic myelopathy.

Conclusion

In patients suffering from cervical myelopathy due to primary spinal stenosis with secondary multisegmental protrusions, decompressive laminectomy with dural plasty improves the clinical symptoms in about 60% of the cases. Laminectomy mainly influences spastic paresis. The results are less favourable with regard to diffuse spinal sensory disturbances or associated radicular symptoms. Duration of neurological deficit and age play the major prognostic roles. Patients over 60 years and with advanced myelopathic symptoms lasting longer than a year are unfavourable candidates for decompressive laminectomy. Very good surgical results can only be expected in cases with early diagnosis, i.e. within the first months of clinical manifestation.

References

- 1. ABOULKER, J., DAVID, M., ENGEL, P., BALLIVET, J.: Les myélopathies cervicales d'origine rachidienne. Neurochir. 11, 87-198 (1965)
- BISHARA, S.N.: The posterior operation in treatment of cervical spondylosis with myelopathy: a long-term follow-up study. J. Neurol. Neurosurg. Psychiat. 34, 393-398 (1971)
- CIBA, K., KÜHNER, A.: Die Diagnostik cervicaler Banscheibenprozesse. I. Die Wertigkeit von Luftmyelographie und Discographie. Nervenarzt 47, 160-164 (1976)
- FAGER, C.A.: Results of adequate posterior decompression in the relief of spondylotic cervical myelopathy. J. Neurosurg. <u>38</u>, 684-692 (1973)
- GORTER, K.: Influence of laminectomy on the course of cervical myelopathy. Acta Neurochir. <u>33</u>, 265-281 (1976)
- GUIDETTI, B., FORTUNA, A.: Long-term results of surgical treatment of myelopathy due to cervical spondylosis. J. Neurosurg. <u>30</u>, 714-721 (1969)
- PESERICO, L., UIHLEIN, A., BAKER, G.S.: Surgical treatment of cervical myelopathy associated with cervical spondylosis. Acta Neurochir. <u>10</u>, 365-375 (1962)
- PIEPGRAS, D.G.: Posterior decompression for myelopathy due to cervical spondylosis: Laminectomy alone versus laminectomy with dentate ligament resection. Clin. Neurosurg. 24, 508-515 (1977)
- 9. THOMALSKE, G., WILD, K., LAMMERT, E.: Zur Behandlung der cervikalen Myelopathie. Nervenarzt 43, 520-524 (1972)



<u>Fig. 1</u>. Cervical air myelography showing multisegmental spinal stenosis

Results of the Treatment of Patients Affected by Chronic Cervical Myelopathy by Surgical Decompression and Ventral Fusion According to Cloward

K. LIEBIG, B. KÜGELGEN, D. HOHMANN, and W. HUK

At the Orthopaedic University Clinic of Erlangen 57 patients affected by chronic cervical myelopathy have been operated on since 1972 acacording to the technique indicated by CLOWARD. Of these patients, 31 were subjected to regular neurological and orthopaedic checks over a period of 4 years. In the following we wish to present the results of these follow-up examinations. The youngest patient was 30 whilst the oldest was 78 years old. The majority of the patients was aged between 50 and 65 years. Men were affected 5 times more frequently than women.

A striking finding was that symptoms pointing to a cervical spine lesion were largely missing or only of secondary importance. Only in 2 patients was it possible to trigger LHERMITTE's sign. The incidence of preoperative complaints is seen in Fig. 1.

All patients were operated on by ventral route according to CLOWARD's technique modified by us.

Preexisting spasticity and central paralysis increased in 10 patients following surgery. These symptoms were interpreted as signs of a temporary hypoxidosis with reactive edema, since regression began within 2 weeks.

In more than two thirds of the patients, the disturbances of gait, the weakness in the legs and arms decreased. Diffuse sensations of discomfort, and radiating pain subsided in about 80% of the patients. In none was it possible to trigger LHERMITTE's sign any more. Only one patient continued to suffer from disturbed micturition (Fig. 2).

Paraspastic and tetraspastic increase in tonus remained unchanged after surgery. The central paralyses in the legs and arms remained unaffected. In one patient central paresis of the arms deteriorated. Radicular pareses of the upper extremity readily improved in 2 patients, remained unaffected in one and degenerated in another. Disturbances of sensibility improved in one third of the patients operated on (Fig. 3, Table 1 and 2).

Considering that chronic cervical myelopathy represents a clinical picture within alternating course, an arrest of the neurological symptoms and a noticeable reduction of the complaints, especially when disordered micturition is largely restaurated, must already be considered a surgical success.

Owing to the age distribution, the mobile segments C4/5 and C5/6 were those most frequently affected by degenerative changes. Thus, fusion took place at the segment C5/6 16 times and at the segment C4/5 5 times. Three times we carried out fusions of two adjacent segments at C4/5 and C5/6.

Table 1. Neurological findings

	Preoperative	Postopera	tive	
		Improved	Unchanged	
Spasticity		· · · · ·		
Paraspasticity	4	-	4	
Tetraspasticity	22	-	22	

Table 2. Neurological findings

Central Paralyses Preoperative		Postoperative			
		Improved	Unchanged	Deteriorated	
In the arms	4	Θ	3	1	
In the legs	4	θ	4		
In arms and legs	7	θ	7		
Radic. arm pary- lyses	4	2	1	1	

None of the patients operated on felt the restricted mobility of the cervical spine to be unpleasant or troublesome. In 29 patients integration of the bone chip was free from complications. We attribute this to the use of aplaster collar which ensured strict immobilization over a period of 6 weeks. Restructuring of the corticospongious chip was largely completed after 6 months. Twice we have seen healing by granulation with spondylitis of the fused segment. Complete healing was achieved by strict immobilization. All in all, we have observed 8 unstable moving segment postoperatively: 6 above the fusion, 2 underneath it. However, three of them existed already at the time of surgery.

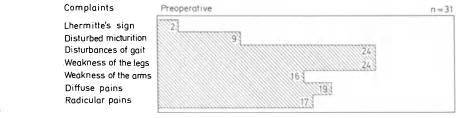
In addition to dorsal osteochondrotic marginal thickening, protruded and slipped disks, KUHLENDAHL (4) considers the pincer mechanism of the plicated ligamenta flava to be a considerable factor in cord compression. DISTELMAIER (1) was able to demonstrate that in two thirds of his patients not the strongly osteochondrotically altered moving segments with reduced mobility, but rather loosened layers of the intervertebral segment above should be considered responsible for such a compression. ERDMANN (2), and later also SEIDEL and SATERNUS (8), pointed out that the unphysiological transverse shifts increase $\overline{\mathrm{in}}$ common with the reduced height of the intervertebral space and the simultaneous arthrotic changes in the joints of the vertebral arches, which narrow the physiological movements of tilt in the involved segments. This change in the motion pattern could furnish an explanation for the narrowing of the spinal canal on dorsal flexion in spines affected by age induced changes, especially in the lower moving segments (2), because it is in this area where the movements reach the greatest deflection in the case of reclination. This pathomechanism can be safely eliminated by ventral decompression and fusion.

The technique modified by us requires that a broad bed for the chip is prepared so that dorsal marginal osteophytes can be readily eliminated. With the folding of two sturdy corticospongious bone chips from the iliac crest inserted sagitally, it is possible to retain the distance of the distracted moving segments without giving rise to a considerable reduction in heigth during the transformation of the chip. Strict external fixation carried out by us for a period of 12 weeks permits additional stabilization of the cervical spine.

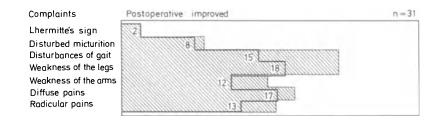
In 32 patients suffering from chronic cervical myelopathy, ventral decompression and fusion according to a modified CLOWARD technique allowed considerable improvement and, in part, stopped the progression of neurological symptoms. On the grounds of the satisfactory experience gained, we feel that the method of CLOWARD is well suited for the management of chronic cervical myelopathy.

References

- DISTELMAIER, P.: Zur Bedeutung der Funktionsaufnahmen der Halswirbelsäule bei der Diagnostik zervikaler Syndrome. Fortschr. Röntgenstr. <u>126, 2</u>, 160-165 (1977)
- ERDMANN, H.: Grundzüge der funktionellen Wirbelsäulenbetrachtung. Manuelle Medizin <u>5</u>, <u>55</u>, <u>6</u> (1967); <u>32</u>, <u>78</u> (1968)
- KRAYENBRÜHL, H., WEBER, G.: Zervikale Diskushernien und in den Wirbelkanal vorspringende knöcherne Randwülste. Erfahrungen mit der ventralen Operationsmethode nach CLOWARD. Münchner Med. Wschr. <u>109</u>, H. 34, 1717-1722 (1967)
- KUHLENDAHL, H.: Pathogenese der sog. zervikalen Myelopathie. Biomechanische und vasozirkulatorische Faktoren. Münch. Med. Wschr. <u>111</u>, 1137-1140 (1969)
- 5. KYSELKA, R., SAUDER, H.-W.: Erfahrungen mit der ventralen Spondylodese nach CLOWARD. Beitr. Orthop. und Traumatolog. <u>24</u>, H. 2, 97-104 (1977)
- MARTIN, G.: Cervical spondylotic myelopathy in canals. New Zealand, Med. Journ. <u>85</u>, 475-476 (1977)
- THOMALSKE, G., WILD, K.v., LAMMERT, E.: Zur chirurgischen Behandlung der cervikalen Myelopathie. Nervenarzt <u>43</u>, 520-524 (1972)
- SEIDEL, K., SATERNUS, K.-S.: Myelographisch anatomische Untersuchungen an Halswirbelsäulenpräparaten Unfallverletzter. Arch. orthop. Unfall Chir. 72, 10-20 (1972)







Complaints Postoperative unchanged n=31 Lhermitte's sign Disturbed micturition Disturbances of gait Weakness of the legs Weakness of the legs Weakness of the arms Diffuse pains Radicular pains

Fig. 2

Fig. 3

Cervical Spondylosis Myelopathy B. GUIDETTI, A. FORTUNA, C. ZAMPONI, and P. P. LUNARDI

For the purpose of treating Cervical Spondylosis Myelopathy (CSM) following etiopathogenetic factors have to be considered: a) spondylosis; b) stenosis of the cervical canal (3); c) mobility of the cervical spine (19). Surgical treatment can affect the various pathogenetic differently (7, 11, 27) and must be tailored to their relative importance in a given patient. These points were not altogether clear when we began to treat patients with CSM. Indeed, in the first series of 92 patients the operation was confined to a posterior laminectomy with section of the dentate ligaments or, in mediolateral spondylosis, with removal of the causal agent (12, 13, 14, 15, 17, 25). The results obtained were, as we shall show, less good than those achieved later with more rational procedures, which we have used in 311 cases.

Our present surgical policy is as follows:

- 1. In patients with spondylosis extending to more than one space and with stenosis of the canal we use the posterior approach: in 66 patients we performed laminectomy from C_1-D_1 and foraminotomy $(\underline{8}, \underline{9}, \underline{22}, \underline{23})$, and in 40 additional cases limited laminectomy and foraminotomy (5-6 laminae). These procedures ensure good posterior transposition of the cord (1), freeing it from ventral mechanical stresses, and, further, they release any affected roots and radiculomedullary arteries. The cord is thus freed from the effect of spondylosis without direct action on the latter. The operation is completed by a tight extensor suture of the neck muscles, which restricts the flexor movements of the spine and reduces the dynamic factor.
- 2. In patients with spondylosis confined to one, two or three spaces, contiguous or not, we use the anterior approach (205 cases) and CLOWARD's technique (4, 5) or that of ROBINSON and SMITH (20, 21), often in combination, if two or three spaces are affected. By these procedures it is possible to remove the degenerated disk material and bony spurs, thereby eliminating the causal factor. They also ensure fusion of the vertebral bodies, which reduces the mobility of the spine at this level and decreases the dynamic factor ($\frac{6}{6}$, $\frac{11}{24}$, $\frac{28}{28}$). In patients with single or double vertebral lesions and not too pronounced stenosis of the canal, we prefer to operate on by the anterior approach and then plan a second operation by posterior approach, if the result is not good when fusion has taken place.

We performed spinal fusion in the first 48 patients using autografts from the iliac crest; since 1967 we have used Kiel bone plugs in 41 cases (2, 16, 26) and since 1971 (in 116 cases) plugs of deproteinised calf bone, which we prepare by a very simple method. These grafts do not result in complete fusion, as an autograft does (18), but the clinical outcome does not differ in the three groups and all the patients present good stability of the spinal column.

Long-Term Results

Of 403 surgically treated patients, 3 died after the operation, one from cardiac infarct on day 3 and the other two from bronchopulmonary complications 9 and 60 days after operation. Long-term follow-up, ranging from 6 months to 26 years, was possible only in 270 patients. This was done either personally or by means of detailed questionnaries completed by the patient's GPs. The others were lost to follow-up, either through change of address or death from various causes (average patient age was high).

In order to evaluate the results, we assessed the patient's preoperative status chiefly in terms of motor symptoms, rating 50% reduction of the use of upper and/or lower limbs as a marked syndrome, 30%-50% reduction as medium grade and less than 25% reduction als mild.

The long-term results (Table 1) have been rated very good (65 cases = 24%) when overall functional recovery was between 85% and 95% of premorbid function. These patients experienced not only a nearly complete regression of paresthesias, pain and twitching but also regained almost full motor function, and had marked reduction of spasticity and of sensory disturbances. All the patients classified as "very good" fully resumed their previous occupations, "good" results were obtained in 75 patients (27.8%)- good meaning between 50% and 80% recovery of function. Most of these patients resumed their previous occupations, but complained of tiring easily when walking, reduced power in one or more fingers but not to the point of being unable to use them. Clinically, some presented increased tone and reflexes in one bone or both legs; a few had radicular sensory disturbances or dysesthesias in one or more upper limb metamers. The results were "fair" in 86 cases (31.9%), with 20%-30% improvement of the preoperative status. This indicates the degree of function, which is directly proportional to the severity of the preoperative clinical syndrome. Thus, patients with a marked syndrome, even with the slight improvement obtained, are to be considered disabled persons with varying degrees of independence, whereas patients whose syndrome was rated as "mild", with even modest benefit of surgery, are perfectly independent and capable of satisfactory social activity. These points apply likewise to the next group: 23 patients (8.6%) were classified as "no change", in that the operation did not modify the preoperative clinical status. In view of the long follow-up in the majority of cases, the "no change" may be regarded as permanent. "Natural deterioration" was observed in 15 cases (5.5%): in this group surgery did not affect the natural course of the disease. After a period of no change lasting from 3-15 months, the motor symptoms deteriorated, especially in the lower limbs, followed by serious sphincter disturbances in some cases. Five of these patients, operated on at first by anterior approach, had a second operation by posterior approach with extended laminectomy plus foraminotomy, the results being good in 2 cases and fair in 3. "Postoperative deterioration" was observed in 6 patients (2.2%).

Factors Affecting Surgical Prognosis

An analysis of the results of surgical treatment in patients with cervical spondylosis myelopathy shows that several factors affect the surgical prognosis and the quality of the result.

Results	N	00 0	
Very good	65	24	
Good	75	27.8	
Fair	86	31.9	
No change	23	8.6	
Natural deterioration	15	5.5	
Postoperative deterioration	6	2.2	

Table 1. Long-term results in 270 cases

1. Type of Operation

Table 2 gives the results by type of operation.

- a) Limited laminectomy with section of the dentate ligaments yielded the lowest rate of good to very good results (16.7%) and the highest rates of fair results (50%) and of natural deterioration (20%).
- b) Limited laminectomy with partial or subtotal removal of degenerate disk material or of the lateral calcified portion yielded better results than the previous procedure (good to very good 35%, fair 47.5%) but less good than the subsequent procedures.
- c) Operations by anterior approach with spinal fusion yielded the best results (good to very good 64.5%) and low rates of no change (5.7%) and deterioration (5.7%).
- d) Extended laminectomy or laminectomy C₁-D₁ with foraminotomy yielded lower rates of good to very good results (51.6% and 55.5% respectively) than the anterior approach, but significantly higher than 1) and 2) and low deterioration rates (3.2% and 6.7% respectively). It should be noted that these operations were performed in patients with severe and diffuse spondylarthrosis and marked stenosis of the cervical canal.

2. Duration of the Disease

Table 3 shows how central this fact is to the surgical prognosis, which, indeed, varied in inverse proportion to the duration. In patients with a history of under 6 months the rate of good to very good results (63.7%) was significantly higher and the rate of fair results (23.5%) and no changes (4.9%) lower. For histories exceeding one year, the good to very good rate drops to 30.1%, the fair rate rises steeply to 45.2% and the "no changes" to 18.7%. This is precisely what one would expect, since the history signifies the length of time that the cord has been damaged, and, obviously, the longer this has gone on the less chance there is of correcting it. This suggests that it is unwise to lose time in conservative treatments, since a possibly reversible damage is likely to become irreversible.

TANTE 2. RESULTS DY LYPE OF OPERATION (2/0 CASES)	ation (2/U cases)			
Type of operation	Results			
	Very good/good	Fair	No change	Deterioration
Limited laminectomy with section of dentate ligaments: 30 cases	5 (16.7%)	15 (50%)	4 (13.6%)	6 (20%)
Limited laminectomy with partial or subtotal removal: 40 cases	14 (35%)	19 (47.5%)	3 (7.5%)	4 (10%)
Revomal by anterior approach with arthrodesis: 124 cases	80 (64.5%)	30 (24.1%)	7 (5.7%)	7 (5.7%)
Laminectomy and foraminotomy: 31 cases	16 (51.6%)	12 (38.7%)	2 (6.5%)	1 (3.2%)
Extended laminectomy C1-T1 with foraminotomy: 45 cases	25 (55.5%)	10 (22.2%)	7 (15.6%)	3 (6.7%)
Noto: The "read " and " not a fine a fine " for a fine a fine " for a fine " for a fine fine a fine a fine a fine a fine a fine		E		

Table 2. Results by type of operation (270 cases)

Note: The "very good" and "good" results are lumped together under "very good/good", and the cases showing natural and postoperative deteriorations are treated similarly.

<u>Table 3</u> . Results by preoperative duration of disease (270 cases)	duration of diseas	se (270 cases)		
Duration of disease	Results			
	Very good/good	Fair	No change	Deterioration
0-6 months 102 cases	65 (63.7%)	24 (23.5%)	5 (4.9%)	8 (7.9%)
7-12 months 95 cases	53 (55.8%)	29 (30.5%)	5 (5.3%)	8 (8.4%)
Over 12 months 73 cases	22 (30.1%)	33 (45.2%)	13 (17.8%) 5 (6.9%)	5 (6.9%)
w mko "word" sod "soond" results are lummed torether "werv good/good". and the	reculte are lumor	todether unde	rery good	/good" and the

Note: The "very good" and "good" results are lumped together under "very good/good", and the cases showing natural and postoperative deteriorations are treated similarly.

108

3. Patient Age

Considering the results by patient age in decades, the very good rate was highest in the under -40s (67.6%), halving in the over -60s (32%). However, putting age against history, we find that of patients of over 60 with a history of less than 6 months (13 cases) 7, i.e. 53.8% had a very good result. This shows that, even in elderly patients, a high rate of good to very good results can be expected as long as the history has been short.

4. Preoperative Clinical Status

The results by clinical syndrome (Table 4) do not vary significantly according to the preoperative status, clearly a less significant factor than duration of disease and type of operation. We found, for example, a high good to very good rate even in marked syndromes when the history has been under 6 months (70.9%) or when patients received the most effective treatment (69.6% for the operation by anterior approach and 50% for extended laminectomy plus foraminotomy).

Conclusions

The results of surgical treatment for cervical spondylosis are significantly influenced by two factors: type of operation and duration of disease. No type of operation can be judged a priori the best: results can be very good with the operation by anterior approach and with the posterior approach including foraminotomy, provided that the choice reflects the importance of the various pathogenetic factors in each individual. A short history is a highly favorable prognostic factor. Age and clinical status are less important: elderly patients with marked syndromes but short history do better than patients with medium grade syndromes but of longer duration. Hence the importance of early diagnosis.

Grade of severity	Results			
50101101	Very good/good	Fair	No change	Deterioration
Mild: 45 cases	28 (62.2%)	11 (24.5%)	2 (4.4%)	4 (8.9%)
Medium: 119 cases	58 (48.7%)	42 (35.3%)	10 (8.4%)	9 (7.6%)
Marked: 106 cases	54 (50.9%)	33 (31.1%)	11 (10.4%)	8 (7.6%)

Table 4. Results by severity of the clinical syndrome (270 cases)

Note: The "very good" and "good" results are lumped together under "very good/good", and the cases showing natural and postoperative deteriorations are treated similarly.

References

1. ABOULKER, J., METZGER, J., DAVID, M., BALLIVET, J.: Les myelopathies cervicales d'origine rachidienne. Neuro-Chirurgie 11, 87-198 (1965)

- CANTORE, G.P., FORTUNA, A.: Intersomatic -usion with calf bone, "Kiel bone splint" in the anterior surgical approach for the treatment of myelopathy in cervical spondylosis. Acta Neurochir. <u>20</u>, 59-62 (1969)
- CHRISPIN, A.R., LEES, F.: The spinal canal in cervical spondylosis. J. Neurol. Neurosurg. Psychiat. <u>26</u>, 166-170 (1963)
- CLOWARD, R.B.: The anterior approach for removal of ruptured cervical disks. J. Neurosurg. <u>15</u>, 602-614 (1958)
- CLOWARD, R.B.: Lesions of the intervertebral disk and their treatment by interbody fusion methods. Clin. Orthop. <u>27</u>, 51-77 (1963)
- CONNOLLY, E.S., et al.: Clinical evaluation of anterior cervical fusion for degenerative disk disease. J. Neurosurg. <u>23</u>, 431-437 (1965)
- 7. DEREYMAKER, A., GHOSEZ, J.P., KENKES, R.: Le traitment chirurgical de la discopathie cervicale. Resultats comparés de l'abord posterieur (laminectomie) et de l'abord ventral (fusion corporeale), dans une cinquantaine de cas personnels. Neuro-Chirurgie <u>9</u>, 13-20 (1963)
- EPSTEIN, J.A., LAVINE, L.S., ARONSON, H.A., EPSTEIN, B.S.: Cervical spondylotic radiculopathy: the syndrome of foraminal constriction treated by foramenotomy and the removal of osteophytes. Clin. Orthop. 40, 113-122 (1965)
- FAGER, C.A.: Results of adequate posterior decompression in the relief of spondylotic cervical myelopathy. J. Neurosurg. <u>38</u>, 684-692 (1973)
- GONZALES-FERIA, L., et al.: Cervical spondylotic myelopathy: a cooperative study. Clin. Neurol. Neurosurg. 78 (1), 19-33 (1975)
- 11. GREGORIUS, F.K., ESTRIN, T., CRANDALL, P.H.: Cervical spondylotic radiculopathy and myelopathy: a long-term follow-up study. Arch. Neurol. 33, 618-625 (1976)
- 12. GUIDETTI, B.: Mielopatie da spondilosi cervicale. Bologna: Ed. Medica 1958
- 13. GUIDETTI, B.: Cervical myelopathy: a complication of cervical spondylosis. Acta Neurol. Latinoam. <u>7</u>, 11-23 (1961)
- 14. GUIDETTI, B., FORTUNA, A.: Trattamento conservativo e chirurgico delle mielopatie da discoartrosi. Relazione al XVI^O Congresso Nationale di Neurologia. Rome, October 23-26 1967. Atti del Congresso, Vol. I, pp. 181-238. Rome: Pensiero Scientifico 1967
- 15. GUIDETTI, B., FORTUNA, A.: Long-term results of surgical treatment of myelopathy due to cervical spondylosis. J. Neurosurg. <u>30</u>, 714-721 (1969)
- 16. MAATZ, R., BAUERMEISTER, A.: A method of bone maceration. Results in animal experiments. J.B.J.S. <u>39-A</u>, 153-162 (1957)
- PIEPGRASS, D.G.: Posterior decompression for myelopathy due to cervical spondylosis: laminectomy alone versus laminectomy with dentate ligaments section. Clin. Neurosurg. 24, 508-515 (1977)
- 18. RAMANI, P.S., et al.: Cervical spinal interbody fusion with Kiel bone. Br. J. Surg. <u>62</u>, 147-150 (1975)
- 19. REID, J.D.: Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. J. Neurol. Neurosurg. Psychiat. <u>23</u>, 214-221 (1960)

- ROBINSON, R.A., SMITH, G.W.: Anterolateral cervical disk removal and interbody fusion for cervical disk syndrome. Bull. Johns Hopkins Hosp. <u>96</u>, 223-224 (1955)
- 21. ROBINSON, R.A., et al.: The results of anterior interbody fusion of the cervical spine. J.B.J.S. 44-A, 1569-1587 (1962)
- 22. SCOVILLE, W.B.: Cervical spondylosis treated by bilateral facetectomy and laminectomy. J. Neurosurg. 18, 423-428 (1961)
- 23. SCOVILLE, W.B., DORHMAN, G.J., CORKILL, G.: Late results of cervical disk surgery. J. Neurosurg. <u>45</u>, 203-210 (1976)
- STRACHAN, W.E.: Cervical myelopathy treated by anterior decompression and fusion. J. Neurol. Neurosurg. Psychiat. 38(8), 823 (1975)
- 25. STOLTMANN, H.F., BLACKWOOD, W.: An anatomical study of the role of the dentate ligaments in the cervical spinal canal. J. Neurosurg. $\underline{24}$, 43-46 (1966)
- 26. TAHERI, E., et al.: Experience with calf bone in cervical interbody spinal fusion. J. Neurosurg. 36, 67-71 (1972)
- 27. VERBIEST, H.: La chirurgie anterieure et laterale du rachis cervical. Neuro-Chirurgie 16, Suppl. 2 (1970)
- 28. WHITE, A.A., et al.: Relief of pain by anterior cervical spine fusion for spondylosis. A report of 65 patients. J.B.J.S. <u>55-A</u>, 525-534 (1973)

Chronic Spondylogenic Myelopathy: Analysis of Data of 62 Patients Operated on by the Anterior Approach

D. FISCHER, H. D. HERRMANN, and F. LOEW

Introduction

The expression "Cervical Myelopathy" covers every illness of the cervical cord. For damages of spinal cord caused by alterations of the spinal column we suggest the term "Spondylogenic Myelopathy". The degenerative spondylogenic myelopathy characterizes a lesion by osteochondrosis or spondylosis - mostly of the cervical spine $(\underline{2})$.

In this paper we present the data of patients with chronic spondylogenic myelopathy. Myelopathy caused by soft disc prolaps was not included.

Patients

From 1974-1978, 62 patients with medullary symptoms caused by cervical spondylosis have been operated on by the anterior approach. There are sufficient follow-up data on 52 patients. The male : female relation in chronic spondylogenic myelopathy is 2 : 1. Onset of symptoms occured mainly in the middle period of life. Fifty percent of all the patients were male and in the 5th decade of life. In 8 out of 10 cases, symptoms dominate on one side. The maximum of radiological abnormalities was found between C4 and C6 in 2/3 of the cases. Fifteen percent of the patients had a congenital narrow spinal canal with an ap-diameter below the standard values (1, 3).

Results and Discussion

We have analyzed the leading clinical symptoms of 62 patients with maximally 3 symptoms per patient. Thus, a total of 26 radicular and 109 medullary findings were collected. The patients were reexamined at regular postoperative intervals. One year after the operation, at the earliest, final results were assessed and qualified as "good", "moderate", "unchanged" or "worse". As the Tables 1-4 show, postoperative results are independant of the duration of the symptom prior to the operation. This is valid for both the radicular and medullary symptoms. This observation is contrary to the general experience with cases of cervical soft disc herniations. An explanation for this discrepancy could be that in chronic myelopathies with slow onset and gradual progression, the pathophysiological complex, consisting of functional disturbances, structural alterations and adapting processes, is either reversible in the early stage of development (up to a certain degree) or not at all. The process of restitution also seems to be limited to a relative short period of time: in most of our cases, the final postoperative state was reached within 1-12 weeks.

Table	1.	Final	results

N = 62	Good	Moderate	Unchanged	Worse
Re-examined = 52	23 (44%)	16 (31%)	7 (13%)	6 (12%)

Table 2. Age and prognosis

	N	Good and moderate improvement	No change and worse	
Below 50 years	37	27	8	
Above 50 years	15	12	5	
Total	52	39	13	

<u>Table 3.</u> Preoperative duration of the leading symptoms and their improvement

		3 1	noı	ntł	ıs	 6 1	nor	ntl	ıs		12	mo	ont	hs	Mo: 12		han ths
Medullary symptoms	N=36	++ 18		-		++ 6		•		N=21	++ 14		-		++ 24	•	

++, good; +, moderate; 0, unchanged; -, worse.

Table 4. Preoperative duration of the leading symptoms and their improvement

		3 months		6 1	noı	ntł	ıs		12	months				han ths
Cervical root compressio	N=0 on		N=17	++ 8			-	N=O			N=9	++ 4		-

++, good; +, moderate; 0, unchanged; -, worse.

One year after operation, the clinical results were satisfactory in about 75% and unsatisfactory in about 25%. Older patients have an evidently worse prognosis than patients below 50 years. Patients with congenital narrow spinal canal and additionally circumscribed spondylogenic spurs have the same results as those with normal spinal canal, provided no additional traumatisation of the spinal cord occurs during surgery.

We used the anterior approach in the operative treatment of chronic spondylogenic myelopathy in all those cases where dorsal spondylotic spurs were "suspicious" of causing the medullary compression in no more than 2 segments, no matter whether a congenitally narrow spinal canal was encountered or not.

Summary

Fifty-two patients operated on for this type of spondylogenic myelopathy by the anterior approach were reexamined. The male : female relation was 2 : 1. Fifty percent were males in the 5th decade. About 20% had a congenital narrow spinal canal with an ap-diameter below the minimal standard values (3). The maximum of spondylogenic narrowing was found between C4 and $\overline{C6}$ in 2/3 of the cases.

<u>Results:</u> good 44%, satisfactory 31%, unchanged 13%, deteriorated 12%, mortality 0.

The duration of the neurological symptoms before operation had no influence on the surgical results. Final neurological restitution was reached within 1 week to 3 months after the operation. The operative results in patients with congenital narrow spinal canal showed the same percentual distribution as the results of the entire group. No factors could be found to explain the groups of unsatisfactory results.

References

- DECKING, D., STEEGE, W. ter: Röntgenologische Parameter der Halswirbelsäule im seitlichen Strahlengang. Die Wirbelsäule in Forschung und Praxis, <u>64</u> (1975)
- 2. HERRMANN, H.D.: Neurologie in Praxis und Klinik. HOPF, H.C., POECK, K., SCHLIACK, N. (Hrsg.). Stuttgart: Thieme (in press)
- 3. PIEPGRAS, U.: Neuroradiologie. S. 88-89. Stuttgart: Thieme 1977

Classification and Prognosis of Cervical Myelopathy E. HAMEL, R. A. FROWEIN, and A. KARIMI-NEJAD

Introduction

Up to the present, 104 patients with cervical myelopathy caused by cervical disk lesions, have been operated on in our clinic. In the earlier years (i.e. up to 1969) when the criteria for operation were much narrower, a posterior decompression by laminectomy was carried out in 19 patients. When the anterior approach was developed, and particularly with the introduction of the operating microscope, the indications were extended. Thus, since 1969, 85 patients with cervical myelopathy have been operated on, after myelographic and discographic confirmation of a disk lesion.

The different clinical manifestations of cervical myelopathy are wellknown from numerous publications. BRAIN, 1952 (1); PIA and TÖNNIS, 1952 (8); TÖNNIS and KRENKEL, 1955 (9); and KUHLENDAHL 1955 (4) had already described "acute" syndromes. In 1952 BRAIN (1) described the "soft extruded disk" and in 1966 MAYFIELD (5) pointed out the possibility of a preformed disk lesion. However, the classification of the different clinical types has not so far been standardized.

We have therefore tried to discover, in our cases of cervical myelopathy, a constant correlation between the length of history, the operative findings and prognosis.

Classification

The length of history in our patients varied from three days to twenty years (Fig. 1). In patients with a short history we frequently found an extruded disk sequestrum (actually in 21 out of 37 patients, or 57%) and an acute episode was often reported in the history (in 18 out of 37 patients, or 47%). These cases, with a history shorter than four months, were designated "acute", as defined in an earlier paper ($\underline{2}$).

However, a restriction of the acute syndrome to the patients with subjective acute episode does not seem justified, since perforated disk lesions were found even without any history on an acute painful onset. Thus, any classification into groups must inevitably be quite arbitrary.

In patients with a history from four months up to one year perforation of disk material are less frequent (in 12 out of 29 patients, or 41%), and acute episodes occured only in 10 out of 29 patients (i.e. 34%). This group was designated as "subacute". In chronic lesions with a history of more than one year, only 5 out of 38 patients described an acute episode. Spinal cord compression was caused mostly by osteophytic spurs, and in only 9 out of 38 patients was a sequestrated disk lesion found at operation.

Prognosis

This subdivision into acute, subacute and chronic syndromes also appeared to be helpful in assessing the prognosis. Since the disturbance of gait was responsible for the greatest disability, this was compared pre- and postoperatively. The disturbance of gait was divided into five grades, following NURICK (6) and PHILLIPS (7) (Fig. 2). If there is a postoperative improvement of one grade (for example from a stage of definite disturbance of gait to a stage of slight disturbance), we called it a slight improvement. A recovery of two or more grades is described as a significant improvement.

In Fig. 3 the results of 85 ventral fusions are summarized and compared with the results obtained in acute syndromes, with perforated disk lesions, and in chronic syndromes, with osteophytic spurs.

The best results were obtained in patients with an acute syndrome who had a perforated disk lesion. These showed a significant improvement of the disability in 44% of the cases, whereas this result was achieved in only 20% of the patients with chronic syndromes associated with osteophytotic spurs. The postoperative result in 25 patients (not shown in this Figure) with subacute onset of symptoms, corresponded approximately to those in the chronic syndromes, in that slightly better results were also seen in those who had a perforated disk.

In the syndrome-time diagram (Fig. 4), the different stages of spinal cord impairment in the preoperative and postoperative course of three patients are demonstrated.

The typical course of *chronic myelopathy* is exemplified by a 60-yearold woman (Fig. 4 "Le"). Her trouble began seven years earlier, with slight disturbance of gait. During the final three years of this period she had complained of increasing leg weakness and, eventually, she developed a tetraparesis. Myelography and discography revealed that calcified osteochondrotic spurs at C3/4 and a cartilaginous protrusion at C4/5 were causing marked ventral compression of the spinal cord. Therefore, both these lesions were operated on. Two years after surgery the patient showed a slight improvement of her paresis but spasticity persisted unchanged. What appears as typical of the chronic syndrome is the slowly progressive deterioration, and, after surgery, an incomplete recovery, even taking into account the longer follow-up.

In contrast to this, there are two separate groups to be distinguished in the *acute forms*.

First of all, those patients whose symptoms developed within a few weeks or months. An example of this type is shown by the clinical course of a 62-year-old man (Fig. 4 "Ke"). Over a period of three months he developed progressive disturbance of gait, combined with radicular pains in his upper limbs. His myelogram revealed a stop of the contrast medium at C6/7. The considerable extravasation of contrast medium at C6/7. The considerable extravasation of contrast at discography revealed lacunar filling defects, typical of extruded sequestra. At operation this suspicion was confirmed. Typical for these types is the rapid, complete and permanent recovery. Postoperatively the pain and paresis diminished within two months and had disappeared after seven months.

In contrast to this favourable prognosis in the acute cases, we must exclude those *hyperacute types*, where the symptoms develop within a few days of even hours.

In this instance, a 40-year-old man (Fig. 4 "En"), after a two day history of radicular pain, developed acute neck and shoulder pain with paraplegia. The operation, performed a few hours after onset, revealed a massive perforated disk lesion at C6/7 with three large extradural sequestra. In spite of the prompt relief of pressure, the transverse cord lesion showed no improvement and the patient died 22 days later, with an ileus and renal failure.

In seven cases with such a hyperacute course, none showed any recovery. Also, in one patient who had endured a chronic illness, there was an acute deterioration shortly before surgery. In this case, also, there was no postoperative improvement, but rather a deterioration to a stage of a complete transverse lesion.

We would like to draw particular attention to this clinical type, about which very little has been described in literature.

References

- 1. BRAIN, R.W., NORTHFIELD, D., WILKINSON, M.: The neurological manifestation of cervical spondylosis. Brain 75, 187-225 (1952)
- FROWEIN, R.A., FRIEDMANN, G., KUNSTEIN-BÖHM, U.: Wahl der Kontrastmitteldiagnostik bei zervikalen Bandscheibenschäden. Röntgen-Bl. <u>27</u>, 538-548 (1974)
- 3. HAMEL, E., FROWEIN, R.A., KARIMI-NEJAD, A., FRIEDMANN, G.: Cervical myelopathy. Neurosurg. Rev. 1, 101-110 (1978)
- 4. KUHLENDAHL, H., HENSELL, V., FELTEN, H.: Durch Halswirbelscheibenveränderungen verursachte chronische Rückenmarksschädigungen. In: Die cervikalen Vertebralsyndrome. REISCHAUER, F. (Hrsg.). Stuttgart: G. Thieme 1955
- MAYFIELD, F.H.: Cervical spondylosis. A comparison of the anterior and posterior approaches. Clin. Neurosurg. 13, 181-188 (1966)
- NURICK, S.: The pathogenesis of the spinal cord. Disorder associated with cervical spondylosis. Brain 95, 87-100 (1972)
- 7. PHILLIPS, D.G.: Surgical treatment of myelopathy with cervical spondylosis. J. Neurolog. Neurosurg. Psychiat. 36, 879-884 (1973)
- PIA, H.W., TÖNNIS, W.: Diagnose und Therapie zervikaler Bandscheibenschäden. Dtsch. med. Wschr. <u>78</u>, 1089 (1952)
- TÖNNIS, W., KRENKEL, W.: Erfahrungen bei der operativen Behandlung des cervikalen Vertebral-Syndroms. In: Die cervikalen Vertebralsyndrome. REISCHAUER, F. (Hrsg.). Stuttgart: G. Thieme 1955

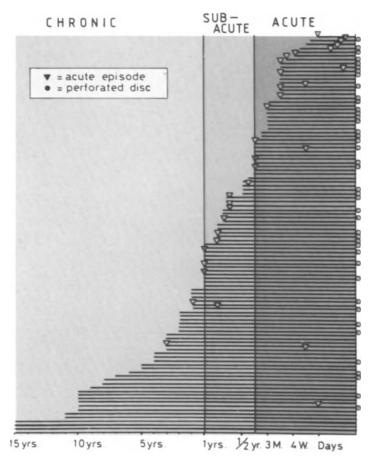
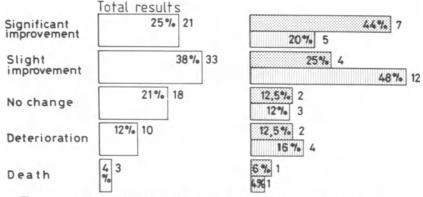


Fig. 1. History of 104 patients with cervical myelopathy

	N	10	20	30	40	50
Begin. med. syndrome	19	2/0	20			
Slight dist. of gait	11.5	5 % 12	2			51
Definite dist.of gait	49	°/o				
Walking only with support	11.5	5 % 12	2			
Unable to walk	9	°/₀ 9				

Fig. 2. Preoperative syndrome in 104 patients with cervical myelopathy. Grades of gait disturbances according to NURICK ($\underline{6}$) and PHILLIPS ($\underline{7}$)



= acute syndrome/perforated disc = chronic syndr./ disc protusion

Fig. 3. Total results of 85 ventral fusion operations in cases of cervical myelopathy as compared to the results obtained in acute syndromes with perforated disk lesions and in chronic syndromes with disk protrusion

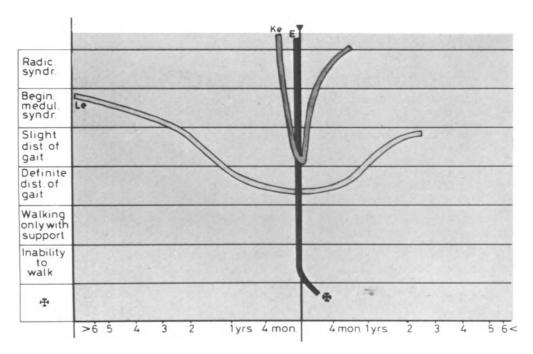


Fig. 4. Syndrome-time diagram of three patients with cervical myelopathy, demonstrating a chronic course (\underline{Le}), an acute course (\underline{Ke}) and a hyperacute course (En)

Therapeutic Results Following Laminectomy and Dural Enlargement in Cases of Cervical Myelopathy

T. DEMIREL, M. MAKSOUD, and W. BRAUN

The present report contains a survey of 50 patients who, after having been diagnosed as suffering from a cervical myelopathy, were operated on in Wuppertal between 1970 and 1978. Of the total group, two patients, one aged 61 and the other 67, died in hospital. Their general well-being and neurological state were very poor. In ten patients a medial disk prolapse at a single level was responsible for the symptoms. In six cases this was removed by an anterior approach, and in four in-stances directly from behind. In ten other cases, a more radicular symptomatology was present, so that the patients, in addition to un-dergoing laminectomy, also had to undergo bilateral foraminotomy at several levels. Despite careful treatment, in two instances we observed a marked postoperative deterioration, so that, subsequently, owing to this deterioration, we did not carry out foraminotomies in this disease. A further negative result which must be mentioned is that on two occasions, apparently owing to the position during surgery, we observed a serious postoperative deterioration of the spinal cord symptomatology and, as a result, have become extremely cautious with regard to maximal bending forward of the head under anaesthesia. Since the above mentioned groups are too small to permit a satisfactory statistical analysis, we will only report on 29 patients, on which the same technique was used. The 30th patient is not included since the dura remained closed.

The 29 patients were operated on in the following manner: Intubation anaesthesia, sitting position, laminectomy which included at least 3 and at most 7 vertebral arches. Median incision of the dura. In most cases a narrowing of the arachnoid space was observed. Also, the spinal cord bulged in dorsally under a certain pressure, so that in all such cases we had to enlarge the dural sac with lyophilized dura. In only five cases were the denticulate ligaments severed. In more recent years, where possible, we have not opened the arachnoid, thus avoiding the escape of cerebrospinal fluid.

All patients were called to follow-up examinations (Table 1). In a few cases we also received reliable information by telephone as to the state of health of the patients, in particular when patients they died as a result of another disease; this occured twice. We were unable to establish the outcome of four patients, two of whom had also died as a result of other causes. Thus, we are only able to report on the results in 25 patients.

In only two patients was the duration of follow-up less than one year. On average it was 4-5 years. The results achieved in 12 patients were good to very good. Two patients were almost completely without symptoms. The others experienced a marked improvement in the paraspastic condition. In seven patients a slight improvement or a standstill in the progression of the neurological disorders was achieved. In the case Table 1. Results of decompressive laminectomy with dural enlargement in cervical myelopathy (1970-1978)

Total	Greatly improved	Slightly improved or no progression	Worse	Unknown
29	12	7	6	4

of six patients one must speak of a progressive deterioration. When one wishes to assess the second group positively, one may say that in 4/5 of the cases the patients profitted from the interventions, and in a few of these one can even speak of a surprising improvement. Here, it must be pointed out that this table does not take into consideration temporary postoperative deteriorations, which improved rapidly with physiotherapy. The follow-up results are taken as a basis. This is also valid for those patients who, after an initial improvement, showed a deterioration.

When one compares the 12 cases in which good operative results were achieved with the six in which poor results were obtained, it is clear that age and, surprisingly enough, severity of the preoperative symptoms did not play an important role. However, patients having a shorter anamnesis have a better chance of a good result.

We have given particular emphasis to this uniformly treated group of patients since it permits the comparison of the results with those treated by other operative procedures, so as to achieve an optimal therapeutic method for this problematic disease. Surgery for Vertebrobasilar Ischaemia in Cervical Spondylosis B. WILLIAMS¹

Introduction

Cervical spondylosis is assuming a greater importance with a growing proportion of elderly persons. The commonest presentation are pain in the neck, radiculopathy and myelopathy but complaint of basilar ischaemic features also occur and such features may sometimes be the sole complaint on presentation. Four cases are presented for discussion.

Case 1

A 53 year old teacher had 18 months of neck and shoulder pain produced by looking to the left. For seven months he had giddiness on looking up and later transient loss of consciousness. These became more severe until he could not look up even slightly without losing consciousness immediately.

On examination, blood pressure was 170/20 in both arms, cranial nerves including visual fields were normal. There was slight sensory loss and weakness of the right hand and slight weakness of the right leg. Extension of the neck was limited to 40° by the onset of giddiness. Radiographs showed osteoarthritic changes at the C5/6 interspace. Arch aortography under anaesthetic (G.A.) showed no generalized vascular disease. The right vertebral artery was displaced laterally by osteophytes at the C5/6 level and the left vertebral artery was similarly affected with a loop of artery out of the inter-transverse canal (Fig. 1).

25.7.78 Operation. The left artery was exposed and the loop identified. The C5/6 interspace was drilled using the CLOWARD apparatus and a bone dowel inserted. The right vertebral artery was not seen.

Afterwards the patient noted improved power of his legs and arms, and three months after operation he had no pain or symptoms of vertebrobasilar ischaemia. He had no sensory deficit; he went back to work and has remained well for a year.

Case 2

A 60 year old man had right tinnitus which came on when he was reversing his car six months before admission. He then had intense

¹ Acknowledgements: I am grateful to Dr. Rod Hughes and Dr. Milne Anderson for referral of these cases and agreement to their publication.

vertigo and briefly lost consciousness. After that if he turned his head to the right he had bilateral blurring of vision.

A blood pressure was 150/90 in both arms. There was no neurological signs. Neck movements were full and pain free except that rotation to the right was limited by blurring of vision.

Radiology showed a wide spinal canal with multiple disc lesion. Arch aortography under G.A. showed a normal left vertebral artery. The right vertebral artery was tortuous but patent with the head straight (Fig. 2) but when the head was turned through 45° to the right there was a narrowing at C6/7. Right vertebral under local anaesthetic (L.A.) showed occlusion and onset of his symptoms with rotation.

23.3.79 Removal of osteophytes was done first. The artery had dense fibrous adhesions which could not be removed therefore the section with two stenoses was resected and anastomosed end to end.

Postoperatively the patient has remained free of symptoms.

Case 3

A 52 year old man suffered the sudden onset of weakness of the left leg with tingling down the left side of the body accompanied by severe vertigo and vomiting. He gradually recovered over three weeks. Thereafter, when looking upwards or to the left he had giddiness and a feeling of faintness.

On examination 11 weeks later blood pressure was 160/90 in the right arm and 145/90 in the left. He had no neurological signs with the head in normal position but if his head was rotated to the left until vertigo occurred, he showed nystagmus.

Radiographs showed a spinal canal of normal size with spondylosis at C4/5. Aortography under G.A. showed poor filling of the left vertebral. Catheterisation showed partial obstruction on turning the head to the left, partly due to atheroma at C2/3. The examination was repeated under L.A. When the patient turned his own head the narrowing of the artery was different from that under G.A. (Fig. 3) being blocked opposite C5/6.

26.6.79 Decompression of left vertebral artery. The anterior bars of the intertransverse foramina were removed from C3,4,5 and 6. There was no narrowing of or fibrous adhesion around the artery. The discs did not protrude and it was thought that the mobilisation of the artery was adequate.

Postoperatively the patient had no neurological complaints and could turn the head fully to the left without discomfort. On angiogram the artery still occluded at C5/6 if he turned his head hard to the left. This was asymptomatic and it was decided to observe the patient rather than re-operate.

Case 4

A 32 year old mechanic had a left hemianopia lasting for 3/4 of an hour followed by frontal and occipital headache. The following day he had an attack lasting 5 minutes after looking to the left.

On examination, 3 months after the onset the blood pressure was 130/90 in the right arm and slightly lower in the left with no limitation of neck movement and no neurological signs.

Radiographs of the neck showed degeneration of the C5/6 interspace. Arch aortography (G.A.) showed a normal vertebral artery on the right and displacement laterally on the left (Fig. 4). Vertebral artery catheterisation (L.A.) showed the artery was wide except when the head was rotated to the left (Fig. 4).

29.6.79 Operation. The artery was exposed and the anterior borders of the intertransverse foramen above and below removed. The artery was displaced laterally and the disc material and osteophytes drilled away. This allowed the artery to drop into its normal position. A routine CLOWARD's fusion was done.

Postoperatively, the patient has remained well.

Discussion

Vertebrobasilar ischaemia are known to occur from atheroma but this is not particularly associated with spondylosis or with the tortuous course around the atlas and axis, according to HUTCHINSON and YATES ($\underline{4}$). When the symptoms follow head movement and are correctable by reversing the position, then main artery occlusion is likely. Either the third part of the artery where it loops backward between foramen of C1 to the foramen magnum may be responsible or the second part of the artery. Symptoms may be associated with a hypoplastic artery on one side or may be produced by narrowing of arteries simultaneously on both sides. BAKAY and LESLIE ($\underline{1}$) believe occlusion of an artery is commoner on the side towards which the head is turned.

Vertebral angiography proves the site of the arterial obstruction. Local anaesthetic allows the patient's conscious level, eye movements and visual fields to be checked. Also if the patient turns the head he may move the vertebrae in a different way from when passively rotated (Fig. 3). Selective catheterisation seems preferable to bilateral simultaneous retrograde brachial injection of VERBIEST (7) because of the quantities of contrast medium and the occasional anomalous origins of the artery.

The anterior approach of HARVEY JACKSON reported by GORTVAI $(\underline{3})$ allows decompression over several levels on one side and may be combined with anterior fusion although access to the opposite side is limited. Many subsequent reports have appeared with operative details $(\underline{1}, \underline{3}, \underline{5}, \underline{6}, \underline{7})$.

The exposure of the vertebral artery is difficult, one feature is venous bleeding because retractors compress the jugular veins. The vertebral artery may be tortuous (Fig. 1) or may not enter the intertransverse foramina until C5 or C4. It may be of normal calibre sometimes but the artery may be narrowed by densely adherent, fibrotic connective tissue. This may be adherent to the discs or to the accompanying veins as well as the adventitia. PASZTOR describes its removal as being "relatively easy" using microsurgical methods but it presented some difficulties in Case 2. The artery was small, inexpansible and friable and a section had to be resected and anastomosed. PASZTOR has suggested that a limited exposure of the vertebral artery immediately opposite the disc may be appropriate but the recommendation that the diseased part should not be dissected until there has been a healthy portion of artery mobilized both above and below seems safer.

Vertebral fusion may allow a limited approach to the medial side of the opposite vertebral artery (1) but complete decompression and clearance of fibrotic tissue is difficult. In Case 1 movement at the disc space may have been occluding both vertebral arteries and an anterior fusion at this joint may have been sufficient. In Case 4 the disease was restricted to one vertebral level on one side and an anterior fusion might have prevented further symptoms. BAKAY and LESLIE (1) point out that absorption of both bone and disc elements, may occur after interbody fusion. In patients with bilateral disease it seems best to approach the side of the maximal disease, decompress the artery, and then fuse the bones rather than to leave the less involved vertebral artery as NAGASHIMA did in 3 out of 5 or to reoperate later on the other side (5). CLOWARD's fusion indicated if there is spondylotic myelopathy. This may be partly caused by compression of branches of the vertebral arteries which accompany nerve roots to supply cord (2). Decompression of the intervertebral foramen following slight distraction and immobilization of the affected joint may relieve this.

Conclusion

Cervical spondylosis may be accompanied by vertebrobasilar ischaemia particularly provoked by turning or extending the head. Vertebral angiography may show the site of compression. Local anaesthesia is safe and produces normal movement.

Decompression of the vertebral artery by anterior approach and removal of disc and bone allows removal of fibrous constrictions which may if severe also be resected.

Anterior fusion is helpful if there is severe arthritis at one joint, associated myelopathy, if there is bilateral disease or subluxation accompanied by bilateral vertebral arterial obstruction on movement.

References

- BAKAY, L., LESLIE, E.V.: Surgical treatment of vertebral study insufficiency caused by cervical spondylosis. J. Neurosurg. <u>23</u>, 596-602 (1965)
- CHAKRAVORTY, B.G.: Arterial supply of the cervical spine cord and its relation to the cervical myelopathy in spondylosis. Ann. Roy. Soc. Coll. Surg. Engl. <u>45</u>, 232-251 (1969)
- 3. GORTVAI, P.: Insufficiency of vertebral artery treated by decompression of its cervical part. Brit. Med. J. 2, 233-234 (1964)
- HUTCHINSON, E.C., YATES, P.O.: Cervical portion of vertebral artery: Clinico-pathological study. Brain 79, 319-331 (1956)
- NAGASHIMA, C.: Surgical treatment of ve-tebral artery insufficiency caused by cervical spondylosis. J. Neurosurg. <u>32</u>, 512-521 (1970)
- PASZTOR, E.: Decompression of vertebral artery in cases of Cervical Spondylosis. Surg. Neurol. <u>9</u>, 371-377 (1978)
- VERBIEST, H.: The lateral approach to the cervical spine. Clin. Neurosurg. <u>20</u>, 295-305 (1973)

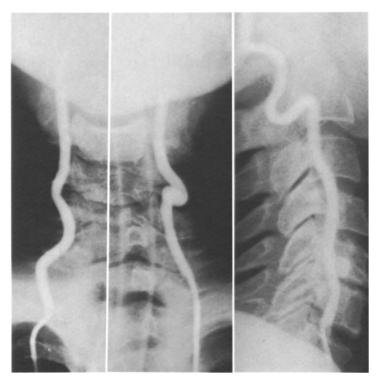


Fig. 1. Case 1. The right vertebral artery is displaced laterally opposite the severely degenerated C5/6 interspace and on the left the artery forms a loop which passes forwards between the intertransverse foramina of C5 and C6



Fig. 2. Case 2. The right vertebral artery was deviated opposite the C5/6 and 6/7 discs but its lumen remained adequate on looking ahead. With the head turned to the right, however, the lumen opposite C6/7 was severely pinched



Fig. 3. Case 3. With the head turned to the left passively under $\overline{G.A.}$ a constriction appears opposite C3/4 interspace. With the examination repeated under L.A. the constriction at 3/4 is accompanied by another opposite C5/6 and generalized narrowing between both sites. The C5/6 compression was complete on turning the head fully, this feature persisted after operation



Fig. 4. Case 4. Pre-operative and post-operative radiographs with the head turned to the left showing correction of the narrowing opposite C5/6 which was exaggerated by head rotation

Differential Therapy of Cervical Radiculopathy and Myelopathy in Degenerative Changes of the Cervical Vertebral Column

P. GRUSS, W. GRÜNINGER, and FR. ENGELHARDT

Introduction

So-called shoulder-hand syndromes representing a radiculopathy and accompanied by pain, paresis and sensory disorders, are observed with increasing frequency as a result of degenerative restructuring processes involving the cervical intervertebral discs, associated with deformative impairment of neighbouring vertebral joints, upper plates and the ligamentous apparatus. Apparently, on account of its particular degree of mobility, the cervical vertebral column is more frequently affected, a misfortune indeed when one considers the functional significance of the nervous system it contains. In the case of monoradicular disturbance, the individual symptoms of the clas-sical triad - namely pain, disturbance of sensation and paresis -can vary considerably in degree and, with further extension of the degeneration, a number of roots can become affected. If, as a result of protrusion, sequester perforation or the formation of marginal serrations, a space-consuming lesion having an impairing effect on the cervical canal develops, a myelopathy may be the result. Symptoms arising in the long tracts of the spinal cord, predominantly spastic pareses of the legs, which, with subsequently occurring sensory disorders, can lead to the transverse syndrome, can be seen. Surgical therapeutic procedures have to be oriented to the origin, nature and extent of the degenerative changes and to the degree of their damaging effect on the nerve roots and spinal cord in the cervical region.

Material and Approach

95 surgical operations using the CLOWARD technique were carried out because of cervical myelopathy: Using the original method introduced by CLOWARD, two levels were fused in 20 cases and 3 levels in 4 cases. In about 25 cases, lyophilized bovine pegs were employed instead of a bone peg autograft obtained from the crest of the ilium. On 8 occasions, employing a ventral approach, only the intervertebral space (IVS) was evacuated, the marginal serrations at the posterior edge of the vertebral body also being removed after spreading of the evacuated IVS. In 6 patients, the evacuation was carried out using a ventrolateral approach then, in two cases, at 2 levels, fusion being effected in 1 case from ventrolateral, using lyophilized pegs. On exposure from dorsal - the FRYKHOLM technique was carried out 20 times - and then only one case at 2 levels. A laminectomy for cervical myelopathy was performed on only 3 occasions. All the patients were followed up. Special care was devoted to an analysis of a group of 45 CLOWARD patients, all of whom had been operated on prior to 1975 (5).

Results

1. The Original Technique Described by CLOWARD

The exposure of the ventral surface of the cervical vertebral column, the trephining of the median hole on a level with the IVS (2) particularly readily permits the removal of marginal serrations and osteophytes that impair the cervical canal in the median region. It is also possible to achieve relief extending into the initial area of the foramina intervertebralia towards lateral by employing oblique punches.

We do not share the opinion expressed by VERBIEST (10) that the method advocated by CLOWARD is unphysiological and dangerous: In our material there is not one single case of additional damage being caused by the surgical procedure.

Admittedly, a number of details must be observed:

- a) We make preferential use of small trephine diameters in order to sacrifice as little of the vertebral body substance as possible.
- b) When the bony peg is drilled out of the iliac crest (under numerous revolutions!), we ensure that the rotational energy acting upon the spongiosa is kept as low as possible by freeing the posterior wall of the upper part of the ilium from its musculature.
- c) We make the peg a little smaller since a difference in circumference of 2 mm between the peg and hole often appears too large, in particular when a narrowed IVS proves difficult to spread.
- d) When being fitted into the prepared hole, the peg must not be compressed by blows in order to avoid a collapse of the spongiosa at the same time, however, the anterior end face of the peg should come to lie 1-3 mm below the surface of the cervical spinal column. By paying attention to a number of details relating to the performance of technical surgical skills, one can, as a rule, prevent the occurrence of a typical CLOWARD complication, namely kyphosis in the operated segment and the slipping forward of the peg. In a group of followed-up CLOWARD patients, we frequently observed more or less marked kyphosis, but, on the other hand, also established that their clinical relevance was only slight (5).

Occasionally, with the CLOWARD method, an increased stressing - and thus possibly premature degeneration - of neighbouring segments appears to manifest (see also (1)): We have seen a 57-year-old patient with a considerable C7 syndrome (Fig. 1) which, following fusion of the involved IVS, appeared to be well into remission, including the paresis, until, two years later, a recurrence occurred which, however, corresponded to the neighbouring segment, one level higher, while the originally surgically treated segment continued to reveal completely free passage (Fig. 2). The subsequently diseased neighbouring IVS was also fused, with the result that the symptoms again disappeared. In our own material, we have seen such a situation so clearly in only 1 case. We are, however, convinced that in the presence of residual symptoms and also in the case of kyphosis, following a CLOWARD operation, possible additional loading of the neighbouring segments must be taken into consideration. Here, however, excellent results are possible conservatively, by employing physiotherapeutic measures (3).

2. IVS Evacuation from the Ventrolateral Approach with and Without Fusion (10)

A number of authors report on attempts to perform solely the evacuation of the degenerated intervertebral disc using the ventral approach in an attempt to eliminate marginal serrations in the floor of the IVS. VERBIEST (9, 10) recommended "lateralizing" the approach, exposing the lateral surface of the cervical spinal column and the anterior surfaces of the transverse processes (care being taken not to injure the vertebral artery) and, in this way, to evacuate the IVS laterally in such a manner that the ventral portions of the longitudinal ligaments remain intact.

We have employed this method with its specific lateral approach in only 6 patients, but in every case with very good results:

We should like to emphasize one particular case with severe pain, disturbance of sensation and paresis in both shoulders and arms (Fig. 3). Following IVS evacuation and fusion at 2 levels employing a ventrolateral approach, an improvement was seen in all the areas of disturbance, including a largely complete elimination of the biceps paresis (Fig. 4).

In our opinion, this technique is particularly elegant in the treatment of cervical myelopathy since it is sparing for the patient. The decision as to whether, after evacuation of the IVS, a "fusing" peq should be fitted, should depend upon the local findings: If the IVS is easily spread, then the decision is likely to be in the affirmative; if, on the other hand, the situation reveals an incipient fusion, then the answer is likely to be in the negative. Here, in view of the fact that the demands in the "fusing" material are not very great, use of the lyophilized bovine peg may certainly be considered.

3. Dorsolateral Exposure (FRYKHOLM)

Using the dorsolateral exposure, cultivated in particular by FRYKHOLM $(\underline{4})$, we achieved good to very good results in all our cases; the indication must, however, be carefully differentiated. This technique would seem ideal in the surgical treatment of monoradicular syndrome, which manifests radiologically as a narrowed intervertebral foramen and a lateral indentation pattern in the contrast medium band at the appropriate level (Fig. 5). Surgically, it is sufficient to expose the vertebral arch adjacent to the lesion, to drill out intervertebral foramen from the dorsal, to remove possible sequesters after lateral incision of the longitudinal ligaments; as a rule, the root has to be elevated. This technique is both easy and avoids structural destruction of the cervical spinal column.

4. Laminectomy

For a number of years, we employed laminectomy in 3 patients presenting with a degeneration of the cervical spinal colums; admittedly, our statistics extend back only until 1971. In one case only very slight improvement was achieved, in another no improvement and in a third case considerable worsening resulted so that, here, a two-level CLOWARD procedure was subsequently performed without, however, achieving any further improvement. In our opinion, in the case of cervical myelopathy where, after all, the space-consuming lesion is more likely to "approach" from the ventral side $(\underline{8}, \underline{7}, \underline{6})$ laminectomy is only rarely indicated

although it should probably not be eliminated entirely from the surgeon's repertoire.

Conclusion

A description is given of the various surgical procedures with the aid of which, in the presence of degenerative diseases of the cervical vertebral column, the nerve roots ("radiculopathy") or spinal cord ("myelopathy") can be relieved. We give preference to the original technique advocated by CLOWARD in cases of unequivocal myelopathy, possibly with radiculopathy whenever, myelographically an appropriate obstruction pattern presents which, with respect to localization, corresponds to the clinical syndrome. The procedure permits an improvement to be achieved even when massive lesions present - complications can be reduced, in particular by exercising care with the technicalities (preparations of the bony peg).

The method for the evacuation of the cervical vertebral column-IVS, carried out more from the lateral aspect, with and without fusing pegs is apparently gaining an ever increasing range of indications. In the case of a strictly delimited monoradicular syndrome, the procedure of foraminotomy under a dorsolateral approach suggests itself while, at the present time, laminectomy is indicated only rarely (Table 1).

References

- ADAMS, C.B.T., LOGUE, V.: Studies in cervical spondylotic myelopathy. II. The movement and contour of the spine in relation to the neural complication of cervical spondylosis. Brain <u>94</u>, 569-594 (1971)
- CLOWARD, R.B.: The anterior approach for removal of ruptured cervical discs. J. Neurosurg. 15, 602-617 (1958)
- CLOWARD, R.B.: Treatment of lesions of the cervical spine by the anterior surgical approach. In: The spinal cord, p. 387. Springfield: C. C. Thomas (1972)
- FRYKHOLM, R.: Cervical nerve root compression resulting from disc degeneration and root-sleeve fibrosis. A clinical investigation. Acta Chir. Scand. Suppl. 160 (1951)
- GRÜNINGER, W., GRUSS, P.: Der Einfluß der Fusionsoperation nach CLOWARD auf die Bewegung der HWS. XXX. Jahrestag. Dtsch. Gesellsch. Neurochir., Essen Sept. 1979
- GRUSS, P.: Indikationen zur Halswirbelfreilegung von vorn. Münch. Med. Wschr. <u>121</u>, 889-890 (1979)
- GRUSS, P., GRÜNINGER, W., ENGELHARDT, Fr.: Möglichkeiten der cervikalen Fusionsoperationen bei engem Wirbelkanal mit Bandscheibendegeneration. Zbl. Neurochir. <u>37</u>, 137-141 (1976)
- KUHLENDAHL, H.: Pathogenese der sogenannten cervikalen Myelopathie. Münch. Med. Wschr. <u>111</u>, 1137 (1969)
- 9. VERBIEST, H.: La chirurgie antérieure et latérale du radis cervical. Neuro-chirurgie <u>16</u>, 154-158 (1970)
- VERBIEST, H.: From anterior to lateral operations on the cervical spine. Neurosurg. Rev. 1, 47-67 (1978)

<u>Table 1</u>				
Surgival method	Clinic	Radiography	Advantages of the method	Disadvantages
Ventral fusion (CLOWARD)	Myelopathy	Marginal spikes, complete or nearly complete contrast- medium block pat- tern	Reliable elimination of ventral space- occupying lesions	Risk of kyphosis
Ventrolateral removal (VERBIEST)	Radiculopathy also mild myelopathy	Incomplete block pattern	Sparing effect: less faulty posture	Narrow cases
(Dorsolateral) foraminotomy (FRYKHOLM)	Monoradicular syndrome	Lateral dent pattern	No structural disorders	(Limited indication)
Laminectomy	(Severe) myelopathy	Narrow spinal cord	(Broad indication)	Dorsal relief only

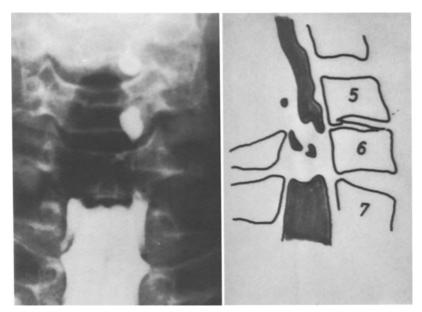


Fig. 1. Pantopaque myelogram in a 57-year-old man presenting with a C7 syndrome and considerable cervical myelopathy. Complete obstruction pattern at the level of the cervical vertebral bodies 6/7. Indication for the surgical procedure as described by CLOWARD

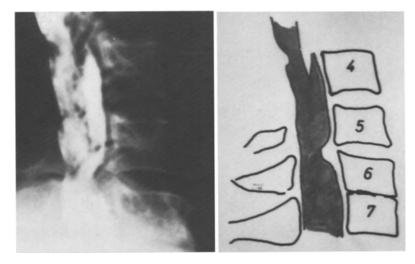


Fig. 2. (The identical case as shown in Fig. 1). Repeat myelography 2 years following surgery with the CLOWARD technique, cervical vertebral bodies 6/7 - notice the good passage at this level! - now, on account of C6 syndrome increase of myelopathy: Incomplete obstruction pattern, cervical vertebral bodies 5/6 (neighbouring segment) Indication for a CLOWARD surgical procedure at this level

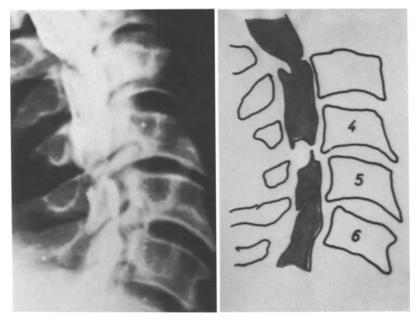


Fig. 3. Myelogram of a 45-year-old man presenting with considerable radiculopathy corresponding to the segments C5 and C6 (with paresis!)

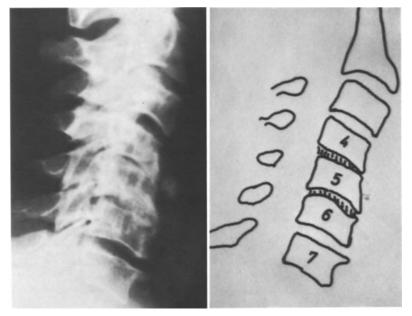


Fig. 4. (The same patient as in Fig. 3). Condition after ventrolateral evacuation and fusion of the cervical vertebral bodies 4/5 and 5/6. Almost complete elimination of the symptoms, including the paresis

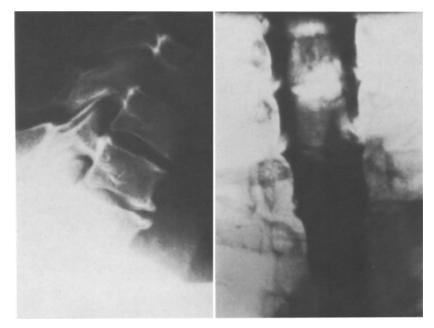


Fig. 5. Left: X-ray image of the cervical vertebral column of a 48year old woman presenting with C7 syndrome: Narrowing of the IVS, cervical vertebral body 7/thoracic 1, marginal serrations also visible. Right: (The same patient as in Fig. 5 left). Indentation pattern in the pantopaque band from the left, lateral, at a level of the cervical vertebral body 7/thoracic 1. In view of the monoradicular syndrome, indication for foraminotomy (FRYKHOLM) The Value of Computed Tomography for the Diagnosis of Spinal Lesions

I. SCHÖTER and J. WAPPENSCHMIDT

CT of the spine, being confronted with adjacent structures of extreme different density, is limited by the physical conditions of the tomograph (1). Although the demonstration of osseous lesions is no problem (9, 15, 20), the visualization of the dural cavity and the branching nerve-roots is of rare incidence (10) (Fig. 1).

Nevertheless, in our opinion CT of the spinal canal is superior to conventional X-ray investigations with regard to:

- scan-sensibility,
- crossectional view,
- visualization of the adjacent region and
- differential diagnosis of contrast stop in myelography.

Among 70 spinal CT's, performed with a Philips Tomoscan 100, respectively 200, some cases were selected to demonstrate this superiority.

Concerning X-ray sensibility, native CT is apt to demonstrate delicate lesions, failing visualization in conventional tomography $(\underline{12})$, as faint metastases, small anomalies $(\underline{8})$, osteomyelitic cavaties, medullary cysts (4) and calcified tumors (Figs. 2, 3).

Regarding the crossectional view, CT reveals the pathological process in a third radiological dimension and is valuable for the localization of space-occupying lesions, especially when computerassisted myelography delineates the outlines of the spinal cord and the surrounding cerebrospinal fluid ($\underline{5}$, $\underline{7}$, $\underline{11}$) in malformations, medullary atrophies ($\underline{17}$), intraspinal tumors ($\underline{13}$).

As to the region adjacent to the spinal canal, CT will visualize spinal lesions extending to the paravertebral area $(\underline{16})$, as hourglass tumors $(\underline{3})$, spinal dysraphism $(\underline{2}, \underline{14}, \underline{21})$, inflammatory and degenerative processes.

Finally computer-assisted myelography is helpful in differential diagnosis of space-occupying lesions $(\underline{6}, \underline{18}, \underline{19})$ causing complete myelographic block. A series of consecutive scans, crossing the level of the pathological process as well as the lower and upper adjacdent region, will reveal specific signs for the differentiation of epidural, para- and intramedullary lesions. The epidural process will reduce the outlines of the contrast-ring and its contents, giving room to the epidural space.

The enlarged contrastring in central position, reducing the epidural space, is pathognonomic for the intramedullary lesion (Fig. 4).

The juxtamedullary space-occupying lesion displaces the deformed spinal cord. Consequently, the contrastring becomes asymmetrical at the level of the upper and lower pole of the process, since the CSF-space is enlarged in the area adjacent to the lesion and diminished in the opposite region (Fig. 5).

As a consequence of our examinations, we feel that CT of the spine allows diagnosis in many cases. However, until further experience has accumulated, spinal CT has to be complemented by conventional X-ray investigations and operative findings.

References

- AMBROSE, J.: Computerized transverse axial scanning (tomography). Part 2. Clinical applications. Brit. J. Radiol. <u>46</u>, 1023-1047 (1973)
- BALERIAUX-WAHA, D., OSTEAUX, M., TERWINGHE, G., DE MEENS, M., JEANMART, L.: The management of anterior sacral meningocele with computed tomography. Neuroradiol. 14, 45-46 (1977)
- BALERIAUX-WAHA, D., TERWINGHE, G., JEANMART, L.: The value of computed tomography for the diagnosis of hourglass tumors of the spine. Neuroradiol. <u>14</u>, 31-32 (1977)
- 4. DI CHIRO, G., AXELBAUM, S.P., SCHELLINGER, D., TWIGG, H.L., LEDLEY, R.S.: Computerized axial tomography in syringomyelia. N. Engl. J. Med. <u>292</u>, 13-16 (1975)
- DI CHIRO, G., SCHELLINGER, D.: Computerized tomography of spinal cord after lumbar intrathexal introduction of metrizamide (computer-assisted myelography). Radiology 120, 101-104 (1976)
- 6. COIN, C.G., CHAN, Y.S., KERANEN, V., PENNINK, M.: Computer-assisted myelography in disc disease. J. of Computer-assisted tomography <u>1</u> (4), 398-404 (1977)
- 7. COIN, G., KERANEN, V.J., PENNINK, M., AHMAD, W.D.: Computerized tomography of the spine and its contents. Neuroradiol. <u>16</u>, 271-272 (1978)
- CLAUSSEN, C.D., LOHKAMP, F.W., v. BAZAN, U.B.: The diagnosis of congenital spinal disorders in computed tomography (CT). Neuropädiatrie <u>8</u>/4, 405-417 (1977)
- 9. GROSSMANN, Z.D., WISTOW, B.W., WALLINGA, H.A., HEITZMANN, E.R.: Recognition of vertebral abnormalities in computed tomography of the chest and abdomen. Radiology <u>121</u>, 369-373 (1976)
- HAMMERSCHLAG, S.B., WOLPERT, S.M., CARTER, B.L.: Computed tomography of the spinal canal. Radiology <u>121</u>, 361-367 (1976)
- 11. HARWOOD-NASH, D.C.F., FITZ, C.R., RESJO, J.M., CHUANG, S.: Congenital spinal cord lesions in children and computed tomographic Metrizamide myelography. Neuroradiol. <u>16</u>, 69-70 (1978)
- 12. HOUNSFIELD, G.N.: Computerized transverse axial scanning (tomography). I. Description of system. Brit. J. Radiol. <u>46</u>, 1016-1022 (1973)
- ISHERWOOD, I., PULLAN, B.R.: Computed tomography on the spine. Applications, limitations and new approach to anatomical registration. Neuroradiol. 15, 47-48 (1978)
- 14. JAMES, H.E., OLIFF, M.: Computed tomography in spinal dysraphism. J. of Computer-assisted tomography <u>1</u> (4), 391-397 (1977)

- 15. KERSHNER, M.S., GOODMANN, G.A., PERLMUTTER, G.S.: Computed tomography in the diagnosis of an atlas fracture. Am. J. Roentgenol. <u>128</u>, 688-689 (1977)
- 16. NAKAGAWA, H., HUANG, Y.P., MALIS, L.I., WOLF, B.S.: Computed tomography of intraspinal and paraspinal neoplasms. J. of Computerassisted tomography <u>1</u>, 377-390 (1977)
- 17. OBERSON, R., AZAM, F.: CAT of the spine and spinal cord. Neuroradiol. <u>16</u>, 369-370 (1978)
- OBERSON, R., AZAM, F., REGLI, T.: Computertomographie des Wirbelkanals mit wasserlöslichen Kontrastmitteln (Metrizamide). Akt. neurol. 4, 195-199 (1977)
- 19. SARTOR, K., RICHERT, S.: Computertomographie des cervikalen Spinalkanals nach intrathekalem Enhancement: cervicale CT-Myelographie. Fortschr. Röntgenstr. <u>130</u>/3, 261-269 (1979)
- 20. SHELDON, J.J., SERSLAND, T., LEBORGNE, J.: Computed tomography if the lower lumbar vertebral column. Normal anatomy and the stenotic canal. Radiology 124, 113-118 (1977)
- 21. WOLPERT, S.M., SCOTT, R.M., CARTER, B.L.: Computed tomography in spinal dysraphism. Surg. Neurol. <u>8</u>, 199-206 (1977)

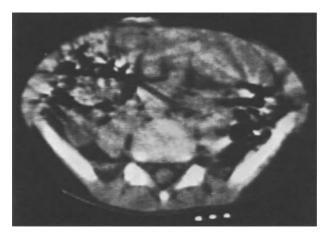


Fig. 1. Native scan (L-5): Visualization of the dural cavity and branching nerve-roots



Fig. 2. Native scan (D-7): Osteomyelitic cavity of a sequestrum

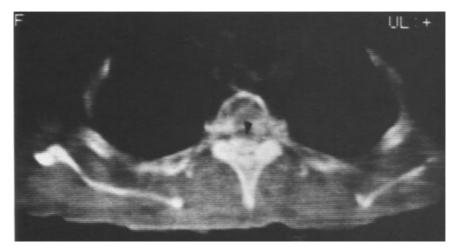


Fig. 3. Native scan (D-3): Calcified meningeoma

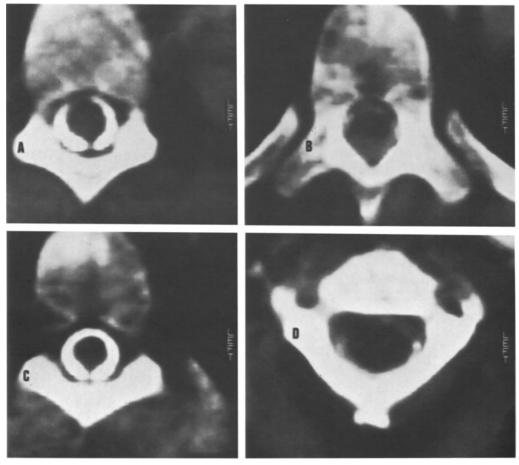


Fig. 4. Computer-assisted myelography in intramedullary spaceoccupying lesion: <u>A</u> (D-11/12) well-shaped contrastring. <u>B</u> (D-11) spinal cord enlarged, CSF-space diminished. Contrastring in central position. <u>C</u> (D-10/11) normally shaped ring. <u>D</u> (C-3) spinal cord enlarged, CSF-space diminished. Contrastring in central position

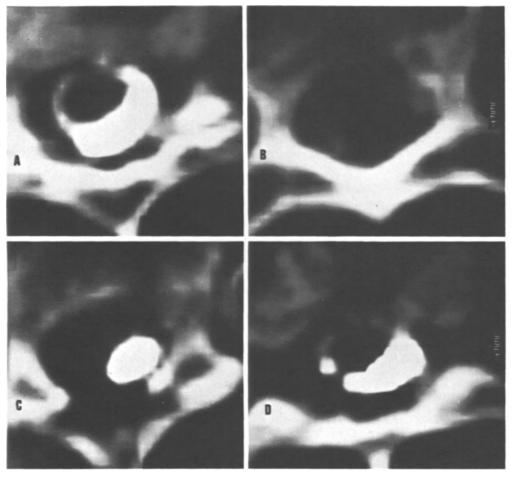


Fig. 5. Computer-assisted myelography in juxtamedullary spaceoccupying lesion: <u>A</u> (D-11) lower tumor pole: CSF-space enlarged, deformed and displaced spinal cord. <u>B</u> (D-11) scan through the tumor: CSF-space almost occluded. <u>C</u> and <u>D</u> (D-10) upper tumor pole: CSF-space enlarged Anterior Cervical Steel Plate Spondylodesis in Mobile or Unreducible Traumatic and Osteolytic Cervical Dislocations

P. C. POTTHOFF

Introduction

The technique of anterior reposition and bone fusion of cervical dislocations according to CLOWARD, even with special modifications (as described in Germany by GROTE (6), BUSCH and SCHÜRMANN (3), LAUSBERG and PIA (9), and others), may not be sufficient to accomplish an adequate and stable reposition of the cervical spine in all cases. In such rare problem cases of extreme cervical instability, bony destruction or also ureducible dislocation an additional cervical steel plate spondylodesis may offer a neurosurgical solution.

Material and Method

In a consecutive group of 177 patients with cervical operations for discs, osteochondrosis, narrow spinal canal and extradural tumors, as well as traumatic dislocations, six patients developed or posed problems of the above mentioned type. One patient with vertebral body necrosis after operation according to CLOWARD (4) and severe instability was operated on elsewhere and is not considered here. The diagnoses and forms of treatment of the other five patients are listed in Table 1.

In case 1, a boy with neurofibromatosis, a 6 cm neurinoma was removed, originating from the 8th cervical root and extending into the thoracic dome on the right side, on March 7, 1978. Hemilaminectomy from C6 to Th 1, and posterior rib resection I to III on the right side, had to be performed to remove the tumor in toto. The boy was discharged in good condition with only a sensory deficit corresponding to C8 on the right side. Sixteen days after surgery he was brought to the hospital for acute spontaneous severe nuchal pain, with no additional neurological deficit. X-rays showed spontaneous dislocation C6 versus C7, the vertebral body C6 having slipped for 3/4 anteriorly over C7. After CRUTCHFIELD-extension a CLOWARD operation C6/7 was done, that proved unstable with a new dislocation C6/7 on the first postoperative day. Prolonged extension achieved no sufficient reposition, therefore on April 20, 1978 an anterior cervical steel plate spondylodesis was performed, resulting in orthograde realignment of the cervical spine in the lateral aspect with stable fusion, in the anterior aspect there remained minor scoliosis. Fifteen months after the last operation, the fusion is remaining stable, but progressive thoracic kyphosis is noted below the fusion, the reason being excessive muscular atrophy of the erector trunci musculature due to neurofibromatous nerve degeneration. There is still no increased neurological deficit (Fig. 1). A similar problem case of cervical, yet traumatic dislocation after upper thoracic spine surgery was described in 1978 by DRENNAN and KING.

I DTOT			
Patient	Diagnosis	OP	Result
1. G.D., male April 28, 1963	Spontaneous dislocation C6/7 after spinalthoracic operation of neurinoma in Neurofibro- matosis RECKLINGHAUSEN	C5-Th ₂ (April 20, 1978)	Stable minor scoliosis, progressive thoracic kyphosis, C8 deficit right, visits school
2. B.K., male April 20, 1921	Osteolytic C4-compression metastasis of adeno - carcinoma of unknown origin pain, in- cipient paraparesis	C3-5 (Sept. 7, 1978)	Died from pulmonary insufficiency (Sept. 14, 1978)
3. C.M., female August 23, 1921	Traumatic dislocation C6/7 with secondary triparesis 5 days posttraumatic after traffic accident Nov. 7, 1978	C6-Th1 (Nov. 14, 1978)	Stable orthograde minor motor deficit right arm walking, independent not working
4. M.H., male July 19, 1951	Traumatic dislocation C6/7 secondary transversal deficit C6, 5 days posttraumatic after traffic accident Dec. 14, 1978	С6-Тh1 (Dec. 20, 1978)	Died quadriplegic after repeated cardiac stand still (Jan. 6, 1979)
5. N.D., male August 2, 1951	Traumatic dislocation C4/5 after sport accident Feb. 9, 1979 with immediate quadri- paresis (paraplegia of legs, no micturition), non-redres- sible by CRUTCHFIELD	C4-6 (Feb. 9, 1979)	Stable , minor kyphosis, finger paresthesias walking independent working parttime

Case 2 presented as a metastatic destruction of the C4 body with rapidly increasing myelopathy and crises of pulmonary insufficiency. The vertebral body of C4 was removed on Sept. 7, 1978, and the defect bridged by a C3 to C5 anterior steel plate (Fig. 2). The patient continued with alternating respiratory insufficiency and died 7 days postoperatively in another hospital, no autopsy being obtained. The histology of the body of C4 revealed an adenocarcinoma of unknown origin.

Case 3 was an instable dislocation C6/7 developing within 5 days after a traffic accident with secondary triparesis. After fusion on Nov. 14, 1978, the neurological deficit regressed within 14 days. There exists radicular C7-pain on the left side and a slight spastic-ataxic gait 6 months after operation (Fig. 3).

Case 4 suffered a traumatic dislocation C6/7 by traffic accident without primary neurological deficit, diagnosed at another hospital. Five days posttraumatic an almost complete transversal syndrome below C6 developed rapidly. A fusion operation one day later did not change the deficit and the patient died quadriplegic in a spinal rehabilitation center 16 days after operation.

Patient 5 came down besides a trampolin board on the ground with his head first when doing hobby sports exercises. He was immediately paraplegic in his legs and showed considerable, distally pronounced paraparesis of his arms. He sensed paresthesias in both hands and showed definite sensory loss from a D3-D5 level downward. No micturition was observed. X-rays two hours after the accident showed traumatic dislocation C4/5 with anterior angulation and anterior oblique compression of the C5 body. Manual manoevers and subsequently reposition manoevers with applied CRUTCHFIELD-extension failed and it was decided to operate on the patient immediately (Fig. 4). At the time of this congress, the patient is back to work parttime as industrial merchant, his functional status being demonstrated in a film, showing complete motion of all extremities. There is perfect control of bladder and bowel function, and only occasional C6 pain alternating in both arms, not incapacitating the patient. Mobility of the cervical spine is very good. (See comprehensive list in Table 1.)

The operative method was similar in all cases: Anterior-lateral exposition of the cervical spine from the right side, identification and excavation of the respective intervertebral disc by drill and rongeurs under image amplifier control, fitting of an autologous bone dowel from the right iliac crest (except case 2), anterior apposition of a steel plate of sufficient size to cover at least the complete ventral surfaces of the neighbouring vertebral bodies. Tight fixation of the steel plate by ASIF-screws (Fig. 5) of sufficient length, i.e. depth of the vertebral body plus thickness of the steel plate plus allowance for minor dorsal penetration through the cortical bone matrix. Wound closure without drainage. Immobilisation with neck plastic bandage for two weeks.

Results

Results were excellent in regard to cervical stability and orthograde alignment at the lesion site in cases 3 and 5, with good residual mobility of the cervical spine in case 5, sufficient in case 3. Cervical stability and alignment is good in case 1, however, mobility of the cervico-thoracic spine is severely reduced by the fixed and slightly progressive thoracic kyphosis due to muscular atrophy with neurofibromatosis RECKLINGHAUSEN, as mentioned on page 3. Neurological deficit is minimal in cases 1 and 3, nil in case 5. Patients of case 2 and 4 died and cannot be evaluated.

Conclusions

Anterior cervical spondylodesis can be achieved by several ways and in special modifications (literature see introduction). The principle of anterior cervical steel plate spondylodesis is known to us also from a personal communication by BURRI (<u>1</u>), but reviewing the literature no publication could be found on this method (compare 1 case of ARMENISE et al., this congress). The principle is similar to anterior cervical interbody fusion combined with onlay bone grafts, fixed by bone screws, as previously described by MINGRINO et al. (<u>10</u>) and by HEMMER (<u>7</u>) in single cases, but appears considerably more stable than the onlay bone graft fusion.

Recently HERRMANN $(\underline{8})$ has described a dorsal metal plate fixation method for dorsal stabilization of the lumbar and thoracic spine, used before by French surgeons. HERRMANN pointed out that "the force which can be applied to the plate by the screws is considerable, so that reposition of a displaced vertebral body is possible".

It is this principle of forced orthograde reduction *and* stabilization that gives advantage to the here described method of anterior cervical steel plate spondylodeses applied in mobile or unreducible dislocations. As shown in case 5, this method was the ultimate way to achieve cervico-spinal realignment, fortunately accompanied by an excellent functional result.

The wider use of this method may have to be confined to very special cases. Several factors still remain unknown with the limited experience evaluated here, e.g., to what degree in the long-time evaluation anterior steel plate spondylodesis will interfere with cervicospinal mobility, if it will be necessary or might be advisable (or contraindicated?) to remove the alloplastic material after what length of time. In spite of these open questions and the limited material, we felt inclined to demonstrate this method having proved so useful to us in the described problematic cases and possibly useful to others in similar conditions.

References

- 1. BURRI, C.: Personal communication. 6th Seminar in Oncology on metastases. University of Ulm, March 24, 1979
- BUSCH, G.: Anterior fusion for cervical spondylosis. J. Neurol. 219, 117-126 (1978)
- BUSCH, G., SCHÜRMANN, K.: Anterior fusion in fracture-dislocation and in spontaneous fractures of the cervical spine. Excerpta Medica Int. Congr. Series <u>242</u>, 340-349 (1971)
- CLOWARD, R.B.: Recent advances in surgery of the cervical spine. Excerpta Medica Int. Congr. Series <u>242</u>, 285-293 (1971)
- DRENNAN, J.C., KING, E.W.: Cervical dislocation following fusion of the upper thoracic spine for scoliosis. A case report. J. Bone Joint Surg. Am. <u>60A</u>, 1003-1005 (1978)
- GROTE, W.: Ventral fusion. Excerpta Medica Int. Congr. Series <u>242</u>, 355-360 (1971)

- HEMMER, R.: Fusion of the cervical spine in childhood. Excerpta Medica Int. Congr. Series 242, 350-354 (1971)
- HERRMANN, H.D.: Transarticular (transpedicular) metal plate fixation for stabilization of the lumbar and thoracic spine. Acta Neurochirurgica <u>48</u>, 101-110 (1979)
- 9. LAUSBERG, G., PIA, H.W.: Stabilizing procedures of the cervical spine. Excerpta Medica Int. Congr. Series <u>242</u>, 365-374 (1971)
- MINGRINO, S., CAPPELLIN, M., MERLI, G.A.: Anterior and posterior vertebral fusion in dislocations and compression fractures of the cervical spine. Excerpta Medica Int. Congr. Series <u>242</u>, 335-339 (1971)

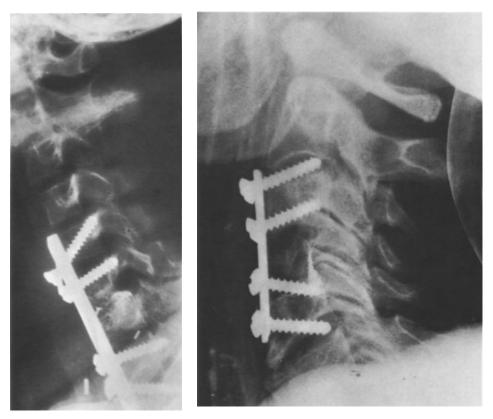


Fig. 1 (left). Anterior steel plate spondylodesis C5 to Th 2 in spontaneous dislocation C6/7 and after instable CLOWARD-procedure at C6/7 (case 1)

Fig. 2 (right). Anterior steel plate spondylodes is C3 to C5 in osteolytic C4-compression by metastasis of adenocarcinoma of unknown origin (case 2)

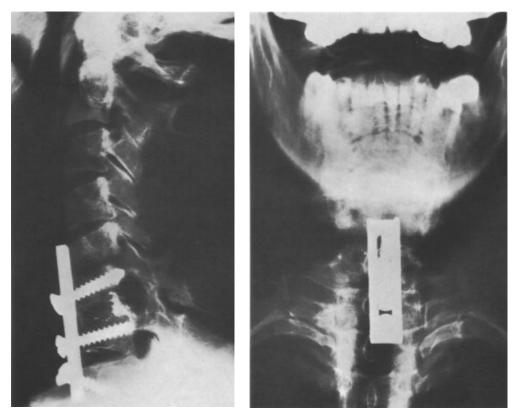


Fig. 3. Anterior steel plate spondylodesis C6 to Th1 in traumatic dislocation C6/7. Left: lateral view; right: anterior-posterior view 3 months postoperative (case 3)

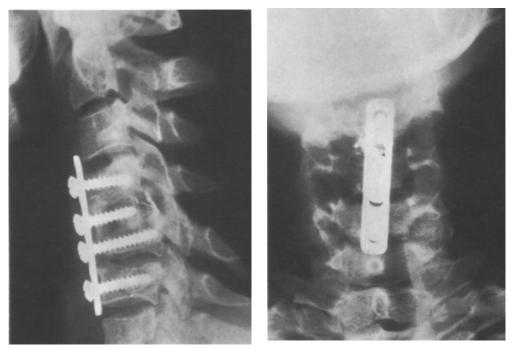


Fig. 4. Anterior steel plate spondylodesis C4 to C6 in unreducible traumatic dislocation C4/5. Forced reduction by screw tightening to anterior steel plate. Slight kyphosis. Full functional recovery. Result 5 months postoperative. Left: lateral view; right: o.-p.-view (case 5)

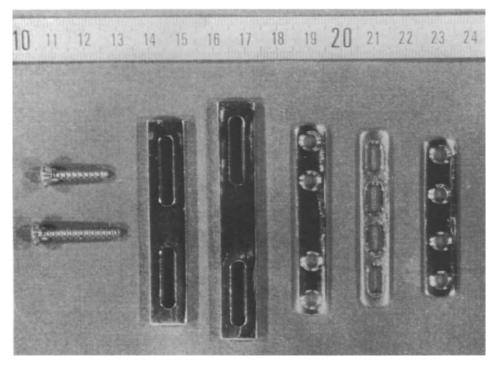


Fig. 5. Material used in anterior steel plate spondylodesis Bone screws from 22 to 29 mm with a diameter of 3.6 mm. Straight slotted contact plates and bone plates with holes or slots of different lenghts and 10 to 12 mm width (supplied by MARTIN) Hydrocephalus in Childhood

Long-Term Results in the Operative Treatment of Hydrocephalus in Children

R. HEMMER

Over two decades after the pioneer work of SPITZ-HOLTER and PUDENZ-HEYER, the treatment of hydrocephalus has reached a point, where a critical assessment of methods, complications, and late results seems useful.

I don't like to enter into a discussion of numerous modifications of the drainage systems. It is certain that hitherto none of the systems used and modifications are able to exclude complications. None of the systems meets the biological demands of a long-term follow-up. Growth processes, obstructions of the catheter, ageing of the material etc. will in due course take place in all shunts implanted in babies.

With that let us proceed to the complications as pointed out in Table 1.

No. of patien (n = 569)	ts with complications	No. of compli- cations	8
	Slipping of ventricular catheter	60	7,8
Ventricular	Obstruction	200	26,1-37,6
	_Other disturbances	28	
Γ	Slipping of cardiac catheter	64	8,3
Cardiac	Obstruction of catheter with- out thrombosis of the vein	82	10,7
catheter {	Obstruction of catheter with thrombosis of the vein	156	20,4 /46,1
	Kinking or other displace- ment within the vein	51	6,7
	Disturbances within the valve or flushing device	75	9,8
	Infection, Sepsis	38	5,0
	Disturbances of peritoneal catheter (n = 92)	12	1,5
		766	

Table 1. Cause of shunt revisions in babies and children 1961-1976 (total of shunts: 752)

Some complications are avoidable or may be minimized. Three of these we pointed out in papers presented 1967 and 1969 $(\underline{3}, \underline{4}, \underline{5})$.

Of primary importance are dislocations of the catheters. Our studies from 1961-1971 (2) have shown that 27% of the complications were dislocations of the ventricular or the heart catheter. After suggesting an innovation for the Holter system and the introduction of simple silicone sleeves with application of a metal bulge to the metallic ends in 1967 and 1968, this complication could be reduced within four years from 16%-1% in ventricular catheters and from 11%-0% in heart catheters. The total amount of slipping catheters from 1961-1976 was reduced to 16%. We suggested a similar innovation for the Pudenz system few years later (Figs. 1, 2).

Another complication is due to the *localisation* of the *ventricular catheter*. Blood, brain tissue, but especially choroid plexus may penetrate into the ventricular catheter and the valve. In the past several specially constructed ventricular catheters have been manufactured. However, a mere change of shape alone cannot prevent the obstruction. The best we can do is to place the catheter into the anterior horn, where there is very little plexus.

A further complication concerns the valve itself (6, 12) which we described as *relative valve insufficiency* in 1969. Either the amount of CSF to be drained is greater than the capacity of the valve, or the capacity of the valve is too large. The checking of the valve during operation should not be limited to forcing water through the valve. The check must include exact flow characteristics. We use a hydrostatic glass manometer filled with 0,9% saline for checking the flow characteristics in drops per minute down to 100 mm H₂O. In a similar way, we proceed with our follow-up patients in the outpatient department.

To a certain extent the formation of a *thrombosis* may be influenced. We know that thrombosis is seen rarely, if the catheter is localised within the auricle of the heart. The early preventive re-implantation of the heart catheter in the clinically healthy can avoid thrombosis. The risk of thrombosis corresponds to the amount of displacement of the distal catheter in the cranial direction and the duration of the displacement. This was clearly shown intraoperatively when performing preventive lengthening of the catheter - first suggested by TSINGOGLOU and FORREST in 1968 (13). We found intraoperatively that thrombosis occurred after indwelling catheters were left in situ for 2 years in 20%, up to 6 years in 67%, and up to 14 years in 85% (Fig. 3). In 100 patients with preventive re-implantation of the heart catheter, we found the end of the catheter within the jugular vein 59 times, within V. brachiocephalica 35 times and in 6 within the V. cava superior. In 37 patients we needed a peritoneal drainage. The shunt could be removed in 4 patients. One fact seems very impor-tant to us. In 37% of children who seemed to be in good health we found an increased intraventricular pressure of 100 to 480 mm water.

Sepsis is an even graver risk. Though it occurred in only 5% at our last investigation in 1976, it was lethal in one-third of the cases. The use of newer aseptic solutions at operation covering of the skin with plastic-foil, perioperative co-trim preparations (Bactrim, Eusaprim) seems to be effective. Repeated revisions increase the incidence of infections. When infections occurs, the shunt must usually be removed before infection can be brought under control. Mortality. Our analysis is based on autopsy findings (41%) and clinical data between 1961-1978. There were 590 children with hydrocephalus excluding hydrocephalus caused by tumors. The total mortality to date amounted to 137 (23%) (Fig. 4), the operative mortality to 0,6%. Neonates accounted for 52% of the deaths, infants for 31%. Children over 6 years of age accounted for only 17%-18% of the deaths.

Statistical evaluation showed in 1961-1973 that the life expectancy after five years of age was 96% ($P_{05} = 0,955$) for hydrocephalus communicans, 83% ($P_{05} = 0,828$) for hydrocephalus with myelocele, and 78% ($P_{05} = 0,779$) for obstructive hydrocephalus. The probable mortality up to the age of ten is seen in Fig. 5.

Mortality amounted to 29% in children who were operated on 16-18 years ago, and to 41 and 47% in children who were operated on 14 and 15 years ago. The sharp drop in the mortality from 1966/67 onward is particularly striking. This is not so much due to new operative technical changes, but rather to a more intensive follow-up of the children and better informed parents. The continued cooperation between surgeon, pediatrician and parents is a prerequisite for successful care.

One word to *shunt dependence:* The question of shunt removal is forced upon physician and parents after few years of good health of the child. The only safe indication for the removal of a shunt is when a disturbance of the drainage is diagnosed and in the course of several weeks or months no signs of increased intracranial pressure appear. So we could remove the drainage system in hydrocephalus communicans in 9%, but after some time 1/3 of these needed a new drainage. In myeloceles we removed the shunt in 10%, with full compensation. The removal of a drainage system without sufficient indication is a serious risk which often leads to emergency situations. For a greater part of the shunted children, the saying is valid: "Once a shunt - always a shunt" (7).

The results relating to the mental development underline the importance of early operation. Impressively we can see this in myeloceles, who were from the beginning of their life medically cared for (Fig. 6). Since 1970 we have performed 605 psychological tests¹ in our outpatient department, and we could confirm our first results. The mental development is significantly higher in children with myeloceles than in the other group of hydrocephalus. Longitudinally we saw in 62% of the tested children the same Intelligence Quotient, in 24% better results, and in 14% worse results. The better results are mostly based upon the stability of the psychological condition, a greater ability to work, and a harmonic maturity. The causes of the decrease of intellectual ability lie partly in the progression of cerebral damage, convulsions etc.

The school situation is marked by the high percentage of teachable myeloceles (99%), in contrast to the ability to attend school in 77% of other hydrocephalic infants. If we consider the group of teens which were operated on 14-18 years ago, the questions arises as to what happens in relation to their apprentice-ship. We found out that in the survival group of 108 registered teens (28 myeloceles, 80 hydrocephalus communicans and occlusus) 25 enter an apprentice-ship, 6 an occupation training year and from a subgroup of 26, 13 enter secondary school, 6 technical school, and 7 grammar school. We estimate that 53% of these teens will have an occupation. This can be a commercial one, handicraft or an occupation of social worker (Table 2). It may happen

¹ With the support of Deutsche Forschungsgemeinschaft.

Table 2. Ages 15-18 in apprenticeship and higher education	apprent	iceship and hig	gher e	ducation		
Commercial employments (10)	(10)	Craftmanship (9)	(6)	Social worker	(9)	Higher education (6)
Salesman	(1)	Shoemaker	(1)	Geriatric nurse	(1)	
Office worker	(2)	Mason	(1)	Infant nurse	(1)	
Office mechanic	(1)	Mechanic	(1)	Kindergarten	(1)	
Bank clerk	(1)	Gardener	(3)	Nursing	(1)	
Civil service	(1)	Stock-keeper	(1)	Housekeeping	(1)	
Postman	(1)	Tailoress	(1)	Domestic science teaching	(1)	
Business administration (1)	(1)	Farming	(1)			
Commercial-school teaching	(1)					
Technical draftsman	(1)					

able :	5.	Ages	151	8	in	apprenticeship	and	higher	education
	1	•							

that some of the patients attending grammar-school choose an academic occupation. 28% of the children are in a special school for physically and mentally handicapped, including 9 children in protected workshops. The rest of 10% are children put out to be nursed. One word about the very severe group of myeloceles. We know from our continued research in mental development that this group has a higher mental development on the average and despite all that physical handicap of being chairbound and incontinent, many of this group are employed full-time. LAURENCE and BERESFORD (11) found that 70% of the non-operated myeloceles - this is a selected group - are in such a situation.

Conclusion

The clinical experience of 590 shunts in newborns and babies of a total of 1246 patients in whom hydrocephalus has been treated by ventriculo-atrial or ventriculo-peritoneal shunts is presented. None of the systems in use excludes the complications due to growth and obstruction etc. Some complications are avoidable: the detachment of catheters has been significantly reduced by modifying the connections, and obstructions of the ventricular catheter reduced by localisation within the frontal horn of the ventricle. Relative valve insufficiency is excluded by checking each valve before implantation for flow characteristics by a glass manometer. Obstruction of the cardiac catheter by venous thrombosis is difficult to eliminate, and appears to increase with the length of time since catheter insertion, and the withdrawal of the catheter from the right atrium. Routine prophylactic lengthening or re-implantation of the distal catheter may keep it patent. Sepsis has been reduced from 9%-5%, but this is still the most dangerous complication. Statistical computations show a decreasing life expectancy from communicating hydrocephalus, to hydro-cephalus with myelocele, and obstructive hydrocephalus. Without doubt the highest probability of mortality in all groups occurs within the first postoperative year. At present, the mortality amounts to 25%-31% in children who were operated on 16 and 28 years ago. The highest mortality (41%-47%) occurred in children who were operated on 14 and 15 years ago, the total mortality within 18 years in 23%. The mental development appears to be significantly higher in children with myelomeningoceles than in other groups of hydrocephalus. The development in relation to the later apprenticeship is directed to commercial employments, craftsmanship and social workers. We estimate that 53% of these will practise an occupation.

References

- BÖHM, B., HEMMER, R.: Die Bedeutung der präventiven Neuanlage des Herzkatheters bei operierten Hydrocephalus-Kindern. Monatsschr. Kinderheilk. <u>127</u>, 357-359 (1979)
- BUSSE, G.: Komplikationen mit der Hirnseitenventrikel-Herzohrdrainage bei 586 Patienten mit Hydrocepahlus. Diss. Med. Fakultät Freiburg 1974
- 3. HEMMER, R.: Complications relating to ventricular-venous shunts: a five-year study. Develop. Med. Child Neurol. 13, 108-112 (1967)
- 4. HEMMER, R.: Komplikationen beim atrio-ventrikulären Shunt und ihre Vermeidung. Z. Kinderchir. 5, 10-24 (1967)
- 5. HEMMER, R.: Dringliche chirurgische Eingriffe an Gehirn, Rückenmark und Schädel im frühen Säuglingsalter. Stuttgart: Enke 1969

- HEMMER, R., POTTHOFF, P.C.: Die Ventilinsuffizienz bei ventriculoauriculären Drainagen. Z. Kinderchir. <u>8</u>, 11-16 (1970)
- 7. HEMMER, R., BÖHM, B.: Once a shunt, always a shunt? Develop. Med. Child Neurol. <u>18</u>, Suppl. 37, 69-73 (1976)
- HEMMER, R.: Surgical Treatment of Hydrocephalus: Complications, mortality, developmental prospects. Z. Kinderchir. <u>22</u>, 443-452 (1977)
- HEMMER, R., CZINK, A.: Lethality of children with hydrocephalus and ventriculo-atrial shunts. Z. Kinderchir. <u>21</u>, 313-326 (1977)
- 10. HEMMER, R.: Therapie des Hydrocephalus. Monatsschr. Kinderheilk. <u>127</u>, 337-341 (1979)
- LAURENCE, K.M., BERESFORD, A.: Degree of physical handicap, education and occupation of 51 adults with spina bifida. Brit. J. Preventive & Social Med. <u>30</u>, 197-202 (1976)
- 12. POTTHOFF, P.C., HEMMER, R.: Valve insufficiency in ventriculoatrial shunts. Develop. Med. Child Neurol. 20, 38-41 (1969)
- 13. TSINGOGLOU, S., FORREST, D.M.: Therapeutic and prophylactic lengthening of distal catheter of the holter ventriculo-atrial shunt. Develop. med. child neurol., Suppl. 16, 35 (1968)

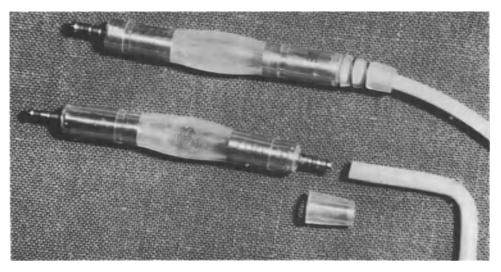


Fig. 1. Connection for Holter system with Hemmer-Sleeve

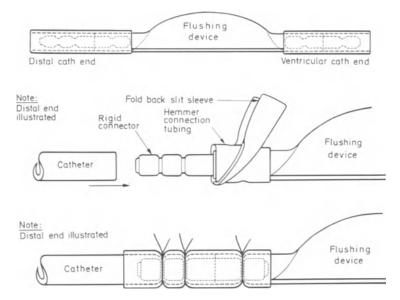


Fig. 2. Connection with Hemmer-Sleeve for Pudenz system

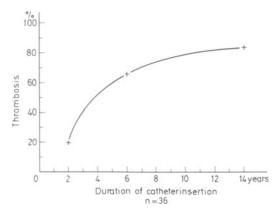
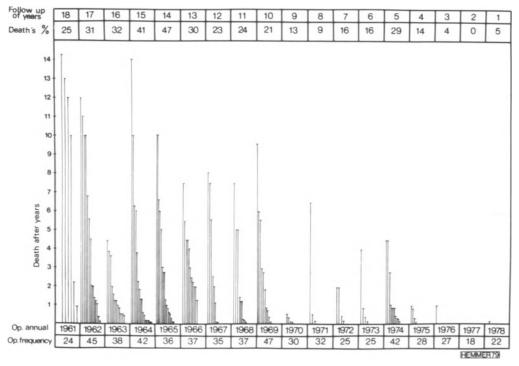


Fig. 3. Percentage of thrombosis in preventive re-implantation in relation to the duration during which the first catheter left in situ



<u>Fig. 4</u>. Death rate 1961-1978 (N = 137)

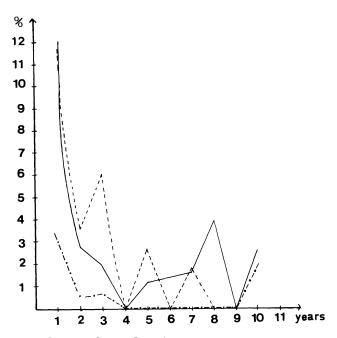


Fig. 5. Statistic likelihood of mortality for 10 years — myelocele, --- hydrocephalus occlusus, -.-.- hydrocephalus communicans

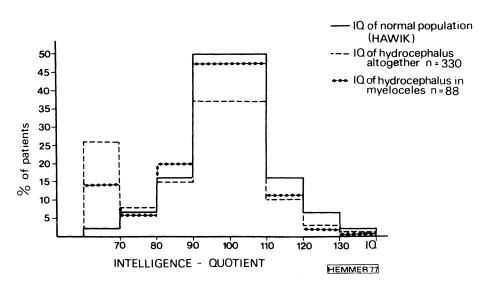


Fig. 6. Intelligence Quotient (HAWIK) of children between 14-18 years of age with hydrocephalus communicans and hydrocephalus occlusus and myeloceles

CT Follow-Up of Hydrocephalus in Children R. SCHÖNMAYR, J. ZIERSKI, and A. L. AGNOLI

The value of computerised tomography (CT) in the evaluation of infantile hydrocephalus has been well established ($\underline{8}$, $\underline{9}$, $\underline{10}$) and typical CT findings in hydrocephalus of diverse etiology have been described ($\underline{3}$, $\underline{5}$, $\underline{7}$, $\underline{8}$, $\underline{11}$). It has been also shown that after shunting, the cerebral mantle surrounding hydrocephalic ventricles becomes thickened over a period of days to weeks ($\underline{2}$, $\underline{13}$). For the evaluation of the ventricles in CT, several methods of determination of ventricular volume have been reported ($\underline{1}$, $\underline{4}$, $\underline{12}$, $\underline{14}$) but their clinical application is cumbersome, particularly if repeated scans in the same patient have to be analysed.

The aim of the present study was the analysis of CT findings in children shunted because of hydrocephalus of different etiology.

Material and Methods

Sixty-seven children with ventricular enlargement of diverse etiology who were subjected to shunting and/or removal of tumor and in whom pre- and postoperative CT were available were analysed. Each child had one to four postoperative CT scans within a period of 2 weeks to one year. Planimetric and volumetric analysis for the follow-up investigations were performed with the help of an Evaluoscope (Siemens Comp.) in selected cases but proved to be of little help because of the difficulty in ascertaining the same superimposition of scans.

Results

The ventricular system became smaller in 38 out of the total of 67 patients (Table 1). Normalisation of the ventricular system was found in only 6 patients, in the majority of cases a more or less pronounced decrease in ventricular size was seen (Fig. 1). The decrease in the ventricular size invariably occurred within 2-3 weeks after shunting. In spite of considerable diminution of the ventricles, the original disfigurement of the ventricular system persisted (Fig. 2). A common feature after shunting, particularly in cases of very large hydrocephalus was the disproportionate atrial dilatation (Fig. 3). With restoration of cerebral mantle and reduction of ventricular size, the disproportion between the frontal horns and trigonum and occipital horns increased or became apparent.

Post-shunting subdural collections were discovered in 6 cases and a calcified subdural haematoma was found in 1 patient. The subdural collections were usually asymptomatic. In some cases they persisted for a long period (over one year) and their disappearance was usually connected with renewed slight enlargement of the lateral ventricles.

Cause of ventricul enlargement	ar	Ventricul	ar size after.	shunting
		Decreased	l Unchanged	Increased
Tumor	18	13	5	-
Perinatal damage (ex vacuo)	13	7	4	2 ^a
Myelomeningocele	10	6	2	2 ^a
Unknown	24	12	9	2 ^a
Trauma	2	-	2	

Table 1. Serial CT for hydrocephalus

a Shunt malfunction.

This should not be confused with post-shunting increase in ventricular size reflecting shunt insufficiency or malfunction, which is easily re-cognisable in serial scans (Fig. 4).

Subarachnoid cysts coexisting with ventricular enlargement may increase in size following successful shunting. In one case shunt revision resulted in a slit ventricle which was detected 3 weeks after the operation and was accompanied by a compensatory increase in the subarachnoid cyst (Fig. 5a, b).

Periventricular lucency was most distinctly seen in 2 cases of posttraumatic ventricular enlargement (Fig. 6a, b). In both the cases pressure recordings showed high pressure waves up to 30-40 mm Hg superimposed on a normal pressure curve with mean ICP of 10-15 mm Hg. Both patients were shunted but in both of them the ventricles did not diminish within 3 months and 3 weeks after shunting respectively (Fig. 7a, b).

Discussion and Conclusions

In a considerable percentage of patients with infantile hydrocephalus of diverse etiology, the shunting procedure which was successful by clinical criteria did not lead to normalisation but only to some decrease in ventricular size. This was seen in all types of hydrocephalus. In some cases, the phenomenon of disproportionate atrio-occipital dilatation (7) was very distinct. This may be explained by the fact that the cerebral gray nuclei become stretched and displaced by dilated ventricle but are not damaged whereas the periventricular white matter, as shown experimentally (13, 15), undergoes axon and myelin loss with final gliosis and atrophy.

Periventricular lucency has been usually interpreted as oedema caused either by transependymal absorbtion or frank disruption of the ependyma and loss of CSF into the white matter. Furthermore a pressure gradient across the ventricular wall has been postulated to explain this phenomenon.

In our material this periventricular oedema could be convincingly demonstrated only in cases of post-traumatic hydrocephalus with originally small compressed ventricles. In spite of the fact that these patients fulfilled the criteria for shunting (progression of ventricular enlargement, pressure waves, failure to improve clinically), shunting did not reverse ventricular dilatation. It seems therefore that the zone of oedema reflects rather the well-known pathological picture of disruption and swelling of axons, empty myelin sheaths with myelin debris (6) with final gliosis and atrophy and the low attenuation values found in the periventricular white matter may correspond to the areas of tissue damage more than to extracellular fluid accumulation. In our experience, shunting had little or no effect upon the reversal of this process.

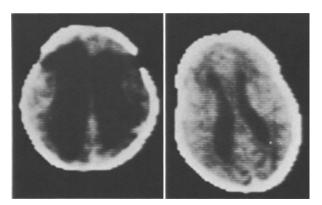
Summary

Findings of CT follow-up in 67 children with hydrocephalus of diverse etiology are presented. Reduction of ventricular size was observed in over 50% of cases but normalisation in less than 10%. The problem of periventricular lucency accompanying enlarged ventricles is discussed in 2 cases of posttraumatic hydrocephalus in children.

References

- 1. BRASSOW, F., BAUMANN, K.: Volume of brain ventricles in man determined by computer tomography. Neuroradiology 16, 187-189 (1978)
- EPSTEIN, F., RUBIN, R.C., HOCHWALD, G.M.: Restoration of cortical mantle in severe hydrocephalus: a new laboratory model. Dev. Med. Child Neurol. Suppl. <u>32</u>, 49-53 (1974)
- 3. GRUMME, Th., SUWITO, S.: Diagnosis of hydrocephalus by computerised tomography. Adv. Neurosurg. 4, 176-182 (1977)
- 4. HACKER, H., ARTMANN, H.: The calculation of CSF spaces in CT. Neuroradiology 16, 190-192 (1978)
- 5. KINGSLEY, D., KENDALL, B.E.: The value of computer tomography in the evaluation of the enlarged head. Neuroradiology <u>15</u>, 59-71 (1978)
- MILHORAT, Th., CLARK, R.G., HAMMOCK, M.K. et al.: Structural, ultrastructural and permeability changes in the ependyma and surrounding brain favoring equilibrium in progressive hydrocephalus. Arch. Neurol. 22, 397-407 (1970)
- 7. NAIDICH, T.P., EPSTEIN, F., LIN, J.P., KRICHEFF, I.I., HOCHWALD, G.M.: Evaluation of pediatric hydrocephalus by computer tomography. Radiology <u>119</u>, 337-345 (1976)
- OSTERTAG, C., HEMMER, R., MUNDINGER, F.: Observations on the differentiation of hydrocephalus occlusus in early childhood using computerised axial tomography. Neuropädiatrie <u>7</u>, 322-326 (1976)
- PALMIERI, A., AMBROSIO, A.: Computerised axial tomography in the diagnosis and control of infantile hydrocephalus. Minerva Pediatr. 30, 155-159 (1978)
- 10. PALMIERI, A., MENICHELLI, F., PASQUINI, V., SALVOLINI, U.: Role of computer tomography in the postoperative evaluation of infantile hydrocephalus. Neuroradiology <u>14</u>, 257-262 (1978)
- 11. RADUE, E.W., KENDALL, B.E.: Computer-Tomographie und Cisternographien beim normal pressure hydrocephalus. Act. Neurol. <u>4</u>, 217-227 (1977)
- 12. ROTTENBERG, P.A., PENTLOW, K.S., DECK, M.D.F., ALLEN, J.C.: Determination of ventricular volume following metrizamide CT ventriculography. Neuroradiology <u>16</u>, 136-139 (1978)

- 13. RUBIN, R.C., HOCHWALD, G.M., TIEL, M., et al.: Hydrocephalus III. Reconstruction of cerebral mantle following ventricular shunting. Surg. Neurol. <u>5</u>, 179-183 (1976)
- 14. WALSER, R.L., ACKERMAN, L.V.: Determination of volume from computerised tomograms. J. Computer Ass. Tomogr. <u>1</u>, 117-130 (1977)
- 15. WELLER, R.O., WISNIEWSKI, H., ISHII, N., et al.: Brain tissue damage in hydrocephalus. Dev. Med. Child Neurol. Suppl. <u>20</u>, 1-7 (1969)



 $\underline{Fig.~1}.$ Hydrocephalus after perinatal bleeding. Reduction in ventricular size after shunting

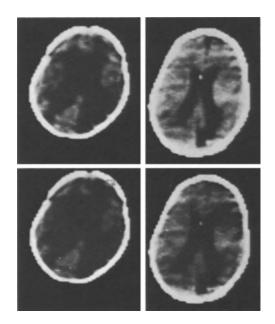


Fig. 2. Hydrocephalus with ventricular deformation. Reduction in ventricular size after shunting. Persisting deformation

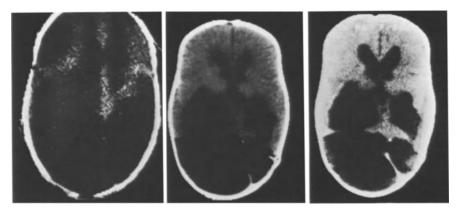


Fig. 3. Congenital hydrocephalus. Disporportionate atrio-occipital dilatation after shunt

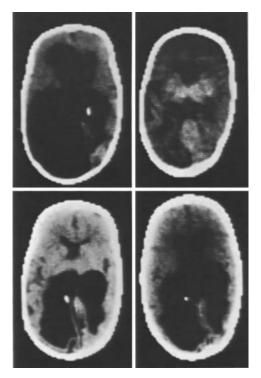


Fig. 4. Myelomeningocele with hydrocephalus. Multiple revisions. Upper row: decrease of ventricular size after revision. Lower row: increase of ventricular size because of malfunction

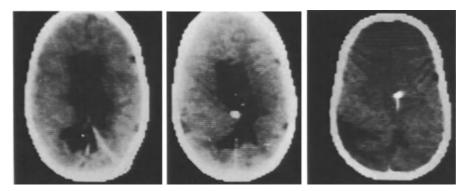


Fig. 5. <u>a</u> Progressive diminution of ventricular size till slit ventricle appears (right picture)

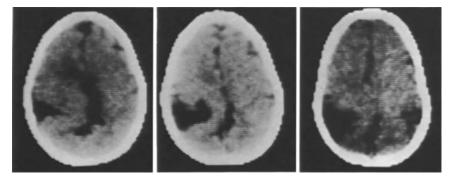


Fig. 5. <u>b</u> Same case. Progressive increase of subarachnoid cyst during the same time (3 months)

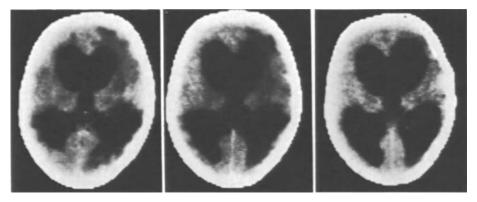


Fig. 6. a Posttraumatic hydrocephalus before shunting

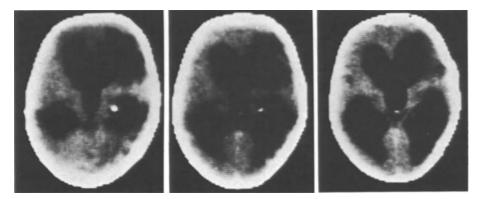


Fig. 6. b Same case. Three months after shunting

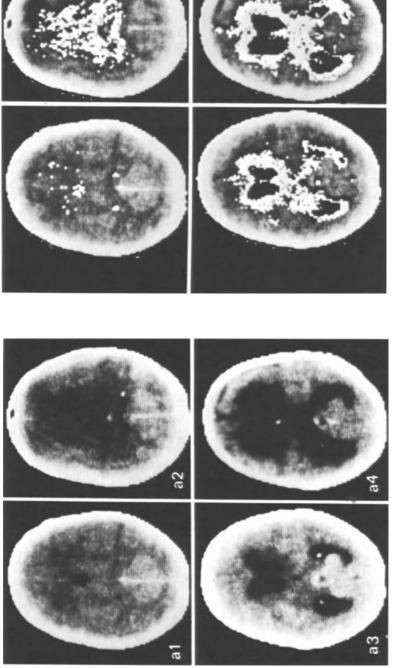


Fig. 7. a Posttraumatic hydrocephalus. a1 hours after trauma; a2 14 days after trauma; <u>a3</u> 4 weeks after trauma; <u>a4</u> 3 weeks after shunting, 2 months after trauma

Fig. 7. b Same case. Display of attenuation values 10-20 HU in the same scans as in Fig. 7a "periventricular oedema"

CT Images of Periventricular Lucency (PVL) in Various Forms of Hydrocephalus

C. SPRUNG and TH. GRUMME

Introduction

The phenomenon of periventricular lucency (PVL) was first described by GREITZ and HINDMARSH 1974 (7) in a patient with normal pressure hydrocephalus and not, as one might have expected, in a case of hypertensive hydrocephalus. For some time PVL was thought to be an artefact (6, 14), but the reports by NAIDICH (13) and GRANHOLM (5) 1976 proved the existence of PVL in different types of hydrocephalus. The pathomechanism, the incidence and the significance of PVL for the outcome of shunt-operations are still controversial in spite of an increasing number of reports from several groups of workers (1, 3, 4, 6, 8-12, 14-16).

Patient Material and Methods

CT-scans of 479 patients with 6 different types of hydrocephalus were reviewed in order to evaluate predisposing factors for the development of PVL. Various factors (incidence of occurrence, site, severity and configuration) were compared, and the significance of PVL for the outcome of shunt operations studied. Table 1 provides a list of the different types of hydrocephalus selected for evaluation and the incidence of the periventricular hypodense zone in each type.

Table 1. Patient material and incidence of periventricular lucency (PVL)

Diagnosis	No. of cases	PVL	8
Infantile hydrocephalus (both aqueduct stenosis and communicating hydroc.)	305	23	7.5
Hydrocephalic children with space- occupying lesions	33	13	39
Idiopathic normal pressure hydro- cephalus	16	4	25
Communicating hydroc. following SAH	26	11	42
Hydrocephalic adults with space- occupying lesions	95	52	55
Neonatal intracranial/intraventricular hermorrhage	4	3	

Results

A periventricular zone of low attenuation was found in only 23 of 305 (7.5%) children with infantile hydrocephalus. Compared with findings associated with space-occupying lesions, the low density zone close to the ventricles in infantile hydrocephalus is found less frequently and is usually less severe. We did not find significant differences between communicating hydrocephalus and aqueduct stenosis - an experience shared by other authors (10, 16). We never observed PVL in the absence of ventricular dilatation even when intraventricular pressure was clearly elevated as in 5 cases of slit-ventricle syndrome. We almost never found PVL in chronic infantile hydrocephalus with extremely distended ventricles. Figure 1 presents a case of communicating hydrocephalus showing the typical site and distribution but unusual severity of PVL for this type of hydrocephalus.

The case of a premature infant with hydrocephalus following neonatal intraventricular hemorrhage is illustrated in Fig. 2. A subependymal hypodense zone appeared in 3 of 4 cases with neonatal intraventricular hemorrhage but was found in only 14 of 58 adults with massive hemorrhage into the ventricles, usually as a sequel of intracerebral bleeding.

Figures 3a and b deal with the third type of hydrocephalus: we observed PVL in 13 of 33 (39%) hydrocephalic children with tumors of the posterior fossa or midline. The fact that PVL was observed in only 1 of 7 cases with blockage at the foramen Monroi despite significant hydrocephalus, and that PVL is much more common in conditions with rapidly progressing stenosis of the fourth ventricle, suggests that incidence may be related to the location of the block.

We found clear evidence of PVL highlighted by metrizamide-reflux in 4 of 16 cases of idiopathic normal-pressure hydrocephalus. Contrary to YAMADA (16), we could not find a pathognomonic site and configuration of PVL in this type of hydrocephalus (Fig. 4). As in all types of hydrocephalus mentioned here, there was a high degree of correlation between decrease of PVL in idiopathic NPH, reduction in size of the ventricles and clinical improvement. In contrast to YAMADA's observations (16), we found PVL in 11 of 26 cases of hydrocephalus following subarachnoid hemorrhage, including 3 cases with normal intracranial pressure.

52 of 95 (55%) hydrocephalic adults with space-occupying lesions also demonstrated PVL. Like WILSON and MOSELEY (15), we found the highest incidence of PVL (65%) in 31 cases of metastasis to the posterior fossa. Slow-growing tumors such as meningeoma and acoustic neurinoma were associated with this phenomenon in only 50% of cases (Fig. 5a, b).

There is a high risk of misinterpretation of various brain lesions associated with hypodense zones adjacent to the ventricles (Fig. 6). In particular, infections of the CSF and brain parenchyma with ependymal inflammation can imitate or intensify PVL. Fig. 6 illustrates various cerebral disorders. The configuration and localisation of the hypodense areas is quite different from PVL for a-d, but the lucent zone in the frontal region of four cases of brain atrophy was very similar to PVL. However, as Fig. 6e shows, the hypodensity located in the region of the cella media does not border directly on the ventricular system but is located in the semioval centers.

Discussion

Our series provides clear evidence that periventricular lucency may occur in all types of hydrocephalus and at any age. Localisation with predominance around the frontal horns - and configuration of PVL are generally independent of the particular type of hydrocephalus, a finding contrary to YAMADAs reports (16). Incidence and severity of PVL vary remarkably with the type of hydrocephalus. In our series the incidence of PVL in infantile hydrocephalus (7.5%) is even lower than the rate reported by KINGSLEY et al. (10). Contrary to the findings of ASADA (1) and KINGSLEY (10), but in agreement with MORI ($\underline{11}$), we never found PVL without at least moderate dilatation of the ventricles. Our experience in 5 cases of slit-ventricle-syndrome without PVL and clear evidence of PVL in cases of NPH prove that intraventricular pressure alone cannot be the decisive factor, as ASADA (1) claimed.

We agree with others (1, 10-13) that maximal ventricular dilatation is very rarely associated with PVL, especially in non-tumorous cases.

One case of infantile communicating hydrocephalus showed clear evidence of PVL 3 weeks post partum, but the parents refused permission for a shunt operation. The low attenuation zone gradually disappeared as ventricular size increased, reaching a maximum at 4 months. As several authors have reported (10, 16), PVL is less common in infantile hydrocephalus than in cases of tumor and adult hydrocephalus, probably because the vault is capable of expansion, thus maintaining intraventricular pressure at moderately increased levels. PVL is also rare in patients with maximally dilated ventricles, presumably because the chronic compensated nature of the hydrocephalus results in only slightly increased pressures. Our findings concur with the hypothesis of DRAYER (4) and others (10, 12, 13) that "it is the acuteness of the hydrocephalus rather than the overall dilatation of the ventricular system that predisposes to PVL".

Although acute hydrocephalus may develop very rapidly after the precipitating event, PVL is usually not demonstrable until at least 12 hours after an acute event such as intraventricular bleeding or blockage of the fourth ventricle by a hemorrhage. This seems to be the main reason for the apparent discrepancy between occurrence of PVL in infants with intraventricular neonatal hemorrhage and that in hydrocephalic adults with massive intraventricular bleeding, usually as a sequel of intraventricular hemorrhage. In most of the latter cases CT was performed immediately after the acute event, and the patients died before developing PVL.

A decrease in PVL was not related to the type of hydrocephalus but correlated well with clinical improvement and was almost always accompanied by a decrease in ventricular size, which is in agreement with all authors using our methods, but conflicts with comparable evaluations reported by BRIGGS (2).

Concerning the pathological mechanism, some authors favor the hypothesis of passive diffusion of CSF into the white matter (1, 3, 11, 14), others favor the idea of active absorption (7, 8, 9) or edema due to hypoxic changes in the periventricular white matter (5, 6). Our observations provide conflicting results. The hypothesis of pressure-dependent passive diffusion through the ruptured ependyma is unlikely for the following reasons:

- 1. PVL is not only seen in cases with increased intraventricular pressure, but also in NPH and even in some cases of atrophy.
- 2. PVL is very rarely seen in cases of infantile hydrocephalus with maximally dilated ventricles and a high probability of rupture of the ependyma.
- 3. PVL is most often seen associated with mild or moderate dilatation of the ventricles and is not only found in the region of the superlateral angle of the frontal horns.

Other facts seem to contradict the theory of an active absorption mechanism:

- 1. Predominance of the frontal region.
- 2. PVL is very rarely seen in chronic conditions, so-called "arrested" hydrocephalus, where CSF is thought to be absorbed through the ventricular wall.

Some factors seem to influence the probability of occurence of PVL:

- a) intraventricular pressure
- b) speed of development of hydrocephalus
- c) overall size of the ventricular system
- d) compliance of the ventricular walls
- e) content of the ventriclesf) localisation of the blockage

In accordance with other reports our findings prove that PVL is not an artefact, but due to a loss of lipids and protein and that it does not represent edema of the ependymal layer following pressure on the ventricular wall. The appearance of PVL in CT-scans is due to the transependymal transit of cerebrospinal fluid into the white matter. The controversy over the pathomechanism can only be settled by neuropathological studies.

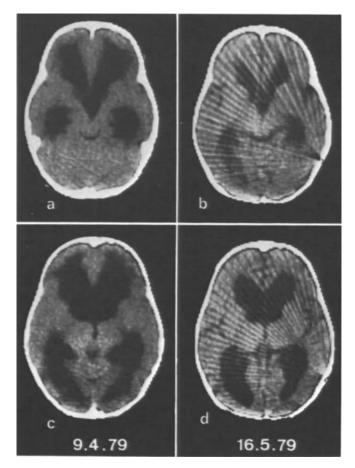
Summary

CT-scans of 479 patients with 6 different types of hydrocephalus were reviewed in order to evaluate predisposing factors, incidence and localisation of periventricular lucency (PVL). There were no significant differences in distribution and configuration, but we found a higher incidence and greater severity of PVL in cases of tumor. The probability of finding PVL increases with increasing intraventricular pressure and depends especially on the speed of development of ventricular dilatation. The danger of misinterpretation in other brain lesions is pointed out. The resolution of PVL was always associated with clinical improvement, regardless of the type of hydrocephalus. The pathological mechanism is discussed.

References

- 1. ASADA, M., TAMAKI, N., KANAZAWA, Y., MATSUMOTO, S., MATSUO, M., KIMURA, S., FUJII, S., KANEDA, Y.: Computer analysis of periventricular lucency on the CT scan. Neuroradiology 16, 207-211 (1978)
- 2. BRIGGS, R.C., ALTEMUS, L.R., LESCHEY, W.H.: Synthesis of criteria for operability in normal-pressure hydrocephalus due to bilateral convexity block. J. Nucl. Med. 19, 1268-1269 (1978)

- DRAYER, B.P., ROSENBAUM, A.E., HIGMAN, H.B.: Cerebrospinal fluid imaging using serial metrizamide CT cisternography. Neuroradiology <u>13</u>, 7-17 (1977)
- 4. DRAYER, B.P., ROSENBAUM, A.E.: Brain edema defined by cranial computed tomography. J. Comp. Ass. Tomography <u>3</u> (3), 317-323 (1979)
- GRANHOLM, L.: An explanation of the reversible memory defect in hydrocephalus. In: Intracranial pressure, Vol. III. BEKS, J.W.F., BOSCH, D.A., BROCK, M. (eds.), p. 173. Berlin, Heidelberg, New York: Springer 1976
- GRANHOLM, L., HINDMARSH, T., LEVANDER, B.: Studies on periventricular hypodensities in hydrocephalus. J. Neurosurg. Sci. <u>22</u> (1978)
- GREITZ, T., HINDMARSH, T.: Computer assisted tomography of intracranial CSF circulation using a water-soluble contrast medium. Acta Radiol. Diagnosis <u>15</u>, 497 (1974)
- HIRATSUKA, H., FUJIWARA, K., OKADA, K., TAKASATO, Y., TSUYUMU, M., INABA, Y.: Modification of periventricular hypodensity in hydrocephalus with ventricular reflux in metrizamide CT cisternography. J. Comp. Ass. Tomography <u>3</u> (2), 204-208 (1979)
- 9. HOPKINS, L.N., BAKAY, L., KINKEL, W.R., GRAND, W.: Demonstration of transventricular CSF absorption by computerized tomography. Acta Neurochir. 39, 151-157 (1977)
- 10. KINGSLEY, D.P.E., KENDALL, B.E.: The value of computed tomography in the evaluation of the enlarged head. Neuroradiology <u>15</u>, 59-71 (1978)
- 11. MORI, K., MURATA, T., NAKANO, Y., HANDA, H.: Periventricular lucency in hydrocephalus on computerized tomography. Surg. Neurol. 8, 337-340 (1977)
- 12. MOSELEY, I.F., RADÜ, E.W.: Factors influencing the development of periventricular lucencies in patients with raised intracranial pressure. Neuroradiology <u>17</u>, 65-69 (1979)
- 13. NAIDICH, I.P., EPSTEIN, F., LIN, J.P., KRICHEFT, I.I., HOCHWALD, G.M.: Evaluation of pediatric hydrocephalus by computed tomography. Radiology <u>119</u>, 337-345 (1976)
- 14. PASQUINI, U., BRONZINI, M., GOZZOLI, E., MANCINI, P., MENICHELLI, F., SALVOLINI, U.: Periventricular hypodensity in hydrocephalus: a clinico-radiological and mathematical analysis using computed tomography. J. Comput. Ass. Tomography <u>1</u>, 443-448 (1977)
- 15. WILSON, J.L., MOSELEY, I.F.: A diagnostic approach to cerebellar lesions. In: The first European seminar on computerised axial tomography in clinical practice. Du BOULAY, G.H., MOSELEY, I.F. (eds.), pp. 123-133. Berlin, Heidelberg, New York: Springer 1977
- 16. YAMADA, F., FUKUDA, S., SAMEJIMA, H., YOSHÜ, N., KUDO, T.: Significance of pathognomic features of normal-pressure hydrocephalus on computerized tomography. Neuroradiology <u>16</u>, 212-213 (1978)



<u>Fig. 1 a-d</u>. N.K., 2 years. Infantile communicating hydrocephalus. <u>a, b</u> preoperative state with evidence of PVL; <u>c, d</u> 5 weeks after Holter drainage operation

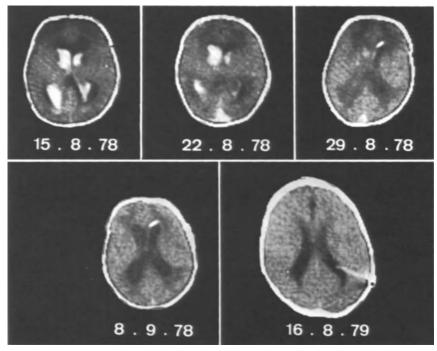


Fig. 2. N.M., 13 days. Neonatal intraventricular hemorrhage. 15.8.78: 13 days post partum. 22.8.78: Resolution of the hemorrhage, increase in PVL. 29.8.78: Continued resolution of hemorrhage. PVL at a maximum in spite of external drainage. 8.9.79: Increase in hydrocephalus despite external drainage. Decrease in PVL. 16.8.79: One year after initial CT studies; Holter drainage; only slight ventricular dilatation; no evidence of PVL

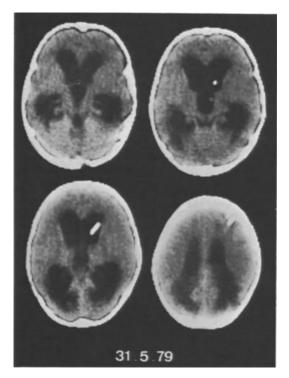


Fig. 3. a S.W., 8 years. Medulloblastoma with occlusive hydrocephalus. Condition with defective external drainage prior to tumor resection

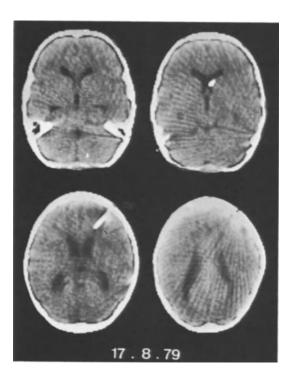


Fig. 3. <u>b</u> 2 1/2 months later following tumor resection. Conversion of external drainage to a Holter shunt

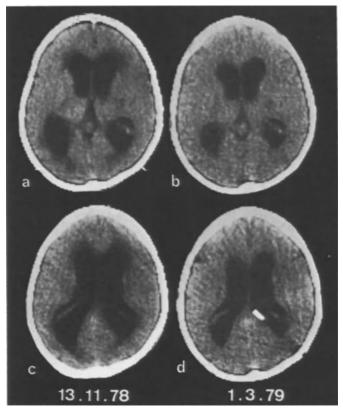
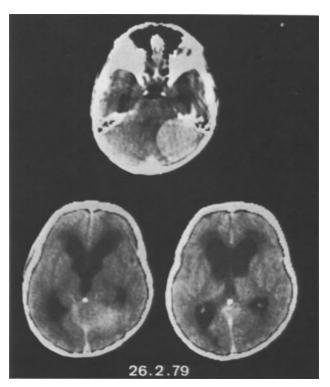
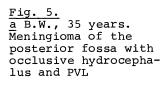


Fig. 4a-d. M.H., 61 years. Idiopathic normal pressure hydrocephalus. a, b Evidence of PVL preoperatively; c, d 3 1/2 months after implantation of a Holter drainage. Note the complete disappearence of PVL, but only slight reduction in ventricle size. Clinically the patient recovered completely





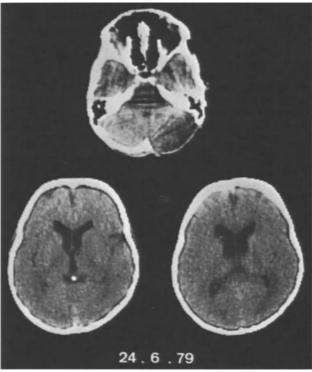
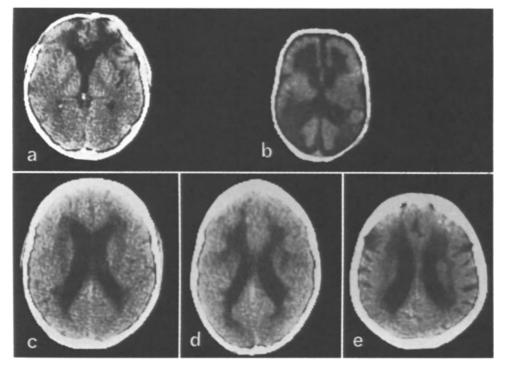


Fig. 5. b 5 months postoperatively after complete excision of the meningioma



<u>Fig. 6 a-e</u>. Cerebral lesions simulating PVL. <u>a</u> F.J., 42 years: Posttraumatic state; <u>b</u> A.H., 3 years: Encephalitis; <u>c</u> K.S., 51 years: Multiple sclerosis; <u>d</u> J.A., 11 years: Leukodystrophy; <u>e</u> H.G., 80 years: Cerebral atrophy. Note the differences in site and configuration as compared to findings in typical PVL

Clinical Observations and CSF Absorption Studies in the Slit Ventricle Syndrome

H. COLLMANN, W. MAUERSBERGER, and G. MOHR

Introduction

The slit-ventricle syndrome (SVS) means a state of intermittent signs of shunt malfunction without substantial ventricular enlargement. It is sometimes observed in shunt-dependent patients with small, slitlike ventricles. It has been attributed to recurrent obstruction of the ventricular catheter, which then causes an increase of intracranial pressure (ICP), before there is time for ventricular re-enlargement (2, 4, 5, 6). However, this interpretation leaves some doubt in view of the great majority of shunted patients with small ventricles, who never present any signs suggestive of SVS. Reviewing our own material we have therefore looked for patients, who had definite signs of shunt malfunction, but did not present gross ventricular dilatation.

Material and Results

During the last 4 years we have observed 5 such patients. The clinical data are summarized in Table 1, and the main points are that all patients were adolescents and had been shunted during infancy because of extensive hydrocephalus, mainly of the communicating type. In view of the above-mentioned hypothesis it is striking that only two patients had previous shunt revisions at all, and in both cases not only the ventricular catheter was found to be blocked. All patients had an at least two-weeks history of intermittent headaches. The increase of intracranial pressure (ICP) was finally proved by the occurrence of papilloedema and depression of the conscious level. At this stage CT controls were performed.

The main findings of CT scanning are illustrated by the follow-up studies of a 9-year-old boy, who had been shunted at 19 months of age for non-absorptive hydrocephalus. He did not require shunt revision over the ensuing 7 years. The first scan (Fig. 1, top), which was taken during this asymptomatic interval, showed nearly collapsed ventricles. The second scan (Fig. 1, bottom) was performed after a two-weeks history of headaches and vomiting, when papilloedema was apparent: In contrast to the duration of symptoms and severity of clinical signs there is only a slight expansion of the ventricles up to a normal size. Yet shunt revision revealed a blocked ventricular catheter. After a short period of clinical improvement the boy deteriorated again, presenting the same periodic symptoms as before, which lasted about 20 weeks until the next scan was performed. Compared with the former one this scan (Fig. 2, above) showed an even less marked response of the ventricles. Shunt failure was again proved, but this time the cardiac catheter was involved. Since then the child has remained asymptomatic, and in a recent scan a slight shrinking of the ventricles is established (Fig. 2, bottom).

Table 1. Slit-ventricle syndrome clinical data of 5 cases

Patients' age	8-13 years
Type of hydrocephalus	Communicating = 4 patients Myelocele assoc. = 1 patient
Age at first shunt	5-22 months
Preceding Shunt revisions	O = 3 patients 4 = 1 patient 6 = 1 patient
Signs of raised ICP	Intermittent headaches = 5 patients Impaired consciousness = 3 patients Papilloedema = 5 patients
Time since onset of headaches	2-52 weeks

Short-term recordings of the CSF pressure, which was performed in all patients, revealed an increased pressure level and/or pressure waves in 4 cases. The remaining patient with a normal pressure level showed, however, a highly abnormal pressure response during the constant flow infusion test (1). A rapid increase of pressure during continuous intraventricular infusion of 0.75 ml/min saline without attainment of a steady state equilibrium proved a severely reduced CSF absorption capacity. In another patient with a moderately elevated baseline pressure, the infusion produced an even steeper rise in ICP indicating a reduced compliance of the brain as well.

Definite and persisting shunt obstruction was found in all of our cases, either at the upper end or at the lower end of the system, and shunt revision was successful in all patients.

Conclusions

Our findings indicate that - at least in the cases presented here intermittent obstruction of the ventricular catheter has little to do with the occurrence of SVS. Both have to be regarded as different problems. According to our findings the essential pathogenetic factor in the SVS is a specific change within the parenchyma resulting in a rigidity of the ventricular walls. This concept finds support in a recent paper by EPSTEIN (3) dealing with the differential diagnosis of the problems associated with chronically shunted slit-like ventricles. Based on the clinical data and the pressure recordings we conclude that in our cases the CSF-brain system responds preferentially with intermittent rises of ICP, when shunt malfunction occurs. Since the clinical picture may be the same as in cases of recurrent shunt obstruction, we suppose that at least some of the previously reported cases of SVS have been misinterpreted.

References

- COLLMANN, H., SPRUNG, Ch.: Patterns of pressure response to intraventricular infusion in shunt-treated hydrocephalic children. J. Neurosurg. Sci. <u>22</u>, 221-226 (1978)
- EPSTEIN, F.J., FLEISCHER, A.S., HOCHWALD, G.M., RANSOHOFF, J.: Subtemporal craniectomy for recurrent shunt obstruction secondary to small ventricles. J. Neurosurg. 41, 29-31 (1974)

- 3. EPSTEIN, F.J., MARLIN, A.E., WALD, A.: Chronic headache in the shunt-dependent adolescent with nearly normal ventricular volume: Diagnosis and treatment. Neurosurgery <u>3</u>, 351-3-5 (1978)
- FAULHAUER, K., SCHMITZ, P.: Overdrainage phenomena in shunt-treated hydrocephalus. Acta Neurochir. <u>45</u>, 89-101 (1978)
- 5. HOLNESS, R.O., HOFFMAN, H.J., HENDRICK, E.B.: Subtemporal decompression for the slit-ventricle syndrome after shunting in hydrocephalic children. Child's Brain 5, 137-144 (1979)
- 6. SALMON, J.H.: The collapsed ventricle: Management and prevention. Surg. Neurol. <u>9</u>, 349-352 (1978)

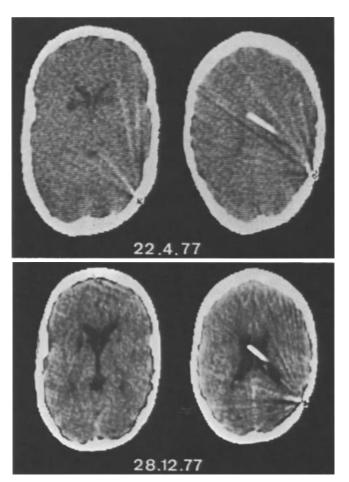


Fig. 1. H.F., 9 years. Slit-like ventricles during the asymptomatic interval (*above*); only slight expansion despite severe signs of raised ICP (*below*)

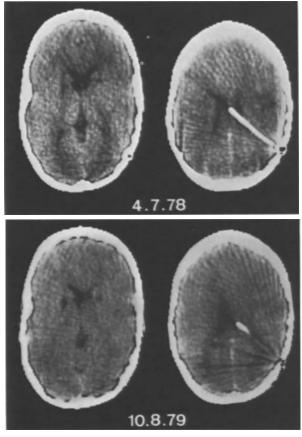


Fig. 2. H.F., 9 years. Same patient. Minimal ventricular dilatation during a second period of shunt malfunction (*above*); CT control following successful shunt revision

Cooperative Study

Long-Term Results of the Operative Treatment of Hydrocephalus in Children

A. AMBROSIO, L. BENVENUTI, E. BIANCHI, S. BRIANI, G. CAGNONI, A. CARTERI, M. COLANGELO, M. FONTANA, S. M. GAINI, M. GEROSA, R. GIUFFRE, G. LIGUORI, I. L. LONGATTI, G. LUCCARELLI, C. MAZZA, F. MIGLIAVACCA, A. MOISE, E. OCCHIPINTI, L. PALMA, A. PASQUALIN, G. PEZZOTTA, G. TOMEI, and R. VILLANI

1. Introduction

The general data concerning the experience of nine Neurosurgical Centers with ventriculo-venous (V-V) and ventriculo-peritoneal (V-P) shunts have been reviewed.

The shunting procedures were performed between January 1960 and December 1978. This study includes 2392 cases; 1820 of these have a follow-up of at least five years. According to the neuroradiological anatomy we classified all these cases into communicating and blocked hydrocephalus. The etiology of the hydrocephalic process was attributed to congenital diseases in about 50%, to infections in 20% and to hemorrhages in 10%. 1247 children underwent V-V and 543 V-P shunting operations.

As a rule the application of systems provided by medium pressure valves were used; high pressure valves were employed in 3%, low pressure in 1.6%.

1820 patients underwent 3232 operations including 1412 revisions; i.e. an average of 1.7 operations per patient.

391 children needed one revision, 179 needed two, 116 patients needed three revisions and 27 needed four. As shown in the Table 1, 291 re-

Time	Revi	sions					Total
<u> </u>	1	2	3	4	5	>	
< 1 month	136	84	26	26	5	4	291
1 - 3 months	111	74	45	6	8	2	256
3 - 6 months	94	49	24	5	6	6	184
6 -12 months	92	49	21	6	3	3	174
1 - 2 years	97	35	16	9	3	3	163
2 - 3 years	77	22	17	9	5	5	135
3 - 5 years	82	30	12	1	5	3	133
> 5 years	53	8	11	1	1	2	76
Total	742	351	172	63	36	48	1412

<u>Table 1.1</u>. Time interval between the first operation and the further revisions

visions were necessary within the first month; 256 patients needed surgical revisions within the second and third month.

62% of the children were submitted to revision within 1 year. These values do not change when each revision is considered separately. The surgical procedures performed at each revision are summarized in Table 1.2.

Operative Death. Among 3232 operations there were 145 deaths (Table 1.3).

The mortality rate of the initial operation and the subsequent revisions was 3.2%, 4.5%, 5.9%, and 10.4%. It is logical to believe that children with many revisions have a higher risk of death. Among the complications reported, which may be the cause of death, infections are particularly frequent. Other complications are related to the use of V-V shunts, to technical errors and sometimes to an incongruous drainage.

Late Mortality. The rate of the late mortality is 261 patients. In this case infections also seem to be the main cause, however, in more than 50% the cause of death remained unknown. The total number of deaths is then 406, the percentage being 22.3 (Table 1.4).

revision
each
at
procedures
Surgical
1.2.
Table

	Revisions	ions					Total
	-	2	m	4	S	^	
Conversion of V-venous into V-P shunt	191	59	22	12	4	m	291
Side conversion of V.V.	54	. 15	7	-	-	4	82
Revision of V.V.	216	113	45	12	ę	12	401
Conversion of V.P. into V.V.	37	26	19	٢	7	7	103
Side conversion of V.P.	35	18	17	6	۲	6	89
Revision of V.P.	155	91	48	16	15	7	332
Extra-intrathecal conversion	2	2	2		-	-	8
Intra-extrathecal conversion	22	Υ	m	7	-	2	33
Shunt pressure changed	21	6	m	-		с	38
Shunts removed	6	15	9	m	2		35
Total	742	351	172	63	36	48	1412

E⊣ 190	Table 1.3. Causes of post	postoperative death							
U I	Causes	First operation	ion	Revisions	ons				Total
1		1		1	2		3	4 and >	
н	Infections	15		6	1	0	10	ε	47
U	Cardiac failure	12		4		2	4		22
д	Pneumonia	m		ß		-		٢	10
д	Pulmonary oedema	9				-		-	7
н	Intracranial hypertension	٣		2		£	1	7	11
н	Intracranial hypotension	2		2		-	7	7	6
Н	Haematoma	٢		-					2
H	Haemorrhage	4		-		+		-	7
D	Unknown	14		10		3	1	2	30
	Total	60 (3.2%)		34 (4.	.58) 2	21 (5.9%)	18 (10.4%)	11	145 (4.5%)
ΕI	Table 1.4								
I		First operation	Revisions	ions			Total		
		Number	٢	2	с	4 or >			
N I	Surgical procedures	1820	742	351	172	147	3232		
S	Subsequent operations	60	33	23	18	11	145	(44.9% of 3	3232)
Ц	Late mortality						261		
1 [4	Total number of mortalities	es					406	(22.3% of 1	1820)
1									

190

2. Mechanical Complication of Shunt Operations

S. BRIANI, L. BENVENUTI, G. CAGNONI, G. LUCCARELLI, F. MIGLIAVACCA, and A. MOISE

In collaboration with nine other Neurosurgical Clinics, we have considered the mechanical shunt complications, which have more or less led to a surgical revision of the drainage system, by evaluating the results of 1820 non-tumoral hydrocephalus in children treated with extrathecal CSF drainage.

Out of 1438 total complications, 541 were mechanical (37.62%). This type of complication does not require the active participation of tissues with which the shunts come into contact and for this reason is called "mechanical". Cases of active participation will be discussed under the title of "biological complications".

It was not possible to find a standardised classification and for this reason we have compiled our own list (Table 2.1).

Table 2.1. Mechanical shunt complications

Disconnection Dislocation Complete migration of the system Shortening of the distal catheter Kinking Rupture Surgical displacement Immediate visceral perforation Damaged organs and tissues Involuntary closure of the system (limited to MPV)

<u>Disconnection</u>. Under such a heading we have included disconnected non-migrating segments of the drainage system. Detachment can occur at any point where there is a connection along the shunt and does not involve, if possible, a one-piece system.

<u>Dislocation</u>. This group includes those cases where a detachment of a segment of the drainage system is followed by a migration of the segment itself, for example when the ventriculo-cardiac and peritoneal catheters have slipped down into the respective cavities. Four cases of cardiac catheter which had migrated into the pulmonary artery are presented in this study.

<u>Complete Migration of the System.</u> Here, only the one-piece system is verified. The whole system migrates in both directions and represents the most frequent complication.

<u>Shortening of the Distal Catheter</u>. Under this heading we have included all those cases where, following the normal body growth of a child, the peritoneal and cardiac catheter leaves its original position. There is a possibility of a continuous function because in some cases the fibrous tube, formed around the catheter, maintains the continuity of the drainage system. These cases will be discussed under biological complications.

<u>Kinking</u>. Obviously, this particular section needs little explanation. We can however add that such an eventuality is noticeably reduced when spring catheter derivatives are used.

<u>Rupture</u>. This section, also self-explanatory, is worth mentioning as many of these ruptures occur at the reservoir and the causes are frequently accidental trauma and injection withdrawal.

<u>Surgical Displacement</u>. This includes all malfunctions caused by surgical techniques such as ventricular catheter which remains, if too short, or penetrates, if too long, into cerebral matter; CSF fistula caused by an over-enlarged drilled hole; distal catheter in a preperitoneal area or a venous catheter which follows an abnormal path.

<u>Immediate Visceral Perforation</u>. Is also part of the above heading and we have only mentioned it to underline the possibility of such a condition.

<u>Damaged Organs and Tissues</u>. This section almost exclusively involves superficial skin diseases which, even if evident along the path of the system, are usually limited to the reservoir area in our investigations with particular attention to the metal wall of the Holter valve and to the dome of the Ames valve, especially when inserted in small children.

Among the mechanical complications some cases of involuntary closure of the system after an error or unintentional mishandling of the Multi-Purpose Valve deserve mentioning. The above situation was verified in 3 cases (5%) out of 60.

Having schematically subdivided the group into ventriculo-atrial (V-A) and ventriculo-peritoneal (V-P) shunts, we calculated a numerical and percent incidence of each mechanical complication group in respect to the type of drainage (Table 2.2).

V-A shuntsV-P shuntsFirst operationa1247543Shunt conversion103291Total1350834

Table 2.2. Number of V-A and V-P shunts

a 30 cases received initially an intrathecal shunt.

The numerical and percentual incidence of a single mechanical complication, respectively referred to as the number of V-A and V-P shunts, is presented in Table 2.3.

Complication	V-A	shunt	V-P	shunt
Disconnection	57	(4.22%)	74	(8.87%)
Dislocation	16	(1.18%)	23	(2.75%)
Complete migration	-		13	(1.55%)
Shortening of the distal catheter	92	(6.81%)	31	(3.71%)
Kinking	17	(1.25%)	41	(4.91%)
Rupture	24	(1.77%)	16	(1.91%)
Surgical displacement	47	(3.48%)	29	(3.47%)
Immediate visceral perforation	-		2	(0.23%)
Damaged organs and tissues	23	(1.70%)	31	(3.71%)
Involuntary closure (MPV)	-		3	(0.35%)
Total	276	(20.44%)	263	(31.53%)

Table 2.3. Numerical and percentage incidence of mechanical shunt complications

The total values of percentage relative to mechanical complications of the V-A and V-P shunts are similar to those presented in the literature (1, 2, 3, 4, 5, 7). From these values the preference for V-A shunts instead of a V-P one is confirmed, at least for mechanical complications. However, we must point out that in our investigation we refer to patients treated more than 5 years ago, a time when V-P shunts were used much less (Table 2.2) and therefore it is possible that with further experience the number of mechanical complications in this type of shunt could be reduced, particularly in the one-piece system ($\underline{6}$). In fact, such a type of shunt is obviously immune to disconnection or dislocation and very rarely presents kinking of a spring catheter is used, even though there is a possibility of complete migration of the shunt into a peritoneal cavity or in the lateral ventricle.

However, there is the inconvenient problem of not being able to control this system as easily as when using the reservoir. Another problem is its rigidity, which can cause visceral perforation or damage tissues more so than any other system. The main indisputable advantage of such a one-piece system is its easy and quick application.

<u>Conclusions</u>. Even if numerical data confirm yet again the superiority of V-A drainage to a V-P one, we consider the latter to be a valid alternative because of the simpler surgical techniques required especially when very small babies have to undergo a drainage operation where it is not always easy to find a vein or perform a lengthy operation.

The final decision depends entirely on the capacity and experience of the surgeon who is given the responsibility of the final analysis and the choice of which drainage system is best suited for each individual.

References

 ASCHER, P.W., OBERBAUER, R.W.: Comparative clinical study of ventricular cardiac and ventricular peritoneal shunts. In: Modern problem in paediatrics, Vol. 18. VILLANI, R., et al. (eds.), pp. 196-197. Basel: Karger 1977

- GIUFFRE, R., PALMA, L., FONTANA, M.: Follow-up study of 360 children subjected to extracranial CSF shunt for hydrocephalus. In: Modern problem in paediatrics, Vol. 18. VILLANI, R., et al. (eds.), pp. 198-202. Basel: Karger 1977
- 3. GUIDETTI, B., OCCHIPINTI, E., RICCIO, A.: Ventriculo-atrial shunt in 200 cases of non-tumoral hydrocephalus in children: remarks on the diagnostic criteria, post-operative complications and longterm results. Acta Neurochirurgica 21, 295-308 (1969)
- IGNELZI, R.J., KIRSCH, W.M.: Follow-up analysis of ventriculoperitoneal and ventriculo-atrial shunt for hydrocephalus. J. Neurosurg. 42, 679-682 (1975)
- LEEM, W., MILTZ, H.: Complications following ventriculo-atrial shunts in hydrocephalus. In: Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 1-5. Berlin, Heidelberg, New York: Springer 1978
- RAIMONDI, A.J., ROBINSON, J.S., KUWARMURA, K.: Complications of ventriculo-peritoneal shunting and critical comparison of threepiece and one-piece system. Child's Brain 3, 321-342 (1977)
- STRAHL, E.W., LIESEGANG, J., ROOSEN, K.: Complications following ventriculo-peritoneal shunts. In: Advances in neurosurgery, Vol.
 WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 6-9. Berlin, Heidelberg, New York: Springer 1978

3. Biological Shunt Complications

E. OCCHIPINTI, M. FONTANA, R. GIUFFRE, and L. PALMA

There are two categories of biological complications:

<u>1. Septic:</u> Septicemia, ventriculitis, meningitis or meningoencephalitis, peritonitis, cutaneous infection of wounds, proximal or distal abscesses, empyema (Table 3.1).

Table 3.1. Biological shunt complications^a

V-A	shunts	V-P	shunts
28	(2.07%)	8	(0.96%)
32	(2.37%)	23	(2.75%)
34	(2.52%)	36	(4.32%)
-		15	(1.79%)
14	(1.04%)	18	(2.16%)
108	(8.00%)	100	(11.99%)
	28 32 34 - 14	V-A shunts 28 (2.07%) 32 (2.37%) 34 (2.52%) - 14 (1.04%) 108 (8.00%)	28 (2.07%) 8 32 (2.37%) 23 34 (2.52%) 36 - 15 14 (1.04%) 18

a V-A shunts: 1350 - V-P shunts: 834.

b Septicemia: 10 endocarditis; 6 shunt-nephritis; 1 purpura necrotica; 19 bacteriaemia.

<u>2. Aseptic:</u> Choroidal-ependymal reactions, phlebothrombosis, peritoneal malabsorption, peritoneal pseudocyst and tissue reactions around the catheter, including glial, cortical or subcortical along the path of the ventricular catheter (Table 3.2).

Table 3.2. Biological shunt complications^a

V-A shunts	V-P	shunts
169 (12.52%) 97	(11.63%)
152 (11.26%) –	
-	110	(13.19%)
-	9	(1.06%)
-	28	(3.36%)
321 (23.77%) 244	(29.26%)
	169 (12.52% 152 (11.26% - - -	169 (12.52%) 97 152 (11.26%) - - 110 - 9 - 28

a V-A shunts: 1350 - V-P shunts: 834.

b All intestinal perforations.

<u>Septicemia</u>: There are two types, acute and chronic, chronic is the most frequent, causing severe clinical and therapeutic problems and sometimes delaying diagnosis $(\underline{3}, \underline{5}, \underline{6}, \underline{7}, \underline{8}, \underline{11}, \underline{12})$. Chronic septicemia is characterized by the syndrome "fever, anemia, splenomegaly"

 $(\underline{8}, \underline{12})$ or subacute bacterial endocarditis or shunt nephritis $(\underline{4})$ and even purpura necrotica hemorrhagica $(\underline{13})$. The shunt infection may show up 4-5 years after the valve has been inserted, which makes one wonder whether the infectious process starts then, or arises after an intercurrent infectious disease due to transitory bacteremia, and bacterial pollution of the shunt apparatus. The first seems more likely, even if the second may not altogether be excluded. Septicemias are more frequent in venous shunts than in peritoneal (28 septicemias in 1,350 V-A shunts i.e. 2.07% against 8 in 834 V-P shunts i.e. 0.96%). Endocarditis is the most troublesome clinical form because of possible cardiovascular complications, dissemination of septic thrombi, and shunt nephritis which may set in, with autoimmune pathological mechanisms, and purpura necrotica hemorrhagica.

<u>Ventriculitis</u>. This is one of the most important biological complications, particularly from a clinical point of view because of the poor prognosis. When considering shunt infections, we should therefore divide them into two categories: ventriculitis and the other infections (<u>10</u>). In this series the rate of ventriculitis is 2.52%, and that of meningitis or meningoencephalitis 3.20%.

Although the pathogenesis of ventriculitis is usually infective, it may also be the result of an aseptic process, with choroidal-ependymal reactions, so intense as to provoke an obliterating productive ependymitis. Cases of ependymitis have been described with multiple ventricular septum formation and even obliteration of both the ventricular cavities and the acqueduct (5, 7, 8). The newly formed tissue was histologically composed of gliomesenchymal granulations with diffuse lymphomonocytic infiltrations, also proliferation in the subependymal glia.

Even if the patient survives the infection, ventriculitis still constitutes a grave threat. Double shunts or repeated revisions may be necessary due to the occlusion of the ventricular cavities, or an evolving choroidal-ependymal process. New infections can easily set in, the original one may return or the process of aseptic ventriculitis may be accelerated.

Actually, treatment is often unsuccessful in these cases, due not so much to the lack of sterilization of the CSF, as to the repeated revisions. Ventriculitis is notably high in hydrocephalus where an iodine compound was used in diagnosis. Both this and the gaseous type help to set off plexo-ependymal reactions.

Other Septic Complications. Infections arising from wounds, proximal or distal abscesses and peritonitis are not so serious. The rate is: abscesses 1.46% and peritonitis 1.79%. Peritoneal infections with possible purulent abdominal collections, are often seen without signs of meningitis.

The CSF from the ventricle may be sterile, and the infection limited to the abdomen, hence the importance of a unidirectional flow of CSF.

<u>Choroidal-Ependymal Reactions</u>. These are by far the most frequent biological complications, and the main cause of obstruction of the ventricular catheter, whichever type is used. Since inert catheters are not yet available, the choice of ventricular catheter does not appear to be very important as far as a possible choroidal-ependymal reaction is concerned. Besides this reaction is common to a-l shunts, V-A, V-P or intrathecal, where a tube is used. In 2184 shunts we found 266 cases, i.e. a rate of 12.18%. This is an active process of biological reactions to the ventricular catheter. It anchors itself to the ventricular wall with neomembranes, or by penetration and vegetation of fragments of tissue in the lumen of the catheter (1, 9).

This is different from the passive process of penetration of material such as fragments of parenchyma, blood clots, fibrin or iodine residue.

<u>Phlebothrombosis</u>. This type of biological complication is also notable, occurring in 152 of 1,350 V-A shunts, a rate of 11,26% of the cases studied. Possible cases of cor pulmonale from repeated pulmonary microembolisms are a severe cardiovascular complication in V-A shunts (14, 15). There is one case of cor pulmonale in this series, a 10-year-old boy, operated upon for post-meningitic hydrocephalus at the age of one, who showed signs of cardio-circulatory failure, following repeated pulmonary embolisms, at school age. The V-A shunts was removed, but later because the boy was shunt-dependent, a V-P shunt had to be performed. At the age of 10, the boy is neuropsychologically normal, but is a severe cardiopathic case, and even with digitalis treatment has only limited autonomy.

<u>Peritoneal Malabsorption</u>. This was found in 110 cases out of 834 V-P shunts, i.e. 13.19%. Although not an apparently serious complication, it obviates the revision of the shunt. Circumscribed peritoneal fluid accumulation (pseudocysts) were found only 4 times. Reactions of tissues, in a sleeve-like form around the peritoneal catheter are more frequent, (28 times in 834 cases). Further complications in peritoneal malabsorption are umbilical fistulas, in 2 cases due to the persistence of the omphalo-mesenteric duct, and hydrocele in six cases due to persistence of the peritoneal-vaginal duct.

Late Visceral Perforations. These complications have only been found in V-P shunts, in a minimal rate (1.08%). One reason may be a local biological reaction, which causes the tip of catheter to be imprisoned, exerting its mechanical action on a restricted area of the visceral wall.

Conclusions. The worst septic biological complications are chronic septicemia with its various clinical forms and ventriculitis. However careful prophylactic precautions may be, septicemia cannot be eliminated, and is more frequent in V-A shunts (a rate of 2.07%). Ventriculo-peritoneal or intrathecal shunts are therefore preferable whenever possible.

Perhaps ventriculitis could be considerably reduced by avoiding ventriculography using an iodine compound.

Among aseptic biological complications, the frequent choroidal-ependymal reactions pose a great problem; obstructing the ventricular catheter they cause malfunction of the shunt. Revisions are troublesome, especially when the ventricles are small.

Phlebothrombosis arises from venous shunts. When using atrial-shunts, the possibility of serious cardiovascular complications must be considered. Tissue reactions around the catheter in its subcutaneous path are at times so intense as to simulate the presence of a catheter which has disconnected itself and migrated. Strangely enough, in some cases the newly formed membranous duct may ensure a certain flow of CSF.

References

- GIUFFRE, R.: Choroidal and ependymal reactions. J. Neurosurg. sci. 20, 123-129 (1976)
- GIUFFRE, R.: Perforation of viscera. J. Neurosurg. sci. <u>20</u>, 149-155 (1976)
- GIUFFRE, R., PALMA, L., FONTANA, M.: Follow-up study of 360 children subjected to extracranial CSF shunt for hydrocephalus. In: Modern problem in paediatrics, Vol. 18. VI_LANI, R. et al. (eds.), pp. 198-202. Basel: Karger 1977
- FONTANA, M., PERRINO, G.: Drammatica risoluzione di un caso di nefropatia da sh-nt liquorale ventricolo-venoso. Reflessioni eziopatogenetiche. Neuropsich. Infant. <u>195</u>, 911-919 (1977)
- GUIDETTI, B., OCCHIPINTI, E., RICCIO, A.: Ventriculo-atrial shunt in 200 cases of non tumoral hydrocephalus in children: remarks on the diagnostic criteria, post-operative complications and longterm results. Acta Neuroch. <u>21</u>, 295-308 (1969)
- 6. GUIDETTI, B., GIUFFRE, R., PALMA, L., FONTANA, M.: Hydrocephalus in infancy and childhood. Child, s. Brain 2, 209-225 (1976)
- OCCHIPINTI, E., RICCIO, A.: Considerazioni su 200 casi di idrocefalo non tumorale dell/infanzia operato di derivazione ventricolo-venosa. Neuropsich. Infant. <u>111-112</u>, 435-447 (1970)
- OCCHIPINTI, E., RICCIO, A.: Considerazioni su alcune complicazioni dopo intervento di derivazione ventricolo-venosa. Min. Neuroch. 15, 156-160 (1971)
- 9. MIGLIAVACCA, F.: Biological complications. J. Neurosurg. sci. <u>20</u>, 137-138 (1976)
- RAIMONDI, A.J., ROBINSON, J.S., KUWAMUSA, K.: Complications of ventriculo-peritoneal shunting and a critical comparison of the three-piece and one-piece systems. Child's brain 3, 321-342 (1977)
- 11. RICCIO, A., OCCHIPINTI, E.: Septicaemias. J. Neurosurg. sci. <u>20</u>, 131-136 (1976)
- 12. RICCIO, A., OCCHIPINTI, E.: Attuali orientamenti nella diagnostica e nella terapia dell'idrocefalo evolutivo dell'infanzia. Parte III. Acta Paed. Lat. <u>23</u>, 514-549 (1970)
- SHERMAN, A.T.: Purpura necrotica as a complication of ventriculoatrial shunts in hydrocephalus. Arch. Dis. Child <u>47</u>, 821-825 (1973)
- 14. SPERLING, D.R., PATRICK, J.R., ANDERSON, F.M., FLYER, D.C.: Cor pulmonale secondary to ventriculoauricolostomy. Amer. J. Dis. Child. 107, 308-315 (1964)
- 15. SYAMANSUNDAR, R., MOLTHAN, M.E., LIPOSW, H.W.: Cor pulmonale as complication of ventriculo-atrial shunts. Case report J. Neurosurg. <u>33</u>, 221-225 (1970)

4. Complications Due to Incongruous Drainage of Shunt Operations A. CARTERI, P. L. LONGATTI, M. GEROSA, C. MAZZA, and A. PASQUALIN

Complications due to incongruous drainage of a shunt are relatively rare, when compared with other undesirable effects of shunt procedures, but certainly constitute a difficult problem in treatment of hydrocephalus.

Complications due to incongruous drainage are presented by Table 4.1, where they have been conveniently divided into 2 categories: underdrainage and overdrainage. It should be noted that underdrainage phenomena are rarely observed in clinical practice. When present, they are generally related to valve insufficiency or to inadequate opening pressure of the valve.

Table 4.1. Complications due to incongruous CSF drainage, on a total of 1820 patients = 13.7%

<u>Underdrainage</u> Valve insufficiency: 33 (1.8%)

Overdrainage: Acute

_				
1.	Cardiac failure:	13	(0.7%)	
2.	Overrapid decompression:	9	(0.5%)	
3.	Acute subdural or epidural hematoma:	6	(0.3%)	
4.	Ascites: •	2	(O.1%)	
<u>0v</u>	erdrainage: Chronic			
1.	Subdural fluid collection:	48	(2.6%)	
2.	Pseudocraniosynostosis:	31	out of 469 (6.6%)
3.	Slit ventricle syndrome:	17	(O.9%)	
4.	Low pressure headache:	5	(0.2%)	
5.	Secondary aqueduct stenosis:	?		

Overdrainage phenomena are more common. They can be observed immediately after operation, or at a certain time following shunt insertion. The responsibility of surgery per se is generally accepted in the first case. In effect, excessive cerebrospinal fluid loss, or uncontrolled passage of cerebrospinal fluid (CSF) to other body compartments, may cause a collapse of the cerebral hemispheres with consequent tearing of the bridging veins (and formation of a subdural hematoma) or, in younger patients, overriding of the sutures with sunken fontanelle. This latter situation (overrapid decompression) may be accompanied by severe neurological deterioration, or even death $(\underline{1})$. Especially in infants, acute cardiac failure can be a deleterious consequence of an excessive amount of fluid passing rapidly to the venous circulation and to the heart.

Acute ascites is a controversial complication of overdrainage; it is more probably due to a preexisting impaired absorption of the peritoneum (10).

Chronic complications of overdrainage are presented mainly by subdural fluid collection ($\underline{6}$, $\underline{8}$, $\underline{11}$) and pseudocraniosynostosis ($\underline{4}$, $\underline{7}$).

A less frequent complication is the so-called "slit ventricle syndrome" described only recently, with the advent of the computerized axial tomography (CAT). Clinically, it presents with recurrent catheter occlusions in the presence of small hyperdrained ventricles $(\underline{3})$.

Low pressure headaches are generally transient and do not constitute a serious problem in children, with preserved elasticity of the dural membranes.

Secondary aqueductal stenosis has been considered by some as a result of overdrainage, even if this seems very unlikely.

We have observed a total incidence of 13.7% of complications due to incongruous drainage in 1820 examined patients.

All 33 cases of underdrainage were easily resolved by the insertion of a new valve at a lower pressure.

Acute complications of overdrainage were generally uncommon (1.6%), but very severe or even fatal. Overrapid decompression and cardiac failure, both occurring in very young patients, were most dreadful.

Chronic complications of overdrainage were relatively frequent (10.3%). As regards pseudocraniosynostosis, we have not considered minor skull changes, that are generally reversible (4, 7). Our evaluation of pseudocraniosynostosis could be extended only to a total of 469 patients.

Our 48 cases with chronic subdural collections are examined in detail by Table 4.2. In these cases, hydrocephalus was generally non-communicating and ventricular enlargement conspicuous. A relationship with the type and pressure of the valve was not clear. It is interesting to note that 15% of subdural collections did not have a related symptomatology. In cases with symptoms, increased intracranial pressure was more commonly noted (57%), followed in incidence by epileptic seizures (30%). There was a high incidence of bilateral collections (55%). At surgery, subdural hematoma constituted the most common finding. Early subdural collections generally consisted of blood or highly xanthocromic fluid, late collections by small hygromas with membrane formation.

Treatment of chronic subdural collections is controversial. In effect, many subdural hematomas are only space filling, and are discovered incidentally, in the last years mostly by CAT. In our experience, the evolution of a space-filling collection can be quite variable. Spontaneous disappearance, within months, is not a rare occurrence. Progressive enlargement to a space-occupying collection with a related symptomatology is also observed. Our patients without symptoms were not treated surgically, but followed clinically and by repeated CAT. In the presence of symptoms, surgical treatment was undertaken with external drainage and temporary closure of the shunt or change in

Type of hydrocephalus		Clinical signs and symptoms					
a) Communicating	= 33%	a) Absent	= 15%				
b) Non-communicating	= 67%	b) Present	= 85%				
Ventricular size		Side of Collection					
a) Mild dilatation	= 22%	a) Same as catheter	= 21%				
b) Significant dilatatio	on = 78%	b) Opposite to catheter	= 24%				
Type of shunt		c) Bilateral	= 55%				
a) Ventriculoatrial	= 48%	Age at surgery					
b) Ventriculoperitoneal	= 52%	a) Less than 1 year	= 38%				
Valve pressure		b) 1-2 years	= 16%				
a) Low pressure	= 6%	c) More than 2 years	= 46%				
b) Middle pressure	= 91%	Surgery (41 cases)					
c) High pressure	= 3%	a) Hematoma	= 72%				
		b) Xanthocromic fluid	= 15%				
		c) Hygroma	= 13%				

Table 4.2. Chronic subdural collections: 48 cases

valve pressure. More complex surgical procedures were reserved for cases not responding to this therapy. Table 4.3 summarizes our surgical experience with chronic subdural collections.

Table 4.3. Surgical treatment of symptomatic chronic subdural collections (41 cases)

External drainage	= 41 cases (100%)
Change in valve pressure	= 22 cases (53%)
Temporary obstruction of the shunt	= 13 cases (31%)
Removal of shunt	= 5 cases (12%)
Insertion of an intrathecal shunt	= 1 case (2%)

In relation to the prevention of this complication, with the use of an antisiphon valve $(\underline{9})$, a subdural collection was noted also in 3 cases with this device. A slit ventricle syndrome was observed in 17 of our patients. In 8 of them, the symptoms resolved spontaneously and did not require treatment. 8 cases needed repeated revisions of the shunt and one case was treated with a subtemporal decompression, as suggested by others $(\underline{2}, \underline{5})$. Generally, a long time was required following the primary shunt insertion (sometimes many years) before development of a slit ventricle syndrome. We have observed a frequent association with pseudo-craniosynostosis (a condition of non-expansible brain) and with chronic subdural collections, mainly bilateral. In Table 4.4 we have summarized our views on the slit ventricle syndrome. We believe that, in the presence of signs of acutely increased intracranial pressure, surgical treatment should be undertaken. Unfortunately, shunt revision is not always successful, and can possibly Table 4.4. Slit ventricle syndrome

A) F	≀adio	logic	al f	ind	lings
------	-------	-------	------	-----	-------

- 1. Slit ventricles with brain not expansible
- 2. Slit ventricles with expansible brain
- Slit ventricles with a subdural collection (mainly bilateral)

B) Clinical findings

- 1. Mild occasional symptoms
- 2. Acutely increased intracranial pressure

C) Surgical treatment

- 1. Change of valve pressure
- 2. Shunt revision
- 3. Subtemporal decompression

cause further problems, as in one of our patients, with intraventricular bleeding resulting from attempted withdrawal of the ventricular catheter. Subtemporal decompression, even if more radical, can be an effective method of treatment.

Prevention of chronic complications of overdrainage implies a detailed knowledge of the physiology of hydrocephalus and of the profound dynamic changes caused by a shunt system. The finding of a persistently negative intracranial pressure in shunted patients has suggested the use of antisiphon valves and volume controlled shunt systems (9). At present, our experience with these methods is still too limited to draw permanent conclusions.

References

- EMERY, J.L.: Intracranial effects of long-standing decompression of the brain in children with hydrocephalus and meningomyelocele. Develop. Med. Child Neurol. <u>7</u>, 302-309 (1965)
- EPSTEIN, F.J., FLEISCHER, A.S., HOCHWALD, G.M., RANSOHOFF, J.: Subtemporal craniectomy for recurrent shunt obstruction secondary to small ventricles. J. Neurosurg. <u>41</u>, 29-31 (1974)
- FAULHAUER, K., SCHMITZ, P.: Overdrainage phenomena in shunt treated hydrocephalus. Acta Neurochir. 45, 89-101 (1978)
- GRISCOLM, N.T., OH, K.S.: The contracting skull. Inward growth of the inner table as a physiological response to diminution of intracranial content in children. Amer. J. Roentgenol. <u>110</u>, 106-110 (1970)
- HOLNESS, R.O., HOFFMAN, H.J., HENDRICK, E.B.: Subtemporal decompression for the slit ventricle syndrome after shunting in hydrocephalic children. Child's Brain 5, 137-144 (1979)
- ILLINGWORTH, R.D.: Subdural hematoma after treatment of chronic hydrocephalus by ventriculocaval shunts. J. Neurol. Neurosurg. Psych. 33, 95-99 (1970)
- KAUFMAN, B., WEISS, M.H., YOUNG, H.F., NULSEN, F.E.: Effects of prolonged cerebrospinal fluid shunting on the skull and brain. J. Neurosurg. 38, 288-297 (1973).

- Mc CULLOUGH, D.C., FOX, J.L.: Negative intracranial pressure hydrocephalus in adults with shunts and its relationship to the production of subdural hematoma. J. Neurosurg. <u>40</u>, 372-375 (1974)
- 9. PORTNOY, H.D., SCHULTE, R.R., FOX, J.L., CROISSANT, P.D., TRIPP, L.: Anti-siphon and reversible occlusion valves for shunting in hydrocephalus and preventing post-shunt subdural hematomas. J. Neurosurg. <u>38</u>, 729-738 (1973)
- ROSENTHAL, J.D., GOLDEN, G.T., SHAW, C.A., HANES, H.A.: Intractable ascites; a complication of ventriculoperitoneal shunting with a silastic catheter. Am. J. Surg. <u>127</u>, 613-614 (1974)
- 11. SAMUELSON, S., LONG, D.M., CHOU, S.N.: Subdural hematoma as a complication of shunting procedures for normal pressure hydrocephalus. J. Neurosurg. <u>37</u>, 548-552 (1972)

5. Neuropsychologic Results of Shunt Operations

A. AMBROSIO, M. COLANGELO, and L. LIGNORI

The results relative to the psychic and intellectual developments, as well as those relative to the eventual presence of neurological deficits, have been evaluated on a group of 910 patients out of the total of 1820 hydrocephalic patients surgically treated and studied in this study. Among all the patients that have been examined, the time limit from the intervention has been at least five years. The quantitative evaluation of the psychomotor development was made in 448 cases using the Gesell scale and the Brunet-Lezine test for the infants. For the preschool and school children the Terman-Merril test was used. For some patients of such age, in particular for those presenting limited deficits, the WISCH scale was utilized. In the exegesis of such tests the overall performance (as well as the various sutests) was prevalently assumed as indicative of sectorial development rather than the absolute value of the I.Q. The above quantitative results are indicated in the Table 5.1.

Intellectual development	No.	90
Normal	210	46.87
Mild retardation	70	15.62
Moderate retardation	81	18.10
Severe retardation	87	19.41
Total	448	100

Table 5.1. Psychological results

In a second group of 462 patients, the development evaluation tests were not effectuated and the global judgement on the psychological assessment stripped of the behaviour of the patients during the neurological examination as well as from the anamnestic date concerning his family life and social life.

In order to evaluate the results, the neurological assessment was accounted for, considering its implication in modifying the intellectual development and the relative performance to the tests.

Table 5.2 reports the incidence of the neurological deficits among the group of patients examined.

An ulterior element taken into consideration as a result of hydrocephalus is epilepsy. In relation with the psychomotor development, the frequency and the incidence of the crisis have been accounted for, as well as its eventual incidence in the mental sphere.

Among the 910 consecutive cases examined, 172 patients with epileptic seizures (Table 5.3) are included.

Neurological deficits	90	
Pyramidal deficits	24.14	
Ataxia	16.26	
Visual impairment	15.45	
Oculomotor deficit	9.99	
Deficit of speech	5.88	
Deficit of hearing	1.73	
Retardation of growth	6.88	
Other deficits	0.77	
Epilepsy	18.90	

Table 5.2. Neurological deficits

Table 5.3. Incidence of epilepsy

	No.	8	
Mild epilepsy	104	60.46	
Moderate epilepsy	42	24.42	
Severe epilepsy	26	15.12	
Total	172	100	

The data relative to the psychomotor evolution as well as the eventual presence of epilepsy enables us to classify the results of the surgical treatment of hydrocephalus into three categories (Table 5.4).

Results	No.	8	
Good	440	48.36	
Mediocre	183	20.10	
Poor	287	31.54	
Total	910	100	

Table 5.4. Overall results

The first category includes 48.35% of the cases and corresponds to patients whose neurological status is normal. The second category collects 20.10% of the cases characterized by mild neurological deficit, moderately retarded and with the occurrence of rare epileptic convulsions, easily controllable with antiepileptic medications.

Finally, the third category combines those patients whose neurological and psychological deficits are of a consistent degree, eventually presenting severe epileptic syndromes. Generally, 31.53% of the above cases are represented by retarded patients below the level of educability. In the long-term evaluation of the overall results, the neurological impairment due to myelomeningocele are not taken into consideration in view of the effects relative to the surgical treatment of hydrocephalus. Generally speaking, a comment can be made about the particular difficulty deriving from the establishment of a given final result in the field or neuropsychological evaluation of hydrocephalic patients. As a matter of fact, one should know the various factors that act upon the psychological development such as the social environment and the family life. From this we can deduct that the various casuistries which constitute any catamnestic study of this kind are not always confrontable (2, 8).

In reference to the intellectual sphere alone, the relationship between hydrocephalus and psychomotor defects is early documented. In fact, a standard deviation is also identifiable (9). In reality the factors that come together in the final result of the surgical treatment of hydrocephalus are multiple (4, 9). Therefore, we related the collected data of the present study with the following factors.

<u>Type of Hydrocephalus</u>. On the average, better results are observable in cases of communicating hydrocephalus, despite the greater percentage of mediocre results in obstructive hydrocephalus. This data is substantially in perfect accordance with the current literature.

<u>Etiology of Hydrocephalus</u>. Good survival is observed in thos patients whose etiology of hydrocephalus was a post-hemorrhagic reaction. Indeed, good results have been achieved in 51.92% of cases with the post-hemorrhagic etiology while in the group of post-inflammatory hydrocephalus good results were found in only 39.17%. Similar good results (i.e. 51.65%) have been obtained in the patients whose hydrocephalus was classified as unknown, although there is large body of evidence that in such cases the etiology was likewise post-hemorrhagic at the time of birth. This is consistent with the general data in the literature (7).

<u>Duration of Hydrocephalus</u>. The patients who have the shunt inserted in the first month when their symptoms began had a better chance (73.42%) of normal intellectual development than those treated with the shunting procedure 3-6 months after the onset of the symptoms (33.67%).

<u>Number of Revisions</u>. The best results have been achieved in those patients who were never reoperated (54.79%). However, in accordance with HEMMER and DILL (2), RAIMONDI and SOARE (5), there seems to be no direct relationship between the number of reoperations and the final intellectual level.

The final factor in the evaluation of the long-term results should have been the degree of the hydrocephalic dilatation. However, we are not able to furnish numerical data because of the difficulty in comparing these data of different origin within such a cooperative study. There is also no general agreement about the prognostic value of the thickness of the cerebral mantle (3, 6, 10).

References

 GUTHKELCH, A.N., RILEY, N.A.: Influence of etiology on prognosis in surgically treated infantile hydrocephalus. Arch. Dis. Child. 44, 29-35 (1969)

- HEMMER, R., DILL, J.: Das Schicksal Hydrozephalus-operierter Kinder. Katamneses über die körperliche und geistige Entwicklung 5 bis 10 Jahre nach Anlage einer Sistemventrikel-Herzrohr-Drainage.
- LAURENCE, K.M., COATES, S.: The natural history of hydrocephalus detailed analysis of 182 unoperated cases. Arch. Dis. Child <u>37</u>, 345-362 (1962)
- LEPOIRE, J., LAPRAS, C.: Traitement de l'Hydrocephalie non tumorale du nourisson par la derivation ventriculo-atriale. Neurochirurgie <u>13</u>, 211-342 (1967)
- 5. RAIMONDI, A.J., SOARE, P.: Intellectual development in shunted Hydrocephalic children. Amer. J. Dis. Child. <u>127</u>, 664-671 (1974)
- 6. SHURTLEFF, D.B., FOLTZ, E.L., CHAPMAN, J.T.: Ventriculo-skull distance: its reliability as an estimate of "cerebral mantle" in the normocephalic child. Am. J. Dis. Child 111, 262-266 (1966)
- 7. SHURTLEFF, D.B., FOLTZ, E.L., LOESER, J.D.: Hydrocephalus, a definition of its progression and relationship to intellectual function diagnosis and complications. Am. J. Dis. Child. <u>125</u>, 688-693 (1973)
- SHURTLEFF, D.B., KRONMAL, R., FOLTZ, E.L.: Follow-up comparison of hydrocephalus with and without myelomeningocele. J. Neurosurg. 42, 61-68 (1975)
- 9. TROMP, C.N., van de BURG, W.: Surgically treated infantile hydrocephalus and predictability of later intelligence. In: Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 31-36. Berlin, Heidelberg, New York: Springer 1978
- 10. YASHON, D., JANE, J.A., SUGAR, O.: The course of several untreated infantile hydrocephalus. Prognostic significance of the cerebral mantle. J. Neurosurg. <u>23</u>, 509-516 (1965)

6. Non-Invasive Instrumental Follow-Up Studies of Shunt Operations S. M. GAINI, G. TOMEI, R. VILLANI, G. PEZZOLA, and E. BIANCHI

Introduction

The postoperative follow-up studies of shunt-treated non-tumoral hydrocephalic children by means of non-invasive diagnostic procedures presents an intriguing and provocative problem. Only computerized axial tomography provides a reliable representation of the size and extent of cerebro-spinal fluid (CSF) spaces and of the possible pathological changes of brain parenchyma (1, 2, 3, 4, 5). Plain films of the skull, chest and abdomen, may document only continuity and correct position of the shunt system, along with the relevant cranial bony changes which commonly follow shunting procedures (6). Echoencephalography offers valuable data on ventricular size, but has little value in the assessment of pathological findings in the cerebral mantle.

<u>Material</u>

Our study includes 212 non-tumoral hydrocephalic patients, 151 males and 61 females; the age ranged from 3-24 years. The follow-up programme extends from 3-15 years after shunting procedures. Besides neurological examination, including psychological and social adjustment, all patients underwent CT scan and X-ray control of the shunt system. No patient showed signs and symptoms of active intracranial hypertension due to shunt malfunction. The roentgenographic study of the skull had confirmed the conclusions we already presented in a 1976 paper, that is the high incidence of basal and vault changes occurring in hydrocephalic children, changes highly suggestive of excessive CSF drainage. The computerized tomographic studies of a similar group of patients have now added valuable data about ventricle size and brain parenchyma. Ventricular size and intellectual status are compared in Table 6.1. Among the 18 patients with slit ventricles: 13 (73%) presented no mental deficit and 5 (27%) had mild mental impairment; however all of the latter cases exhibited neurological deficits following perinatal meningitis (3 cases), severe malformation hydrocephalus (1 case) and severe intracranial hypertensive episodes with a functioning shunt (1 case). Intelligence, as determined by social adjustment, is not damaged in 36 (78%) out of the 46 patients with normal ventricular size; in 6 a moderate impairment is present (4 of these had meningo-ventriculitis in the history, another had several deep coma episodes due to shunt malfunction, and the last case presented with a malformation hydrocephalus); in 4 cases severe mental impair-ment accompanied grave neurological deficits (subdural calcified haematomas were present in 2 cases and infantile meningo-encephalitis in the history of the other 2 patients). Moderate dilation was found in 43 patients; of these 28 (65%) have normal intellectual status, 11 (25%) moderate intelligence and 4 (10%) severe handicaps. In the group of patients with great dilation (grade 3, 41 patients or grade 4, 20 patients), intelligence was normal in 17 (42%) and in 6 (30%), respectively. The incidence of serious mental deterioration was 22% and 50% of the cases respectively.

Ventricular size		Mental development					
	Cases No.			Medium impairment		Handi- capped	
Collapsed	18	13 (73%)	5	(27%)	-	
Normal	47	37 (78%)	6	(13%)	4	(98)
Dilation (grade 1)	43	28 (65%)	11	(25%)	4	(10%)
Dilation (grade 2)	44	23 (52%)	17	(38%)	4	(9%)
Dilation (grade 3)	40	17 (438)	14	(35%)	9	(22%)
Dilation (grade 4)	20	7 (30%)	4	(20%)	10	(50%)
Total	212	124 (58%)	57	(27%)	31	(15%)

Table 6.1. Ventricular size and mental development in shunt treated hydrocephalic children

A comparison between intelligence and size of extracerebral spaces is shown in Table 6.2. Only 12% of the patients with normal or slightly enlarged sulci are affected by severe mental impairment. On the contrary 45% of patients with markedly increased volume of subarachnoid spaces (a sign of brain mantle damage) are seriously mentally impaired. A clear positive relation is evident between normal intellectual status and normal size of extracerebral spaces. The overall consideration of the CSF spaces of the 31 handicapped patients reveals that: 9 (28%) show a considerable dilation both of the ventricles and of the extracerebral spaces. The same finding is present in 6 (10%) of the cases showing moderate mental impairment and only in 2 of the 124 cases with normal intelligence. Among 12 seriously retarded patients who present normal or slightly increased ventricular size, 6 (50%) have enlarged cerebral spaces, a sign shown by only 13 of the 80 patients with normal mental development. The comparative study of mental development, ventricular width and subarachnoid spaces size had confirmed that the evaluation of the extracerebral spaces volume possesses more neurological significance than the evaluation of the ventricular volume alone. In fact, 23 patients with marked dilation of the cerebral ventricles but normal cerebral sulci have adequate intellectual development, 13 have a mild mental impairment and only 10 are seriously retarded. Correlation of mental development and cerebral mantle features reveals (Table 6.3) that 94 of 149 patients with a normal mantle densitometric appearance have a normal intellectual status, 39 (24%) a moderate impairment and only 20% are retarded.

Of the 12 cases with cerebral atrophy, 7 can be rated as normal, 3 present slight impairment and only 2 present serious mental deterioration (neurological deficits were present in the last 2 patients).

Ventricular focal dilation or porencephalic features were found in 24 patients; among these, 9 patients with a mild dilation of frontal or occipital horns have a normal intellectual status and 10 a moderate impairment. The 5 severely retarded patients show bilateral frontal atrophy (2 cases), parieto-occipital bilateral cystic cavities (2 cases) and an unilateral cyst (1 case).

The pathophysiology of the calcifications exhibited by 5 patients might be as follows: a scar develops due to the cerebral trauma of ventricular catheterization (3 cases) with no deficits and due to inflammatory processes in one case which is moderately impaired. In the remaining case, calcifications are spotted over both hemispheres, and severe mental impairment is present.

External CSF space		Mental deve	lopment		
	Cases No.	Normal	Medium impairment	Handi- capped	
Absent	77 (36%)	39 (51%)	27 (35%)	11 (14%)	
Normal	54 (25%)	39 (72%)	9 (17%)	6 (11%)	
Dilation (grade 1)	42 (20%)	28 (66%)	9 (22%)	5 (12%)	
Dilation (grade 2)	28 (13%)	16 (57%)	· 9 (32%)	3 (11%)	
Dilation (grade 3)	11 (5%)	2 (18%)	3 (27%)	6 (55%)	
Total	212	124 (58%)	57 (27%)	31 (15%)	

Table 6.2. External CSF spaces width and mental development in shunted hydrocephalic children

Table 6.3. Cerebral mantle features and mental development in shunted hydrocephalic children

Cerebral mantle		Mental development				
	Cases No.	Normal	Medium impairment	Handi- capped		
Normal	149	94	39	19		
Atrophy - focal - hemispheric	9 3	5 2	3 -	1 1		
Porencephaly	24	9	10	5		
Cyst - supratentorial - infratentorial	3 3	2 2	- 1	1		
IV. vent. dilation	2	1	-	1		
Calcification	5	3	1	1		
Tumor	2	2	-	-		
A-V malformation	2	-	2	-		
Extracerebral fluid collection	10	3	3	4		

Extracerebral haematomas were observed in 10 patients: 3 are symptomfree whereas voluminous and calcified extracerebral fluid collections, suggestive of a chronic process, are present in 4 retarded patients.

Discussion and Conclusion

Our experience confirms that CT scan is the best single procedure to provide the largest amount of information in order to evaluate shunt-treated patients. Comparison between the dimensions of CSF spaces, cerebral mantle and assessment of intellectual status reveals a high percentage of adequately developed patients with a normal CT. Mental impairment of patients with normal CT is mainly due to inflammatory processes at the origin of the hydrocephalus itself. Ventricular size seldom correlates directly with the mental development status. In fact even extreme ventricular hydrocephalus may be associated with a normal mantle and normal intellectual development. Also asymptomatic intracranial hypotension, highly likely in patients with collapsed ventricles, does not seem to play a fundamental role in the development of intellectual functions.

The findings we presented, based on CT examination and clinical evaluation of the patients, have substantial practical interest for the clinical management of the hydrocephalic patients with shunts. While CT scan is a valuable tool for demonstrating morphological changes (and thus explaining clinical deterioration by demonstrating regional atrophies, haematomas, arachnoid cysts, tumors), functional data in hydrocephalic conditions may be only assumed from subependymal periventricular density alterations, and assessment of correct functioning of the shunt system must be deduced mainly from the clinical status. CT then, cannot and must not be the only means of evaluating hydrocephalic patients, as it must always be strictly correlated to clinical conditions and pathogenetic causes. We must also be reminded that even the definite correlation emerging between morphological findings and mental status presents exceptions: the occasional altered scan in the mentally unimpaired patients and the fully normal scan in the retarded one.

As in many fields, the questions raised by the findings which have emerged in the course of our investigation are more numerous than one can answer. Thus the problem of infantile hydrocephalus and its mangement awaits many further contributions of study and investigation and is certainly still quite far from being solved.

References

- HOCKELEY, A.D., HOLMES, A.E.: Computerized axial tomography and shunt dependency. In: Advances in neurosurgery, Vol. 6. WÜLLEN-WEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 48-51. Berlin, Heidelberg, New York: Springer 1978
- MEESE, W., LANKSCH, W., WENDE, S.: Diagnosis and postoperative follow-up studies in infantile idrocephalus using computerized tomography. In: Cranial computerized tomography. LANKSCH, W., KAZNER, E. (eds.), pp. 424-429. Berlin, Heidelberg, New York: Springer 1976
- 3. NAIDICH, T.P., EPSTEIN, F., LIN, J.P., KRICHEFF, I.I., HOCHWALD, G.M.: Evaluation of pediatric hydrocephalus by computerized tomography. Radiol. <u>119</u>, 337-345 (1976)
- 4. PALMIERI, A., MENICHELLI, F., PASQUINI, U., CALVOLINI, U.: Role of computed tomography in the postoperative evaluation of infantile hydrocephalus. Neurorad. <u>14</u>, 257-262 (1978)
- RAYBAND, C., FARNARIER, P., PALMIERI, P., PINSARD, N., CHOUX, M.: CAT in the diagnosis and management of childhood hydrocephalus. In: Computerized axial tomography. De BOULAY, G.H., MOSELEY, I.F. (eds.), pp. 168-172. Berlin, Heidelberg, New York: Springer 1977
- 6. VILLANI, R., GAINI, S.M., GIOVANELLI, M., TOMEI, G., ZAVANONE, M.L., MOTTI, E.D.F.: Skull changes and intellectual status in hydrocephalic children following CSF shunting. Develop. Med. Child Neurol. <u>18</u>, Supp. 37, 78-81 (1976)

Investigations of Factors Influencing the Development of Hydrocephalic Children

F. ZAUNBAUER, K. GLONING, E. HIFT, W. KOSS, and M. SUNDER-PLASSMANN

Introduction

Following the diagnosis of an internal hydrocephalus in a child, parents and doctors alike are primarily concerned about the future development of the little patient. It is for this reason that we have investigated a number of factors that might be of assistance in obtaining the above information.

Within the scope of this paper¹ we have tested the following factors: time elapsed since onset, age at time of surgery, thickness of the brain mantle at the time of surgery, circumference of head, re-operations, functioning of the shunt at follow-up examination.

Patients

Within a period of one year all children who had undergone a shunt operation at the Vienna Neurosurgical University Hospital because of an internal hydrocephalus within the last 6 months were asked to come for an outpatient follow-up examination (all patients having been provided with a Pudenz-Heyer shunt system). Those children who had undergone palliative surgery because of an inoperable brain tumor were not included in the study. Letters were sent out to the parents of 76 children; 52 actually came - 20 girls and 32 boys; they were accompanied by either mother and father or both parents. At this point in time the children's age ranged between 10.5 months and 12 years and 4 months (the average age being 2 years and 9.5 months).

With regard to etiology, the following findings were of interest: 7 cases of meningocele, 4 cases of meningitis, 2 cases of each cerebral malformations, Rhesus incompatibility and craniotabes and toxoplasmosis in one case; the remaining 34 cases were due to unknown causes. The age at the time of surgery ranged between 2 weeks and 8 years and 3 months (average 5.7 months). The time span between the occurrence of the primary symptom and the operation ranged from 2 weeks to 3 years and 8 months - the average being 6 weeks.

At the time of surgery the cortex thickness was 1.3 to 35.00 mms (mean value 13.75 mms). 26 children (= 50%) required at least one reoperation. 43 children still had a functioning shunt when palpitated, nine did not.

¹ The paper was largely written in cooperation with Prof. Dr. K. GLO-NING from the Neurological Institute of the University of Vienna who unfortunately passed away a while ago.

Method of Examination and Evaluation

Every child was subjected to a psychological (development) a clinical neurosurgical and a neurological examination. The psychological test varied according to age and development level: age group 0-6 years -KRAMER Intelligence Test or Infant Test by BÜHLER and HETZER; age group 4-6 years - Intelligence Test for Pre-school Children, the WECHSLER Pre-school- and Primary-Scale of Intelligence Test (WPPSI); age group 6-15 years was given the Hamburg WECHSLER Intelligence Test.

For the joint evaluation, the results of the psychological tests were converted to development quotients and then classified into four groups: a) not affected (IQ ≥ 0.90), b) slightly affected (IQ 0.70-0.89), c) severely affected (IQ 0.60-0.79) and d) most severely affected (IQ < 0.60).

Following the neurological examination, the children were subdivided into non-specific, slightly affected (for instance strabism or reflex difference) or severely affected (this implied at least a monoparesis).

The numerical data obtained in no way corresponded to a normal distribution; evaluation was therefore continued and the Lienert procedure (independent of parameters) was used. For this purpose the data were transformed into alternative characteristics:

The quantitative characteristics (time elapsed since onset, age at time of operation, thickness of brain mantle, circumference of head, reoperations, IQ, neurological disorder) were divided in half; between the factors listed, four-field correlations were then computed.

Results

Table 1 shows the distribution of incidence of the children examined with regard to the severity of the disturbed psychological development. The IQ's amounted to 0.6-1.12 (mean value 0.64).

Table 1. State of mental development at follow-up

Not affected	6	11.5%
Slightly affected	7	13.5%
Severely affected	23	44.2%
Very severely affected	16	30.8%

Table 2 shows the correlations of the different factors examined and the *mental development*. Mention had to be made of the fact that five out of the seven children with meningoceles were severely disturbed; this percentage more or less corresponds to that of the remaining children - it is therefore not necessary to evaluate them separately.

17 children showed no specific neurological symptoms (= 32.7%), 9 (= 17.3%) presented minimal deficiencies.

The two children who had brain malformations were severely impaired both mentally and neurologically.

	r	Significance
Time elapsed since onset of operation	0.00	nsa
Age at time of surgery	-0.04	ns
Thickness of brain mantle at operation	0.13	ns
Reoperation	0.12	ns
Head circumference at follow-up examination	0.04	ns
Severe neurological impairment	-0.35	p < 5%

<u>Table 2.</u> Four-field correlation coefficient (r) between mental development and the factors listed

a ns = not significant

In 43 children the shunt worked adequately, in 9 it no longer functioned. Of the latter group, one child had a slight development disorder, the others were severely retarded in their development.

Discussion

As stated in other studies (GEISZ and STEINHAUER), the children seen at a follow-up examination in our clinic - when viewed as a group are markedly impaired in their mental development. However, about 25% show none or only very slight impairment in their development.

Neurological disorders and mental handicap have a low but significant correlation. This result is of no special interest since the above symptoms are both manifestations of the cerebral disorder due to the hydrocephalus.

LAURENCE, UNGER et al. as well as INGRAM and NAUGHTON have arrived at similar results. In agreement with LAURENCE, HEMMER and DILL; GUIDETTI et al.; IVAN et al. and SEEGER, we found that the correlations between mental development and head circumference, brain mantle thickness and reoperations are not significant. The correlations are around zero. As an impressive example it ought to be mentioned that a child with a brain mantle thickness of 5 mms was found to be developed well above average at the follow-up examination.

Contrary to the authors mentioned above, we found no significant correlation between mental development, time elapsed since onset and age at which surgery was performed. This may be partly due to the fact that we used methods that were independent of parameters and that such techniques tend to level matters. We found, however, that these methods were the only ones that turned out to be appropriate for the group. The correlations computed for instance by GEISZ and STEINHAUER are not high either (around -0.35).

From this we may conclude that neither the time elapsed since onset nor age at the time of the operation nor thickness of brain mantle nor reoperations, etc. are very important in terms of prognosis. Of course, surgery ought to be done as early as possible.

Two factors are probably relevant in terms of prognosis but because of low incidence they are not statistically significant in our study:

- 1. Both children with malformations of the brain are severely retarded in their mental development.
- 2. 7 out of the 8 children with a non-functioning shunt discovered at the time of the follow-up examination are severely or very severely retarded in their mental development.

Summary

Fifty-two children who had undergone a shunt operation because of internal hydrocephalus, were followed-up at least six months later; the investigators recorded psychological development, and examined the neurosurgical and neurological results of the operation. 25% of the patients examined were little or not all affected in their mental development. 50% had no neurological symptoms or presented only slight deficiencies. Time elapsed since onset, age at the time of operation, brain mantle thickness at the time of operation, reoperations and head circumference do not seem to be parameters on the basis of which a prognosis may be made. Patients with skull malformations and children with a non-functioning shunt discovered at the time of the follow-up are more likely to have an unfavourable prognosis.

References

- BÜHLER, Ch., HETZER, H.: Kleinkindertests. München: J.A. Barth, 1966
- GEISZ, D., STEINHAUSEN, H.C.: Zur psychologischen Entwicklung von Kindern mit Hydrocephalus. Prax. Kinderpsychol. Kinderpsychiatr. 23 (4), 113-118 (1974)
- 3. GIUDETTI, B., OCHHIPINTI, E., RICCIO, A.: Ventriculo-atrial shunt in 200 cases of non-tumoral hydrocephalus in children: remarks on the diagnostic criteria, postoperative complications and longterm results. Acta neurochir. (Wien) 21, 295-308 (1969)
- 4. HARDESTY, F.P., PRIESTER, H.J.: Hamburg-Wechsler-Intelligenztest für Kinder. Stuttgart, Bern: H. Huber 1950
- HEMMER, R., DILL, J.: Das Schicksal hydrocephalusoperierter Kinder. Dtsch. med. Wschr. <u>27</u>, 1149-1155 (1971)
- INGRAM, T.B., NAUGHTON, J.A.: Pediatric and psychological aspects of cerebral palsy associated with hydrocephalus. Dev. Med. Child Neurol. <u>4</u>, 287-292 (1962)
- IVAN, L.P., et al.: Surgical treatment of infantile hydrocephalus: ten years experience in the use of ventriculo-atrial shunts with the Holter-valve. Canad. Med. Ass. J. 98, 337-343 (1968)
- LAURENCE, K.M.: Neurological and intellectual sequelae of hydrocephalus. Arch. Neurol. (Chicago) <u>20</u>, 73-81 (1969)
- LIENERT, G.: Verteilungsfreie Verfahren in der Biostatistik.
 Aufl. Meisenheim am Glan: A. Hain 1973
- 10. KRAMER, I.: Intelligenztest. 3. Aufl.. Solothurn: Antonius-Verlag 1965
- 11. SEEGER, W.: Eigene Erfahrungen in der Behandlung des kindlichen Hydrocephalus mit der Pudenz-Heyer-Drainage. Med. Klinik <u>35</u>, 1422-1426 (1963)

- 12. UNGER, R.R., ALLMANN, S., SCHMITZ, W., et al.: Neurologischpsychiatrische Nachuntersuchungsergebnisse nach Shuntoperationen (Spitz-Holter) wegen frühkindlichem Hydrozephalus. Dtsch. Gesundh. <u>24</u>, 1427-1435 (1969)
- 13. WECHSLER, D.: Wechsler Preschool and Primary Scale of Intelligence. New York: The Psychological Corporation 1967

Long-Term Results of Shunt Operations over a Period of 10 Years M. KLINGER, G. GROHMANN, W. HAUBNER, S. KUNZE, and H. ELM

In order to study the long-term results of CSF-shunts in children, the charts of 287 patients operated at the Neurochirurgische Universitätsklinik in Erlangen between 1968 and 1978 were analyzed. This group was subdivided according to the type of shunt used: 260 children had received ventriculo-atrial shunts, chiefly of the SPITZ-HOLTER type and 37 children were treated with ventriculo-cisternal shunts as described by TORKILDSEN.

Figure 1 presents a survey of the causes of hydrocephalus in the 260 children with ventriculo-atrial (VA) shunts. The hydrocephalus was congenital or of indeterminate origin in about two-fifths of the cases, while tumors, infections and myelo-meningoceles were each responsible for a further one-fifth of the cases.

Marked differences become apparent when the complications within these etiological groups are studied (Fig. 2). As was to be expected, the mortality was highest in the group due to tumors, where all 16 patients (33%) succumbed to the underlying disease within a period of one year. The second-highest mortality, 14%, occurred in children with hydrocephalus accompanying myelomeningoceles. All these patients also died within a year of the shunt operation, but here too, the basic malformations with paralysis of the legs, as well as of colon and bladder function play an important role. In contrast, the mortality of children with congenital hydrocephalus is considerably lower, namely 4%. The top of each column in Fig. 2 indicates the patients without complications. This shows that the most favorable results are obtained in cases of myelomeningocele and congenital hydrocephalus, where no less than 64% and 62% respectively remained free of disturbances in function.

The study showed that 84 of the 269 patients with a VA-shunt had to be reoperated because of a complication, making a percentage of 31.8%. However, many patients had to have several revisions of the shunt so that a total number of 126 operations were performed resulting in a total complication rate of 48.5%. Classified according to the etiological groups, the complication rate was highest in the infectious group (48%) where almost every second child had to have a shunt revision. In cases of congenital hydrocephalus more than one-third of the patients were reoperated in the course of 10 years.

Figure 3 indicates which section of the shunt system was affected by the complication. Disturbance of function occurs most frequently at the site of the cardiac catheter, where 35.7% of the cases were found to have either a thrombotic occlusion of the catheter or shortening of the catheter due to growth of the child. The ventricular catheter was replaced in 36 patients corresponding to about one-quarter (25.7%) of the cases, while the valve itself was removed and a new one reinserted in 32 patients (22.8%). General complications were usually of an infectious nature and the percentage of these complications at 15.7% corresponds to the literature. These infections included local wound healing problems, ependymitis and CSF-fistula. Valve sepsis was observed 4 times in our patient material.

A further question of interest for a long-term survey is, "How many patients were reoperated how many times, and *when* is a complication most likely to occur?" In Fig. 4 the graphic illustration indicates that two-thirds of the patients never had any complications. 20% needed one reoperation because of a complication and this was usually necessary within the first year of the primary operation. A second revision had to be performed in 9% of the patients and here, too, the time of reoperation for 18 of the 24 patients was within the first year after the shunt revision. Only 2%, i.e., 5 patients had to have 3 shunt revisions.

Obtaining data on the mental function of these patients is much more difficult according to our experience. For one thing, patients, particularly children, are loathe to come to a follow-up examination in the previously traumatic hospital environment, especially if they are free of symptoms. Another factor is the lack of information on the follow-up charts regarding the developmental status of these children.

Table 1 shows the long-term evaluation of the development and intelligence of 164 patients. 19% corresponding to 31 patients, were judged to be severely retarded. About one-third of the cases, 28% to be exact, were moderately retarded. More than half of the children - 53% - were able to attend school or showed an intelligence level corresponding to their actual age at examination. Many adolescents had begun an apprenticeship or were employed full-time. Since there is an obvious symple error due to inadequate patient response, the percentage of normal children is probably considerably higher.

Table 1	<u>l</u> .	Late	results	of	164	patients

Severely retarded	31
Moderately retarded	46
Normal	87

Information on the function of the valve was found in 139 patient charts. In 127 of these 139 cases, the valve functioned perfectly. In 12 cases, making up 8.6%, valve function was impaired.

The second major group of the hydrocephalic children comprising a total of 37 cases, was treated with a TORKILDSEN ventriculo-cisternal shunt. In Table 2 the results of these TORKILDSEN shunts are presented. Since the majority were performed for cases of midline tumor, it is not surprising to find a mortality of 16% in the tumor group. In the comparable group of tumor patients treated with a SPITZ-HOLTER shunt, the mortality was 33%.

A transient disturbance in the CSF flow as indicated by headache and general worsening of the patient's condition occurred in 3 tumor patients and in 2 non-tumor patients with hydrocephalus. Far more serious are cases of a permanent impairment of CSF flow through the shunt which make a ventriculo-atrial shunt necessary. The total of 6 tumor patients and 2 non-tumor patients who required this measure result in a complication rate of 21.6%. The comparable percentage in cases with VA-shunts was 31.8% in our case material.

No. of cases		Complication			
		Transient disturbance	Permanent disturbance	Mortality (%)	
Hydrocephalus due to tumor	25	3 = 12%	6 = 24%	4 = 16%	
Hydrocephalus due to non-tumorous disease	12	2 = 18%	2 = 18%		

Table 2. Results of hydrocephalus treatment with a TORKILDSEN shunt

In summary, the most important data will be emphasized. Surveying a total of 260 children and adolescents treated over a period of 10 years, no complications occurred in 68% of the patients. In 31.8% of the cases, corresponding to 84 patients, a total of 126 shunt revisions were performed, resulting in a total complication rate of 48.5%. The disturbances of function affected the cardiac catheter most often, then the ventricular catheter and finally the valve itself in order of importance. Usually these complications occur in the first year, so that it seems reasonable to schedule follow-up examinations at short intervals in this period. More than half of the patients on whom such data was available were in school or employed, while one-fifth was severely retarded. In the great majority of patients, valve function was good.

Patients treated with a ventriculo-cisternal shunt of TORKILDSEN were found to have a permanent disturbance of function in 21.6% of the cases. Such a complication made it necessary to perform a ventriculoatrial shunt in addition to the TORKILDSEN shunt. Despite the relatively small number of TORKILDSEN shunts, the lower complication rate on a long-term basis is remarkable.

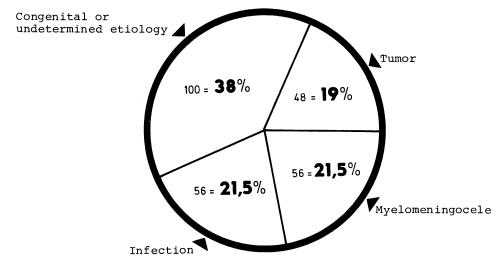


Fig. 1. Etiology of hydrocephalus in 260 patients

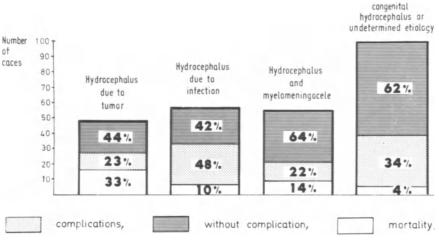


Fig. 2. Complication rate and mortality in cases of hydrocephalus due to various etiologies

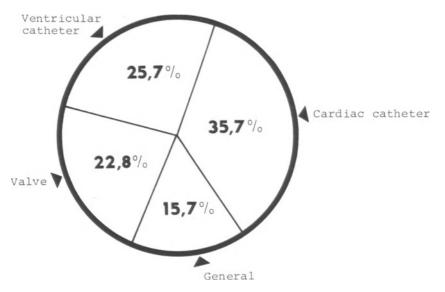


Fig. 3. Complication rate of the parts of the shunt system

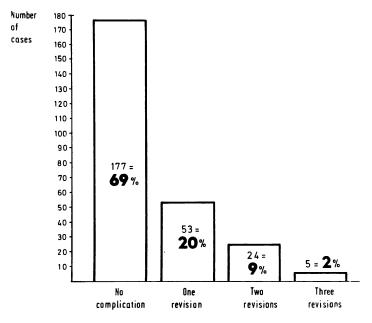


Fig. 4. Revisions of ventriculo-atrial shunts

Complications Following Shunt Operations in Children J. LIESEGANG, E. W. STRAHL, and H. R. STREICHER

This communication deals with a series of 287 children up to the age of 6, who were provided with a ventriculoatrial or ventriculoperitoneal shunt during the years 1968-1977. With a few exceptions only the Pudenz-systems were used. The primary treatment has normally been ventriculocardiac shunting. Only 17 patients (i.e. 6%) were initially provided with a ventriculoperitoneal shunt and 25 received a peritoneal catheter as a replacement for their atrial one. In most cases there was no special indication for the insertion of the peritoneal catheter (n = 17), often the lack of a venous entrance was the reason for using a peritoneal catheter (n = 13), mainly during atrial catheter revisions. Other causes included cardiac diseases (n = 4), septicaemia (n = 2) and superficial skin diseases of the neck (n = 2).

All operations were performed under general anaesthesia. Using a ventriculoatrial shunt, the atrial catheter was advanced under fluoroscopy control through the right common facial vein or the internal jugular vein until the tip of the catheter was lying in the right atrium. Peritoneal catheters were inserted with the help of a peritoneal trocar, the length of the intraperitoneal section of the catheters being 20-30 cm. The ventricular catheters and flushing reservoirs were inserted through a right frontal approach in 228 cases, through a temporo-occipital one in 59 patients.

The main diagnoses and causes for shunting are listed in Table 1.

HC ^a and myelocele	88
Congenital HC	79
HC after meningitis	32
HC due to tumor obstruction	31
HC of other or unknown origin	39
Subdural effusion	18
Total	287

Table 1. Cause for shunt operation

a HC = hydrocephalus.

The age of the infant patients at the time of the initial shunt operation is shown in Table 2, more than 50% of them being babies, 0-3 months old, at that time. Of the 287 patients provided with a shunt, 159 (55%) later on needed a revision of the shunt system, the incidence of complications decreasing with advancing age.

Age	No. of patients	No. of patients with shunt revisions
0-3 months	158	110 (69%)
4-6 months	39	20 (51%)
7-12 months	29	12 (41%)
1-3 years	46	14 (30%)
4-6 years	15	3 (20%)
Total	287	159 (55%)

<u>Table 2</u>. Time of initial shunt operation and number of patients with shunt revisions

Altogether 284 revisions were performed on these 159 children, an average of 1.79 revisions per patient (Table 3).

Number of revisions	No. of patients
1	83 (52%)
2	41 (26%)
3	25 (16%)
4 or more	10 (6%)
Total 284	159 (100%)

Table 3. Number of revisions per patient

Table 4 shows that most of these revisions were necessary because of complications of the distal part of the shunt system, the atrial or the peritoneal catheter, so that 44% of all 276 patients provided with an atrial catheter needed a revision of the delivery cannula and 41% of the 43 patients with a ventriculoperitoneal shunt. Insofar as the small number of patients with a ventriculoperitoneal shunt allows a deduction, the percentage of complications seems to be about the same with atrial as with peritoneal catheter. In 23% of all 287 patients, complications occurring at the ventricular catheter caused a revision, while complications at the flushing device led to a revision operation in only 5% of all children.

Table 4. Number of patients with shunt revisions and number of revisions

	No. of patients	Number of revisions
Ventricular catheter	65 (23%)	82
Flushing device	14 (5%)	14
Atrial catheter	123 (44%)	163
Peritoneal catheter	18 (41%)	25
Total	159	284

The most common cause for a revision of the ventricular catheter was the obstruction of the cannula by choroid plexus or brain tissue in 57 patients, the incidence being the same regardless whether a frontal or temporo-occipital approach to the right lateral ventricle was used. In 11 cases wrong positioning of the ventricular catheter made an early revision necessary, but this complication occurred twice as often when the catheter was inserted through the temporo-occipital approach than through the frontal one. Seven infections due to shunt colonisation are listed here including 5 cases of meningitis and 2 cases of septicaemia which led to a removal and reinsertion of the shunt system later on. Other causes for a revision of the ventricular catheter were intracerebral hemorrhage and disconnection of the catheter in two patients each. In 3 cases the reason for a ventricular catheter revision was a suspected obstruction of the cannula which could not be verified during the operation (Table 5).

Table 5. Ventricular catheter (VC) - cause of revision

Obstruction of VC by choroid plexus or brain tissue	57	
Wrong position of VC	11	
Infection	7	
Intracerebral hemorrhage	2	
Other	5	
Total	82	

Half of the small sum of 14 complications at the flushing device were local wound infections in 7 cases. One patient had a meningitis and another one a septicaemia due to a CSF-fistula at the site of the reservoir. 3 times the valve had to be replaced because of leakage on account of repeated tapping of CSF and twice the reservoir was obstructed by brain tissue or blood (Table 6).

Table 6. Flushing device (FD) - cause of revision

Infection	9
Defects of FD by puncturing	3
Obstruction of FD	2
Total	14

The most frequent complication causing a revision of the atrial catheter was a shortening of the catheter because of the patient's growth: 127 times in a total of 87 children.

At this point it should be mentioned that in most cases the catheter was replaced by a longer one as a precaution when X-ray controls showed a withdrawal of the catheter from the right atrium or the superior vena cava. That is probably why the shortening of the catheter was combined with a thrombotic occlusion in only 22 of these 127 cases. Infections originating from the atrial catheter occurred 11 times in 10 patients. These infections included 6 cases of septicaemia, 2 cases of meningitis and 3 local wound infections. A relative valve insufficiency because the opening pressure was too high led to a revision in 7 cases, the opening pressure was too low in 5 patients, 2 of them developed a subdural effusion that needed a drainage. Five catheters disconnected, one of them slipped into the pulmonary artery and had to be removed by thoracotomy. Other causes for revision were CSF-fistulas (n = 3), kinking of the catheter (n = 3) and disturbances of heart rhythm (n = 2) as shown in Table 7.

Table 7. Atrial catheters (AC) - cause of revision

AC too short due to growth	127	
Infection	11	
Opening pressure too high	7	
Opening pressure too low	5	
Disconnection of AC	5	
CSF-fistula	3	
Other	5	
Total	163	

Table 8 shows the reasons for revision of peritoneal catheter. A frequent cause for such a revision was infection which occurred in 7 cases. These infections included 3 cases of septicaemia, 2 cases of meningitis, following catheter colonisation and 2 cases of local wound infection. Disconnection of the peritoneal catheter occurred in 7 patients, 2 of them requiring a laparotomy in order to remove the detached catheter which had slipped into the abdominal cavity. The detachment of the catheters occurred only with the RAIMONDI spiral type which we used in the beginning. In our experience, the silicone rubber of the RAIMONDI spiral catheter is not as elastic as that of other types and tends to tear more easily at the connecting ligatures. For this reason we have stopped using RAIMONDI catheters and since then have observed no such complication any more. Other causes of revisions were kinking of the catheter (n = 3), obstruction of the catheter (n = 3), peritoneal adhesions and malabsorption of CSF (n = 3) and a relative valve insufficiency because the opening pressure of the valve was too high. Different placement of the catheter in the abdomen had no influence on the type of complication nor on the time of their onset.

Table 8. Peritoneal catheters (PC) - cause of revision

Infection	7	
Disconnection of PC	7	
Kinking of PC	3	
Obstruction of PC	3	
Peritoneal adhesion and mal-		
absorption of CSF	3	
Opening pressure to high	3	
Total	25	

The number of infections rose to 34, that is 12,2% of all children provided with a shunt. 9 of these patients died due to a meningitis

or septicaemia (Table 9). No child in this series died because of other complications as far as we know. The fatal outcome in 1/4 of the patients who developed an infection, that is a total mortality of 3,1%, therefore shows that infection continues to be the most dangerous complication following shunt operations. Therefore everything must be done to reduce the occurrence of infections and to treat them adequately. In our opinion this means a temporary removal of the shunt system in due time in addition to antibiotic therapy in most cases. Complications due to growth or obstruction, on the other hand, can be avoided only to some extent like the obstruction of a shortened cardiac catheter by prophylactic lengthening for instance, presuming a close postoperative follow up. Complications such as disconnection of catheters, wrong positioning of the ventricular catheter or relative valve insufficiency can be reduced significantly by improved operative technique. Reducing the tapping of the reservoir to the indispensible number helps to diminish defects of the valve and infections, too.

Table	9.	Number	of	infections

			,		
	Ventri- cular catheter	Flushing device	Atrial catheter	peri- toneal catheter	Total
Local infection		7	3	2	12
Meningitis	5 (1퐈)	1 (1中)	2 (1뇬)	2 (1⊕)	10
Sepsis	2 (1판)	1	6 (3班)	3	12
Total	7 (2표)	9 (1퍞)	11 (5垂)	7 (1軠)	34 (9⊕)

Thus the total complication rate of about 60% in 1969/1970 could be lowered to 26% in 1977 in this series.

References

- BEKS, J.W., TER WEEME, C.A.: Results of the treatment of hydrocephalus with ventriculojugular shunts. Neurochirurgie <u>18</u>, 118-120 (1975)
- HEMMER, R.: Surgical treatment of hydrocephalus: Complications mortality, developmental prospects. Z. Kinderchir. 22, 443-452 (1977)
- 2a.IGUELZI, R.J., KIRSCH, W.M.: Follow-up analysis of ventriculoperitoneal and ventriculoatrial shunts for hydrocephalus. J. Neurosurg. <u>42</u>, 679-682 (1975)
- JEFFREYS, R.V., CHIR, M.: Complications of ventriculoatrial shunting in hydrocephalus. Adv. Neurosurg. 6, 17-22 (1978)
- LEEM, W., MILTZ, H.: Complications following ventriculo-atrial shunts in hydrocephalus. Adv. Neurosurg. <u>6</u>, 1-5 (1978)
- RAIMONDI, A.J., ROBINSON, J.S., KAWANUERA, K.: Complications of ventriculo-peritoneal shunting and a critical comparison of the three-piece and one-piece systems. Child's Brain 3, 321-342 (1977)
- 6. STRAHL, E.W., LIESEGANG, J., ROOSEN, K.: Complications following ventriculo-peritoneal shunts. Adv. Neurosurg. <u>6</u>, 6-9 (1978)

Surgical Treatment and Long-Term Results in Children with Hydrocephalus

B. RAMA and O. SPOERRI

Introduction

From 1959-1968 hydrocephalus in 189 consecutive patients was treated surgically. Children with hydrocephalus due to intracranial tumor are not included in this series. The follow-up period for the youngest patient is now 10 years and 20 years for the oldest. How have these patients, some of whom are already teenagers, developed?

Material

Of the original 189 children 82 are alive. They are now between 10 and 34 years old (mean = 13.9 years). The mean observation period between primary operation and follow-up examination is 13.8 years; and 6.6 years between last operation and follow-up examination. 74 children died.

Thirty-three cases are not included in this presentation, as 26 of them had failed to return to the outpatient service following primary surgery, and as the observation period for another 7 children was less than ten years before they got lost to further follow-up.

Cause of Hydrocephalus

Idiopathic hydrocephalus was the largest etiological group (55 children), followed by spina bifida (53 children). In 22 patients hydrocephalus had developed following bacterial infection of the CNS. Aqueduct stenosis and perinatal traumatic intracranial hemorrhage were the cause of 11 cases of hydrocephalus each. 4 times hydrocephalus was due to toxoplasmosis.

Some Aspects of Surgical Treatment

The first ventriculo-atrial shunting procedure in this clinic was performed in 1959; a HOLTER valve was used. Until 1962 the HOLTER valve was inserted. From 1963 onwards the PUDENZ valve was used exclusively $(\frac{7}{2})$.

The 156 patients underwent a total of 369 operations. Sixty-eight children had one operation; 4 times a TORKILDSEN procedure was carried out; 10 patients had a HOLTER valve and 54 times a PUDENZ valve was inserted. 88 children had two or more operations, two of these children were operated on 11 times. There is no apparent relationship between the number of shunt revisions and the type of drainage system used nor of operating technique. Prognosis with regard to the number of operations was most favorable for hydrocephalic children with spina bifida, and hydrocephalus that had developed due to a bacterial infection of the CNS. The average was 1.9 operations per child. It must be born in mind, however, that the mortality of the neonate and the small child was highest in this group. But those 35 children who survived required a surprisingly small number of operations. Children with hydrocephalus following toxoplasmosis and those with idiopathic hydrocephalus needed 2.6 operations per child. Then followed the patients whose hydrocephalus had complicated perinatal intracranial hemorrhage; they required 2.9 operations per child. The unluckiest were the patients with aqueduct stenosis, who underwent 3.4 operations on the average.

The most frequent cause for shunt revision was non-functioning of the ventricular or cardiac catheter and bacterial colonization of the shunting system. The next most frequent indication for shunt revision was increased intracranial pressure in spite of apparent patency of shunt (Table 1).

Table 1. Causes of shunt revision

Ventricular catheter		Wound infection	3,7%
mechanical obstruction	31,8%	Bacterial colonization	
PUDENZ pump deficient	3,3%	of shunting system	14 , 9%
Cardiac catheter obstruction	28,0%	Increased intracranial pressure despite	0 0 0
Peritoneal catheter		functioning shunt	8,9%
obstruction	0,9%	No information	8,4%

Survival - Mortality

Seventy-four of the 156 children died after a mean survival of 2,5 years. Overall mortality was 47%. Mortality in the spina bifida group was 54%, and 63% in children who had aqueduct stenosis. Hydrocephalus due to meningitis and toxoplasmosis showed a mortality of 50% each. Life expectancy was best for patients who had developed following birth injury (27% mortality) (Table 2).

Hydrocephalus			No. of operations per patient		Follow-up period (years	
- Etiology -	Alive	Dead	Alive	Dead	Alive	Dead
Idiopathic	33	22	3,1	1,9	13,3	2,0
Spina bifida	24	29	1,9	1,8	13,4	1,7
Bacterial infection	11	11	2,4	1,4	13,7	1,8
Aqueduct stenosis	4	7	2,5	4	16,7	3,3
Intracranial hemorrhage neonatal	8	3	3,1	2,3	12,7	2,8
Toxoplasmosis	2	2	3	1	13,0	3,5

<u>Table 2</u>. Mean number of operations and mean follow-up period of 156 patients

What did the patients die of?

Twenty times death was due to bacterial colonization of the shunting system and septicemia. Fifteen children died of increased intracranial pressure due to malfunction of the shunt. The death of 4 patients was related to primary surgery: one had an epidural hematoma; one died of intraventricular hemorrhage and in two cases autopsy did not reveal the cause of death. Eleven children died of a cause not directly related to hydrocephalus. Twenty-four times precise information with regard to the cause of death could not be obtained. These children had died away from the hospital. There is no relationship apparent between cause of death and number of operations or etiology of hydrocephalus.

Assessment and Development

It was attempted to evaluate the state of development. Psychological tests were not done systematically in all patients, and the results of such tests are therefore not discussed here. The criteria were school performance, judgement by parents and jugdement on the basis of clinical examination. 37 children are attending regular school: 23 of them are attending primary school, 10 children have reached a secondary level and 4 of them are now at high school. One patient is working as a commercial employee. Twenty-one children are attending a variety of special schools. Another 21 children are handicapped to such a degree that schooling of any sort is not possible. They either live with their family, others in day-homes, and a third group became home-children.

From two patients no information regarding schooling could be obtained. The number of children attending school of any sort is comparable with the figures in LORBER's 1969 series ($\underline{6}$). 32% of the children with normal intellectual performance had a functioning shunt. In 15% of the cases it could not be decided whether or not the shunt was functioning. In the remaining 53% surprisingly a malfunction of the shunt was found. As these children showed no signs of increased intracranial pressure it is thought that the system is functioning at least partially. Otherwise independence of the shunt in this group of patients would have to be assumed, thus confirming LAURENCE's ($\underline{4}$) claim of 47% of arrested hydrocephalus. There is no apparent relationship between the origin of hydrocephalus and intellectual development ($\underline{2}$). Only in the group of perinatal intracranial hemorrhage just 25% of the patients are intellectually normal compared with 50% of all other hydrocephalics (Table 3).

Modern treatment of hydrocephalus, has it really been progress? Before the time of ventriculo-atrial shunting 20% to 35% of the patients were still alive after a follow-up period of 5 until 10 years (1, 2, 3, 5, 6). Thank to modern neurosurgical treatment using CSF shunting procedures, survival rate has improved to 60% (own results) and 86% (6) in the group of idiopathic hydrocephalus. The long term problem with the spina bifida children and hydrocephalus is much greater (2); this may explain why survival rate in a nonselected group of children presents with 53% (own results) to 61% (2).

Purely quantitative life expectancy is unimportant. Surgical treatment has raised the number of educable patients from 5% ($\underline{2}$) and 27% ($\underline{4}$) up to 72% (own results) and 76% ($\underline{1}$, $\underline{2}$, $\underline{3}$, $\underline{5}$, $\underline{6}$).

Hydrocephalus	In- educable			Educable		Total
		Special	Primary	Second- ary	High- school	
Idiopathic ^a	10	5	6	6	4	33
Spina bifida	4	9	9	2	-	24
Bacterial infection	3	2	4	2	-	11
Aqueduct stenosis	1	1	1	1 ^b	-	4
Intracranial hemorrhage neonatal	3	3	2	-	-	8
Toxoplasmosis	-	1	1 .	-	-	2

Tabelle 3. Intellectual development

a No information on 2 patients.

b Commercial employee.

References

- BACHS, A., WALKER, E.: Surgical clinic of hydrocephalus. Surg. Clin. N. Amer. <u>32</u>, 1347-1361 (1952)
- FOLTZ, E.L., SHURTLEFF, D.: Five-year comparative study of hydrocephalus in children with and without operation (113 cases). J. Neurosurg. 20, 1064-1078 (1963)
- HAGBERG, B., SJÖGREN, I.: The chronic brain syndrome of infantile hydrocephalus. Amer. J. Dis. Childh. <u>112</u>, 189-196 (1966)
- 4. LAURENCE, K.M.: The natural history of hydrocephalus. Lancet II, 1152-1154 (1958)
- 5. LAURENCE, K.M., COATES, S.: The natural history of hydrocephalus. Arch. Dis. Childh. 37, 345-362 (1962)
- LORBER, J.: Die Behandlung des primären kongenitalen Hydrocephalus. Mschr. Kinderheilk. <u>117</u>, 66-73 (1969)
- 7. STOLZ, Ch., WENKER, H.: Vergleichende Untersuchungen über Frühergebnisse nach Spitz-Holter- und Pudenz-Heyer-Operationen bei Kindern und Jugendlichen. Z. Kinderchir. 2, 25-39 (1965)

Behavior of Hydrocephalus after Shunt Procedures

K. TORNOW, W. PIOTROWSKI, and H. G. LENARD

Please don't expect me to reveal spectacular new findings about hydrocephalus. What I wish to do is to simply report on the practical experience we have gained with shunt operations.

Our patient group consisted of 43 infants or children with a roughly uniform sex distribution, suffering from hydrocephalus, who had been operated on in Mannheim during the last six years (Table 1). Aetiologically speaking (Table 2) congenital hydrocephalus was predominant in 25 cases, fifteen of them presenting with additional spinal malformations; the remaining forms were acquired either perinatally or postnatally not including brain tumour cases.

Table 1

University Clinic Mannheim Paediatrics - Neurosurgery - Neuroradiology Hydrocephalus in early infancy $n = 43 (21 \sigma^7, 22 \Omega)$

Table 2. Causes of hydrocephalus

Congenital <	with		15
	without /	<pre>spinal malformation;</pre>	10
Acquired		- perinatal	15
		- postnatal	3
			n = 43

Most patients (Table 3) - they accounted for 67% or 29 cases were operated on during the first three months of life. There was one child unable to undergo surgery before the age of eight and another where operation had to be put off until the age of thirteen. Initially, we preferred the HOLTER shunt - nine children underwent it, but later we resorted almost exclusively to the HAKIM shunt. Our operating technique differs only slightly from the original method.

In 23 cases, the postoperative course (Table 4) was completely free from complications, which means only half of the patients. The operation itself was in no case fatal, but three patients died during the first month following the operation from the sixth day onwards, and

Months		
1	12	
1- 3	17	
3-6	7	
6-12	3	
12	4	
	n = 43	-

Table 3. Hydrocephalus. Age at which operation was performed

Table 4. Hydrocephalus. Postoperative course (n = 43)

Free from complications	23
Postoperative death	9
Control	45 x

another 6 children died later from septic complications being almost exclusively the cause of death.

It is impossible to ascertain reliably for each case whether the operation proper had caused the exitus, but certainly it can be taken for granted that shunt operations prvent the fatality rate from being much higher.

Operative revisions (Table 5) had to be carried out 45 times, in one case even 9 times. This was mainly due to an obliterated catheter.

Table	5.	Hydrocephalus	(n =	43)

Controls	Patients	
1	8	
2	7	
3	1	
4	1	
7	1	
9	1	

Case Reports

After we had started using our computerized tomograph one and a half years ago, we have largely managed without making use of a pneumoencephalogram. The first case (Fig. 1) was a female infant who underwent surgery on the 26th day of life. You see here the pre-operative and postoperative CT. We managed to reduce the extreme form of hydrocephalus almost entirely within a period of 29 days. The same course was observed in nine other patients.

The next picture shows (Fig. 2) the behaviour of hydrocephalus in an infant with postnatal subarachnoid haemorrhage. At the top, we can discern the increasing dilatation of the ventricle which was stopped temporarily by a HAKIM shunt, but which progressed again as a result of meningitis. As demonstrated by the pictures below, the size of the ventricle doubled within 12 days. The infant died.

The last figure demonstrates (Fig. 3) a severe form of an internal hydrocephalus on the left side, which remained unaffected by the shunt operation even 6 weeks afterwards. Moreover, a bifrontal subdural hygroma developed as a surgical complication. Eight days following the necessary valve replacement - see picture in the middle the ventricular system had already become normal. The hygroma now situated above the two hemispheres was largely eliminated by punctures.

Despite our operating technique, which we believe to be fully developed, the clinical success rate including a favourable influence on the skull growth, only amounted to roughly 65% (= 28 cases) in our small group of patients. From this we conclude that for all eligible cases - selection of these is another problem to be dealt with early surgery should be the objective, for we have seen that an effectively functioning shunt is likely to induce even the most severe forms of hydrocephalus to decrease within a few days.

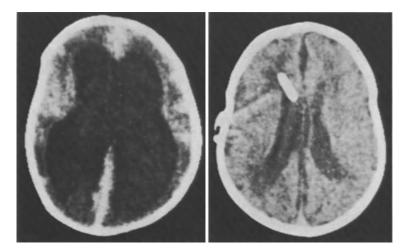


Fig. 1. Computer tomograms of a female infant. Regression of the internal hydrocephalus 29 days after HAKIM shunt operation

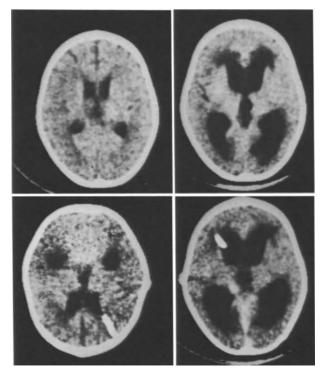


Fig. 2. Computer tomograms of a female infant. *Above:* Increasing enlargement of the ventricle following postnatal subarachnoid haemorrhage. *Below:* Increasing enlargement of the ventricle resulting from postoperative meningitis

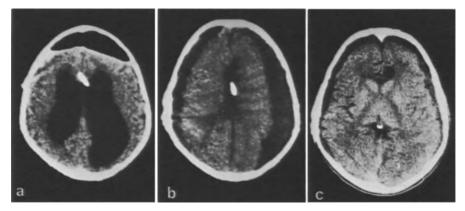


Fig. 3 a-c. Computer tomograms of a 13-year-old girl. a Six weeks after HAKIM shunt, hydrocephalus remained unaffected. Bifrontal subdural hygroma. <u>b</u> Eight days after valve replacement, ventricular system back to normal. Subdural hygroma now situated above the two cerebral hemispheres. <u>c</u> Subdural hygroma was largely removed by puncture

Early and Late Mortality Following Shunt Procedures in Early Infancy P. C. POTTHOFF

Introduction

Several investigations have been carried out on the question of how hydrocephalic children shunted in early infancy *live*, but only little is known about the question of how these children *die* ($\underline{6}$, $\underline{16}$).

Patient Group

Out of a series of 140 shunt insertions or revisions performed at the Neurosurgical Clinic Günzburg/Ulm between January 1971 and June 1978, a consecutive group of 50 children with non-tumoral hydrocephalus shunted in early infancy, below the age of six months, was evaluated (Table 1). Survival has been assessed up to August 1979. Only those patients that died will receive further evaluation here.

Results

<u>1. Mortality</u>. Early mortality up to 6 months after the initial shunt operation was 10% (5 children), and late mortality was 14% (7 children), bringing the global mortality rate to 24%.

<u>2. Causes of Death.</u> The causes of death are listed in Table 2, column 4. Here 4 more cases with primary shunt operation beyond the age of 6 months were added for analysis. These were not included in the calculation of mortality rates. The causes of death in these 16 patients have been categorized and evaluated for their interrelations in Table 3, which forms the basis for the discussion of the causes of death.

There were four main categories of causes of death:

- 1. Primary deficiencies
- 2. Other causes unrelated to hydrocephalus and shunt treatment
- 3. Shunt failure of technical, organic or/and infectious origin
- 4. Iatrogenic factors concerning shortcomings in the intensity or continuity of medical treatment

Whereas the relation of death to one of the causes is usually clear in early mortality, interaction of different causes commonly accounts for late mortality.

Early mortality ensued from primary defects in case 73, with massive occlusive hydrocephalus and postnatal asphyxia on the day of operation, due to cardio-pulmonary insufficiency with multiple heart defects. Also in case 119, with massive occlusive hydrocephalus. This child was born by Caesarean section and had postnatal asphyxia. Death occurred 6 days <u>Table 1.</u> Controlled survival time of 50 non-tumorous hydrocephalic \triangleright children with ventriculo-atrial shunt operations below the age of 6 months

Columns

- 1 Months before shunt
- 2 Patient number and diagnosis
- 3 Survival years after shunt
- 4 Actual condition in August 1979
- 5 Years of survival

Abbreviations

H, hydrocephalus; O, occlusive; C, communicating, perm permangnus; ASP, asphyxia; MMC, myelocele; ECELE, encephalocele; CYST, cystic malformation of the brain; PREM, premature birth; TOXO, toxoplasmosis; AQU, aqueductal obstruction; MEN, meningitis; PIN EP, pineal epidermoid, part partially; HAEM, cerebral haemorrhage; RET, retarded; ICT, seizures; PARASP, paraspasticity; ERETH, erethism; TETRAPAR, tetraparesis; (GOOD)in brackets, time too short to judge final outcome.

Actual conditions

GOOD,	child	lives in family with development according to age;
FAIR,	child	lives in family with satisfactory development, but one
	major	or several minor defects;
RET ,	child	severely retarded, often living in institution

postoperatively, in continuous respiratory insufficiency, probably of central origin, combined with a recent basal meningitis.

Early death on the day of operation and on the third day after operation, due to brain collapse and intracranial hemorrhages, occurred as a consequence of the technical cause of overshunting in cases 111 and 113, in spite of all acute measures to compensate overshunting when the condition was recognized. The types of valve applied have not been used in our clinic following these two incidents in 1971. Such critical overshunting appears to be avoidable by intraoperative testing of valve function prior to implantation, as described by POTTHOFF and HEMMER (10) and HEMMER and POTTHOFF (7) to prevent undershunting. To avoid overshunting (2), valve perfusion capacity during intraoperative testing should probably not exceed four drops per minute at a hydrostatic pressure of 100 mm H₂O in patients with large hydrocephalus. The early death of case 62 is considered to be due to non-recognition as a form of undertreatment, thereby related to a *iatrogenic* factor in combination with a hydrocephalus - and shunt-independent other cause: The child died from an incarcerated inguinal hernia 5 days after shunt insertion, detected too late for a surgical repair.

It will have to be accepted for early mortality that children with multiple primary defects will die despite the most intensive and ingenious neurosurgical care. They are often born premature and endure a difficult postnatal period with asphyxia, brain hemorrhages and possibly central nervous system infection. Causes of early mortality from technical shunt failure and from other treatable unrelated causes should be brought under control, and require continuous attention from the neurosurgeon and his pediatric colleagues (Table 3).

Late mortality is more difficult to evaluate. As to primary defects, patient 103 had a primary polycystic hydrocephalus and Listeria-

CONTROLLED SURVIVAL TIME

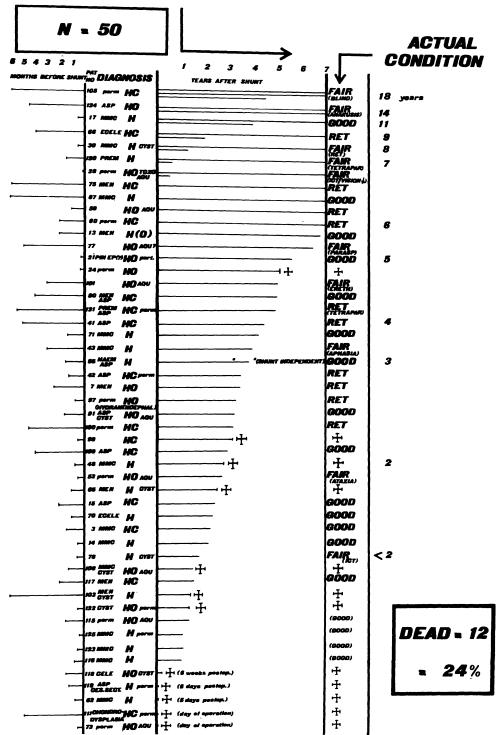
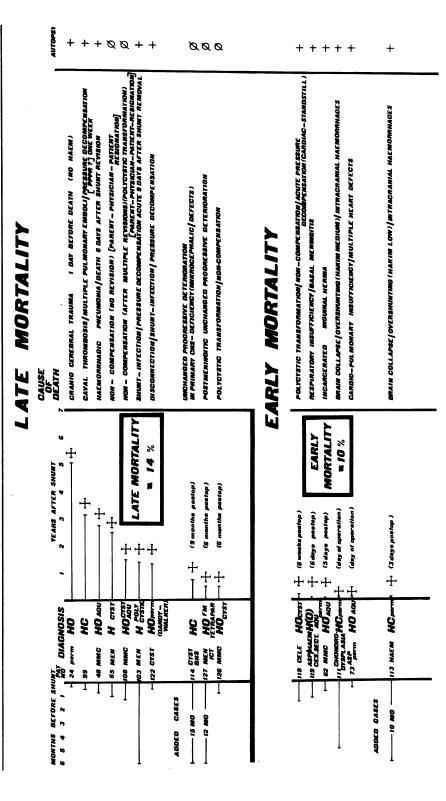


Table 2. Causes of death in late and early mortality (column 4). Other columns and legends as in Table 1



238

meningitis. The child died from non-compensation after shunt removal because of septicaemia.

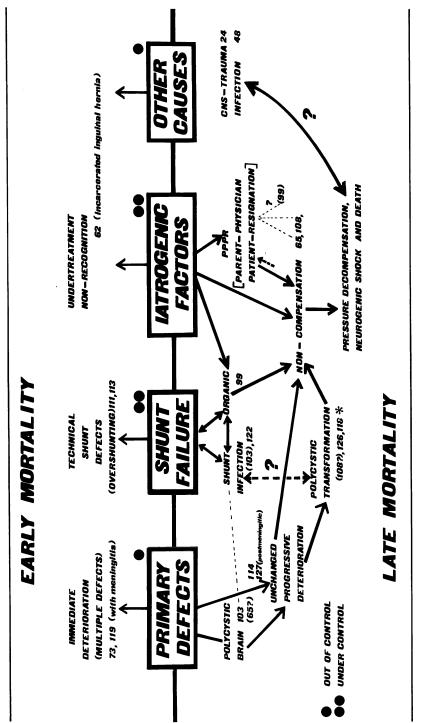
Case 114 was a primary microcephalic child with severe retardation and BNS-epilepsy with a large left temporal cyst, shunted without effect. The child continued to deteriorate and died at home.

Patient 127 suffered from Hemophilus-meningitis, causing epilepsy, severe spasticity and hydrocephalus with occlusion at the Foramen of MAGENDIE. Shunt, shunt revisions and posterior fossa exploration opening the occlusive membrane of the Foramen of MAGENDIE 2 months before death, failed to alter the progressive deterioration.

In a probable combination of primary defects with infection and shunt failure, a remarkable clinical development was observed in cases 118 and 126, and most likely in case 108. We have termed it Polycystic transformation of hydrocephalus (see Figs. 1-4). On the course of patient 118 and 126, both showed certain similarities: Child 118 with an occipital meningocele and child 126 with a thoracic-lumbar myelocele were operated shortly after birth. Both developed hydrocephalus. After shunt insertion ventriculitis of unclear origin with intermittent increase of intracranial pressure (due to partial shunt malfunction?) was observed. CT showed progressive polycystic transformation of the brain. While infection could be controlled, at least for certain periods of time, non-compensation of intracranial pressure rises (compare Table 3) continued in spite of several decompressive measures. Pressure decompensation with complete derangement of CNS-functions ("neurogenic shock") and death ensued (Table 3). The interrelation of these influences is not clear, especially between infection and polycystic transformation. Perhaps intermittent shunt malfunction induced by infectious CSF-changes plays a part in this process. -Case 108 was born with a large myelocele. She developed progressive hydrocephalus with a large left frontal cyst. Despite multiple shunt revisions this condition still showed raised intracranial pressure. Signs of infection were not present. This child, however, was lost to control, her parents becoming restless and dissatisfied, blaming the physicians bitterly that no progress was achieved, taking the child to other hospitals without success. This finally led to resignation of parents as well as physicians as well as to a "resigning" of the patient-child to thrive on any of the therapeutic measures. It was from this child that we derived the term of *Parent-Physician-Patient-Resignation (= PPPR)* to describe this difficult, progressively deteriorating situation that was observed in two more cases. Child 108 died at home.

Patient 65 developed polycystic hydrocephalus after septicaemia and meningitis with Serratia, a shunt was done at the age of 4 weeks, but the parents had been counseled that the situation was hopeless from the medical side, so nothing was done to investigate or treat the child further. He died 2 1/2 years later, an outcome of necessity or of parent-physician-patient-resignation?

PPPR may also have played a role in the outcome of child 99 who died from *organic causes with shunt failure*. This child had warning signs of shunt failure one week prior to death with increasing headache, and it must be taken for some form of PPPR that neither parents nor physician thought it necessary to send the child in for neurosurgical diagnosis and treatment. The child had had a shunt for communicating hydrocephalus. It was known that the cardiac catheter was short at the last check-up 6 months before death, and prophylactic lengthening of this catheter might have avoided the outcome (physician-resignation?).





The child died after one week of progressive headache from non-compensation and acute pressure decompensation at the home hospital.

Shunt failure because of infection led to death in case 122, after multiple revisions. Its death is considered the outcome of prolonged shunt infection with intermittent non-compensation.

Infection also played a role in case 48 that presumably died from pneumonia acquired 4 days after exchange of a blocked ventricular catheter. Though a terminal pressure increase may have caused respiratory insufficiency and so aggravated pneumonia, the clinical diagnosis was pneumonia leading to categorization of this death under other causes.

An unrelated cause of death applied also to child 24 whose death is still not understood and whose fate gave the initial incentive for this examination. G.K. was born on August 24, 1971, by Caesarean section with a head circumference of 51 cm. Contract studies showed a severe, almost hydranencephalic hydrocephalus. A HOLTER shunt (medium pressure) was inserted. Astonishingly, the child began to thrive. In 1974 an exchange of the valve system had to be done because of shunt septicaemia that was controlled by these measures. The child continued to develop, starting to talk and walk, and going shopping with his mother. On September 21, 1976, the patient accidently fell backwards on a stone floor with his occiput. He became unconscious for 15 minutes, awakened again, then had a general seizure and became unconscious again. Examination showed no fracture of the skull, no intracranial hemorrhage, no elevated intra-ventricular pressure with normal CSF. The child remained unconscious, developed respiratory insufficiency with cyanosis, progressed - in spite of all intesive therapy - into pulmonary edema and died 20 hours after the accident. Autopsy failed to prove any signs of intracranial trauma or hemorrhage, not even in serial cuts of the brain stem. The brain showed enlarged lateral ventricles joined together to a monoventricular system, there was aqueductal stenosis and the cerebellum was small and deformed. This death from a clear-cut cranio-cerebral trauma without morphological sequalae remains poorly understood. It is categorized under other causes, but it is possible that infratentorial pressure increase (16, 12) or a functional brain stem disorder related to the pre-existing cerebral malfunction may have been provoked by the trauma, leading to neurogenic shock (13) and death.

Discussion

A total mortality rate of 24% corresponds roughly to figures in the literature (4, 5, 8, 14, 15), showing that every third to fourth hydrocephalic child will die in the course of time. The figures of 3% deterioration plus 2% death due to complications plus 3% death due to other causes, a total of 8% in the collective study of LEEM and MILTZ (lit. see 17) of "Complications following ventriculo-atrial shunts in hydrocephalus" are low, and are in contradiction to figures of JEFFREYS and CHIR, of DE VILLIERS et al. and of VAN VEUSEKOM (lit. see 17) that are higher and more closely resemble the figures presented here.

Causes of death rarely have been listed in detail $(\underline{6}, \underline{15})$ (also compare literature under $(\underline{17})$) but show many influences. It is evident that an attempt to classify causes of death - especially in late mortality - will simplify these influences and be subject of a possibly contro-versial discussion. Primary defects with severe cerebral malfunction,

also in combination with multiple body defects, such as well as hydrocephalus-unrelated causes like trauma or general infection, may lead to death directly or concomitantly with cerebral pressure decompensation out of control of therapy. One of the other main lethal complication groups - shunt failure - may occur from different reasons, such as mechanical in maladapted valves, organic by valve system obstruction or disconnection, and also infectious, the shunt as foreign body being the primary nocitive agent for the development of shunt infection. In a reappraisal of the facts presented here, it appears that infection in general - be it shunt infection, CNS-infection, respiratory or urinary tract infection, or a combination of these and others - is one of the major and possibly lethal hazards for the shunted hydrocephalic child (compare OCCIPINTI, this congress). It is remarkable that in 9 of the here described lethal courses (cases 119, 103, 127, 118, 126, 65, 122, 48, 24) infectious processes of various origins and at various times have been observed. This leads to the conclusion that infection of any kind demands major attention of the physician. Here may be one of the *iatrogenic* factors that can be improved. Others are further improvement in parent couselling, counteracting parent-physician-patient-resignation, and intensification of postoperative surveillance to detect undertreatment.

Conclusions

The hydrocephalic child shunted in early infancy remains a problem patient in many instances. Close supervision, active therapy and excellent parent-physician-relations may diminish these problems. Death - often after many and intensive attempts of therapy - remains tragic in these children. Causes of death have been analysed in 16 patients. A mechanism of non-compensation - Polycystic Transformation of Hydrocephalus - has been described and discussed. Infection has been considered as one of the major hazards for these children. A difficult socio-psychological constellation that may arise around the hydrocephalic child - Parent-Physician-Patient-Resignation - has been named.

- CZINK, A.: Über die Ursachen der Letalität bei Kindern mit ventrikulo-aurikulärer und subduro-aurikulärer Drainage. Med. Fak. Univ. Freiburg 1976 (Diss.)
- FAULHAUER, K., SCHMITZ, P.: Overdrainage phenomena in shunt treated hydrocephalus. Acta Neurochirurgica 45, 89-101 (1978)
- 3. HEMMER, R. (ed.): Erfahrungen mit der modernen operativen Hydrocephalus-Behandlung. Stuttgart: Enke 1964
- HEMMER, R.: Surgical treatment of hydrocephalus: complications, morbidity, developmental prospects. Z. Kinderchir. <u>22</u>, 443-452 (1977)
- 5. HEMMER, R.: Long-term results in the operative treatment of hydrocephalus in children. This volume
- HEMMER, R., CZINK, A.: Die Letalität bei Kindern mit Hydrocephalus und ventrikulo-aurikulärer Drainage. Z. Kinderchir. <u>21</u>, 313-326 (1977)
- 7. HEMMER, R., POTTHOFF, P.C.: Die Ventilinsuffizienz bei ventrikuloaurikulären Drainagen. Z. Kinderchir. 8, 11-16 (1970)

- HEMMER, R., WEISSENFELS, E., HÄNSEL-FRIEDRICH, G., FRIEDRICH, H.: Körperliche und geistige Entwicklung nach Frühoperation der Myelocelen. Neurochirgia <u>20</u>, 7-19 (1977)
- 9. MAWDSLEY, T,, RICKHAM, P.P.: Improvement in the post-operative mortality following early operation for open myelomeningocele. Dev. Med. Child Neurol. Suppl. <u>25</u>, 68-70 (1971)
- POTTHOFF, P.C., HEMMER, R.: Valve insufficiency in ventriculoatrial shunts. Dev. Med. Child Neurol. Suppl. 20, 38-41 (1969)
- 11. POTTHOFF, P.C., HEMMER, R.: The biventricular, the bilateral and the dual-unilateral shunt. Dev. Med. Child Neurol. Suppl. <u>22</u>, <u>12</u> 127-136 (1970)
- 12. SALAH, S., SUNDER-PLASSMANN, M., JELLINGER, K.: Late death in cases of treated internal hydrocephalus caused by encephalo-cranial disproportion. Mod. Probl. Paediat. Vol. 18, pp. 149-151. Basel: Karger 1977
- 13. SCHMIDT, K., POTTHOFF, P.C. (eds.): Neurogener Schock. Stuttgart: Schattauer 1976
- 14. SHARRARD, W.J.W., ZACHARY, R.B., LORBER, J.: Survival and paralysis in open myelomneingocele with special reference to the time of repair of the spinal lesion. Dev. Med. Child Neurol., Suppl. 13, 35-50 (1967)
- 15. SHURTLEFF, D.B., KRONMAL, R., FOLTZ, E.L.: Follow-up comparison of hydrocephalus with and without myelomeningocele. J. Neurosurg. 42, 61-68 (1975)
- 16. SUNDER-PLASSMANN, M., SALAH, S., JELLINGER, K.: Late death in cases of internal hydrocephalus treated during early childhood. Neuropädiatrie 6, 162-174 (1975)
- 17. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.): Treatment of hydrocephalus. In: Advances in neurosurgery, Vol. 6, pp. 1-56. Berlin, Heidelberg, New York: Springer 1978

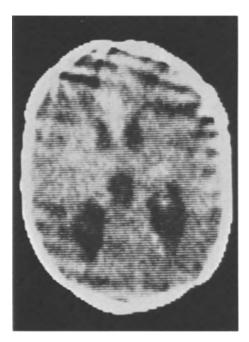


Fig. 1. Almost normal ventricular system (CT Oct.20, 1978)

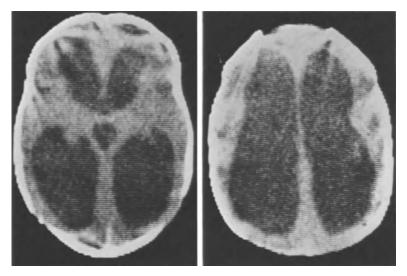


Fig. 2. Rapidly developing hydrocephalus (CT Nov. 10, 1978)

Figs. 1-3. Polycystic transformation of hydrocephalus in case 118

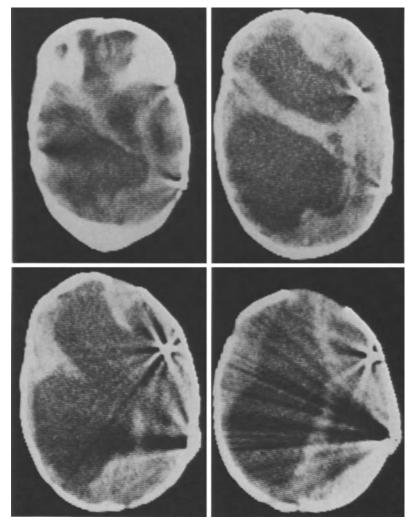


Fig. 3. Polycystic transformation (CT Jan. 1, 1979)

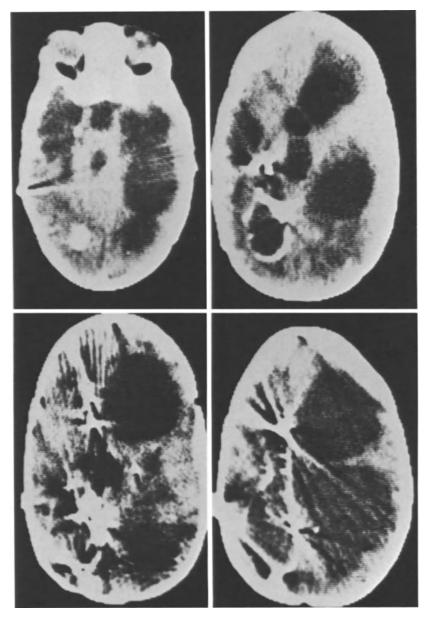


Fig. 4. Polycystic transformation of hydrocephalus, late phase in case 126. Progressive polycystic transformation especially on the right side, after newly inserted left sided shunt (CT Jan. 24, 1979)

Long-Term Follow-Up Studies in Hydrocephalus Patients with Spina Bifida or Encephalocele

E. W. STRAHL, M. DÜCHTING, H. C. NAHSER, and H.-E. NAU

Little information is available about EEG-changes in children suffering from hydrocephalus of different origin. Only some data have been published about the incidence of epileptic seizures in children with myelomeningoceles (MMC) or encephaloceles (EC). For this reason we have analysed the charts of 202 patients treated for these diseases over a long period during hospitalisation or as out-patients.

Patients and Method

Since 1969 we have treated 297 patients with spina bifida in the Department of Neurosurgery of the University of Essen. In 248 patients EEG long-term follow-up investigations have been performed. Forty-six infants under 1 year of age were excluded from this study.

The remaining patients, 105 girls and 97 boys with an average age of 5.8 years, presenting a more or less severe hydrocephalus, were divided into the following groups:

A = MMC, normal EEG, no seizures (n=90) B = MMC, pathological EEG, no seizures (n=46) C = MMC, pathological EEG, seizures (n=45) D = all EC (n = 21)

We have analysed only those EEGs which had been recorded during routine examination and not during acute episodes of shunt dysfunction or other diseases.

Results

Patients with normal EEGs had shunt procedures in 76% (n=68), while in groups B and C only 4% (n=2) were left without cerebrospinal fluid shunt. Six patients of group D had to have shunt operations.

The incidence of the different complications due to CSF-shunts is presented in Fig. 1.

Among 181 patients with MMC we found 45 presenting epileptic seizures. In group D, patients with EC, the number of epileptic seizures was comparatively higher (10 out of 21).

The analysis of pathological EEG alterations in the groups B, C and D revealed that severe general slowing was more frequent in the group of patients suffering from convulsions than in the others (Table 1). Multifocal or generalized spikes corresponding to the clinical symptoms, were found in group C and D almost exclusively. Seven out of 21 children suffering from EC had a normal EEG.

Group (N)	A (90)	в (46)	C (45)	D (21)	Total (202)
EEG (%)					
Normal	100	0	0	38	49
Slowing - Generalized - Focal	0 0	43 20	53 16	24 14	24 9
Spikes - Generalized - Focal	0 0	4 46	36 33	19 29	11 21

Table 1. EEG findings in 202 patients with MMC and EC

Computerized tomography (CT) was evaluated in 171 patients (Table 2). The high percentage of normal size of ventricles in group A corresponds to the good EEG-findings. In the others we found either symmetric or asymmetric enlargement of the ventricles. However in group B the symmetric hydrocephalus was predominant (63%), while asymmetric enlargement was as common as the symmetric one (44%) in group C.

Group (N)	A (75)	B (41)	C (41)	D (14)	Total (171)
CT (%)					
Ventricular siz	e				
- Normal	45	15	12	43	30
 Symmetric dilatation Asymmetric 	35	63	44	29	37
dilatation	20	22	44	65	30
Absence of corpus callosum	7	7	17	21	11
General atrophy	8	7	15	7	11
Porencephaly	0	5	10	7	4
Subdural effusion	0	0	5	0	1

Table 2. CT findings in 171 patients with MMC and EC

In a similar fashion, defects of brain tissue evaluated by CT-scan, such as agenesis of corpus callosum, generalised atrophy of the brain, porencephalic cysts and subdural effusions were more frequent in the groups of patients suffering from seizures.

We did not find any strong relationship between focal EEG changes and the side of VC or local brain lesions seen by CT-scan. In 29 cases the EEG focus was ipsilateral, in 25 cases contralateral to the VC. After several VC-revisions in the same hemisphere the number of EEG foci on the side of operation increased.

Nearly half of the patients with convulsions had their first seizure within the first two years of life, the maximum occurring in the first six months (Fig. 2). The average age at the onset of seizures was 2.07 years.

Twenty-one out of 45 patients with MMC had their first epileptic seizure when acute complications occurred, such as infections of the CSF-shunt systems with ventriculitis or meningitis (9 cases), VC-revisions (7 cases), raised intracranial pressure in cases of other shunt dysfunctions (4 cases) and febrile systemic infections (1 case). Only 2 out of 10 EC-patients with convulsions had such an origin of their first seizure.

Eleven of 55 epileptic patients had one attack only, 14 had repeated attacks in spite of anticonvulsive drugs. In 15 patients we could derive EEGs before the first seizure had occurred. Seven of them were normal, two had focal sharp waves, one showed generalised spikes and waves. In the other 6 children we found general slowing in EEG.

Discussion

Reports concerning EEG follow-up investigations in hydrocephalic children suffering from MMC or EC are very rare. Dealing with the incidence of seizures, HOSKING (3) found epileptic seizures in 26% of 100 children with MMC and hydrocephalus. HUNT et al. (4) observed attacks in 20.5% of the patients with MMC who were investigated. Analysing a mixed collection of 152 patients with spina bifida, BLAAUW (1) found 35 patients with seizures (23%).

In our long-term follow-up study of 181 patients with MMC, 25% of these patients presented with seizures. For the 21 patients with EC this percentage was 48. Although 80% of our 202 patients had a shunt-operation, we found with a pathological EEG only 55% of all patients. The related data given by WÜNSCHE ($\underline{6}$) with 68% and by GRAEBNER ($\underline{2}$) with 74% are much higher. Our findings are also in contrast to those of GRAEBNER ($\underline{2}$) who had marked a lateralisation of EEG alterations corresponding to the site of VC. Our results are in accordance with PAMPLIONE's ($\underline{5}$) who found only a small increase of marked EEG signs ipsilateral to the shunt.

It is remarkable that the group of patients with convulsions and with more or less marked EEG alterations is as large as the group of those who never had any attack despite similar EEG alterations. EEG and CTscan can not exactly predict anything about the prognosis of patients with spina bifida.

Conclusions

- 1. Nearly a quarter of our patients with spina bifida suffers from convulsions.
- The first epileptic seizure can be due to complications of the shunt system, esp. infections and crisis of high intracranial pressure, in half of the cases.
- 3. At intervals remote from complications, routine EEG-investigation of all patients with spina bifida do not give any diagnostic clue in patients without seizures which would be followed by therapeutical consequences. Therefore we think of them as an unnecessary burden for patients and their parents.
- CT-scan is more convenient for diagnosing complications of hydrocephalus and the course of its treatment, but it can not give individual prognostic clues.

5. Only patients with epileptic seizures should be treated with anticonvulsive drugs and be controlled regularly by EEG.

- BLAAUW, G.: Hydrocephalus and epilepsy. In: Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M. (eds.), pp. 37-41. Berlin, Heidelberg, New York: Springer 1978
- GRAEBNER, R.W., CELESIA, G.G.: EEG findings in hydrocephalus and their relation to shunting procedures. Elektroencephalogr. Clin. Neurophysiol. 35, 517-521 (1973)
- HOSKING, G.P.: Fits in hydrocephalic children. Arch. Dis. Child. 49, 633-635 (1974)
- 4. HUNT, G., LEWIN, W., GLEAVE, J., GARDNER, D.: Predictive factors in open myelomeningocele with special reference to sensory level. British Mdecial J. <u>4</u>, 197 (1973)
- PAMPIGLIONE, G., LAWRENCE, K.M.: Electroencephalographic and clinicopathological observations in hydrocephalic children. Arch. Dis. Child. 37, 491-499 (1962)
- WÜNSCHE, W.: Elektroencephalographische Befunde bei mit Hydrocephalus internus nach auriculo-ventrikulärer Ableitung. Kinderärztl. Praxis 10, 455-467 (1970)

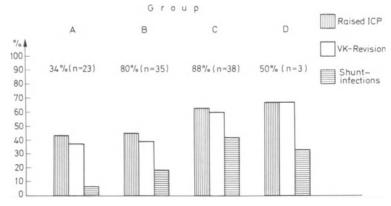


Fig. 1. Incidence of complications in CSF-shunts in 161 patients with MMC (n=181) and EC (n=21)

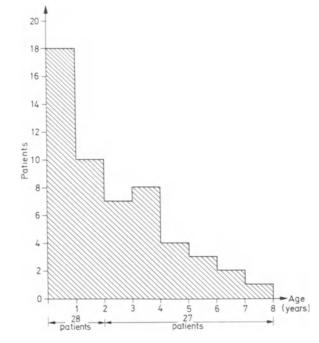


Fig. 2. Age of first seizure in MMC (n=45) and EC (n=10)

Unusual Long-Term Complications Due to Ventriculo-Cardiac Shunt Material

E. ZANDER and R. CAMPICHE

In 1959 we started implanting ventriculo-cardiac shunts in Lausanne. The material we used was manufactured by the HOLTER Company and it is with this material that we have had the most experience. In general this material has given us complete satisfaction so we continue to use it in a great number of our shunt operations.

On three occasions we have encountered an unusual late complication which consisted of a dislocated HOLTER valve which occurred 9, 10 and 11 years after the implantation of the drainage system. In each of the three cases, the complication occurred in children in whom the drainage material had been implanted during the third month of life and in whom the complication due to the dislocation of the valve manifested itself clinically by increased intracranial pressure, nausea and vomiting, papillary oedema and vertigo. In the three cases, the scanner showed a shrunken ventricular system without signs of hydro-cephalus under tension. X-rays of the head and the chest showed a drainage system which seemed in good functioning order with no disconnection. It was only on examining the skull X-rays closely that it became apparent that the valve was too long, that is to say, that the distance between the two metal components was too large, even when taking into consideration the factor of X-ray enlarging (Figs. 1, 2). During the subsequent operation which consisted of implanting a new valve, we were able to remove the dislocated valves without difficulty. One part which was withdrawn contained only the metal component whereas the other portion consisted of the other metal component with the plastic portion of the valve. The thread which attached the plastic segment had completely disappeared (Fig. 3). The dislocation probably occurred because the valve, incrusted in the bone, had been drawn out by the growth of the skull, the two metallic extremities of the valve serving as anchor points. With the new valves which are completely covered with plastic, dislodging is less likely.

In conclusion, if there is a malfunction of a drainage system, one must not forget to examine, by means of the skull X-rays, the dimensions of the valve so as not to overlook an eventual dislocation.

Another inconvenience that we met with concerns the plastic tube of the cardiac catheter. In the beginning the HOLTER Company furnished the catheters impregnated with barium, which gave a smooth surface but which was unfortunately not very opaque to X-rays. Later, drains impregnated with silver which were more opaque to X-rays were introduced. These catheters have a rougher surface and do not seem to be tolerated as well by the surrounding tissues. In fact, we have met with some difficulty in changing or elongating these drains during operations designed to adapt the length of the tube to the growth of the child. These catheters, impregnated in silver, became more easily and more frequently covered by a fibrous, gritty sheath than those impregnated with barium and this prevents any mobilisation of the drain in the vein and it cannot be withdrawn from the vein except by a very strong traction (Fig. 4). We believe therefore, in light of this experience, that the silver impregnated catheters should be reserved for adults, in whom it is very useful to have opaque drains for X-ray controls. If adhesions to the vein develop, it is not very important since thre is little chance that one will be led to change the system. In children, however, it is better to use a barium catheter which causes less adverse reactions and which can be changed without difficulty. Its opacity is sufficient in the child, but this is not the case in the adult.

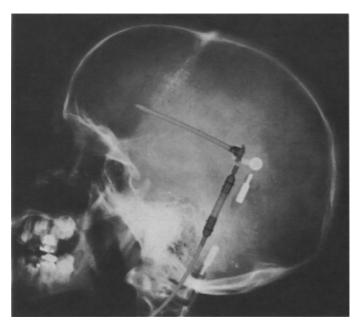


Fig. 1. X-ray of the skull in profile with the HOLTER valve next to its excessively large X-ray image

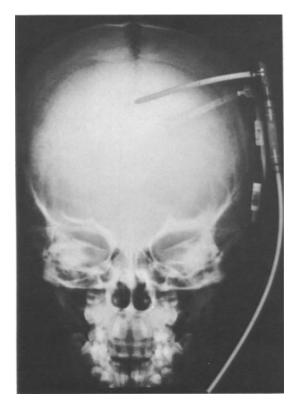


Fig. 2. Idem, frontal view

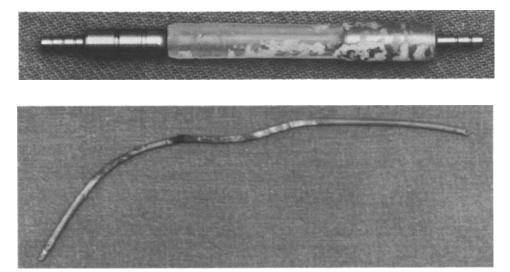


Fig. 3 (*above*):Valve without fixing thread as it was upon removal during the operation Fig. 4 (*below*).Cardiac catheter with its fibro-calcarious sheath

Neurological Impairment Caused by Ventricular Shunt Insertion K. WEIGEL and B. ВÖHM

Computed tomography (CT) is very helpful in demonstrating the presence and extent of substantial brain defects as a consequence of ventricular catheter insertions.

Material and Methods

In the past 12 months 185 patients with a CSF shunting system were checked by computer tomography and neurological examination. These patients had been operated on with CSF shunting systems in a number of hospitals because of a hydrocephalus of varying etiology. In the follow-up examination, particular attention was paid to neurological deficits, that were not caused by the primary disease, but could be solely attributed to catheter placement.

Results

In patients with high frontally implanted ventricular catheters no neurological deficits due to catheter placement were observed. On the other hand considerable neurological defects were found in more than 80% of the patients (Table 1) in whom ventricular catheters had been inserted in the temporo-parietal or occipital region as a result of a catheter placement.

Table 1. Functional deficit due to ventricular catheter insertion

Localization of ventricular catheter	No. of cases	Neurological deficit (No. of cases)
Frontal lobe	150	O (O%)
Central region	7	6 (86%) (hemihypaesthesia, hemiparesis)
Temporo-parietal region	21	19 (90%) (dyspraxia, aphasia, agnosia)
Occipital lobe	7	4 (57%) (visual field defects)

Illustrative Cases

Case 1. A 52-year-old female patient with an occlusion of the foramen of Monroe was operated on using a bioccipital shunt. Five days after the operation, a homonymous lower left quadrant anopia was observed (Fig.1),

as a consequence the ventricular catheter (Fig. 2) which was implanted in the right occipital region.

Case 2. In a 12-year-old boy with a communicating hydrocephalus a moderate paresis of ocular movement was present preoperatively. At a control examination no defect of the visual field was found. After a shunt-revision a further follow-up examination revealed a moderate hemianopia to the left as a result of a partial atrophy of the right occipital pole due to catheter insertion (Fig. 3).

Of special importance are catheter insertions in the temporo-parietal region as we were able to establish in 21 cases.

Case 3. A patient who is now 53 years old, was first treated with a TORKILDSEN drainage because of a post-meningitis occlusive hydrocephalus. However, this drainage had to be removed after three months as it did not work properly. At that time no neurological defects could be established. An atrio-ventricular shunt on the right was carried out, the ventricular catheter was inserted in the temporoparietal region (Fig. 4). In the following months the left handed patient developed a left-right impairment and pronounced dyspraxia, accompanied by moderate bradykinesia. These defects were caused by the catheter insertion in the right parietal region (Area 39, 40).

Case 4. A 12-year-old boy suffered from postmeningitis hydrocephalus. Preoperatively no neurological deficits were observed. Postoperatively a moderate hemiparesis of the left leg and a right-sided hemihypaesthesia occurred which were again caused by the ventricular catheter insertion. The catheter perforated first the left postcentral region, its tip reaching across the midline and then penetrating the right inner capsule (Fig. 5).

Case 5. A 13-year-old boy with a long history of convulsions due to subdural empyemas of both sides with subsequent hydrocephalus had a shunting operation with a catheter placement precentrally on the left (Fig. 6). It perforated the inner capsule which resulted in a paresis of the right leg.

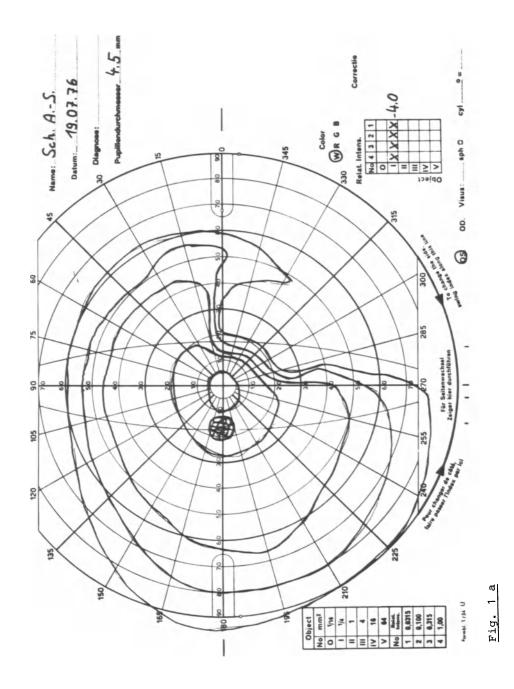
Case 6. This 17-year-old patient suffered from occlusive hydrocephalus caused by occlusion of the aequeductus mesencephali. During a shunting operation the ventricular catheter was inserted through the right central region, which caused a moderate left hemiparesis in the patient who had not displayed any neurological defects before (Fig. 7).

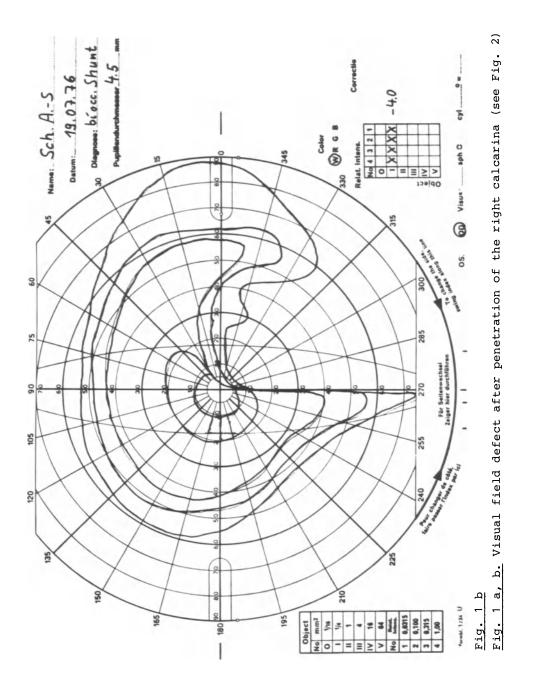
Discussion

These examples demonstrate clearly that ventricular catheter placement may lead to functional impairment. The neurological deficits found at the follow-up examinations and the computer-tomographic controls correlated with catheter placement in functionally important fields of the cortex (<u>1</u>). The placement of the catheter is safe in the frontal lobe and into the frontal horn of the lateral ventricle. The region in front of the coronal suture at a distance of approximately 2,5-3 cm from the midline is a good place for trepanation. In the lateral projection the external auditory canal provides the target point in the anterior-posterior projection parallel to the midline. The length of the catheter measured from the dura is not to exceed 6 cm as has again been described in SEEGER's anatomical atlas (<u>4</u>). Otherwise the shunting system may damage structures such as the capsula interna, the anterior basal ganglia or basal structures such as the optic chiasm. Occipital insertion should only be performed in special cases such as those cases where transventricular removal of space-occupying lesions in the third ventricle is planned. Also in these cases the insertion should be performed at the right distances from the midline. The best location for a trepanation has proved to be 7,5 cm above the external occipital protuberance and 3 cm next to the midline. In this case the catheter is aimed at the middle of the nasion, the length of the catheter measured from the dura may not exceed 5,5 cm.

The result of our follow-up examinations on patients with parietal, temporal or medio-occipital shunt systems has led us to emphasize again the importance of catheter insertion through the frontal lobe into the frontal horn. It has already been described by a number of authors (2, 3, 4, 5). This placement seems to be the only way to avoid damage to functionally important areas of the cortex and possible additional neurological defects.

- GADO, M., HANAWAY, J., FRANK, R.: Functional anatomy of the cerebral cortex by computed tomography. J. of Computer Assisted Tomography <u>3</u> (1), 1-19 (1979)
- HEMMER, R.: Dringliche chirurgische Eingriffe am Gehirn, Rückenmark und Schädel im frühen Säuglingsalter. Stuttgart: Enke 1969
- HEMMER, R.: Surgical treatment of hydrocephalus: complications, mortality, developmental prospects. Z. f. Kinderchir. <u>22</u>, 443-452 (1977)
- SEEGER, W.: Atlas of topographical anatomy of the brain and surrounding structures. Wien, New York: Springer 1978
- 5. YONMANS, J.R.: Surgical neurology. Philadelphia: Saunders Comp. 1973





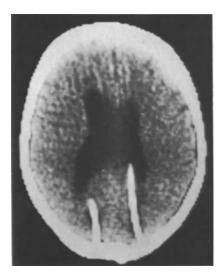


Fig. 2. Placement of bioccipital ventricular catheters penetrating the right calcarina

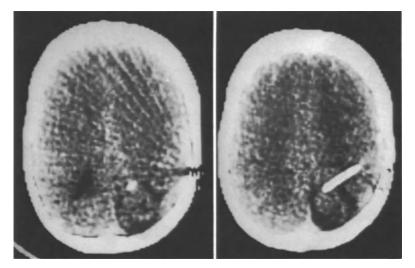


Fig. 3. Partial atrophy of the right occipital lobe due to ventricular catheter insertion

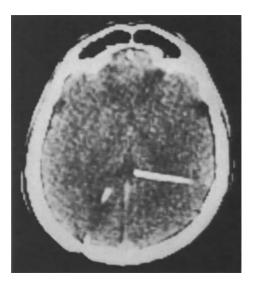


Fig. 4. Catheter insertion through the right posterior parietal region

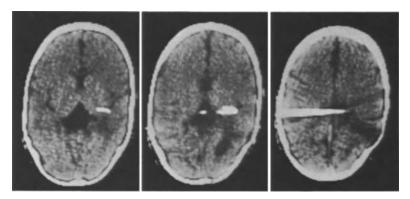


Fig. 5. Ventricular catheter perforating the left postcentral region with the tip across the midline in the right inner capsule

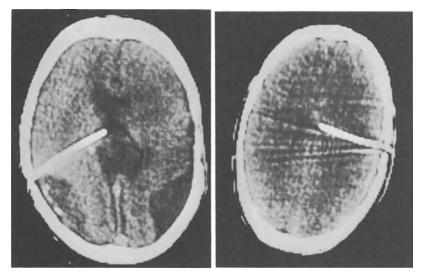


Fig. 6 (*left*). Penetration of the left inner capsule by a ventricular catheter

<u>Fig. 7</u> (right).Ventricular catheter placement through the right central region

Immune Complex Disease Associated with Chronic Infection of Ventriculoatrial Shunts

H. ARNOLD, F. BLÄKER, and B. HILLENKAMP

Since first described by BLACK et al. $(\underline{2})$ in 1965 and by STICKLER and co-workers $(\underline{11})$ in 1968, one of the manifestations of immune complex disease (ICD), namely nephritis, is known to be a special complication of atrial shunts colonized by coagulase negative Staphylococcus albus or - less commonly - by other bacteria, most of which were thought to be nonpathogenic. Hitherto about 40 cases have been reported; 25 of them were reviewed by LABRUNE and his colleagues ($\underline{5}$). We established a few additional constituents of ICD coinciding with nephritis.

There were 25 infections in a series 398 ventriculoatrial (VA) shunts inserted from 1967 until 1978. Seven were first found because of low grade septicemia occurring one-half to two years after operation. Intermittent fever and anemia were found as a rule, often lasting for several months or even years. Hepatosplenomegaly was present in 5 out of 7 children. Without exception urinalysis revealed signs of glomerulonephritis, i.e., mild proteinuria and microhematuria. Occasionally macrohematuria occurred. BUN and creatinine were elevated in 4 out of 7 patients. Juvenile hypertension had the same incidence (Table 1). Only in one child did oliguria point directly to an impairment of kidney function.

<u>Table 1</u>. Symptoms of chronic infection causing immune complex disease

Signs of infection	
Intermittent fever Hepatosplenogemaly Anemia	6/7 5/7 7/7
Signs of nephritis	
Proteinuria/hematuria BUN and creatinine elevation Juvenile hypertension Hypoproteinemia Nephrotic syndrome	7/7 4/7 4/7 2/7 1/7
Signs of immune complex disease	
Hypocomplementemia Nephritis Pulmonary hypertension Arthralgia Anemia	4/4 7/7 3/7 2/7 7/7
	Intermittent fever Hepatosplenogemaly Anemia Signs of nephritis Proteinuria/hematuria BUN and creatinine elevation Juvenile hypertension Hypoproteinemia Nephrotic syndrome Signs of immune complex disease Hypocomplementemia Nephritis Pulmonary hypertension Arthralgia

Besides nephritis other symptoms of ICD appeared, some of which predominated for a time. Dyspnea arising during effort was found to be an effect of pulmonary hypertension as a result of arteries rather than of thrombo-embolism (see below). In 2 children arthralgia was the first sign. Anemia, which was thought to be a result of iron deficiency caused by infection, is to be classified as a component of ICD, too. Hyocomplementemia was detected whenever it was searched for (Table 1). Most distinctly it was demonstrated by the finding of a significant serum C₃ depression.

The incidence of the different manifestations of ICD varies; all combinations seem to be possible. While nephritis and anemia proved to be present in every case, arthritis and pulmonary arteritis developed less frequently. Slight pulmonary arteritis, however, may have been overlooked. Only one child was affected by all manifestations of ICD (Table 2).

chron	ic ventri	culo-atrial shu	int infection		
No. o	f case	Nephritis	Arthritis	Pulmonary arteritis	Anemia
1	К.	+	+	+	+

Table 2. Manifestation of immune complex disease associated with

				arteritis	
1	К.	+	+	+	+
2	S.	+	+	-	+
3	W.	+	-	+	+
4	Ρ.	+	-	-	+
5	н.	+	-	-	+
6	м.	+	-	-	+
7	St.	+	-	+	+
Tota	1	7/7	2/7	3/7	7/7

As a rule the course of the illness was determined by a series of misinterpretations. Recurrent banal infection, rheumatoid arthritis, and anemia were diagnosed and treated accordingly by means of antibiotics, corticoids, and transfusion, respectively. Several times, if knowledge of the disease was lacking, Staphylococcus albus grown from the blood culture was dismissed as a contaminant. Hence the time which elapsed between the first symptoms and actual diagnosis, varied from 5 months to 9 years. One child died from pulmonary hypertension following a diagnostic procedure performed in order to exclude congenital heart disease. Postmortem examination of the lung disclosed medial thickening and intima proliferation with thrombotic appositions thus indicating diffuse pulmonary arteritis. Another child required dialysis for kidney failure. She had a reasonably good recovery after shunt replacement, and renal function normalized. One boy suffering from pulmonary hypertension with severely impaired respiration as well as from nephritis and arthritis was freed of his symptoms following conversion of the atrial to a peritoneal shunt. Another one, on the contrary, developed juvenile hypertension secondary to chronic glomerulonephritis, because a part of an infected disrupted atrial catheter was left in the subclavian vein 5 years ago.

Since the infections were caused by Staphylococcus albus, we replaced the shunts immediately as was proposed by SCHOENBAUM and coworkers $(\underline{10})$. The operation was performed at the earliest three days after anti-

biotic management was started according to bacteriological results. Generally the new device was placed in a different anatomical location. In three cases intraoperative ventricular perfusion was performed from the new to the first ventricular access using a gentamycin dilution. If practicable, a peritoneal shunt was inserted.

Discussion

The diagnosis of shunt-induced ICD can seldom be derived from clinical signs. The delay from first symptoms to diagnosis - as is also reported by others (2, 5, 10, 11) - may be due to both the great variety of symptoms and the generally long interval from shunt insertion to the onset of the disease. Furthermore, the onset may be insidious and even asymptomatic. Apparently, nephritis is an obligatory constituent of the disease. Urinalysis should be done routinely to detect it. However, other manifestations of ICD may predominate by far, pulmonary arteritis being the most conspicuous and most threatening of them. Probably that is why particulars about the kidneys are missing in almost all reports dealing with pulmonary complications. Vice versa, slight ICD of pulmonary vasculature may have taken an unnoticed course, if anemia and nephritis came to the fore.

Whether or not shunt infection may be a condition of thrombo-embolism still is a point of controversy. Like NUGENT et al. (8) and ANDERSON (1) we feel that infection is a major factor in the development of pulmonary thromboembolic complications in shunted hydrocephalics. This view seems to be justified by the findings of FOKES (4), that shunt colonization may exist even though clinical signs of infection are lacking and blood culture is negative.

Hitherto, in all publications (1, 3, 6, 7, 8, 9) multiple emboli originating from the catheter tip were thought to cause pulmonary disease following VA shunting. In all those cases, however, postmortem studies or biopsies were performed which demonstrated arteriolar medial thickening and intima proliferation presumably resulting from ICD.

Cases to demonstrate this are found in several cases of the series of EMERY and HILTON (3), the case of RAO et al. (9), one case of NOBLE and coworkers (7), and at least one case of NUGENT and associates (8). As was emphasized by ATERMAN (6) discussing the case of MCMAHON (6), alterations in the arterioles are not to be expected secondary to pulmonary embolism. Likewise periarteritis such as seen in 8 cases by EMERY and HILTON (3) did not fit to thrombo-embolism; therefore the authors postulated that periarteritis might be an autoimmune reaction of pulmonary vessels to cerebrospinal protein. To us it seems more likely that the immune reaction was elicited by immune complexes derived from bacteria, which had colonized the shunt.

Undoubtedly the vessel wall alterations arising from immune reactions are prone to general thrombosis which may be difficult to differentiate from a postembolic state. Our observations suggest that at least a part of what was supposed to be pulmonary thrombo-embolism may be explained readily by immune complex induced pulmonary arteritis. Pulmonary arteritis is a known manifestation of ICD such as are membranoproliferative glomerulonephritis, immune complex anemia, and polyarthritis. Since it was observed to develop together with other manifestations of ICD just mentioned and to disappear after removal of an infected shunt, a common pathogenesis may be assumed. The only effective therapy of shunt-induced ICD is early and definite removal of the infected shunt. To avoid a recurrence of ICD, the conversion to a peritoneal shunt is to be recommended, for the immune complexes cannot pass the peritoneal barrier. Almost always shunt removal results in normalisation of renal and pulmonary function, and anemia and arthritis disappear. Serum C_3 increase is a reliable indicator of recovery.

Summary

Membranoproliferative glomerulonephritis, pulmonary arteritis, immune complex anemia, and polyarthritis resulting from low grade infection of ventriculoatrial shunts represent a pathogenetic entity. The *shunt induced immune complex disease* should be treated by early and definite shunt removal. The conversion to a peritoneal shunt is to be preferred to the renewal of an atrial one.

- ANDERSON, F.M.: Ventriculo-auriculostomy in treatment of hydrocephalus. J. Neurosurg. 16, 551-557 (1959)
- BLACK, J.A., CHA_LACOMBE, D.N., OCKENDEN, B.G.: Nephrotic syndrome associated with bacteraemia after shunt operations for hydrocephalus. Lancet II, 921-924 (1965)
- EMERY, J.L., HILTON, H.B.: Lung and heart complications of the treatment of hydrocephalus by ventriculoauriculostomy. Surgery <u>50</u>, 309-314 (1961)
- FOKES, E.C.: Occult infections of ventriculatrial shunts. J. Neurosurg. 33, 517-523 (1970)
- 5. LABRUNE, B., LEVY, M., BAUBY, C., GUBLER, M.C., MALLET, R.: Les glomßerulonephrites secondaires aux dérivations ventriculoatriales. Arch. franc. péd. <u>30</u>, 793-797 (1973)
- MCMAHON, D.P., ATERMAN, K.: Pulmonary hypertension due to mulitple emboli. J. Pediatrics 92, 841-845 (1978)
- 7. NOBLE, T.C., LASSMAN, L.P., URQUHART, W., AHERNE, W.A.: Thrombotic and embolic complications of ventriculo-atrial shunts. Develop. Med. Child Neurol. 12, Suppl. 22, 114-122 (1970)
- NUGENT, G.R., LUCAS, R., JUDY, M., BLOOR, B.M., WARDEN, H.: Thrombo-embolic complications of ventriculatrial shunts. J. Neurosurg. <u>24</u>, 34-42 (1966)
- 9. RAO, P.S., MOLTHAN, M.E., LIPOW, H.W.: Cor pulmonale as a complication of ventriculoatrial shunts. J. Neurosurg. 33, 221-225 (1970)
- SCHOENBAUM, S.C., GARDNER, P., SHILLITO, J.: Infections of cerebrospinal fluid shunts: epidemiology, clinical manifestations, and therapy. J. Infect. Dis. 131, 543-552 (1975)
- 11. STICKLER, G.B., SLIM, M.H., BURKE, E.C., HOLLEY, K.E., MILLER, R.H., SEGAR, W.E.: Diffuse glomerulonephritis associated with infected ventriculoatrial shunt. New England J. Med. <u>279</u>, 1077-1082 (1968)

Histological Investigations and Clinical Considerations on Shunt Dysfunctions

V. REINHARDT and H.-E. NAU

Introduction

Complications following shunt procedures have been investigated clinically by many authors $(\underline{4}, \underline{5}, \underline{7}, \underline{9}, \underline{12}, \underline{14})$. According to the large statistical material of nine German neurosurgical clinics, occlusions of these shunts are very common with obstructions of peritoneal catheters occurring in 11% and obstructions of atrial catheters in 22%. These obstructions occurred in cases of atrial catheters in 83%, in cases of peritoneal catheters in 77% during the first year after the primary shunt procedure $(\underline{9}, \underline{14})$.

Although reports in the literature on these the complications are very numerous (2, 8, 10, 11, 13, 15), there are only a few investigations to be found dealing with the histological findings and the mechanisms by which these obstructions develop (1, 2, 3, 6, 13). This, however, seems to be necessary in order to avoid these frequent obstructions. That is why we examined patients with shunt dysfunctions clinically and morphologically.

Patients and Method of Investigation

Among the 38 patients investigated the age ranged from 0,5-50 years (average 7.18 years). Twenty-two of them were male, 16 female. They were investigated clinically by neurological examination, computer tomography, and in 22 cases by RHISA test, plain X-ray and EEG. The revised shunt systems were examined histologically after embedding with paraffin and staining with HE and Elastica-van Gieson.

Results

The reason for the primary shunt procedure was hydrocephalus in patients with lumbar myelomeningoceles in 9 cases, hydrocephalus following meningitis in 9 cases, occlusive hydrocephalus due to tumors in 6 cases, and hydrocephalus due to other and unknown causes in 14 cases. In 6 cases the shunt system was revised completely in 17 cases we had to revise the ventricular catheter, in 10 cases the atrial one, in 2 cases the peritoneal catheter, and in 2 cases the drainage of a cyst. The time interval from the primary shunt operation to the first revision of ventricular catheters ranged from 6 days to 10 years, in cases of atrial catheters this interval was 2 months.

The symptoms leading to clinical admission were vomiting in 31.58%, headache in 26.32%, impaired appetite in 18.42%, and an increase in head circumference in 28.95%.

The head circumference was normal in older patients, but had shown an increase during the last days or weeks before admission in all young children. The large fontanelle bulged in 8 of 10 patients aged up to 1 year. Computer tomography was performed on all patients except 3. In 24 cases (68.57%) an enlargement of the ventricular size was found in comparison to the findings after the primary shunt procedure. In 16 cases plain skull and/or chest X-rays were performed. In 5 cases only, there was a shortening of the atrial catheter, in 7 cases a separation of sutures. The RHISA test demonstrated a peripheral stop in 12 cases, but was found to be normal in 5 cases. In 17 cases EEG investigations were carried out and showed an increase in general slowing in 12% before shunt revision, in 5 cases a larger number of seizure potentials was found.

The morphological cause for the obstructions of the ventricular catheters was tumor tissue in one case, poor organizing and scar tissue as well as foreign body reactions in 27 cases, glial and ependymal tissue in 14 cases, plexus tissue in 7 cases, and brain tissue in 11 cases, fibrin and coagulated blood in 10 cases. Positive morphological findings were observed in a smaller number of atrial catheters (2 cases) although a large number was revised. In these cases we found acute and organized thrombosis inside and outside the catheters. In addition fibrin and protein material were found occasionally. In some cases the surrounding surface tissue (endothelial and mesothelial tissue) grew like a tunnel on the surface of the catheter, entering the lumen through the foramina from the outside. The mechanisms of obstruction could be divided into four categories: (1) obstruction by growth of the surrounding tissue, (2) obstruction of the foramina by invasion of the external tissue or elements of the internal fluid, (3) internal obstruction of the lumen by internal growth of surrounding tissue, (4) displacement of tissue particles into the lumen of the catheter. The mechanisms of obstruction in atrial and peritoneal catheters belonged mainly to categories (1) and (2). Ventricular catheters were different. Here the mechanisms (3) and (4) prevailed.

Discussion

Our results showed that it is possible to explain some of the different mechanisms of obstruction. These can be due to the reaction of the surface tissue in cases of atrial and peritoneal catheters. This in in accordance with findings of BECKER and NULSEN (2) and ALTHER (1). The avoidance of such complications lies in the reduction of the mechanical and chemical irritation of the surface tissue, because this is a stimulus for growth. This is the morphological reason for changing the atrial and peritoneal catheters as early as possible in a growing child. This fact is confirmed by our observations that the majority of lengthened atrial catheters did not show any morphological obstruction.

The mechanism of the obstruction of ventricular catheters is different. Obstructions by the choroid plexus are due to its active mobility, the chemotaxis, and the high amount of vascularisation. It is therefore advisable to avoid close contact with the plexus. Therefore the frontal position of the catheter is more favourable than the occipital one. The posterior parts of the lateral ventricles contain a large mobile tail of the plexus, but in the frontal region, the plexus is fixed to the foramen of Monroe. This explains why there is a much lower percentage of revisions in the frontal position of the catheters. The incidence of obstruction of ventricular catheters by brain tissue was surprising. The tissue core inside the catheter (Figs. 1 and 2) showed mainly large particles of original brain tissue containing pieces of

ventricular surface with ependymal lining or pieces of white matter with myelin and glia including parts of the original vascular supply. Therefore, it has to be accepted that these pieces of original brain tissue enter the lumen through the foramina possibly by the pressure difference between catheter lumen and the surrounding brain tissue or the cerebrospinal fluid. We believe that these pieces of brain tissue usually stay in contact with the surrounding brain tissue by the vas-cular supply. This opinion is supported by time, caliber, and texture of the vessels inside the catheter lumen. This mechanism apparently acts mainly in the moment when the catheter is inserted. As in most cases, the vascular supply is not interrupted, the tissue core reacts very quickly with the organizing reaction and even phagocytosis of blood and lipid. Continuing growth leads to a high pressure of the tissue core inside the catheter which can be demonstrated by the circular texture of the core and the prolaps-like outgrowth of tissue through the pores (Fig. 2). To avoid this, the catheter must be inserted into the frontal horn.

Summary

Thirty-eight patients suffering from shunt dysfunctions were analysed clinically and histologically. We found four types of obstructions:

- 1. Those by growth of the enveloping tissue
- 2. Obstruction of the foramina by invasion of the external tissue or elements of the internal fluid.
- 3. Internal obstruction of the lumen by internal growth of enveloping tissue.
- 4. Displacement of tissue particles into the lumen of the catheter.

The first two possibilities were observed mainly in atrial and peritoneal catheters, whereas the others could be found in ventricular catheters. On the basis of the histological findings we recommand the frontal insertion of the ventricular catheter, a modified implantation of the ventricular catheter, and early lengthening of atrial catheters in the growing child.

- ALTHER, E.: Die Reaktion der Gewebe bei ventrikulo-kardialer Ableitung des Liquors durch Ventilsysteme. Mschr. Kinderheilkunde 118, 585-591 (1970)
- BECKER, D.P., NULSEN, F.E.: Control of hydrocephalus by valveregulated venous shunt: avoidance of complications in prolonged shunt maintenance. Neurosurg. 28, 215-226 (1968)
- EMERY, J.L.: Fibrin and thrombosis in the central nervous system in children with particular reference to congenital hydrocephalus. J. clin. Path. <u>17</u>, 348-352 (1964)
- 4. GROTE, E., ZIERSKI, J., KLINGER, M., GROHMANN, G., MARKAKIS, E.: Complications following ventriculo-cisternal shunts. Advances in neurosurgery, Vol. 6, pp. 11-17. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.). Berlin, Heidelberg, New York: Springer 1978
- 5. HEMMER, R.: Therapie des Hydrocephalus. 75. Tagung der Dtsch. Ges. f. Kinderheilkunde. 4.-6. September 1978, Freiburg, Abstracta
- ILZUKA, J.: Zerebraldiagnostik im Kindesalter mittels Stereoencephaloskopie. Z. Kinderchir. 10, 2-13 (1971)

- 7. JEFFREYS, R.V., CHIR, M.: The complications of ventriculo-atrial shunting in hydrocephalus. Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 17-23. Berlin, Heidelberg, New York: Springer 1978
- JENSEN, H.P., AMADOR, L.V.: Ventrikulo-aurikulostomie zur Behandlung des Hydrocephalus. Neurochirurgia 4, 99-113 (1961)
- LEEM, W., MILTZ, H.: Complications following ventriculo-atrial shunts in hydrocephalus. Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 1-5. Berlin, Heidelberg, New York: Springer 1978
- LORENZ, R.: Komplikationen nach TORKILDSEN-Drainage. Acta Neurochir. 14, 246-253 (1966)
- SCARFF, J.E.: Treatment of hydrocephalus: an historical and critical review. J. Neurol. Neurosurg. Psychiat. <u>26</u>, 1-26 (1963)
- 12. SHURTLEFF, D.B., FOLTZ, E.L.: Clinical observations on twenty hydrocephalic children subjected to ventriculo-atrial shunt: a preliminary report. A.M.A.J. dis. Child. 98, 467-470 (1959)
- 13. SPERLING, D.R., PATRICK, J.R., ANDERSON, F.M., FYLER, D.C.: Cor pulmonale secundary to centriculo-auriculostomy. A.M.A.J. Dis. Child. <u>107</u>, 308-315 (1964)
- 14. STRAHL, E.W., LIESEGANG, J., ROOSEN, K.: Complications following ventriculo-peritoneal shunts. Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 7-9. Berlin, Heidelberg, New York: Springer 1978
- STRENGER, L.: Complications of ventriculovenous shunts. J. Neurosurg. 20, 219-224 (1963)

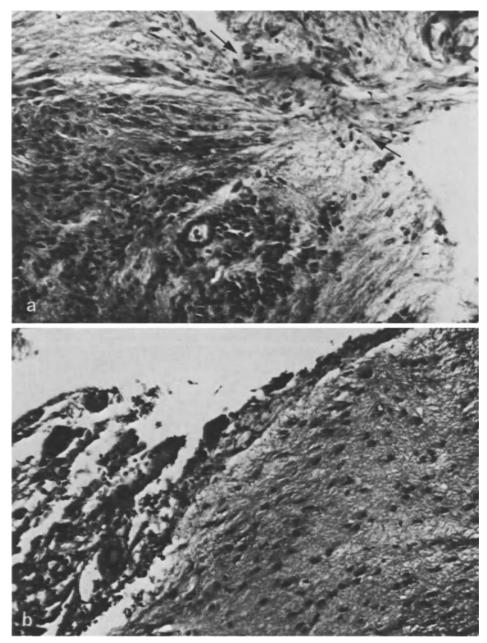


Fig. 1 a, b. H.E., 225 x. Part of the obstructing tissue core with ependymal cells and fibrillary astrocytes; the tissue protruding through one of the pores of the catheter \rightarrow (a). Foreign-body giant cells at the border of the tissue core (b)

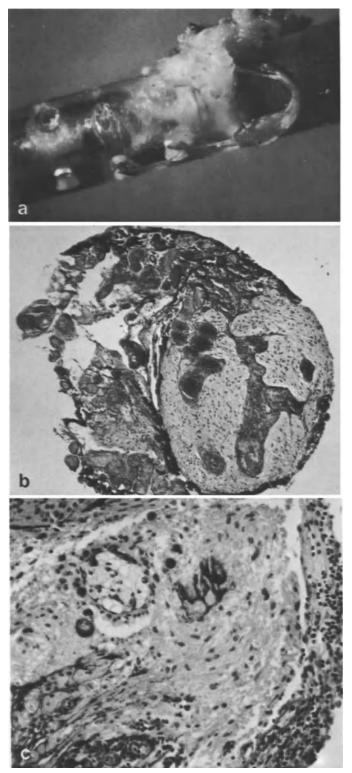


Fig.2 a-c. Macrophotograph of the ventricular catheter with obstructing tissue protruding out of the pores (a). Tissue core of the catheter with glial and marginal collagenous scar tissue and many blood vessels (b). Marginal organising tissue, blood vessels and philidphagocytosis within a predominantly glial core (c). a 15x; b EvG, 90 x; c EvG, 225 x

Free Topics

Transoral-Transpalatine-Transclival Approach to Aneurysms of the Vertebral and Basilar Artery

H. W. PIA and R. LORENZ

The transoral approach to the clivus and the upper cervical spine in cases of trauma, tumours and malformations has become the procedure of choice and means an important contribution to these lesions.

The transoral transclival intradural approach to aneurysms of the vertebro-basilar junction and the lower half of the basilar artery (2, 3, 4, 5, 6, 7) seems to be a very good solution. This approach aims directly at the artery and aneurysm. Manipulation with the cranial nerves and brainstem is not necessary. The medial localisation and ventral projection of the aneurysms of vertebro-basilar junction, upper posterior inferior cerebellar artery and anterior inferior for this "direct" approach.

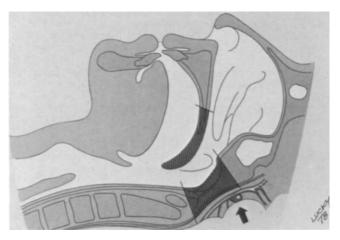
The problem is the danger of CSF-fistula because of the impossibility of a proper closure of the dura. One case with fistula, meningitis and death led DRAKE (1) to abandon the operation. He wrote: "Since then it has been learned that all aneurysms overlying the lower third of the clivus, even in the midline, can be exposed through the posterior fossa via an unilateral suboccipital opening". On the other hand he stresses the possibility of injury from below to the 10th and 11th nerves and from above, in a single or combined operation to the 5th, 6th, 7th and 8th nerves. Since none of us has such an experience as DRAKE and is able to perform these operations frequently we agree with SANO (5, 6) that the transclival approach has many advantages, especially for the less experienced "aneurysm neurosurgeon", that we do not necessarily have to share his fear.

Transseptal-transsphenoidal approach to the hypophysis has become a routine, and there is no reason why there should not be routine indications for transclival operation.

In our case of a lower basilar aneurysm with duplication of the basilar artery, subtemporal transtentorial approach 10 years ago was unsuccessful. A second bleeding indicated the closure by the transclival approach. The movie demonstrates the different steps of the operation (Fig. 1) including the pre- and postoperative angiograms. It is essential to have adequate instruments especially a spreader with special blades, because the length and angle of the blades have to be changed while exposing and drilling away the hard palate, the mucosa of the pharynx and the clivus. The defect of the dura was closed by a piece of lyophilised dura and muscle and some sutures of the mucosa. An open lumbar CSF drainage was used for one week. The course was uneventful.

We believe that, in spite of our limited experience, the transclival intradural access to special aneurysms has advantages and should be performed.

- 1. DRAKE, C.G.: Treatment of aneurysms of the posterior cranial fossa. Progr. Neurol. Surg. 9, pp. 122.194. Basel: Karger 1978
- FOX, J.L.: Obliteration of midline vertebral artery aneurysm via basilar craniectomy. J. Neurosurg. <u>26</u>, 406-412 (1967)
- 3. PIA, H.W.: Classification and treatment of aneurysms of the vertebro-basilar system. Neurol. Med. Chir. (Tokyo) 19, 575-594 (1979)
- 4. PIA, H.W.: The transoral transclival approach to basilar aneurysms. In: Cerebral aneurysms. Advances in Diagnosis and Therapy. PIA, H.W., LANGMAID, C., ZIERSKI, J. (eds.). Berlin, Heidelberg, New York: Springer 1979
- 5. SANO, K., JIMBO, M., SAITO, I.: Vertebro basilar aneurysms with special reference to the transpharyngeal transclival approach to the basilar artery aneurysms. Brain and Nerve (Jap.) <u>18</u>, 1197-1203 (1966)
- 6. SANO, K., SAITO, I.: Transoral transclival approach to aneurysms at the vertebro-basilar junction. In: Cerebral Aneurysms. Advances in Diagnosis and Therapy. PIA, H.W., LANGMAID, C., ZIERSKI, J. (eds.). Berlin, Heidelberg, New York: Springer 1979
- WISSINGER, J.P., DANOFF, D., WISIOL, E.S., FRENCH, L.A.: Repair of an aneurysm of the basilar artery by a transclival approach. Case report. J. Neurosurg. 26, 417-419 (1967)





Investigations on the Pathogenesis of Syringomyelia B. WILLIAMS

Introduction

GARDNER $(\underline{2})$ has shown the association between syringomyelia and abnormalities at the foramen magnum and improvement after craniovertebral decompression, not only for symptoms of the craniovertebral malformation but also those of the associated syringomyelia.

In a series of 118 patients (10) possible causes included spina bifida ($\underline{6}$), meningitis ($\underline{3}$) and cerebral tumour ($\underline{2}$). Other possible factors were severe head trauma in 21 cases and history of difficult birth in 62. Head trauma has been suggested as a causative factor by earlier authors including HERTEL and HILD ($\underline{3}$) but the relevance of such an incident is difficult to determine. Head injury at any age may through cerebral anoxia and swelling, sub-arachnoid bleeding, hydrocephalus or basal arachnoiditis cause herniation of the cerebellum. If injury takes place during birth moulding of the skull and overlapping of bones of the vault permit compression of the brain. Invagination or occipital osteodiastasis around the foramen magnum may occur because the expulsive force of the uterus is applied to the base of the skull by the cervical spine. The finding that 62 out of the 118 patients reported some factor predisposing to injury in their birth was of interest. Results of this enquiry and comparison with 200 controls have been published (10).

CHIARI (1) suggested that the hind brain hernia in both the CHIARI type I deformity and the CHIARI type II was due to hydrocephalus, although it is now known that the CHIARI type I malformation may commonly occur without it. In a recent series of 80 patients with syringomyelia in whom ventricular size was assessed by WEST and WILLIAMS (6) 54 were normal and only 3 showed severe hydrocephalus. Symptoms of high pressure in the head are rare in syringomyelia, and it seems that pressure differences across the foramen magnum may be intermittent (7).

In a study of CHIARI type II malformation it has been noted $(\underline{8})$ that the important differences in pressure were those in which the intracranial pressure was higher than the intraspinal; this has been called craniospinal dissociation. Pressures in the tissues are important but cerebrospinal fluid (CSF) pressures were more accessible to measurement, therefore only CSF pressure was considered.

Four types of pressure recordings were found:

 Normal: In these the baseline pressures were equal in the head and the spine (baseline pressures refers to the average pressure ignoring the pulsations due to heart beat, coughing, crying and so on).

- 2. Baselines equal with valvular effect: In these cases the head pressure rose higher than the spinal pressure but equalised at rest after a few seconds.
- 3. Baseline dissociation with valvular effect: pressure differences between the head and spine were present at rest and were aggravated by exertion.
- 4. Total dissociation: In this state, always associated with rapidly progressive hydrocephalus, the pressure events in the head were separate from those in the spine, indicating a watertight blockage at the foramen magnum.

The present results concern a study of adults only.

Materials

Over 100 adult recordings of intracranial and intraspinal pressure measurements gave adequate normal values. Also included were 43 patients with proven syringomyelia in whom 83 recordings have been done of these 38 patients have had preoperative studies and 36 postoperative (31 both pre- and postoperative).

Methods

Ventricular puncture was made through the right frontal twist drill hole $(\underline{4})$ using an 8 cm JEFFERSON cannula. Pressure recordings were made from CSF in the sitting position (Fig. 1) and from a mouthpiece.

The pressures were recorded with the patients at rest, during QUECKENSTEDT's manoeuvre, during coughing and during a modified VALSALVA manoeuvre produced by blowing into the mouthpiece.

Results

In 32 cases the results were normal. In the majority of abnormal cases after CSF had been forced from the spine into the head by the epidural veins, it was delayed by valvular action at the foramen magnum and then slowly returned to the "baseline equal" state. This was best seen after two types of event.

<u>Coughing:</u> After a sharp rise in abdominal pressure such as a cough the pressure rises faster, higher and sooner in the spine than in the head and during recovery the CSF drops back rapidly from the head into the spine ($\underline{9}$). In cases with a valve the CSF pressure did not equalise rapidly and dissociation was visible after a single cough and often more marked after a sequence (Fig. 4).

<u>Straining</u>: After a VALSALVA's manoeuvre produced by blowing (Figs. 2, 3) there is usually an increase in cardiac output and blood pressure with a bradycardia (Post Valsalva Rebound) and if this occurred when the CHIARI malformation was tightly impacted then the rise in CSF pressure caused by the increased pulsation in the cerebral vascular bed might be only in the head. This exacerbated the dissociation and pressure differences over 30 mm Hg were seen. This post VALSALVA rebound (PVR) was frequently associated with pounding headache for from 2 seconds to 30 seconds after straining a characteristic complaint accompanying CHIARI type I malformation.

Of 31 cases who had recordings before and after cranio-vertebral decompression the majority showed improvement in both the craniospinal pressure dissociation and clinical state after operation. There was not perfect correlation between the clinical progress and the pressure tests (Table 1). In particular, 9 of the cases with proven syringomyelia did not have dissociation although the anatomical form was suitable to produce valvular action in all of them.

Pressure state	Clinical state				
	Improved by operation	Not improved by operation			
Pressure dissociation tests normal before and after operation	7	2			
Full correction of pressure dissociation	10	1			
Improvement of pressure dissociation	7	1			
Dissociation worse or un- changed	0	3			

 $\underline{Table \ 1}.$ Numbers of cases with pressures assessed before and after craniovertebral decompression

Out of six adult patients who had pressure recordings after valved ventriculoatrial shunts none had correction of craniospinal pressure dissociation although all have shown some improvement in the lowering of the baseline pressures and lessening of the dissociation during the PVR.

Discussion

The morphology of the CHIARY type I malformation is always compatible with craniospinal dissociation. Clearly, such dissociation is present with lesions such as head injury, birth injury or posterior fossa tumours which cause a hind brain hernia. In chronic hind brain herniation however progression is very slow and a CHIARI malformation, once caused, can itself cause further pressure differences thus exacerbating its own progression. This study may thus have demonstrated the mechanism by which slight degrees of craniospinal pressure dissociation repeatedly occurring over many years may eventially cause marked deformities which are usually thought of as "congenital".

The failure of a proportion of patients with CHIARI type I malformation to show dissociation is not surprising. The testing itself is limited, energetic actions such as lifting cannot be done as shown in Fig. 1. Many of these syndromes have long periods of remission. Cough headache may be present for years and then suddenly stop. Thus it seems probable that some syringomyelia patients may not have been impacting their CHIARI malformations at the time of testing, even on maximal exertion.

It seems that craniospinal pressure dissociation secondary to the CHIARI malformation is common in syringomyelia, this does not however prove the causative mechanisms of the syrinx. Perhaps the most serious objection to the idea that craniospinal pressure dissociation causes filling of a syrinx along a communication from the floor of the fourth ventricle lies in the difficulty in demonstrating such a communication. WEST and WILLIAMS (6) in a recent investigation of 61 cases of syringomyelia by ventriculography were able to show it in only 7 patients. This supports the contention of RICE-EDWARDS (5), that a significant communication is rare. Possibly at the time that patients present a communication which may have been quite sizeable while the syrinx was developing could have been narrowed down, may be by external pressure from the tonsils at the site of the CHIARI malformation.

The nature of improvement in syringomyelia patients after craniovertebral decompression together with the dangers have been discussed (11).

The demonstration of craniospinal pressure dissociation has practical importance. While patients are impacting their hind brains in the foramen magnum then they are lowering the fourth ventricle, distorting its floor and compromising its outlets, causing descent of the stem and angulating the lower cranial nerves, narrowing the subarachnoid spaces around the cisterna magna and possibly filling a syrinx. Thus even in patients who present with a chronic complaint surgical treatment may be advocated more confidently when there is craniospinal pressure dissociation.

Clearly many patients have intermittent craniospinal pressure dissociation for years without danger. It seems likely that many patients with CHIARI malformations may have such dissociation all their lives. Thus it is not reasonable to regard craniospinal pressure dissociation as always being an indication for operation. If the patient has hydrocephalus with a valved shunt in the first instance and to observe progress even though a valved shunt does not correct craniospinal pressure dissociation.

Testing the pressures may confirm the success of operative treatment or indicate the necessity for further observation and possibly reinvestigation.

Conclusion

The foramen magnum abnormalities in syringomyelia are compatible with transient higher pressure in the head than in the spine. It is suggested that birth injury may be a cause of the CHIARI type I malformation. Transitory pressure difference has been called craniospinal pressure dissociation and clinical measurements have confirmed pressure differences after coughing and straining. Craniospinal pressure dissociation may also be found with cough headache and features such as attacks of unconsciousness after exertion, ataxia and oscillopsia. Its demonstration may help in assessing decompressive operations at the foramen magnum

- 1. CHIARI, H.: Über Veränderungen des Kleinhirns infolge von Hydrocephalie des Großhirns. Dtsch. med. Wschr. 17, 1172-1175 (1891)
- GARDNER, W.J.: The dysraphic states from syringomyelia to anencephaly. Amsterdam: Excerpta Medica 1973

- HERTEL, G., HILD, J.: Syringomyelia und trauma. Arch. Psychiat. Nervenkr. 216, 393-408 (1972)
- KAUFMANN, G.E., CLARK, K.: Emergency frontal twist drill ventriculostomy. J. Neurosurg. <u>33</u>, 226-227 (1970)
- RICE-EDWARDS, J.M.: A pathological study of syringomyelia. J. Neurol. Neurosurg. Psychiat. <u>40</u>, 198-199 (1977)
- WEST, R.J., WILLIAMS, B.: Radiographic studies of the Ventricles in syringomyelia. (In press)
- 7. WILLIAMS, B.: Current concepts of syringomyelia. Br. J. Hosp. Med. <u>4</u>, 331-342 (1970)
- WILLIAMS, B.: Cerebrospinal fluid pressure gradients in spina bifida cystica, with special reference to the Arnold-Chiari malformation and aqueductal stenosis. Dev. Med. Child Neurol. Supp. <u>35</u>, 138-150 (1975)
- 9. WILLIAMS, B.: Cerebrospinal fluid pressure changes in response to coughing. Brain <u>99</u>, 331-346 (1976)
- 10. WILLIAMS, B.: On the pathogenesis of the Chiari malformation. Z. Kinderchir. <u>22</u>, 533-553 (1977)
- 11. WILLIAMS, B.: A critical appraisal of posterior fossa surgery for communicating syringomyelia. Brain 101, 223-250 (1978)



Fig. 1. Disposition of patient with "minitransducers" on a ventricular cannula, a lumbar puncture needle, a venous catheter into the subclavian vein and attached to a mouth piece and anaeroid monometer. The method of referring all the fluid pressures to a common zero level is indicated

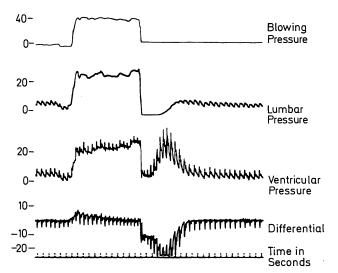


Fig. 2. Recordings from a preoperative patient with syringomyelia and syringobulbia coming on 36 years after severe birth injuries. The recordings are read left to right, vertical scale is pressure in millimetres of mercury. The "blowing" trace on top shows when the patient blew a "square wave" of 40 mm Hg for 12 s. During this period the CSF pressure rose in the lumbar region smartly by about 20 mm Hg and rose somewhat less rapidly in the ventricles. When the patient suddenly stopped blowing the lumbar pressure fell to 10 mm Hg lower than the ventricular. The seconds after stopping the blow the Post Valsavla Rebound (PVR) started and produced a craniospinal pressure dissociation reaching 35 mm Hg. The pressures in the head and spine had equalised 9 s after the patient stopped blowing

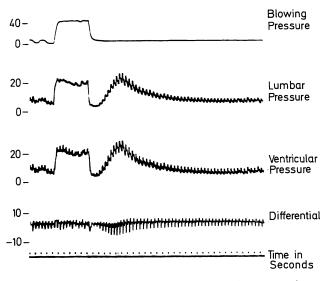


Fig. 3. Postoperative pressure recordings in the same patient as in Fig. 2, after craniovertebral decompression for CHIARI type I malformation; the arachnoid was not breached and the tonsils not disturbed. The "square wave" produced by blowing and also the PVR were almost equal in the spine and in the ventricles. Compare the differential trace on the bottom of the recording in Fig. 2 with that in Fig. 3. That in Fig. 3 is almost normal showing only a slight downward bowing of the baseline pressure at the height of the PVR which indicates a minimal degree of encroachment upon the CSF pathways by the persistent malformation

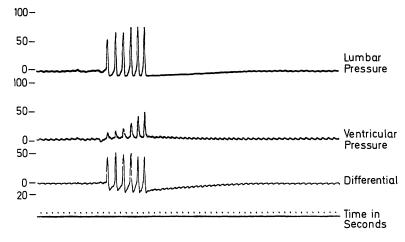


Fig. 4. Preoperative recording from a patient with syringomyelia and cerebellar ectopia taken during 6 successive coughs. Note that progressively more CSF went through to the head with the 6 coughs and that the baseline pressures were thereby forced progressively further apart. Note that it took about 15-18 s for the baselines to return to equal after the last cough. This phenomenon is cough summation of craniospinal pressure dissociation, it is characteristic of severe deformity and is easily corrected by operation

Technique and Results of the Cervical Discectomy J. GILSBACH and H. R. EGGERT

Stimulated by the positive reports on the ventral cervical disc operation without fusion (1, 2, 3, 5) we perform pure discectomy since 1977, combined with the removal of the dorsal spurs.

In 44 patients (31 male/13 female, aged 31-76 years) the pathology was a hard disc or spondylosis in 25 cases and a soft disc in 19 cases. The duration of symptoms was more than one year in 53% of patients, and from four weeks to one year in 45%. Only one had a history shorter than four weeks. The main symptom was radicular pain in 27 patients, often together with minor neurological deficits, and in marked neurological deficits 17 cases (7 medullar/10 radicular) with or without pain.

The diagnostic procedure consisted in least one contrast investigation, namely myelography or discography, often both (21x). Myography was additionally carried out in 28 patients. Often one single affected level could not be defined neither clinically, nor electromyographically, or by contrast investigation. In these cases of unclear findings we operated on the most impressive level(s) (Table 1).

Single			26
	3/4	1	
	4/5 5/6	14	
	6/7	8	
	7/1	2	
Two			15
	3/4 + 4/5	1	
	4/5 + 5/6 5/6 + 6/7	6 6	
	6/7 + 7/1	2	
Three			2
Infec	3/4 + 4/5 + 5/6	1	2
	4/5 + 5/6 + 6/7	1	
Four			1
Four			1
	3/4 + 4/5 + 5/6 + 6/7		

Table 1. Operated levels in 44 patients

We use the CLOWARD approach and the especially designed instruments. The correct level is controlled by a lateral fluoroscopy at least once. The procedure on the spine is performed under the surgical microscope: the anterior longitudinal ligament or the spurs are opened 1.5 cm wide and after partial removal of the disc the interspace is gently spread up to 3-4 mm by a single intervertebral spreader. No attempt is made to remove the upper or lower plate of the vertebra and the latero-ventral portions of the disc. In hard discs or spondylotic spurs the space-occupying parts, including the processus uncinati, are drilled away but the posterior longitudinal ligament is left intact. It is resected only if there are visible defects.

Postoperatively we use no supporting or immobilizing measures.

Follow-up evaluation in 41 patients, mean 11 months (2-32) (Table 2):

Table 2

Main symptom - Radicular	deficit	(n=9)
Markedly improved Improved Unchanged	33% 55% 11%	
Main symptom - Myelopathy	Y	(n=6)
Improved	67%	
Unchanged	338	
Main symptom - Radicular	pain	(n=26)
Relieved	278	
Markedly improved	38%	
Improved	198	
Unchanged	16%	

Clinical evaluation of the operative result is problematic because of the underlying degenerative process. We restrict our evaluation to the effect on the main symptoms or signs which led to operation. The radicular pain and the usually accompanying minor neurological deficits are both greatly improved whereas major neurological deficits (medullar and/or radicular) - often without pain - are not as well influenced by an operative therapy. More or less pronounced local complaints were reported postoperatively by 85%, 15% showed no local complaints (Table 3).

Kyphosis		No. of patients
5 ⁰	63%	24
5 ⁰ -15 ⁰	32%	12
15 ⁰	5%	2

Table 3. Early postoperative findings (n=38)

Postoperative X-ray check-up revealed angular kyphosis of $5^{\circ}-6^{\circ}$ on average. We found no marked difference between one- or multi-level operations (5° in 1 level operation, 5.6° in 2 level operation). Complications referable to discectomy itself were not observed in the group of 44 patients.

Summarizing, we confirm the positive aspects of the cervical disc operation without fusion: the procedure is quick, safe, uncomplicated, can be used for more than one level without problems, does not pose the problems of obtaining and implanting the bone graft, and has the expected effects on the radicular pain and neurological deficit. The results are as good as after fusion procedures. Instability was not observed postoperatively; normally we saw a fusion with the effect as reported in literature (3, 5). Whether the local complaints often reported are attributable to the kyphosis or to the subluxation (4)of the corresponding intervertebral joints and could be better influenced by another operative method, is doubtful in the presence of a degenerative process of the cervical spine.

- HANKINSON, H.L., WILSON, Ch.B.: Use of the operating microscope in anterior cervical discectomy without fusion. J. Neurosurg. <u>43</u>, 452-456 (1975)
- MARTINS, A.N.: Anterior cervical discectomy with and without interbody bone graft. J. Neurosurg. <u>44</u>, 290-295 (1976)
- MURPHY, M.G., GADO, M.: Anterior cervical discectomy without interbody bone graft. J. Neurosurg. <u>37</u>, 71-74 (1972)
- SUNDER-PLASSMANN, M.: Kann die vordere Halsbandscheibenoperation ohne Verblockung als Standardmethode empfohlen werden? Wiener Med. Woschr. Suppl. <u>20</u>, 3-11 (5/1974)
- WILSON, D.H., CAMPBELL, D.D.: Anterior cervical discectomy without bone graft. J. Neurosurg. <u>47</u>, 551-555 (1977)

Contribution to Ventral Microsurgical Foraminotomy in Case of Cervical Nerve Root Compression

H. BAUMANN, M. SAMII, and K. VON WILD

We report on 40 patients operated on because of a cervical radiculomyelopathy since October 1977. Surgical intervention was indicated on account of definite radicular deficit with or without spinal syndromes. In 90% of the cases, the segmental clinical findings were verified by electromyography.

As a rule plain X-rays of the cervical spine in the region of the affected segments showed reduction of the intervertebral space and osteochondrotic spurs with narrowing of the intervertebral foramen. In more than 90%, cervical myelography with AMIPAQUE confirmed the clinical finding of root compression and/or narrowing of the spinal canal by osteochondrotic spurs, touching the spinal cord.

In less than 10% the myelogram showed no pathological findings despite of definite clinical symptoms and nerve root compression confirmed at surgery (Fig. 1).

Surgical procedure consists in a ventral discectomy and bilateral ventral opening of the intervertebral foramen. The foraminotomy is performed under the microscope, using a diamond air drill. Thus, an optimal ventral opening of the foramen, by most careful treatment of the nerve root, will be possible. The dorsal spurs, if necessary, can also be removed.

As recommended by GROTE and co-workers, we use an alloplastic interponate (PALLACOS R) for preservation of the intervertebral space and blocking of the adjacent vertebrae (Fig. 2).

The importance of this procedure was already pointed out by SUNDER-PLASSMANN in his comparative study of cervical disk operations. In case of very reduced intervertebral space we use no dowel because one has only to count with a minimal flexion of the segment. In these cases the bilateral foraminotomy is of high importance for sufficient ventral decompression of the nerve root (Fig. 3).

During the last 2 years we operated on 40 patients and performed bilateral foraminotomies in 65 segments. The intervertebral space C5/6 was afflicted in more than 50%.

In total, we performed 22 monosegmental and 16 bisegmental surgical interventions; 3 or 4 segments were operated on in one case. The most positive follow-up occurred in cases of monosegmental interventions. During follow-up of 2-22 months a complete remittance of the complaints was observed in 6 patients. In 30 patients the neurological deficit reverted by improvement of pain. In 3 patients there was no improvement of the neurological symptoms. One patient showed increased neurological disturbances. In this case a diabetic neuro-

Table 1. Postoperative results in the 40 patients operated on

1. Full recovery	6 patients
 Improved neurological symptoms 	30 patients
3. No improvement	3 patients
 Impaired neurological symptoms 	1 patient

pathy was found additionally to the cervical radiculopathy. Until present, 63% of the patients could resume their former profession.

Complications noticed, were profuse intraoperative bleeding of a paravertebral vessel in one case. Additional disturbances resulting from this were not observed.

A Horner-syndrome occurred postoperatively in 3 patients. Two patients temporarily complained of severe dysphagia. One patient died at the 10th postoperative day in consequence of a myocardial infarction, although postoperative recovery was satisfactory. By blocking the operated segment with an alloplastic interponate, an optimal axial position of the cervical column could be attained in more than 85% of the surgical interventions.

During the first postoperative months we saw a minimal axial mobility, ascertained by plain X-ray controls taken in anteflected and retroflected position, in 13% of the blocked segments. In these cases, during further follow-up, we could observe an increased tendency for complete blocking. In 8 cases we saw plate or vertebral infractions. However, these were patients with a more distinct osteoporosis. Follow-up was not affected by this.

The very satisfactory follow-up results in these 40 patients make this surgical method, in our opinion, an alternative to the classical ventral fusion $(\underline{1}, \underline{3}, \underline{9})$ or to the exclusive discectomy $(\underline{5}, \underline{6}, \underline{8})$ for treatment of cervical radiculo-myelopathy.

- CLOWARD, R.B.: Anterior approach for removal of ruptured cervical discs. J. Neurosurg. <u>15</u>, 602-617 (1958)
- CLOWARD, R.B.: Recent advances in surgery of the cervical spine. Proceedings of the German Society for Neurosurgery, Vol. 1, 285-293 (1969)
- DEREYMEKER, A., MULIER, J.: La fusion vertébrale par voie ventrale dans la discopathie cervicale. Revue neurologique, Tome 99. No. <u>6</u>, 597-616 (1958)
- 4. GROTE, W., BETTAG, W., WÜLLENWEBER, R.: Acta Neurochirurgica <u>22</u>, 1-27 (1970)
- HANKINSON, H.L., WILSON, C.B.: Use of the operating microscope in anterior cervical discectomy without fusion. J. Neurosurg. 43, 452-456 (1975)
- 6. HIRSCH, C., WICKBOM, I., LIDSTRÖM, A., ROSENGREN, K.: Cervical disc-resection. J. Bone and Joint Surg. 46-A, No. <u>8</u>, 1811-1821 (1964)

- 7. MURPHY, M.G., MOKHTAR GADO, m.B.: Anterior cervical discectomy without interbody bone graft. J. Neurosurg. 37, 71-74 (1972)
- ROBINSON, R.A., SMITH, G.W.: Anterolateral cervical disc removal and interbody fusion for cervical disc syndrome. Bull. Johns Hopkins Hospital 96, 223-233 (1955)
- 9. ROBINSON, R.A., WALKER, E., FERLIC, D.C., WICKING, D.K.: The results of anterior interbody fusion of the cervical spine. J. Bone and Joint Surg. 44-A, 1569-1587 (1962)
- 10. ROOSEN, C., GROTE, W., BETTAG, W.: Komplikationen ventraler Fusionsoperationen. Neurochirurgia <u>18</u>, 1-11 (1975)
- 11. SUNDER-PLASSMANN, M.: Vordere Halsbandscheibenoperation ohne zusätzliche Verblockung. Fortschr. Med. 94, Nr. 6 (1976)

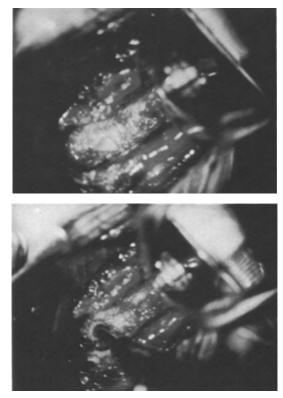


Fig. 1. Ventral discectomy (*above*) and opening of the intervertebral foramen with the aid of a diamond air drill (*below*)



<u>Fig. 2.</u> Preservation of the interspace and blocking of the adjacent vertebrae by an alloplastic dowel (PALLACOS R)

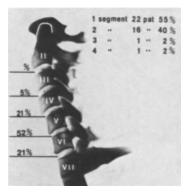


Fig. 3. Levels operated on in our material (40 patients, 65 segments)

The Influence of the Cloward Fusion Operation on the Motility of the Cervical Spine

W. GRÜNINGER and P. GRUSS

Introduction

Pathogenesis of cervical myelopathy has not been explained up to now. A relevant component in its development is the direct mechanical lesion of the spinal cord by degenerated lumbar disc tissue or by osteophytes, congenital stenosis of the cervical canal being a decisive predisposing factor. Several authors (5, 15, 16) have stressed the importance of local vascular disturbances as another predisposing factor. In early literature (1, 13, 14) the motility of the cervical region of the vertebral column was discussed as relevant for the pathogenesis of cervical welopathy. According to O'CONNEL (9), maximal motion of the cervical vertebral column in the sagittal plane leads to a difference of length of up to 5 cm. At the same time retroflexion causes a severe transverse stenosis of the horizontal plane by the folding of the ligamentum flavum (7).

KUHLENDAHL $(\underline{12})$ therefore states biomechanical and vasculo-circulatory factors as components of the pathogenesis of cervical myelopathy. We investigated the influence of the fusion of two or more cervical vertebrae on the motility of the cervical part of the vertebral column in the sagittal plane.

Material and Approach

Thirty-eight patients were examined 1-3 years after the fusion operation according to CLOWARD's technique. One control group consisted of 33 healthy persons of the same age as the patients, and another control group consisted of 26 juvenile persons in the age of 13-30 years. Standardised X-rays of the cervical spine in maximum ante- and retro-flexion were made. They were evaluated with measuring technique of ADAMS et al. (2). Using BAKKE's method (6), the motility of individual segments of the cervical spine was determined (Fig. 1).

Results

The fusion of two or more vertebral bodies causes a strong restriction of the movement of the cervical spine in the sagittal plane. The difference is significant between the older control group, and the patients who underwent the CLOWARD operation. A comparison of the juvenile control group - with an average age of 23 years - and the older control group - with an average age of 56 years - demonstrated the age dependant decrease of cervical spine motility (Table 1).

Selection of subgroups of patients in regard to location and number of fusioned vertebral bodies shows the decrease of motility of the

Table 1

	No.	Age	Angle I-II ^(O)
Younger control	26	22,9 (13-30)	53,3 2s 38,9-67,7
Older control	33	55,6 (31-64)	40,6 2s 16,0-65,2
CLOWARD patients	38	52,7 (34-66)	23,3 2s 5,1-43,3
Older control/CLOWARD pa	tients	t	6,5

cervical spine in the sagittal plane with an increased number of fusioned vertebrae. There was no difference in findings, however, between 9 patients with a fusion of the segment C5 and 11 patients with a fusion of the segment C6 between the 6th and 7th cervical vertebral bodies (Table 2).

Table 2

Fusion	No.	Angle I	-II ⁽⁰⁾
<u> </u>		Mean	Range
1 Segment	22	26,3	7-40
2 Segments	13	20,0	13-37
3 Segments	3	12,2	7-19
Localisation			
C4/5 C5/6 C6/7 C4/5/6 C5/6/7 C4/5 + C6/7 C4/5/6/7 C5/6/7/Th1	2 9 11 5 7 1 2	- 27,0 27,2 19,1 20,0 -	18-38 13-40 7-37 13-29 13-32 - 37 9-19 - 7

In the physiological motion of the cervical spine, retroflexion is a much larger component than anteflexion. In our control groups, the proportion of retroflexion/anteflexion was almost 2:1, corresponding with published data (19). The individual position at rest is not considered with the measuring technique used, upright position of the cervical spine being assumed. Anteflexion and retroflexion are about equally affected by the restriction of cervical spine motility, increasing with age. The fusion of vertebrae, however, restricts mainly retroflexion (Table 3).

A comparison of individual segment movement of the cervical spine after surgery shows a shifting of maximum motility into the upper segments of the cervical region of the spinal column. Pathological mobility was seen only in two patients in the upper neighboring segment of the block vertebra, contrary to observations of WOESNER et al. (<u>18</u>). In a few cases, we found a strong gliding movement of the upper vertebrae against the block vertebrae with a maximum edge displacement of

Table 3

	No.	Anteflexion $k-\bar{x}$ (mm)	Retroflexion l-x (mm)
Younger control	26	9,6	16,5
Older control	33	7,6	13,9
CLOWARD patients	38	7,4	7,7

2,5 mm (Fig. 1). In the same way as VERBIEST $(\underline{17})$ and ADAMS et al. $(\underline{4})$, we more often observed an increased osteochondrosis with immobilisation by the development of ventral exostoses and bone bridges in the neighbour segments of the block vertebra (Fig. 2).

ADAMS et al. $(\underline{4})$ found the best postoperative results in patients showing a restriction of cervical spine motility below 40° . We also saw an improvement of preoperative symptoms in 10 of 18 patients with movement restriction less than 30° , whereas only 2 out of 8 patients with a postoperative motility of above 30° showed any clinical improvement. We therefore consider immobilisation of the cervical spine, especially the restriction of retroflexion, as the decisive therapeutic effect of the CLOWARD operation. Comparative results were published by surgeons preferring mere discectomy and fusion of cervical vertebral bodies without removal of exostoses in the treatment of cervical myelopathy (HIRSCH et al. (<u>11</u>), HANKINSON et al. (<u>10</u>)).

Summary

Thirty-eight patients were examined one to three years after an operation by CLOWARD's technique. The X-rays of the cervical spine in maximum anteflexion and retroflexion were compared with those of a control group of corresponding age and with those of a younger control group.

The fusion of two or more vertebrae leads to considerable limitation of sagittal movement of the cervical spine on an average of $23,5^{\circ}$ in comparison with $40,6^{\circ}$ in the corresponding age control group. With the increasing age, anteflexion and retroflexion deteriorate equally, whereas the fusion operation restricts mainly the retroflexion of the cervical spine. In fact, the therapeutic effect of the CLOWARD operation seems to lie in the restriction of movement of the cervical spine.

- ABOULKER et al.: Les myelopathies cervicales d'origine rachidienne. Neuro-Chirurgie 11, 89 (1965)
- ADAMS et al.: I. Movement of the cervical roots, dura and cord, and their relation to the course of the extrathecal roots. Brain 94, 557 (1971)
- 3. ADAMS et al.: II. The movement and contour of the spine in relation to the neural complications of cervical spondylosis. Brain $\underline{94}$, 569 (1971)
- ADAMS et al.: III. Some functional effects of operations for cervical spondylotic myelopathy. Brain <u>94</u>, 587 (1971)

- ALLEN: Neuropathies caused by bony spurs in the cervical spine with special reference to surgical treatment. J. Neurol. Neurosurg. Psychiat. <u>15</u>, 20 (1952)
- 6. BAKKE: Röntgenologische Beobachtungen über die Beweglichkeit der Wirbelsäule. Acta radiol. (Stockh.) Supp. 13, 1 (1931)
- BREIG et al.: Effects of mechanical stresses on the cervical spondylosis. J. Neurosurg. <u>25</u>, 45 (1966)
- CLOWARD: The anterior approach for removal of ruptured cervical discs. J. Neurosurg. <u>15</u>, 602 (1958)
- 9. O'CONNELL: Cervical spondylosis. Proc. R. Soc. Med. <u>49</u>, 202 (1956)
- HANKINSON et al.: Use of operating microscope in anterior cervical discectomy without fusion. J. Neurosurg. <u>43</u>, 452 (1975)
- 11. HIRSCH et al.: Cervical disc resection: a follow-up myelographic and surgical procedure. J. Bone Joint Surg. (Am) 46, 1811 (1964)
- 12. KUHLENDAHL: Pathogenese der sogenannten zervikalen Myelopathie. MMW <u>111</u>, 1137 (1969)
- 13. REID: Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. J. Neurol. Neurosurg. Psychiat. <u>23</u>, 214 (1960)
- 14. STOLTMANN et al.: The role of the ligamenta flaca in the pathogenesis of myelopathy in cervical spondylosis. Brain 87, 45 (1964)
- 15. TAYLOR: Vascular factors in the myelopathy associated with cervical spondylosis. Neurology (Minneap.) <u>14</u>, 62 (1964)
- 16. UMBACH: Diagnostik und Therapie cervico-brachialer Syndrome. Nervenarzt <u>45</u>, 169 (1974)
- 17. VERBIEST: La chirurgie anterieure et latérale du rachis cervicale. Neuro-Chirurgie <u>16</u>, Suppl. 3 (1970)
- 18. WOESNER et al.: The evaluation of cervical spine motion below C2: a comparison of cineroentgenographic and conventional roentgengraphic methods. Amer. J. Roentg. <u>115</u>, 148 (1972)
- 19. ZAUNBAUER et al.: Röntgenologische Bewegungsanalysen der HWS bei gesunden Kindern und Jugendlichen. Röfo <u>96</u>, 87 (1962)

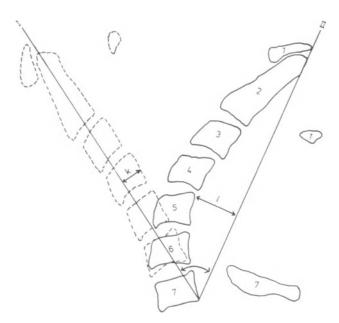


Fig. 1. Measuring technique according to ADAMS. The angle I-II remarks the total movement, k the anteflexion, l the retroflexion



Fig. 2. L.K. (male, 63 years), 23 months after fusion at C4/5/6. Gliding of the vertebral bodies above the block vertebra. Note the osteochondrosis of the uncovertebral joints and the restriction of retroflexion

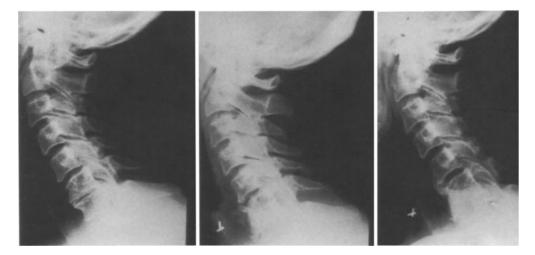


Fig. 3. S.F. (male, 48 years), development of ventral osseous bars in the adjoining segment to the block vertebra C6/7. Left: before operation; middle:2 months after operation; right: 41 months after operation

Discitis After Discography P. DISTELMAIER, I. VLAJIC, and J. WAPPENSCHMIDT

Introduction

In our mind discography is the most reliable method to verify cervical disc protrusions (2, 3, 4). We rarely see complications (14). Discitis after discography is the most severe complication we are afraid of. That is why we want to describe its incidence in our patients.

Material and Approach

During the last 6 years we performed more than 800 discographies on nearly 500 patients. After thorough disinfection of the skin (aether, alcohol, desinfectants) we performed the puncture of the disc under X-ray biplane fluoroscopy in general without local anaesthesia. Only in the case of severe pain during the puncture did we use local anaesthesia. We used 0,25-0,6 cm³ of a water soluble contrast medium (2, 4, 7).

We observed that discitis occurred 3 times after discography, that is in 0,6% of our patients. Two patients only suffered from pain in the neck after the discography. Discitis was discovered accidently during operation, which was discontinued. One patient had symptoms which indicated sepsis, with temperature between 38° C and 39° C, leucocytosis and 780/3 cells in the cerebrospinal fluid. The patient suffered from severe pain in the neck and back, and shivering spells.

Blood cultures were negative in all cases. Clinical symptoms vanished with antibiotic treatment in all cases. The clinical symptoms which had led to discography persisted and did not change.

We would like to present the clinical and radiological aspects of two typical cases.

<u>Case 1.</u> A woman, 53 years old, with a left-sided brachialgia and hypaesthesia at C6 and C7 showed degenerative alterations in both segments (Fig. 1a). Myelography and discography showed degenerative alterations in both segments and a disc protrusion mainly at C5/C6 and, to a lesser extent, at C6/C7 (Fig. 1b, c). Two days later the patient developed temperatures of 39° C and suffered from pain in the neck and the back. A plain X-ray of the cervical spine at that time showed no alterations. CSF contained 778/3 but no organisms were found either in the blood or in the cerebrospinal fluid. Antibiotic treatment was instituted. No operation was undertaken. The pains and febrile episodes disappeared. A follow-up 6 months later showed a discitis and osteomyelitis which had caused a fusion of the vertebrae C5/C6/C7. The spatium retropharyngicum was still enlarged (Fig. 1d). Another antibiotic treatment was instituted. Six months later the X-ray picture showed the fusion of the vertebrae C5/C6/C7 with a gibbus C4/C5 (Fig. 1e). The clinical situation was the same as before the discography and discitis. A foraminotomy of C5/C6 produced good result.

<u>Case 2.</u> A 52 years-old patient showed symptoms of a cervical myelopathy (Fig. 2a). Myelography and discography indicated a protrusion of the disc C5/C6 (Fig. 2b). Three days after discography the patient was operated. Interbody fusion had been planned. The operation had to be discontinued because inflammatory tissue was discovered (Fig. 2c).

Antibiotic treatment was instituted. X-ray follow up one month later indicated a fusion of C5/C6 (Fig. 2d). Angiography and myelography 1 year later showed complete fusion of C5/C6, the vertebral canal was not involved, the vertebral arteries were slightly compressed but not occluded (Fig. 2e, f). The clinical situation of the patient did not change.

Discussion

Discography is generally accompanied by little risk (9, 14). Discitis is the most frequent complication (0,6% of our patients). Adequately treated with antibiotics, it generally leads to a fusion of the vertebral bodies with disc destruction (9). We observed no neurological sequellae, nor dangerous involution of other organs. We were unable to cultivate bacteriae. Nevertheless we think the discitis were bacterial in etiology. The result of an interbody fusion is of no value in case of radicular symptoms, the foramen intervertebrale in general remains narrowed. In the case of cervical myelopathy, however, it is perhaps worthwhile to study aseptic cervical nucleolysis (8).

Conclusion

Discitis is a complication of discography which occurred in about 0,6% of our patients. This percentage can be compared to that of other authors (4, 9, 14). When pain in the neck or fever occurs after discography it should be investigated with X-rays of the cervical spine. If the results are normal, the investigations should be repeated after a short period of time. In general the first radiological sign is a broadening of the retropharyngeal space. Antibiotic treatment should then be initiated. The result is an interbody fusion and a destroyed disc, as described in all published cases of discitis (9).

The risk of discography seems to be tolerable and not to impair fundamentally its value $(\underline{12})$, especially since we did not find neurological sequellae ($\underline{9}$, $\underline{13}$). As far as we are aware, there is only one lethal complication of discography described in literature ($\underline{15}$).

- CLOWARD, R.B.: Surgical treatment of traumatic cervical spine syndroms. Wiederherstellungschirurgie und Traumatologie <u>7</u>, 148. Basel, New York (1963)
- DISTELMAIER, P., LINS, E.: Technik und Aussagekraft der Diskographie bei der Diagnostik zervikaler Syndrome. Röntgenbl. <u>29/4</u>, 178-183 (1976)

- DISTELMAIER, P.: Zur Bedeutung der Funktionsaufnahmen der Halswirbelsäule bei der Diagnostik zervikaler Syndrome. Fortschr. Röntgenstr. <u>126/2</u>, 160-165 (1977)
- 4. FROWEIN, R.A., FRIEDMANN, G., KEUNSTEIN, U.: Die Kontrastmitteldiagnostik bei zervikalen Bandscheibenschäden. Röntgenbl. <u>27/11</u>, 538-548 (1974)
- 5. GROTE, W., WAPPENSCHMIDT, J.: Technik und Indikation zur zervikalen Diskographie. Fortschr. Röntgenstr. <u>106</u>, 721 (1967)
- HOLT, E.P.: Further reflections on cervical discography. J. Amer. Med. Ass. <u>231/6</u>, 613-614 (1975)
- LINDBLOM, K.: Diagnostic puncture of intervertebral discs in sciatica. Acta orthop. scand. <u>17</u>, 231 (1948)
- 8. NCNEILL, T.: Chemonucleolysis. Lancet 1/7914, 1022 (1975)
- 9. ROOSEN, K., BETTAG, W., FIEBACH, O.: Komplikationen der zervikalen Discographie. Fortschr. Röntgenstr. <u>122/6</u>, 520-527 (1975)
- 10. SCHAERER, J.P.: Cervical discography. J. int. Coll. Surg. <u>42</u>, 287 (1964)
- 11. Sèze, S.: Les accidents de la discographie. Rev. rheumat. Paris <u>19</u>, 1027 (1952)
- UMBACH, W.: Diagnostik und Therapie zervikaler Syndrome. Nervenarzt <u>45</u>, 169 (1974)
- VOGELSANG, H.: Discitis intervertebralis cervikalis nach Diskographie. Neurochirurgia <u>16</u>, 80 (1973)
- 14. VOGELSANG, H.: Die zervikale Diskographie: Nervenarzt <u>46/7</u>, 337-342 (1975)
- 15. WELLAUER, W.: Tödliche Komplikation bei Diskographie. Persönliche Mitteilung in: ROOSEN, BETTAG und FIEBACH: Komplikationen der zervikalen Diskographie. Fortschr. Röntgenstr. <u>122/6</u>, 520-527 (1975)



Fig. 1 a-e. Woman (53 years), leftsided brachialgia a X-ray at admission: osteochondrosis C5/C6/C7 b Myelography: narrowing of the spinal canal at C5/C6 c Discography: positive result at C5/C6, negative at C6/C7 d Control 6 months later, no operation: discitis and osteomyelitis e Control one year later: synostosis of the vertebrae C5/C6/C7, dislocation of C4/C5



- <u>Fig. 2 a-e.</u> Man (52 years), cervical myelopathy
 <u>a</u> X-ray at admission: osteochondrosis, especially at C5/C6
 <u>b</u> Discography: positive result at C5/C6
 <u>c</u> Control after operation, discountinued because of inflammatory signs: osteomyelitis, discitis
 <u>d</u> X-ray one year later: fusion of C5/C6 osteophytes
 <u>e</u> Angiography of both vertebral arteries: slight narrowing C5/C6
 <u>f</u> Myelography: no narrowing of the vertebral canal

Considerations on 100 Anterior Cervical Discectomies Without Fusion S. GIOMBINI and C. L. SOLERO

Since 1974 anterior discectomy without fusion for cervical discopathy has been adopted at the Istituto Neurologico of Milan, according to some technical modifications suggested by HIRSCH $(\underline{4})$, BOLDREY $(\underline{1})$, SUSEN $(\underline{9})$, MURPHY and GADO $(\underline{6})$. In most cases we have employed the operating microscope.

The subject of this paper is the evaluation of the results of the operations performed with this technique, particularly in relation to the sagittal diameter of the cervical spinal canal.

Material and Method

In the last 5 years (February 1974 - February 1979) 100 operations have been performed on 99 patients, whose age ranged from 23-69 years (average age: 46,5); 71 were males, 28 females.

Preoperative clinical picture was pure radiculopathy in 55 cases, pure myelopathy in 13 cases and radiculo-myelopathy in 32 cases. Two patients had been previously operated on elsewhere, they underwent a posterior cervical discectomy and a cervical laminectomy, respectively. Myelography was performed in all of the cases and it was always consistent with the clinical picture: in 22 cases, all with findings of radiculopathy, one or several radicular pouches were found to be amputated, whilst in 78 cases transverse bars were evident, at the level of one or more discs. Fifty-four operations were performed on one disc, 41 on two discs and 5 on three discs; in 4 cases two not adjacent discs were operated upon (Table 1).

The approach to the anterior aspect of the cervical vertebral bodies was carried on according to the technique described by SMITH and ROBINSON ($\underline{8}$), always from the right side. After radiological identification of the appropriate level, the disc was partially removed and, after spreading the adjacent vertebral bodies, the discectomy was completed. The posterior osteophytes were removed thus exposing the posterior longitudinal legament, that sometimes was opened. At this point the dura of the cord and the roots were free of encroachment. The wound was closed without a drain.

The operating microscope was employed in 70 cases.

The second day after the operation the patient was usually allowed to leave his bed, fitted with a stiff cervical collar that he was instructed to wear for four weeks, only while standing. In uncomplicated cases, the patient was discharged 5 days after the operation. All of the patients of this series have been examined at the time of discharge, after a month and later on, with an average follow-up period of 26 months (5-64 months).

Levels operated	No. of operation	ons	
Single	5	54	
C3-4	3		
C4-5	4		
C5-6 C6-7	19 24		
C7-D1	4		
Тwo	4	1	
C3-4, C4-5	3		
C4-5, C5-6	8		
C5-6, C6-7	26		
C3-4, C5-6 ^a	3		
C5-6, C7-D1 ^a	1		
Three	,	5	
C3-4, C4-5, C5-6	2		
C4-5, C5-6, C6-7	2 2		
C5-6, C6-7, C7-D1	1		
Total	10	0	

Table 1. Operated levels in 100 operations

a Operations performed on not adjacent levels.

Results

Intra-operative complications were: cerebrospinal fluid leakage (3 cases), bleeding from epidural veins (4 cases), fracture of the anterior aspect of one vertebral body (1 case). Early post-operative complications are shown in Table 2: shortly after the operation; HORNER's syndrome did not improve; radicular or medullary motor deficits caused or worsened by the operation did not improve in 4 patients, whilst were transitory in 4 more cases.

Table 3 shows early and late operative results, according to preoperative syndrome.

Three patients died: one of them was operated on for a pure radiculopathy (36-year-old woman), the two other for a severe pure myelopathy (60-year-old woman and 69-year-old man). No intraoperative complication happened. In one case tetraparesis was noted at recovery from anaesthesia and worsened rapidly; in the other two cases tetraparesis appeared 12 hours after operation. These two were re-explored, and one of them underwent also a decompressive laminectomy, but nothing abnormal was found. The re-operation did not change the neurological picture. The third patient was not re-operated on because a slight improvement was noted with corticosteroid therapy. All of them died after a month because of respiratory complications.

The other 96 patients of this series have been periodically followed as out-patients and the results of the follow-up study has been classified as:

Complication	Transitory	Permanent	
Dysphagia	1		
Recurrent laryngeal palsy	9		
HORNER's syndrome		4	
Opposite radicular pain	4		
Opposite radicular motor deficit	1	1	
Worsening of myelopathy	3	2	
Hemiparesis		1	
Total	18	18	
	26		

Table 2. Early operative complications occurring in 23 patients

<u>Table 3</u>. Immediate and late operative results as per clinical syndrome

	Oper- ations	Excellent	Good	Poor	Bad	Deaths
Pure radiculopathy	55	22	30	1	1	1
Pure myelopathy	13	1	7	3		2
Radiculo-myelopathy	32	6	14	3	9	
Total	100	29	51	7	10	3
<u> </u>		80				

<u>excellent:</u> preoperative symptoms relieved, abnormal signs improved or abated;

good: minimum persistence of preoperative symptoms, abnormal signs unchanged or improved;

poor: symptoms and signs unchanged;

 $\underline{bad:}$ preoperative symptoms unchanged or worsened, abnormal signs worsened and/or appearance of new deficits.

The operation was successful in 80 cases (excellent and good results); in 7 cases it did not change the pre-operative signs and symptoms (poor results); ten patients became worse.

Best results have been achieved in the group with a pure radiculopathy, while most failures were found among patients with myelo-radiculopathy.

After the first 22 operations, a radiological control of the cervical spine in neutral position and in flexion-extension was performed; this control was repeated 4 and 15 months after the operation (Table 4). A satisfactory alignment of the vertebral bodies, with complete fusion between them in the cases with the longest post-operative follow-up, was evident in the neutral position in all of the cases except in one, where a mild asymptomatic spondylolystesis was found. Dynamic films often showed reduction in flexion and extension movements of the operated interspaces, without any clinical relevance.

Table 4. Radiological for	ollow-up i	in 22	cases
---------------------------	------------	-------	-------

Fusion	Complete Incomplete	10 12
Alignment	Normal Abnormal	21 1
Dynamic films	Normal Abnormal	3 19

Discussion

HIRSCH (4) in 1958 adopted the anterior cervical discectomy without fusion. BOLDREY (1), SUSEN (9), MURPHY and GADO (6), MARTINS (5) reported good results using a similar technique. ROBERTSON (7) achieved better results with this procedure than with interbody fusion.

The removal of the disc and of posterior and lateral osteophytes became easier after the introduction of the operating microscope, due to good light and magnification; for this reason, at the Istituto Neurologico of Milan was performed, since 1974, most of the operations with microsurgical technique, as recommended by others $(\underline{3}, \underline{7})$.

The incidence and type of operative complications, the average postoperative hospital period and the percentage of excellent and good results in our series are comparable to the data reported by other authors. In our series the percentage of patients that became worse and the mortality rate are definitely higher than the figures given by other authors.

Ten patients became worse: in 5 of them the cervical discopathy was documented by myelograms and was consistent with the signs and symptoms found pre-operatively, but it was not the cause of neurological picture. These signs and symptoms were due to other diseases involving the spinal cord or the roots, whose nature became clear later: two cases had amyotrophic lateral sclerosis, the remaining 3 had multiple sclerosis, syringomyelia and Pancoast's syndrome respectively.

Some of the worse, unexpected post-operative results induced us to take into consideration the diameter of the cervical spinal canal. As stated by EPSTEIN et al. (2), "... the sagittal diameter of the canal, ... normally is from 13-18 mm as measured from the dorsal aspects of the C3-C7 segments to the white line indicating the junction of the laminae to form the spinous processes ... When a sagittal diameter of 13 mm is present, a borderline situation exists. Values less than 10 or 11 mm are important".

All of our cases have been re-examined on the basis of this parameter. In two patients out of the three that died the diameter of the cervical spinal canal measured less than 13 mm. The third patient who died did not have a narrow cervical canal; he was the oldest patient in the series (69 years): this factor may have influenced the outcome.

Out of the ten patients that became worse, five had a correct indication to surgery; two of them had the sagittal diameter of the cervical spinal canal less than 13 mm; in two other it measured between 13 and 14 mm. In the 5th case we were not able to find any cause for the worsening. Table 5. Operative failures according to sagittal diameter of cervical spinal canal

Sagittal diameter	Cases	Bad results	Deaths
Less than 13 mm	7	2	2
Between 13-14 mm	16	2	
More than 14 mm	71 ^a	1	1

a Figure not including 5 patients with wrong indication for discectomy.

In Table 5 we correlated the number of the patients and the operative failures to the sagittal diameter of the cervical canal. Out of 7 cases in whom it was less than 13 mm, two died and two became worse; the incidence of failures is less relevant but nevertheless consistent in patients whose diameter measured between 13 and 14 mm (2 out of 16 became worse), while this incidence is lower when the diameter is normal.

We believe that the most probable cause of the failures that we had among the patients that we operated upon with a correct indication was the fact that we did not consider their narrow cervical spinal canal. In patients with a cervical discopathy and stenosis of spinal canal an anterior discectomy without fusion may be highly dangerous; in these cases we suggest to perform a decompressive laminectomy and, if the symptoms and signs are not relieved by this procedure, to perform later on an anterior discectomy with interbody bone graft to ensure the stability of the spine.

Conclusions

Anterior cervical discectomy without fusion in cases of cervical radiculopathy and/or myelopathy offers good results without major complications provided that it is performed on correct clinical indication, in patients without a narrow cervical spinal canal. The microsurgical technique makes the procedure safer and more delicate.

- BOLDREY, E.B.: Anterior cervical decompression (without fusion). Presented at the American Academy of Neurological Surgery, Key Biscayne, Florida 1964
- EPSTEIN, B.S., EPSTEIN, J.A., JONES, M.D.: Anatomico-radiological correlations in cervical spine discal disease and stenosis. Clin. Neurosurg. 25, 148-173 (1978)
- HANKINSON, H.L., WILSON, C.B.: Use of the operating microscope in anterior cervical discectomy without fusion. J. Neurosurgery 43, 452-456 (1975)
- 4. HIRSCH, C., WICKBOM, I., LINDSTRÖM, A., ROSENGREN, K.: Cervical disc resection. A follow-up of myelographic and surgical procedure. J. Bone Joint Surg. <u>46A</u>, 1811-1821 (1964)
- 5. MARTINS, A.N.: Anterior cervical discectomy with and without interbody bone graft. J. Neurosurg. 44, 290-295 (1976)

- MURPHY, M.G., GADO, M.: Anterior cervical discectomy without interbody bone graft. J. Neurosurg. 37, 71-74 (1972)
- ROBERTSON, J.T.: Anterior removal of cervical disc without fusion. Clin. Neurosurg. <u>20</u>, 259-261 (1972)
- SMITH, G.W., ROBINSON, R.A.: Anterior lateral cervical disc removal and interbody fusion for cervical disc syndrome. Bull. Johns Hopkins Hosp. <u>96</u>, 223-224 (1955)
- 9. SUSEN, A.F.: Simple anterior cervical discectomy without fusion. Presented at the American Academy of Neurological Surgery, San Francisco, California 1966

Posterior Selective Spinal Thermorhizotomy for Control of Intractable Pain Syndroms

F. COLOMBO, A. ALEXANDRE, D. CURRI, and A. BENEDETTI

Introduction

In cases of chronic pain involving rather few metamers or roots with a minor functional relevance, posterior rhizotomy has become a widely performed operation. However, its inevitable consequences, such as complete anesthesia, marked hypotonia and, sometimes, its uneffectiveness on relieving patient's suffering (anesthesia dolorosa), engendered the research of selective techniques aiming at a good analgesia without disadvantages due to the suppression of the other kinds of sensibility (epicritic tactile and positional senses) (1, 2, 10, 13, 14).

Following MELZACK and WALL's theory $(\underline{6})$, complete section of all root nerve fibers is replaced by selective section of the small diameter A₀ and C fibers in a series of new surgical techniques. SINDOU $(\underline{9}, \underline{10})$ performs an incision in the ventrolateral region of posterior spinal cord-rootlets junction; SWEET (<u>11</u>), UEMATSU (<u>12</u>), LAZORTHES (<u>4</u>), following experimental results of LETCHNER and GOLD-RING (<u>5</u>), employ, controlled thermocoagulation by selective heating of the small diameter fibers for percutaneous rhizotomy; larger diameter fibers are left relatively intact. Histological demonstration of the selectivity of the thermic lesion is lacking.

Controlled thermocoagulation of the trigeminal ganglion and rootlets is nowaday a widely accepted practice in trigeminal neuralgias (1), on the other hand, percutaneous radiofrequency rhizotomy at spinal level (12, 14) does not always allow successful results, and exposes to the risk of vascular or motor nerve lesion. SIEGFRIED (8) proposes radiofrequency thermic lesion of the ventrolateral region of the spinal cord-rootlets junction in open surgery in addition to surgical incision. We do not know if such refinement has ever been applied.

In three cases of intractable pain syndromes we have performed a selective posterior rhizotomy by means of radiofrequency current (thermorhizotomy) through open surgery; clinical results are reported.

Material and Approach

We have decided to perform the thermic lesion on each posterior root 1 cm from the spinal cord through open surgery because:

- it is thus possible to avoid the risks and complication of percutaneous approach;
- 2. no manipulation of the cord is requested;

- 3. at 10 mm there is no danger of damaging the small perforating vessels which enter the spinal cord ventrolaterally to the rootlets and the lesion of which would cause ischaemic damage; and
- at this site the fibers of different diameter are mixed and homogeneously exposed to the radiofrequency heating.

A widened hemilaminectomy is performed under general anesthesia. After dural incision and root identification we carry each rootlet on the tip of the thermoelectrode (bare tip 3,5 mm long, made by Fisher, Germany). Tip and rootlet are coated by a drop of saline (Fig. 1). Temperature is then rised to 70⁰ C and maintained for 60 sec.

<u>Case 1</u>

S.D. (63-years-old male) suffered from postherpetic pain involving the upper thoracic roots (T1-T2-T3-T4) on the left side. On admission, a zone of cutaneous hyperalgesia was found on the territory of the first four thoracic roots. Any kind of stimulus caused an intense pain, and sensibility testing was thereby impossible. His excruciating continuous pain has induced abuse of morphinic drugs. Herpetic pain began six months prior and kept worsening. On March 13, 1977 selective posterior thermorhizotomy was performed at T1-T2-T3-T4 on the left side. On the following days spontaneous pain progressively disappeared and, at discharge, on March 27, only at not too troublesome zone of paresthesia at T4 was left. Almost complete analgesia was present in the four segments, together with mild tactile hypoesthesia. One year later, the surgical result is unmodified.

<u>Case 2</u>

B.L. (54-years-old female) underwent right-sided mastectomy for mammarian duct carcinoma in 1974. Postoperatively a wound infection caused osteitis of the fifth rib. A pain syndrome arose shortly after surgery, growing unbearable, with sensation of painful pressure and rent involving the chest wall at the level of the fifth and sixth dermatomes on the right. After having experienced several kinds of treatment (infiltration of intercostal nerves, transcutaneous stimulation, acupuncture), a left high cervical cordotomy was performed on January 1977 but even this operation was useless. She was admitted to our department in September 1977. After hemilaminectomy and exposure of the T4-T5-T6-T7 right posterior roots, thermorhizotomy was performed. When discharged, 12 days later, she did not feel pain; analgesia of the involved skin district was almost complete, together with tactile hypesthesia. Six months later the patient complained of variable paresthesias with mild pain sensation. Transcutaneous nerve stimulation is now helpful.

<u>Case 3</u>

P.E. (74-years-old male) was admitted to our department suffering intractable postherpetic pain at T6-T7 level on the right for three years. Neurological examination revealed only painful disesthesia in the involved dermatomes. On February 4, 1978, surgery with thermocoagulation of the posterior roots T4-T5-T6-T7-T8-T9 on the right is undertaken. On the third postoperative day the patient is symptomfree and at discharge the hypoesthetic area T5-T8 is completely analgesic. Six months later the results are unchanged.

Discussion

Our clinical experience is limited (three cases) and follow-up is short. Nevertheless it seems worthwhile to observe that:

- 1. Analgesia was beyond our expectancies: it appeared on the third to fourth postoperative day. Therefore it is surely not due to suggestion or to simple root manipulation.
- Tactile sensitivity, even if impaired, was not significantly affected.
- 3. Thermocoagulation seems to involve also fibers conducing visceral pain: patient No. 1, who was also a cardiopath, was free from angor after surgery, even through clinical situation and ECG remain unmodified. This may represent a potential risk for him.
- 4. While clinical data fully confirm the selectivity of the thermic lesion, histological examination (hematoxilin-eosin and luxol fast blue) of cat safenous nerve previously heated to 70° C failed to demonstrate any specific lesion of smaller A_o and C fibers.
- 5. Our three patients were in precarious general condition: surgical trauma was strictly limited. In case 1 and 2 we have renounced to coagulate two roots below and two roots above as suggested by neurophysiological observation made by SHERRINGTON (7) and FOERSTER (3). The thermic lesion was confined to those roots directly innervating the involved area. The fact that the analgesic result was always good leads us to suppose that there is a relative persistence of metamerization at the thoracic level.

Conclusion

In cases of intractable pain syndromes selected to undergo open posterior rhizotomy, our selective technique (thermorhizotomy) can be performed. Although histological demonstration of the selectivity of the thermic lesion is still lacking, the experience of percutaneous radiofrequency rhizotomy demonstrates that it is possible to abolish pain by controlled thermocoagulation without damaging other forms of sensibility.

- ABBE, R.: Intradural section of the spinal nerves for neuralgia. Mes. Surg. J. <u>135</u>, 329-335 (1896)
- BEENET, W.H.: A case in which acute spasmodic pain in the left lower extremity was completely relieved by subdural division of posterior roots of certain spinal nerves. Med. Chir. Trans. <u>72</u>, 329-348 (1889)
- 3. FOERSTER, O.: The dermatomes in man. Brain 56, 1-39 (1933)
- LAZORTHES, Y., VERDIE, J.C., LAGARRIGUE, J.: Thermocoagulation percutanée des nerfs rachidiens à visée analgésique. Neurochirurgie 22, 445-454 (1976)
- LETCHNER, F.S., GOLDRING, S.: The effect of radiofrequency current and heat on peripheral nerve action potential in the cat. J. Neurosurg. <u>29</u>, 42-47 (1968)
- 6. MELZACK, R., WALL, P.D.: Pain mechanism: a new theory. Science 150, 971-979 (1965)

- 7. SHERRINGTON, C.S.: Experiments in examination of the peripheral distribution of the posterior roots of some spinal nerves. Proc. R. Soc. 60, 403-411 (1896)
- SIEGFRIED, J.: Douleur et neurochirurgie fonctionelle. Neurochirurgie <u>22</u>, 571-578 (1976)
- 9. SINDOU, M., FISHER, G., GOUTELLE, A., MANSUY, L.: La radicellotomie postérieure sélective. Premiers résultats dans la chirurgie de la douleur. Neurochirurgie 20, 391-408 (1974)
- SINDOU, M., FISHER, G., MANSUY, L.: Posterior spinal rhizotomy and selective posterior rhizidiotomy. Prog. Neurol. Surg. <u>7</u>, 201-250 (1976)
- SWEET, W.H., WEPSIC, J.C.: Controlled thermocoagulation of trigeminal ganglion and rootlets for differential destruction of pain fibers. J. Neurosurg. 39, 143-155 (1974)
- 12. UEMATSU, S., UDVARHELYI, G.B., BENSON, D.W., SIEBENS, A.: Percutaneous radiofrequency rhizotomy. Surg. Neurol. <u>2</u>, 319-325 (1974)
- 13. WHITE, J.C.: Posterior rhizotomy. A possible substitute for cordotomy in otherwise intractable neuralgias of the trunk and extremities of non malignant origin. Clin. Neurosurg. <u>13</u>, 20-41 (1966)
- 14. WHITE, J.C., SWEET, W.H.: Pain and the neurosurgeon: a forty year experience. Springfield, Ill.: Thomas 1969



Fig. 1. (Case 2). Widened hemilaminectomy of T4-T5-T6 and the upper part of T7.T posterior root of T6 is lying on the bare tip of the thermoelectrode, coated by a drop of saline

Intramedullary Ependymomas: Long-Term Results of Surgical Treatment

B. GUIDETTI, S. MERCURI, and R. VAGNOZZI

The authors report on the late functional results in a series of 48 operated ependymomas of spinal cord with a follow-up ranging from 1-27 years.

The purpose of this communication is to present the long-term results of intramedullary ependymomas surgically treated.

Our series comprises 528 primary spinal tumours operated on at the Neurosurgical Department of the University of Rome from May 1951 to May 1978. One hundred and fifty-nine were intramedullary tumours of which 48 were ependymomas (Table 1).

Table 1. Primary spinal tumors

Extramedullary	
Inclancaulaly	
Neurinoma	136
Meningioma	113
Primary benign bone tumors (giant cell	
tumors, chondroma, aneurysmal bone cyst, etc.)	32
Sarcoma	42
Neuroblastoma and ganglioneuroma Chordoma	16
Lipoma and angiolipoma	8 8
Juxtamedullary glioma	6
Epidermoid, dermoid and teratoma	4
Melanoma	1
Extramedullary haemangioblastoma	3
	369
Intramedullary	
Glioma	
Ependymoma	38
Ependymoma cauda	10
Astrocytoma	53
Spongioblastoma	13
Glioblastoma	5
Oligodendroglioma	1
Unclassified glioma	5
unverified glioma	4
Haemangioblastoma	12
Lipoma	8
Dermoid, epidermoid Unclassified tumors	5 4
Neurinoma	4
Neurrioma	
	159

The surgical treatment does not differ from the one we have reported in 1964 and 1967 (<u>6</u>, <u>7</u>), except for the fact that we took advantage of a greater experience and of the use of the operating microscope in this type of surgery, which led to better results and to a total absence of operative mortality in the last 11 years. The mortality rate during the patient's stay in the Clinic, which lasted from several days to 3 months, is of 8,3% (4 cases) (Table 2). From the 4 patients that died following surgical management 1 died on the 10th postoperative day of pulmonary embolism, 2 died of respiratory paralysis, and 1 of purulent meningitis 3 months after the operation. This last patient had had decompressive laminectomy in another hospital 1 year prior to the second operation, and was treated with extensive X-ray radiation. Due to post-irradiation changes in soft tissues, satisfactory closure of the wound was never accomplished. In all these cases the tumour was located in medulla or cervical region.

We accurately followed up all the operated patient. We could not obtain informations of 1 patient (2%). The remaining 43 (89,5%) have had a follow-up ranging from 12-310 months (Table 2).

To evaluate long-term results we checked the patients conditions as follows:

Good (90%-100%): No neurological abnormality or very little deficit such as some sensory disturbance or slight sphinteric troubles; normal gait; normal life.

Slight (70%-90%): Slight neurological abnormality, slight deficit such as weakness of the limbs, sensory disturbance or slight sphincteric troubles; walking without assistance; autonomous life.

Moderate (50%-70%): Disability due to moderate neurological deficit; walking with cane or crutches; possibility of autonomous life.

Poor (30%-50%): Gross neurological deficit, gross motor and sphincteric disturbances; walking with support; no autonomous life. The postoperative results have been divided into 4 groups: (1) improvement; (2) no improvement; (3) worse; and (4) died. The neurological improvement is evaluated in %.

Total removal was performed in 9 of the 10 ependymomas of the cauda equina, subtotal in 1. All the operated patients are living and in satisfactory conditions with an average survival of 145 months after surgery (Table 2).

From the 21 intramedullary ependymomas who had radical removal of the tumour and a follow-up ranging from 12-310 months (average survival: 124 months), 11 are living well without serious neurological deficit and with perfect use of all their extremities; 8 are well with slight neurological deficit and normal use of their extremities and 2 are ambulant with the help of the cane (Table 2).

Out of 9 patients who had only subtotal removal of the tumour (followed in 8 by X-ray radiation), 2 patients died, one after 2 years, probably from the tumour, and one after 7 years from a heart-attack. One patient was lost at the follow-up. From the 6 living patients with a follow-up ranging from 25-240 months (average survival: 103 months), 1 did not benefit from the operation, while the others showed a 20% improvement as compared to the preoperative state (Table 2).

0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0			Neuro state	rolog	Neurological state		Neurol. %)	-ωτ әл	лє Dєзቲр	γerapy	Jsol qu	Post	oper	Postoperative results			pəq	Lavivaus (
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$		Number	poog	ͻ ϥϐτͺϛ	.Yoder.	Poor		oberati	ijsr9q0	д Хел-Х	-wollow-	га	qII	III ^c	dd	элтти	кеорега	айтала) гиотада
$ \begin{bmatrix} 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \\ 1 & 1 &$	Preoperative Radical	70	I	m	15	6	50	л С		ъ		5				5		, c
9 - 2 6 1 45 9 2 5 1 1 5 0 2 2 2 1 1 5 0 1 2 2 1 1 65 20 1 5 0 1 2 2 1 1 55 - 5 1 1 2 1 4 2 55 - 2 1 2 1 2 10 - 4 2 55 - 2 1 2 1 2 10 - 4 2 55 - 2 1 2 1 2 1 1 2 1 1 2 1 2 1 1 2 1 1 1 2 1 1 1 2 1 1 1 2 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 </td <td>Postoperative</td> <td>-</td> <td>[</td> <td>ω</td> <td>7</td> <td>I</td> <td>85</td> <td>n n</td> <td>r</td> <td>r</td> <td>i</td> <td>1</td> <td>I</td> <td></td> <td></td> <td>_</td> <td>ı</td> <td>7</td>	Postoperative	-	[ω	7	I	85	n n	r	r	i	1	I			_	ı	7
7 2 2 1 1 65 20 1 2 1 1 2 1 1 2 1 1 2 1 1 1 2 1 1 1 1 2 1	Preoperative subtotal	đ	I	7	9	-	45	C	I	a		u	c			ų		100
2 2 2 55 1 - 1 - 1 2 5 2 1 - 1 - 1 2 2 	Postoperative	n	7	7	-	~	65	0	I	0	-	n	D			٥		2
	Preoperative Bionsv-Mielot	~	I	I	7	I	55	لر ا	I	~		~		.		~	I	ά
- 4 4 2 55 10 35 - 2 - 8 2	Postoperative	1	I	I	-	-	50	7		1		-		-		1		
10 35 - 2 - 8 2			ī	4	4	2	55											
	Cauda	10						35	ı	2	1	8	7			0	ı	145

Table 2. Intramedullary ependymomas

a Improvement. b No improvement. c Worse. d Died.

From the only 2 patients in whom a posterior myelotomy and biopsy were performed by mistake, 1 showed an improvement that lasted almost 2 years and then a progressive worsening; the other showed a slight steady improvement for 12 years (Table 2).

Discussion

When we evaluate the long-term resulgs and compare them with those of other series $(\underline{1}, \underline{2}, \underline{3}, \underline{4}, \underline{5}, \underline{8}, \underline{9})$, we can draw the following conclusions:

- The best functional long-term results are obtained in patients that had total tumour removal. It is interesting to note that average survival does not differ significantly between patients who had either total or only partial removal or myelotomy of the lesion (radical removal: 124 months; subtotal removal: 103 months; posterior myelotomy: 98 months) while the neurological functional recovery is highly favourable in the patients who had total removal.
- 2. Histological differences among ependymomas do not seem to have any bearing on the long-term results. There is no doubt that in the past some tumours, when looked at with the naked eye, seemed infiltrating, and thus discouraged radical removal. The use of the operating microscope has cleared the perplexity.
- 3. Location and extension of the tumour still have a prognostic value, but less than in the past. Bulbar or high cervical ependymomas are associated with greater surgical risk and postoperative complications. The use of the microscope has improved prognosis even for tumours of this region.
- 4. Functional recovery is strictly related to the preoperative neurological state of the patient. The best results have been obtained in patients with slight neurological deficit and with preoperative symptoms not exceeding 2 years.
- 5. Postoperative radiation treatment seems to help some of these patients, particularly those who have had posterior mielotomy.

- BROAGER, B.: Spinal intramedullary glioma, subtotal or partial excision and X-ray treatment in five cases. Minerva Neurochir. 9, 1-5 (1965)
- CHIGASAKI, H., PENNYBACKER, J.B.: A long follow-up study of 128 cases of intramedullary spinal cord tumours. Neurol. Medicochir. <u>10</u>, 25-66 (1968)
- 3. FISCHER, G., TOMMASI, M.: Spinal ependymomas. In: Handbook of Clinical Neurology, Vol. 20. VINKEN, P.J., BRUYN, G.W. (eds.), pp. 353-387. North-Holland 1976
- 4. FISCHER, G.: Les ependymomes intrarachidiens. Neurochirurgie <u>23</u> (Suppl. 1), 149-215 (1977)
- 5. GREENWOOD, J.: Intramedullary tumors of spinal cord. A follow-up study after total surgical removal. J. Neurosurg. <u>20</u>, 665-668 (1963)
- GUIDETTI, B., FORTUNA, A., MOSCATELLI, G., RICCIO, A.: I tumori intramidollari, pp. 1-408. Roma: Il Lavoro Neuropsichiatrico 1964

- 7. GUIDETTI, B.: Intramedullary tumours of the spinal cord. Acta Neurochir. 17, 7-23 (1967)
- 8. NITTNER, K.: Raumbeengende Prozesse in Spinalkanal (einschließlich Angiome und Parasiten). In: Handbuch der Neurochirurgie, Bd. 7/2. KRENKEL, W., OLIVECRONA, H., TONNIS, W. (eds.), pp. 1-606. Berlin, Heidelberg, New York: Springer 1972
- 9. SLOOFF, J.L., KERNOHAN, J.W., Mac CARTY, C.S.: Primary intramedullary tumours of the spinal cord and filum terminale, pp. 1-255. Philadelphia: W.B. Saunders 1964

Diagnostic Problems of Neurosurgical Interest in Tick-Borne Encephalitis

J. SZÁNTŎ

In Central Europe tick-borne encephalitis is one of the most frequent viral diseases of the central nervous system. There are geographic areas where these ticks occur in masses, and frequent visitors to such forests are most likely to be infected. The vector belongs to the genus Flavivirus of the Toga family.

From 1955 through 1975 the total number of cases encephalitis and serous meningitis recorded in Hungary amounted to 18,774. Blood serum could be studied in 4,711 patients. Of these, 1361 contained HI antibodies against the virus of tick-borne encephalitis.

A strong seasonal preponderance is characteristic, the incidence being the highest during the summer months.

In Hungary, Zala county seems to be a geographical predilection site.

During the last 20 years, the clinical diagnosis was established on the basis of the HI tests in 644 cases recorded in Zala county. In recent years there is a definite rise in incidence.

Incubation time is 1 or 2 weeks. In most cases the disease presents with general symptoms such as headache and fever. It manifests itself either as a slight cold with temperature, but without any sign of CNS involvement, or in the form of a mild meningitis. It may also take a severe course with occasional unilateral paralysis and disturbed consciousness. The first, so-called viraemic phase, may end in a short afebrile period. This is followed by the second phase, in which the central nervous system becomes actually involved. The stiff paralysis of the muscles innervated by the cervical and upper thoracic cord is a typical but not indispensable sign.

Prognosis depends on the clinical picture. From the meningeal form the patient usually recovers within 1-2 weeks. In general, however, severe paralyses required prolonged treatment. In these cases, residual symptoms are frequent. Mortality is estimated at about 1%; in Zala county 5 of the 644 patients died.

Diagnosis is based primarily on the clinical symptoms, on CSF findings, as well as on the HI tests. Those clinical forms associated with CNS involvement can not be differentiated with certainty from intracranial space-occupying lesions during the acute phase. The latter usually produce papilloedema which makes the immediate CSF test questionable. In 6 of our cases carotid angiography was necessary to clarify the diagnosis.

The following is a characteristic case:

A 37-year-old male had a tick removed from his skin 18 days before the manifestation of the first symptoms. He was admitted to our hospital on account of headache of 2 days duration, nausea, visual and speech disturbances, somnolence and weakness in the left extremities. Physical examination disclosed a hyperaemic fundus left central facial paresis, severe paralysis of the left extremities with reflex disorders characteristic of central nervous lesions. The patient was somnolent throughout. Due to the strong suspicion of an intracranial space-occupying lesion, also supported by the echogram (showing slight dislocation), and by positive EEG findings, carotid angiography was performed. This revealed only signs of edema of the right hemisphere. CSF findings unequivocally indicated an inflammatory process.

The diagnosis of tick-borne encephalitis was established on the basis of the 3 HI tests (1:40, 1:160, 1:80). The patient died after deepening of the coma, with tetraplegia and bronchopneumonia, on the 30th day of the disease.

The histological changes were typical of tick-borne encephalitis, and could be observed in the Ammon's horn, thalamus and cerebellum (Figs. 1, 2). The inferior olive and the basal part of the pons also displayed characteristic alterations.

It is of particular interest that this is the first human case in which the virus of tick-borne encephalitis could be identified electron microscopically in the brain, i.e. in the thalamus, cerebellum and substantia nigra.

- KULI, V., MOLNIR, E.: Szerologiailag igazie kallanci-enephalitis esceek epidemiologiai elemzek (in Hungarian). Medicus universalis XI/3, 139-141 (1978)
- KORNYCY, St.: Contribution to the histology of tick-borne encephalitis. Acta Neuropathol. 43, 179-183 (1978)
- MAZLO, M., SZAULO, I.: Morphological demonstration of the virus of tick-borne encephalitis in the human brain. Acta Neuropathol. <u>43</u>, 251-253 (1978)

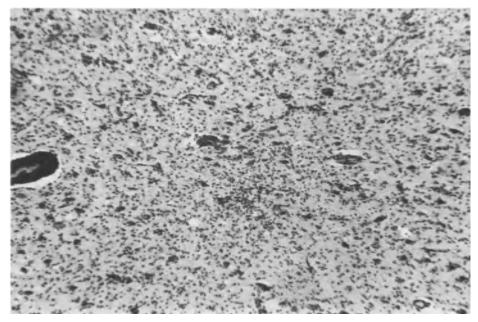


Fig. 1. Thalamus: infiltrated vessel walls, diffuse glial proliferation with accumulation in the middle of the picture

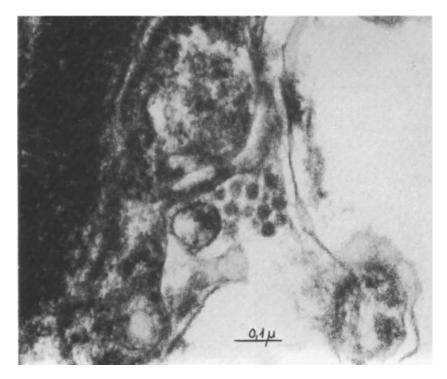


Fig. 2. Group of dark, round particles, diffuse glial proliferation with accumulation in the middle of the picture $% \left(\frac{1}{2} \right) = 0$

Experimental Investigation on the Development of Brain Abscess in Cats

T. WALLENFANG, J. BOHL, K. KRETZSCHMAR, and M. MAYER

The prognosis of brain abscess is determined by brain edema, intracranial pressure and capsule formation (1, 3, 12).

The aim of the present investigation was to study the alterations of these factors concomitant with the sequence from the acute to the chronic abscess. The measurements of the intracranial pressure are correlated with the biochemical and morphological findings.

Materials and Methods

The right hemispheric white matter of 56 cats, anesthetized with ketamine (20 mg/kg) was inoculated stereotactically with 0,03 ml of a mixture of agar and Staphylococcus aureus-containing bouillon (ratio 1:3) (2, 6, 11, 13). The amount injected contained between 1 and 1,5 million Staphylococcus aureus. The contralateral hemisphere of a group of 12 animals, who had received the same quantity of mixture only (without Staphylococcus aureus) served as control. The extent of edema was visualized by intravenous injections of 2% Evans Blue (1 ml/kg).

The development of the acute abscess and its encapsulation was traced by means of repeated computed tomography (CT). Intraventricular pressure (IVP) measurements were performed over two hours under steadystate conditions of blood gases and blood pressure in six animals after 2, 4, 7, 10 and 20 days. Brain tissue samples around the abscess and from the control were removed to determine water and electrolyte contents.

After establishing the presence of an abscess with the help of computed tomography in 18 animals, treatment with dexamethasone¹ (0,5 mg/kg body weight) and cefazedon² (50 mg/kg body weight) was instituted on the seventh day. Eight animals with brain abscess were perfused with 3.9% glutaraldehyde and examined with the light microscope as well as under the electron microscope.

Results

After inoculation with Staphylococcus aureus, an abscess developed in all cases accompanied by a macroscopically visible edema in the white matter of the inoculated right hemisphere.

¹ Decadron (MSD Sharp + Dohme, München).

² Refosporin (Merck, Darmstadt).

By means of CT the formation of capsules was visualized distinctly. In the first days, a maculated poorly defined area of increased density occurred after injection of contrast medium as a sign of inflammatory reaction and circumscribed disturbance of the blood-brain barrier (14). The maximum circumference of the abscess was measured between the seventh and tenth day. At this date, the abscess appeared as a mixed dense ring structure with central necrosis. Subsequently, both diminution and homogenous density of the ring structure corresponding to a capsule were demonstrable (Fig. 1).

Neurological symptoms were most evident in the acute stage of the abscess. Progressive deterioration of consciousness was observed from the fourth to the eighth day in all cases. Mydriasis and hemiparesis in 83% was most evident around the seventh day. While encapsulation was developing, these symptoms recurred on the subsequent 3-4 days; only the animals with a herniation into the tentorial notch showed no or an extremely delayed recovery.

With the tissue necrosis produced by the Staphylococcus aureus, the edema around the acute abscess increased rapidly. The maximum water accumulation was found from the seventh until the tenth day (82.3 g/ 100 g w.wt.) and was over 12% higher than in the contralateral hemisphere and in the control group which had only received agar and bouillon. Corresponding to the progressive extent of the edema in the extracellular space, the water content later increased in the remote areas. On the seventh day, the water accumulation was nearly as high (80.4 g/100 g w.wt.) as adjacent to the abscess. With the development of encapsulation, the water content decreased in the remote areas (73.1 g/100 g w.wt.) but only slightly in the areas adjacent to the abscess (80 g/100 g w.wt.) (Fig. 2).

However, the increasing space-occupying mass of the abscess, above all the spreading edema in the white matter and towards the lateral ventricle, resulted in an increase in intraventricular pressure (9). On the seventh day the intraventricular pressure was nearly eight times as high as in the control group. When intracranial pressure exceeded ten times the normal level in this most critical time of abscess development, it always resulted in a herniation in the tentorial notch, which ordinarily leads to a progressive neurological impairment. With encapsulation of the abscess, the intracranial pressure decreased. However, it remained significantly increased analogous to the slow decrease of edema (Fig. 3).

The therapy with dexamethasone (0.5 mg/kg body weight) and cefazedon (50 mg/kg body weight) beginning at the time of maximal edema and highest intracranial pressure brought about a significant reduction of the water content in all areas of the white matter in the first three days, whereas a high level of edema persisted in the adjacent areas during the following days, also during the encapsulation. In the remote areas, the water content continued to decrease slowly (7, 8) (Fig. 2).

In accordance with these changes of water content, there was a striking decrease of intraventricular pressure in the first three days after therapy but IVP remained higher than normal in the phase of encapsulation (Fig. 3).

Histological and ultrastructural studies on the seventh day revealed a massive exudation of a protein-rich fluid into the greatly enlarged extracellular space with deposition of fibrin and erythrocytes and accumulation of macrophages. In the neighborhood of the abscess (until the tenth day), we found narrow gaps in the endothelial layer in many small blood vessels as well as a widening of the intercellular clefts between the endothelial cells and an increase of basal lamina material.

On the twentieth day, the abscess was already surrounded by a thick, dense capsule of connective tissue with numerous blood vessels, with chronic inflammatory perivascular infiltrations and with an intense foreign body reaction. The perifocal chronic edema and massive loss of nerve fibers had led to a remarkable reactive astrocytosis in the surrounding white matter $(\underline{2}, \underline{4}, \underline{5}, \underline{8})$ (Figs. 4, 5).

Discussion

In accordance with clinical experience, the level of consciousness depended on the size of the abscess and associated edema in our experimental model $(\underline{1}, \underline{12})$. The greatest mass effect could be observed in the encephalitic stage. The appearance of contrast ring enhancement in the CT around the seventh day after a diffuse increase of density in the area of encephalitis cannot be equated with abscess capsule formation. Parallel to the increase of the contrast enhancement and the subsequent ring appearance, the edema spread in all areas of the right hemispheric white matter $(\underline{10})$. When a shifting of the ventricular system could be observed in the CT, the IVP was extremely high. Between the acute and chronic stage of the abscess, there is a period in which it is not possible to differentiate contrast enhancement caused by abnormal permeability of capsule vessels from that caused by progressive inflammation.

Corresponding to the increasing morphological density of the capsule, the abscess dimensions seem to decrease in the CT, perhaps due to the diminishing perifocal inflammation. Further investigations into the effect of antibiotics and corticosteroids are required in order to handle the problems in clinical practice.

- CAREY, M.E., CHOU, S.N., FRENCH, L.A.: Experience with brain abscesses. J. Neurosurg. 36, 1-9 (1972)
- FALCONER, M.A., McFARLAN, A.M., RUSSEL, D.S.: Experimental brain abscesses in the rabbit. Br. J. Surg. 30, 245-260 (1943)
- 3. GARFIELD, J.: Management of supratentorial intracranial abscess; a review of 200 cases. Brit. Med. J. 2, 7-11 (1969)
- HIRANO, A.: The fine structure of brain edema. In: The structure and function of nervous tissue, Vol. 2, pp. 69-135. BOURNE, G.H. (ed.). New York 1969
- 5. LONG, D.M., MAXWELL, R.E., FRENCH, L.A.: The effects of glucosteroids upon cold induced brain edema. II. Ultrastructural evaluation. J. Neuropathol. Exp. Neurol. 30, 680-697 (1971)
- 6. MARKLEY, G.M.: A method for the experimental production of brain abscess. Proc. Soc. Exp. Biol. Med. <u>47</u>, 171-175 (1941)
- MAXWELL, R.E., LONG, D.M., FRENCH, L.A.: The effects of glucosteroids on cold induced brain edema. Gross morphological alterations and vascular permeability changes. J. Neurosurg. <u>34</u>, 477-487 (1971)
- QUARTEY, G.R.C., JOHNSTON, J.A., ROZDILSKY, B.: Decadron in the treatment of cerebral abscess. J. Neurosurg. 45, 301--10 (1976)

- 9. REULEN, H.J.: Vasogenic brain edema. Br. J. Anaesth. <u>48</u>, 741-752 (1976)
- 10. REULEN, H.J., GRAHAM, R., SPATZ, M., KLATZO, J.: Role of pressure gradients and bulk flow in dynamics of vasogenic brain edema. J. Neurosurg. <u>46</u>, 24-35 (1977)
- 11. THOMAS, L.: A single stage method to produce brain abscess in cats. Arch. Pathol. 33, 472-476 (1942)
- 12. WALLENFANG, Th., REULEN, H.J., SCHINDLING, H.: Investigation on the prognosis of brain abscess. In: Advances in Neurosurgery, Vol. 4. WÜLLENWEBER, R., BROCK, M. (eds.), pp. 296-299. Berlin, Heidelberg, New York: Springer 1977
- 13. WALLENFANG, Th., BOHL, J., SCHREINER, G.: Experimental brain edema in acute and chronic brain abscess in rabbits and its morphological alterations. In: Advances in Neurosurgery, Vol. 7. MARGUTH, F., BROCK, M., KAZNER, E., KLINGER, M., SCHMIEDEK, P. (eds.), pp. 304-310. Berlin, Heidelberg, New York: Springer 1979
- 14. ZIMMERMANN, R.A., BILANUIK, L.T., SHIPKIN, P.M., et al.: Evolution of cerebral abscess: Correlation of clinical features with computed tomography. Neurology <u>27</u>, 14-19 (1977)

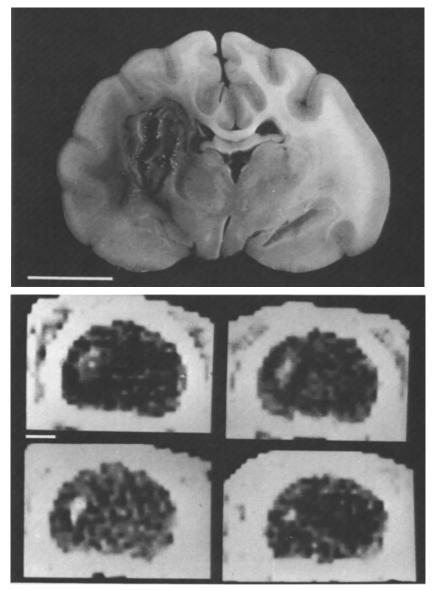


Fig. 1. Above: Acute abscess, seven days after inoculation. Necrosis and extensive edema in the right hemisphere result in an extreme shifting of the midline (white line = 1 cm). Below: Computed tomography of the same animal 8, 11, 18 and 25 days after inoculation (white line = 1 cm)

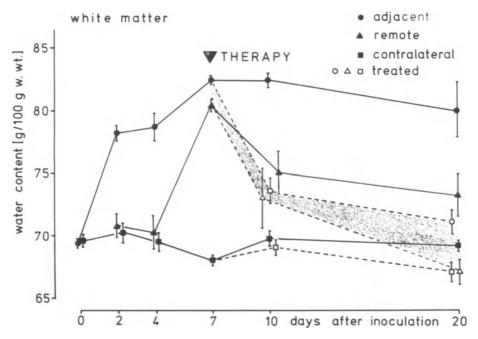


Fig. 2. Changes of water content (g/100 w.wt.) in the various areas of the white matter. When encapsulation developed from the seventh day onwards, water content decreased significantly in the areas remote from the abscess. After beginning therapy on the seventh day, edema decreased in all areas until the tenth day. The right and left hemisphere of the abscess group with and without therapy and of the sterile control group (agar+ bouillon without Staphylococcus aureus) are compared. x + Sx are given

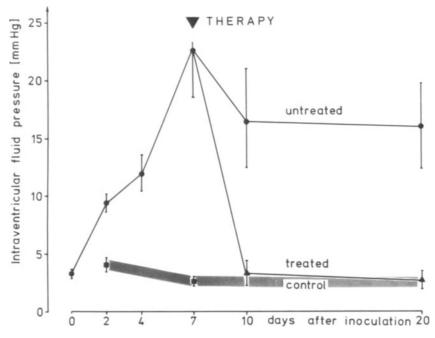


Fig. 3. Intraventricular fluid pressure, measured in the contralateral ventricle on the different days, during development of the abscess in the right hemisphere. Therapy was performed from the seventh day onwards. IVP remained high after encapsulation, in contrast to the treated animals. $\bar{x} + S\bar{x}$ are given

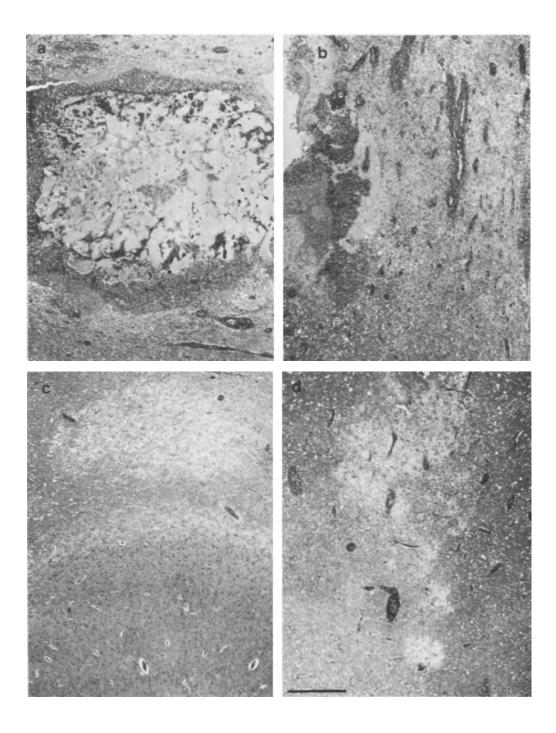


Fig. 4 a-d. Acute brain abscess on the seventh day (paraffin embedding, HE). a Acute abscess in the cerebral white matter with a mixture of bouillon, agar and clumps of bacteria in the center, with cell debris and a numerous leucocytes at the border and with severe perifocal edema. b Intensive inflammation in the cerebral white matter in the close vicinity of the abscess, especially in enlarged perivascular spaces. c Focally pronounced edema reaches subcortical white matter, while the cortex seems unchanged. d Patchy distribution of brain edema even at some distance from the abscess with acute inflammatory perivascular infiltration (black line = 0.5 mm)

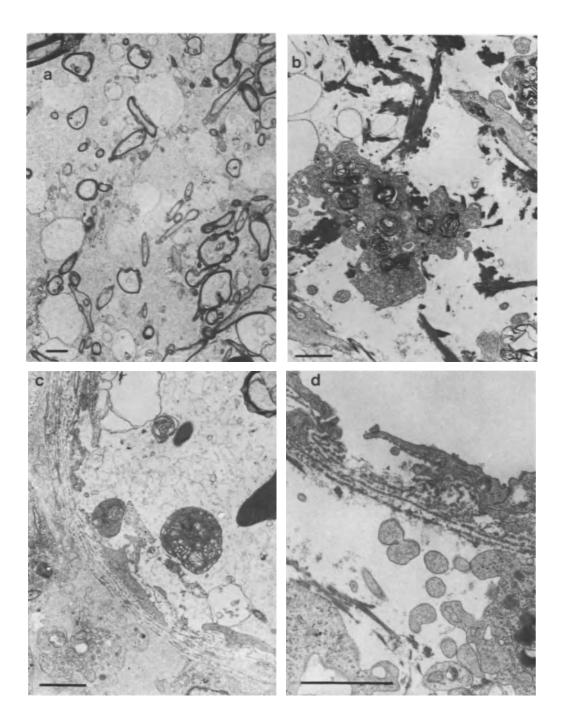


Fig. 5 a-d. Acute brain abscess on the seventh day (ultrastructure). a Severe brain edema with protein-rich fluid in the extremely enlarged extracellular space and with swelling of astrocytic processes. b Macrophage and fibrin deposition in the perifocal zone of severe inflammatory edema. c Wall of small blood vessel (venule) in close proximity to the abscess with several gaps in the endothelial cell layer and with duplicating and splitting basal lamina material. d Wide gap between two endothelial cell processes with fibrin deposition in perivascular space. (Black lines = 2 µm) Study of Various Types of Experimental Brain Edema Using the Electrical Impedance Technique and the Electron Microscope A. SCUCCIMARRA, V. PENSABENE, N. PANDOLFO, and F. DE BLASI

Introduction

The aim of our study was to examine whether the more representative models of experimental brain edema are either purely vasogenic or cytotoxic, or whether, instead, both of these types of edema are present, one prevailing over the other. The methods of the electrical impedance technique and the electron microscope were used.

Material and Methods

Our experiments, carried out on 9 dogs, are still in progress. We induced brain edema using three methods which we believe are the principal models of the various types of brain edema, and those wich most closely approximate the clinical picture:

- 1. clamping of a carotid artery;
- 2. supratentorial extradural compression (technique advanced by REID and CONE in 1939) $(\underline{3})$;
- 3. localized cold induction (KLATZO, 1958) (1).

During the experiments, the dogs were maintained under general anesthesia with endotracheal intubation.

The values of the electrical impedance were monitored by an impedanzometer which we had made (it is at a constant current of 1,8 μA and at the frequency of 50 Khz).

The exploring electrode (a stainless steel wire insulated with teflon for its entire length except 2-2,5 mm at one tip which is slightly sharpened) was fixed in the cerebral cortex. The other (a common 23 Ga 1/2 needle) in the muscles of the animal's front leg.

The values of the electrical impedance of the control cortex, measured for about 6 hours, showed variability between 900 and 1100 ohms.

The electrical impedance is inversely proportional to the amplitude and to the state of hydroelectrolytic repletion of the extracellular space of the tissue. Consequently, every time that a flux of ions is directed from the extracellular compartment to the intracellular one, with the resulting reduction of the former, the electrical impedance of the tissue increases. Every time a flow of ions is directed from the blood vessels in the extracellular space, the impedance of the tissue decreases. From this the importance of recording the impedance in cases of brain edema can be deduced, aiming at obtaining useful information on the movements of the ions in the various phases. The samples of cerebral tissue for microscopy were taken far from the electrodes. They were seen under the optic and under the electron microscope. For the latter the fixation was carried out in OsO_2 , either immediately or following prefixation with glutaraldehyde.

Results

Let us now examine separately the results obtained in each of the three types of brain edema.

Figure 1a shows a graphic demonstration of the variation in the electrical impedance in dogs in whom ischemia and thus brain edema had been induced by occluding a carotid artery.

As can be seen, at varying moments of half-an-hour to one hour after the moment of closure, the impedance values increase, reaching their maximum at 2 - 21/2 hours.

After 3-4 hours, however, the impedance values decrease progressively to very low values.

In supratentorial extradural compression, the graphs of the variations of electrical impedance (Fig. 3) show how, half-an-hour from the end of compression, this progressively decreases.

Later, however, about 2-3 hours later, the impedance increases. The graphs of the variations in electrical impedance in edema caused by localized induction of cold are similar to those of the preceding type of edema (Fig. 4). It should be noted that the later phase of increase in impedance values is less pronounced.

Discussion

In the first phase of ischemic edema, the increase in the impedance can be explained by a flow of water from the extracellular space (which thus decreases) towards the intracellular compartment, which is logically the first to be involved by ischemia: probably the energy systems of the cell are influenced, with the consequent accumulation of catabolites in the cytoplasm which attracts water and ions by osmosis (cytotoxic edema).

In the second phase of ischemic edema, the impedance values decrease, although the brain remains edematous. Indeed, it appears more swollen; and this cannot be interpreted as a regression of the cytotoxic edema, but as the overlapping of a vasogenic, extracellular phase over the intracellular one. This is caused by the break-down of the haematoencephalic barrier. The images obtained on both the optic and the electron microscope confirm this biphasic character of ischemic edema (Fig. 2a,b). In the first phase, when the impedance values reach their maximum, we note remarkable hydration of the cytoplasm and the nucleus of the cells with evident alteration of the cell-organelles (mitchondria) (Fig. 2a).

In the second phase, when the impedance values decrease, next to these signs of intracellular edema (cytotoxic) we note an increase in the extracellular space, particularly around the vessels (vaso-genic edema) (Fig. 2b).

In supratentorial extradural compression, half-an-hour after the end of compression, electrical impedance progressively decreases: this can be interpreted as an increase in the extracellular space owing to the early break-down of the haemato-encephalic barrier (vasogenic edema).

In a second phase, in this edema, the impedance increases although the brain remains swollen: here too, the phenomenon cannot be explained in terms of regression of the edema, but only by the flow of water and ions from the extracellular space towards the intracellular one, i.e. a cytotoxic phase is added to the original vasogenic edema. Moreover, it is conceivable that in this type of edema too, the cellular compartment is damaged both by the length of the compression period and the difficulty of exchange between cells and vessels owing to the increased extracellular space. In these cases, too, our electron microscope studies supported the concepts suggested by the variations in electrical impedance. At about half-an-hour after the end of compression, there is involvement of the extracellular space without affecting the intracellular one (Fig. 2c). Instead at three hours, we see how the intracellular space is now involved (Fig. 2d). The considerations on the edema caused by localized induction of cold are similar to those of the preceding type of edema. Only, the second phase, in which electrical impedance increases, is less pronounced: i.e. the cytotoxic phase is therefore less evident and the edema is more clearly vasogenic.

Our study thus affirms that the first (in 1967) KLATZO's classification (2) in vasogenic and cytotoxic edema is not absolute, that is, it does not appear that only vasogenic or only cytotoxic edemas exist, but that in all the models of brain edema which we investigated there are two phases, even though one phase prevails over the other: in effect, the cytotoxic type prevails and appears first in ischemic edema; on the contrary, the vasogenic type is more precocious and prevails in extradural compression and in localized cold-induced edema. The first phase prevails over the second because it is the one on which the edema-producing factor acted most. Naturally our study is not complete, further experimentation is necessary.

Conclusion

On the basis of their experiments with dogs the authors affirm that KLATZO's distinction between vasogenic and cytotoxic edema is not absolute: all the models of brain edema investigated are biphasic with the first phase (vasogenic or cytotoxic) prevailing over the second, because that is the one on which the edema-producing factor acted chiefly.

- KLATZO, I., PIRAUX, A., LASKOWKI, E.J.: The relationship between edema, blood brain barrier and tissue elements in local brain injury. J. Neuropath. Exp. Neurol. 17, 548-564 (1958)
- KLATZO, I.: Neuropathological aspects of brain edema: Presidential address J. Neuropath. Exp. Neurol. <u>26</u>, 1-14 (1967)
- REID, W.L., CONE, W.V.: The mechanism of fixed dilatation of the pupil resulting from ipsilateral cerebral compression. J. Amer. Med. Ass. <u>112</u>, 2030-2034 (1939)

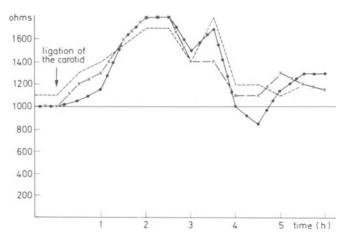


Fig. 1. Variations of electric impedance in ischemic brain edema

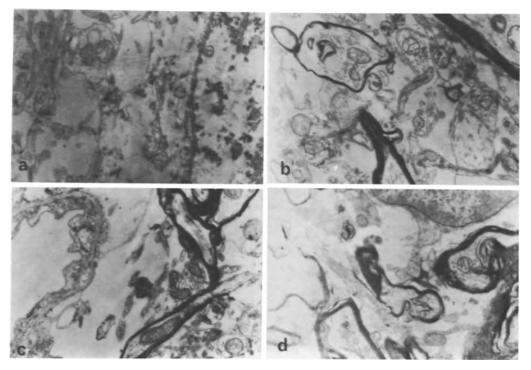


Fig. 2. a, b ischemic brain edema; \underline{c} , d brain edema produced by supratentorial extradural compression

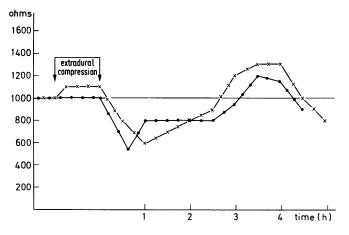


Fig. 3. Variations in electric impedance in brain edema produced by supratentorial extradural compression

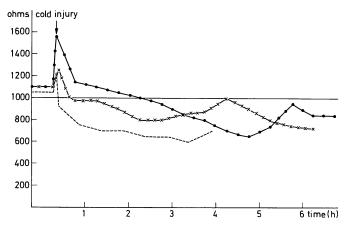


Fig. 4. Variations in electric impedance in cold-induced brain edema

Osmoregulation, Brain Damage and Prognosis M. GAAB, H. A. TROST, I. HAUBITZ, K. W. PFLUGHAUPT, and E. HALVES¹

Introduction

In recent years, many reports have appeared on the *central regulatory* disturbances following brain trauma, in which hyperglycemia (9, 22), azotaemia (5) and hyperthermia (20) have been interpreted as the consequences of a diencephalic trauma. The regulation of the water balance is also a diencephalic hypothalamic function. Consequently, diabetes insipidus is not uncommon after brain injury (26). Similarly, a malfunction in ADH-secretion with water intoxication, the so-called SCHWARTZ-BARTTER-Syndrome (7, 21), has also been described. There is, however, little quantitative data on water balance disturbances following brain trauma.

An important parameter in the control of the water balance is the maintenance of a constant *osmolality* $(289 \pm 8 \text{ mmol/kg}^2)$ in all body fluids, with the exception of excreta $(\underline{18}, \underline{29})$. In our study we examined clinically, the behaviour of this *osmoregulation* in patients with severe brain injury, and experimentally, in dogs, the reactions of disturbances in osmolality on the diencephalon.

Patients, Materials and Methods

Clinical Measurements

In 102 patients with severe brain injury (at least 48 h coma), the serum osmolality was measured by cryoscopy, at least once daily, and *electrolyte* and *metabolite* contents were determined simultaneously in an 18-channel autoanalyser, so that the computation of osmolality was possible (osmolality (mmol/kg) = $1.86 \text{ Na}^+ (\text{mmol/l}) + \text{Gu} (\text{mg/dl})/18 + \text{BUN} (\text{mg/dl})/2.8; 10,30)$. The intracranial pressure (ICP) was recorded by miniature force transducers (14). In patients without ICP disturbance, the corresponding CSF values were measured. The clinical development was defined according to a modified Glasgow-Coma-Scale (Table 1).

All Patients received similar high caloric diets through naso-gastric tubes and routine i.v. infusions (8) and also underwent a high dosage dexamethasone therapy programme $(\underline{17})$. Water and electrolyte losses were balanced. Patients suffering from diabetes insipidus over 24 h, kidney damage, pretraumatic diabetes mellitus or hyperthermia greater than 39° C over 48 h, were excluded. The data was statistically evaluated by a TR 440-computer. For control purposes, similar measurements were made on 30 patients with damaged intervertebral discs. All data is expressed as mean values + SEM.

¹ We would like to express our thanks to the Fresenius Company for provision of the Knauer osmometer.

² mmol/kg (SI) = mosmol/kg.

С	Eyes	spontaneously	4 points				
0		to verbal command	3				
N	opening	to pain	2				
S		no response	1				
С	best	obeys commands	6				
I		localizes pain	5				
0	motor	flexion bilateral	4				
U	resp.	extension unilateral	3				
S	- ·	extension bilateral	2				
N		no response	1				
E	best	oriented and convers.	5				
Ş	Dese	disoriented and convers.	4				
S	verbal	inappropriate words	3				
5		incomprehensible sounds	2				
	resp.	-	2				
-		none	1				
Р	pupil	normal, L +	1				
U	form	normal or small, L (+)	6				
Р		enlarged, T +	5				
I	and	small, L -	4				
L	reaction	enlarged, round, L -	3				
		distorted, L -	2				
		bilat. max. mydriasis	1				
Bes	Best score = 22, worst score = 4						

Table 1. Clinical assessment according to the point scale (in accordance with Glasgow Coma Scale)

Experimental Investigations

To test the reaction of hyperosmolality on the diencephalon, we increased the serum osmolality in 9 dogs, to $343 \pm 8 \text{ mmol/kg}$, for a period of 7 days, by dehydration. Following perfusion with glutaral-dehyde (12), the hypothalamic-hypophyseal system was prepared "en bloc" and examined by light and electron microscopy after frontal section. H.E. stains, cell pictures and neuro-secretion stains were carried out according to the procedures of BOCK, STERBA and ROSSBACH (13), and electron microscopy was performed with a Zeiss EM 9 S2/ Siemens 101.

Results

Clinical Measurements

While the serum osmolality in the 30 controls lay in the normal range (286 ± 13 mmol/kg), patients with severe brain damage showed considerable variations in osmolality. The individual values (Fig. 1a) deviated far beyond the accepted normal range, with a preponderance of hyperosmolar values, especially in non-survivors. The regression also lay above the normal limit. Only occasionally, were hyposomolar values recorded i.e. less than 245 mmol/kg, particularly in non-survivors (Fig. 1a).

When we compare survivors and non-survivors (Fig. 1, 2), we see that, in the latter, the osmolality is significantly higher, with mean values in the critical range of 320 mmol/kg $(\underline{4}, \underline{24})$ which correlates with the marked deterioration of their clinical condition. The formal calculation for osmolality does not give this information despite correction (5-8 mmol/kg: 10,30), as this correction is invalid when the value exceeds normal ranges. Correspondingly, the serum-Na⁺ (likewise Cl⁻), which is directly related to the osmolality, is not significantly higher in non-survivors, generally lying in the normal range. Glucose is certainly elevated in non-survivors but only exceeds the therapy threshold values (9) of 200 mg/dl between the 12th and 14th days. Its increase corresponds to that of the osmolality. Urea-N shows a similar increase but, like glucose, does not provide a sufficient explanation for the considerable disturbance of osmolality.

The close *relationship* between the disturbance of *osmolality and prognosis*, which does not exist for glucose and BUN, is shown in Fig. 1b. We observed a high mortality rate (>80%) when osmolality values exceeded 300 mmol/kg for 4 days, 320-340 mmol/kg for 2 days and >340 mmol/ kg for 1 day.

In contrast to predominant conditions of hyperosmolality, conditions of hypo-osmolality occur much less frequently. This water intoxication can however result in acute brain edema, which we have observed in 4 children in the last 2 years, following brain trauma (Fig. 3): Between the 3rd and 6th day after trauma, the osmolality and Na⁺ decreased sharply, despite a positive fluid balance, and an uncontrollable increase in ICP appeared simultaneously (Fig. 3a). We were able to save 3 children by decompressive trepanation and water restriction (Fig. 3b).

Experimental Investigations

All dogs showed signs of disturbance of consciousness when osmolality was high: In the supraoptic nucleus and in the neurohypophysis, scarcely any neurosecretion was evident (13). Together with these indications of functional exhaustion, almost 30% of the ganglia in the supraoptic nucleus, were found to be necrotic with predominantly osmophilic cell pyknosis (Fig. 4a). We also found glia degeneration with the typical, so-called "Verney's vesicles" (Fig. 4b); (13, 28) for the supraoptic nucleus in dehydration.

Discussion

After severe brain injury, considerable disturbance of osmoregulation occurs with predominantly hyperosmolar disturbances. In part, these run parallel to hyperglycemia and azotaemia, attributed to diencephalic damage (5, 9, 22). This hyperosmolality, nevertheless, possesses considerable prognostic value. Consistently high osmolality values were recorded in the majority of cases just prior to death from increased ICP. The calculated osmolalities were consistently lower than those determined directly and only the latter were shown to correlate with a true disturbance of osmoregulation by multiple regression. As osmolality is regulated by the hypothalamus, which contains the ADH-secreting cells with their own osmoreceptors (28), we therefore conclude that, as with the control of glucose and urea concentrations, the disturbance in osmoregulation is the result of diencephalic malfunction. Such hyperosmolar disturbances were described as early as 1939 (1) in brain tumors and particularly in the diencephalic tumour, and often without diabetes insipidus (3, 11). Similar disturbances have also been reported in cases of injury to the 3rd ventricle by endoscopy $(\underline{2})$. The absence of diabetes insipidus may be explained by a simultaneous malfunction of the anterior hypophyseal lobe and a subsequent thyroid deficiency (19, 25). This requires further endocrinological investigation.

The experimental investigations which indicated considerable ganglia and glia cell necrosis in the supraoptic nucleus induced by hyperosmolality, are comparable to those described in cholera patients, and suggest irreversibel hypothalamic damage perhaps resulting from attempted maintenance of the osmolality (13).

In contrast to the above, conditions of hypo-osmolality resulting from over-secretion of ADH, occur much less frequently and appear to be more prevalent in children $(\underline{3}, \underline{27})$, incurring the risk of acute brain edema $(\underline{6}, \underline{27})$.

Our results indicate the importance of direct osmolality measurements after severe brain trauma. Hyperosmolar values of around 320 mmol, which result in a disturbance of the blood-brain-barrier (24), should be treated immediately; values >350 mmol/kg, which may lead to severe neurological disturbances (46), must however be reduced with great care due to the high risk of subsequent brain edema. Values below 260 mmol/kg must also be treated carefully because of the danger of diffuse brain swelling (water restriction and negative balance).

Osmolality disturbances after brain trauma present a further argument against the indiscriminate use of osmotherapy. In cases of hyperosmolality it may lead to acute toxic values $(\underline{15})$. The chronic use of hyperosmolar solutions further increases the osmolality by progressive Na⁺-retention (<u>16</u>).

Conclusion

Disturbances in osmoregulation occur after severe brain injury. More frequent are cases of hyperosmolar disturbances which correlate with a deteriorating prognosis and run parallel to hyperglycemia and azotaemia. Hypo-osmolar disturbances, due to excessive ADH secretion, occur less frequently, but may result in acute brain edema. These disturbances are interpreted as diencephalic damage and, in extreme cases, are indicative of "hypothalamic death". Direct measuring of osmolality, after severe brain trauma, is essential as the calculated values are invalid. Hyperosmolar disturbances of >310 mmol/kg, similarly hypoosmolar disorders of <260 mmol/kg require treatment but sudden changes in osmolality must be avoided due to the danger of brain edema. Our results provide a further argument against the indiscriminate use of osmotherapy in the treatment of brain trauma.

- 1. ALLOTT, E.N.: Sodium and chloride retention without renal disease. Lancet I, 1035-1037 (1939)
- AMACHER, A.L.: Endoscopic ventricul ostomy. Letter to the editor. Surg. Neurol. <u>9</u>, 450 (1978)
- ANDLER, W., ROOSEN, K., REINHARDT, V.: Hypothalamisch bedingte Störungen der Osmoregulation im Kindesalter. Neurochirurgia <u>22</u>, 56-68 (1979)
- 4. ARIEFF, A.I., GUISADO, R., LAZAROWITZ, V.C.: Pathophysilogy of hyperosmolar states. In: Disturbances in body fluid osmolality. ANDREOLI, Th.E., GRANTHAM, J.J., REUTER, F.C. (eds.), pp. 227-250. Bethesda/Baltimore: Williams and Wilkins 1977
- 5. AUER, L., HOLZER, H., TRITTHART, H., GELL, G.: Azotaemia in severe head injury - central dysregulation or renal failure? Acta Neurochir. <u>41</u>, 355-361 (1978)

- BAETHMANN, A.: Das Hirnödem mechanischer, zirkulatorischer, osmotischer, metabolischer und toxischer Genese. In: Der bewußtlose Patient. AHNEFELD, F.W. (ed.). Berlin, Heidelberg, New York: Springer 1978
- BARTTER, F.C., SCHWARTZ, W.B.: The syndrome of inappropriate secretion of antidiuretic hormone. Amer. J. Med. <u>42</u>, 790-806 (1967)
- BERG, G., MATZKIES, F.: Fortschritte auf dem Gebiet der künstlichen Ernährung. Deutsch. Ärztebl. 74, 2439-2441 (1977)
- 9. DELOOF, Th., BERRE, J., GENETTE, F., Van de STEENE, A., MOCCAWAD, E.: Disturbances of the carbohydrate metabolism in acute head trauma. Acta Neurochir., Suppl. <u>28</u>, 113-114 (1979)
- DORWART, W.V., CHALMERS, L.: Comparison of methods for calculating serum osmolality from chemical concentrations, and the prognostic value of such calculation. Clin. Chem. <u>21</u>, 190-194 (1975)
- 11. EMMRICH, P.: Das hyperosmolare Syndrom. Notfallmed. <u>3</u>, 515-521 (1977)
- GAAB, M., ENGELHARDT, F., SCHIMRIGK, K.: Fixation of the canine brain by carotis perfusion. Mikroskopie <u>31</u>, 107-110 (1975)
- 13. GAAB, M., ENGELHARDT, F.: Zur Genese der "Verney'schen Cysten" im Nucleus supraopticus. Verh. Anat. Ges. <u>69</u>, 579-586 (1975)
- 14. GAAB, M., KNOBLICH, O.E., DIETRICH, K., GRUSS, P.: Miniaturized methods for monitoring intracranial pressure in craniocerebral injury before and after operation. In: Advances in neurosurgery, Vol. 5. FROWEIN, R.A., WILCKE, O., KARIMI-NEJAD, A., BROCK, M., KLINGER, M. (eds.), pp. 5-11. Berlin, Heidelberg, New York: Springer 1978
- 15. GAAB, M., GRUSS, P., RATZKA, M., WODARZ, R.: Critical intracranial effecte of osmotherapy. In: Advances in neurosurgery, Vol. 6. WÜLLENWEBER, R., WENKER, H., BROCK, M., KLINGER, M. (eds.), pp. 193-205. Berlin, Heidelberg, New York: Springer 1978
- 16. GAAB, M., TROST, H.A., PFLUGHAUPT, K.W.: The prognostic value of osmolality within the first week of sustaining head injury. Acta Neurochir., Suppl. 28, 115-119 (1979)
- 17. GOBIET, W., BOCK, W., LIESEGANG, J., GROTE, W.: Treatment of acute cerebral edema with high doses of dexamethasone. In: Intracranial pressure III. BEKS, J.W.F., BOSCH, D.A., BROCK, M. (eds.), pp. 231-235. Berlin, Heidelberg, New York: Springer 1976
- 18. HENDRY, E.B.: The osmotic pressure and chemical composition of human body fluids. Clin. Chem. <u>8</u>, 246-265 (1962)
- 19. KLEEMAN, Ch.R.: Water metabolism. In: Clinical disorders of fluid and electrolyte metabolism, 2nd ed. MAXWELL, M.H., KLEEMAN, Ch.R. (eds.), pp. 215-295. New York: McGraw-Hill 1972
- LAUSBERG, G.: Zentrale Störungen der Temperaturregulation. Acta Neurochir., Suppl. 19 (1972)
- 21. PENNEY, M.D., WALTERS, G., WILKINS, D.G.: Hyponatraemia in patients with head injury. Intens. care Med. <u>5</u>, 23-26 (1979)
- 22. PENTELÉNYI, T., KAMMERER, L., PÉTER, F., FELUTE, M., KORÁGYI, L., STÜTZLE, M., VERESS, G., BEZZEGH, A.: prognostic significance of the changes in the carbohydrate metabolism in severe head injury. Acta Neurochir., Suppl. 28, 103-107 (1979)

- 23. ROBERTSON, G.L., ATHAR, S., SHELTON, R.L.: Osmotic control of vasopressin function. In: Disturbances of body fluid osmolality. ANDREOLI, Th.E., GRANTHAM, J.J., RECTOR, F.C. (eds.), pp. 125-148. Bethesda/Baltimore: Williams and Wilkins 1977
- 24. STERRETT, P.R., THOMPSON, A.M., CHAPMAN, A.L., MATZKE, H.A.: The effects of hyperosmolarity on the blood-brain barrier. A morphological and physiological correlation. Brain Res. <u>77</u>, 281-295 (1974)
- 25. SCHMIDT-NIELSEN, B.M., MACKAY, W.C.: Comparative Physiology of electrolyte and water regulation, with emphasis on sodium, potassium, chloride, urea, and osmotic pressure, In: Clinical disorders of fluid and electrolyte metabolism, 2nd ed. MAXWELL, M.H., KLEE-MAN, Ch.R. (eds.), pp. 45-93. New York: McGraw-Hill 1972
- 26. SHUCART, W.A., JACKSON, J.: Management of diabetes insipidus in neurosurgical patients. J. Neurosurg. <u>44</u>, 65-71 (1976)
- TRUNIGER, B.: Hyponatriämie, Hypoosmolalität und Wasservergiftung. Med. Klin. <u>70</u>, 1071-1079 (1975)
- 28. VERNEY, E.B.: The antidiuretic hormone and the factors which determine its release. Proc. Roy. Soc. B <u>136</u>, 25-106 (1947)
- 29. WARHOL, R.M., EICHENHOLZ, A., MULHAUSEN, R.O.: Osmolality. Arch. Intern. Med. <u>116</u>, 743-749 (1965)
- 30. WEISBERG, H.F.: Osmolality-calculated, "delta", and more formulas. Clin. Chem. 21, 1182-1185 (1975)

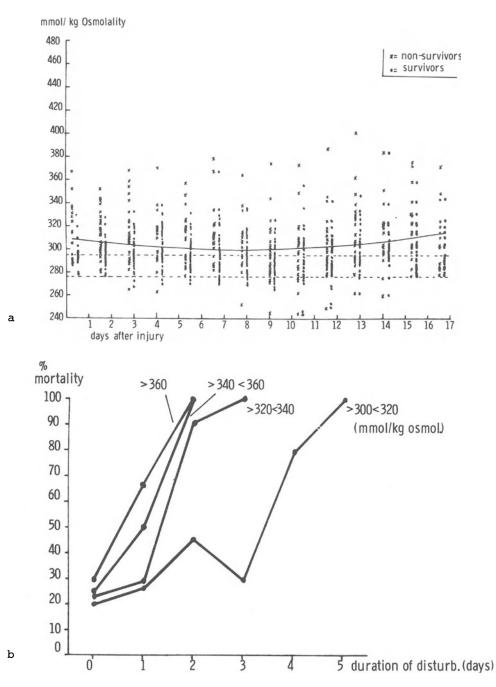


Fig. 1 a, b. Osmolality following severe brain injury and the effect on prognosis (n = 102). Osmolality disorders are more marked in non-survivors than in survivors. Normal range ---. Increasing mortality with increasing level and duration of hyperosmolality (\underline{b})

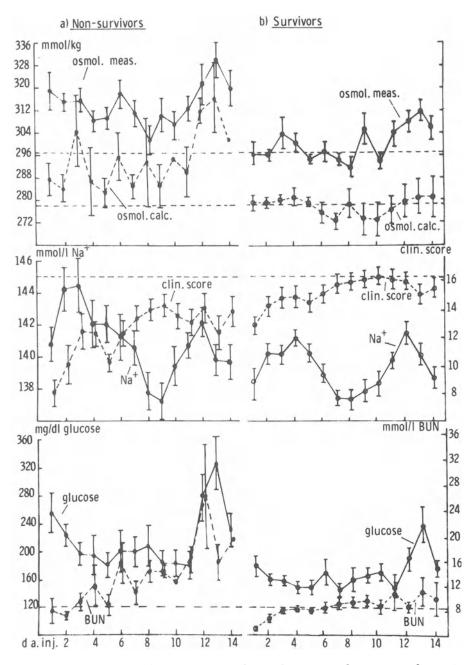
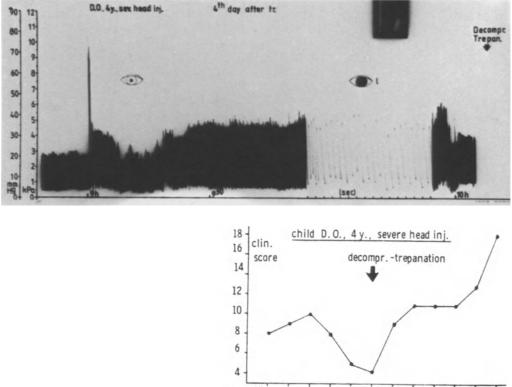


Fig. 2 a, b. Osmolality, clinical condition and serum values in non-survivors (a) and survivors (b) with severe head injury

344



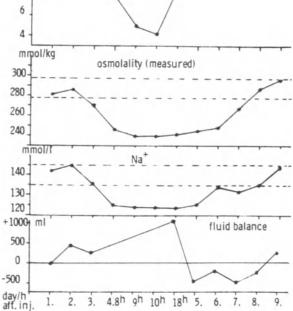


Fig. 3. Hypo-osmolar disturbance through inappropriate ADH-secretion following brain trauma: The drop in osmolality is accompanied by clinical deterioration and increasing ICP (*Avobe*; inserted: pressure transducer, used size compared to matchstick). Decompressive trepanation and water restriction result in a quick improvement (*below*)

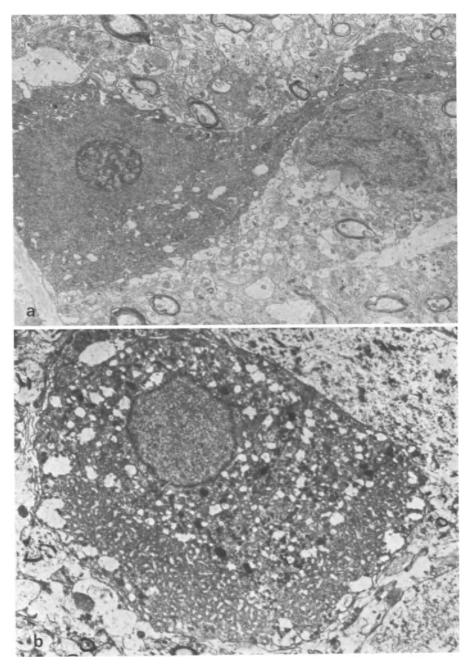


Fig. 4 a, b. Osmophilic, pycnotic ganglion cell necroses (a earlier, b later stage) in dogs with artifically increased osmolality in dehydration. Electron micrographs, magn. <u>a</u> ca. 2000 x; <u>b</u> 4000 x Brain Ischemic Disorders: Role of the Perfusional Brain Scanning in the Neurosurgical Diagnosis

M. BORTOLUZZI and G. MARINI

The value of the clinical investigation of the brain's arterial bed in cerebral ischemic disorders by microemboli injected into the brain arteriolar tree has been already reported (VERHAS et al., 1976, BORTO-LUZZI et al., 1979). The present report deals with the significance of the pathologic patterns observed during Perfusional Brain Scanning (PBS) and with the role of this procedure in the assessment of the small lesions of the brain microcirculation. Importance has been assigned to this investigation in view of planning a surgical revascularization (BORTOLUZZI et al., 1977).

Material and Method

Human albumin microspheres of $30+5 \ \mu$ m in diameter labeled with Tc₉₉ for one μ Ci were injected by a 19 G percutaneous needle into the internal carotid artery of 35 patients with supratentorial ischemic lesions. Twenty of these had transitory ischemic attacks (TIAs) while fifteen presented completed strokes (CS). All patients were submitted to EEG, brain scan and cerebral angiography. In cases of special interest, cerebral blood flow studies and CT scan were performed as well. A homogeneous microembolization of the supratentorial brain region was obtained following the distribution of the cerebral blood flow. This method allows the visualization of the 30 μ m arteriolar bed. The diffusion of the radionuclides was detected by a high resolution parallel hole collimator of a Pho-Gamma IV Nuclear Chicago linked to a computer and the display recorded with a polaroid camera. Five views are avaliable: lateral, vertex, anterior, mesial and posterior. A transitory worsening of the neurological conditions was noted in one old patient submitted to angiography, cerebral flow study and PBS at the same session.

Results

In all cases a pathologic scan was observed which allowed us to recognize the site and the extent of the ischemic lesions. Further information about the interhemispheric arterial supply and the degree of the arteriolar bed damage were obtained. On morphological grounds, we were able to identify 3 pathologic patterns: (1) large cold areas, (2) multifocal small lesions, (3) uniform decreased radioactivity over the hemisphere. The first type (Fig. 1) is the result of a thromboembolic episode of a major cerebral artery, since evidence of a thromboembolic stenosis, especially of the middle cerebral artery, was found at angiography. The second type (Fig. 2a) is probably related to an occlusive process of small peripheral arteries. With angiographic magnification in some instances, we were able to detect an occlusion of small arteriolar branches. The third type (Fig. 3) seems to be the effect of rather widespread vascular-parenchymal damage. In these cases our clinical investigations were not clear enough to explain this uniform but not severe damage of the arteriolar bed. By means of this classification, we observed (Fig. 4) that presented of CS first pattern, large cold areas was present in all cases associated with multifocal small lesions of the 2nd type in 46% of cases and with the 3rd type in 6,5%. The TIAs showed small lesions of the 2nd type in most cases, 65% and in the 20% uniform damage of the arteriolar bed. The foirst pattern was observed in 3 patients only, but the cold areas were in these instances smaller than those found in the CSs. From these figures it emerges that the associated patterns, expression of a more severe ischemic damage, are observed in the 53% of the CSs, always linked with large ischemic lesions, while these are uncommon in the TIAs, which present a single pattern in 95% of the cases.

Discussion

Our findings with this group of patients demonstrate that there is a significant correlation between the severity of the brain ischemic lesions observed at the PBS and the neurological status. Large ischemic damage became worse in most cases of CS, while lesser ischemic impairment and singular pathologic patterns are common in the TIAs. It is noteworthy that in all cases of CSs as well as in TIAs, ischemic damage was found. In the TIAs in more instances the PBS showed a very high sensitivity in proving the small lesions of the arteriolar field which were never demonstrated in the CT scan or at cerebral blood flow studies (Fig. 2b). Therefore this procedure of microembolization seems to be the method of choice in the diagnosis of microcircle damages related to the episode of TIAs. This surprisingly high percentage of ischemic lesions in patients with transitory neurological dysfunctions agrees with the pathologic investigations of COLE and YATES (1967). In 100 hypertensive and 100 normotensive people who had died in a general hospital for a wide variety of causes, they found many small and recent brain lesions which could have been the basis for a clinical non-persistent attack, even though no reference of such an event was recorded. Since our investigations with follow-up studies favour the thesis that the ischemic areas observed at the PBS are anatomical rather than functional lesions, it seems more suitable from a pathophysiological point of view, to plan a surgical anastomotic shunt to the still partially functioning area than to the region where the arteriolar bed cannot undergo any improvement. Many even rather large ischemic lesions were found in the so-called silent areas, e.g. in the frontal region without neurological dysfunctions. These findings underline how the usual clinical parameters (motor, sensory, speech, visual disorders) may be unreliable for a comprehensive evaluation of the ischemic damage. A more interested attitude for these asymptomatic lesions seems reasonable as obvious clinical disabilities were observed following the enlargement of these ischemic areas.

Conclusion

Perfusional brain scanning possesses a very high sensitivity in detecting the small lesions of the arteriolar bed, which never appeared in the CT scan or in cerebral blood flow studies especially in the TIAs. Information about the interhemispheric arterial supplies and the site, extension and the degree of the ischemic lesions were obtained. Three pathologic patterns are recognizable on the scans which seem to be the result of different pathophysiological events. A valuable correlation between the cerebral damage observed at the PBS and the neurological status was found, since some patterns are peculiar to the CSs while others are common in TIAs. The pathophysiological significance of the lesions allows us to put forward some suggestions as to surgical revascularization.

References

- BORTOLUZZI, M., GIUNTA, F., GUERRA, P., MARINI, G.: The Tc₉₉ human radioalbumin microspheres intracarotid injection in cerebrovascular accidents. In Proceedings of the X^O European Symposium of the International College of Surgeons. Milan 1977
- BORTOLUZZI, M., MARINI, G., GIUNTA, F.: Critical evaluation of the hemispheric brain scanning using Tcgg labeled human albumin microspheres in cerebral ischemic disorders. Preliminary report. J. of Neurosurgical Science (in press).
- 3. COLE, F.M., YATES, P.O.: Intracerebral microaneurisms and small cerebrovascular lesions. Brain <u>90</u>, 759-760 (1967)
- 4. VERHAS, M., SCHOUTENS, A., DEMOL, O., PATTE, M., RAKOFSKY, M., SRUYVEN, M., CAPON, A.: Use of ^{99m} Tc-labeled albumin microspheres in cerebral vascular disease. J. Nucl. Med. <u>17</u>, 179-184 (1976)

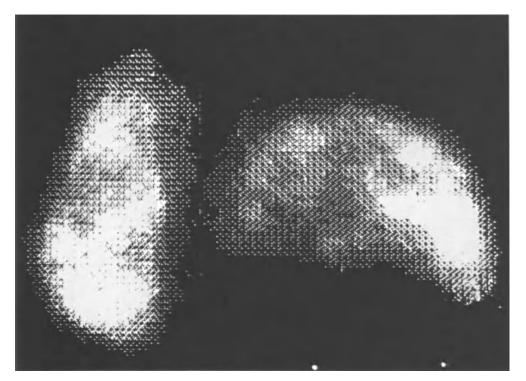
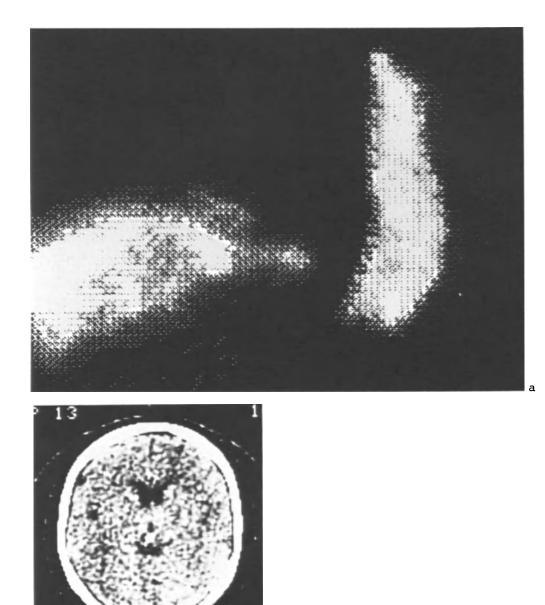


Fig. 1. Left PBS in a 37-year-old patient with right hemiplegia and permanent speech disturbances. Vertex and lateral views: large fronto-temporo-parietal cold area. Cerebral angiography showed thromboembolic stenosis of the middle cerebral artery



b

Fig. 2 a, b. Right PBS in a 34-year-old woman with transitory left brachial paresis and paresthesias. Vertex and lateral views: multifocal small lesions in the temporo-parietal area. CT scan in the same patient (b) showed normal findings. The regional CBF was in the normal range

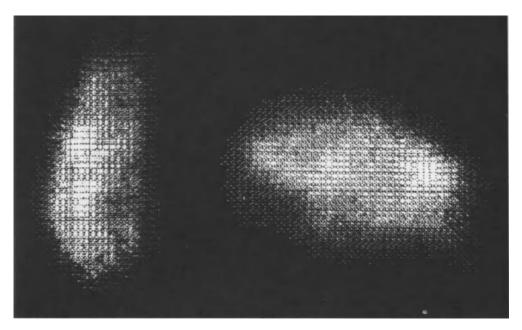


Fig. 3. Left PBS in a 33-year-old woman with cheiro-oral paresthesias and transitory speech impairment. Vertex and lateral views: rather homogenous radioactivity decreasing over the hemisphere

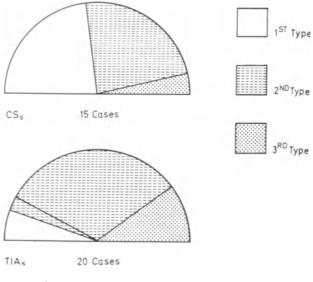


Fig. 4

Intracranial Pressure Time Course in Massive Ischemic Brain Infarction I. PAPO, G. CARUSELLI, M. SCARPELLI, and A. LUONGO

Intracranial pressure (ICP) after massive brain ischemia has been extensively studied in animal experiments. Conversely, no data concerning post-ischemic ICP variations in humans are available in the medical literature.

It might be assumed, however, that the ICP time course can be derived indirectly from multiple data, like clinical evolution, angiography and CT scan pictures and, finally, in patients with a fatal outcome, from the neuropathological findings. To verify this assumption and to quantify the intracranial hypertension, we have studied the ICP behavior in a group of selected patients harboring massive brain infarctions with increasingly severe disturbances of consciousness and definite neuroradiological evidence of a mass lesion.

Method and Clinical Material

ICP was recorded continuously by LUNDBERG's intraventricular technique in 11 patients. ICP recording was commenced on the second day after the stroke in 5 patients and on the third in the remainder. The aver-age duration of ICP measurement was 5.5 days. The age of the patients ranged from 42-72 years (average 59). In 10 patients the ischemic infarction was located in the perfusion area of the middle cerebral artery (on the right side in 6 cases and on the left in 4), in the last patient there was a large infarction involving most of the perfusion area of the posterior cerebral artery territory. In 8 patients CAT was performed whereas the remainder underwent only angiographic examination. In the first 48 hours after the stroke all patients exhibited mild disturbances of consciousness (more than 10 points on Glasgow Coma Scale). On the third day they began deteriorating and on the fourth or fifth day all of them were deeply comatose (3-5 points on G.C.S.). ICP time course was parallel to clinical evolution: so, pressure was slightly elevated, ranging between 15 and 25 mm Hg, when ICP recording was started and began rising as soon as consciousness deteriorated. The peak of intracranial hypertension was attained between the fourth and the sixth day after the ictus. All patients became deeply comatose with mild levels of intracranial hypertension, ICP baseline ranging from 25-40 mm Hg.

Altogether 8 patients were operated upon: in 3 bilateral cases a subtemporal decompression was carried out and 4 underwent the same procedure unilaterally. The last patient with a large infarction in the posterior portion of the right hemisphere, a parieto-occipital bone flap was removed and the dura left open. 5 patients in whom ICP declined steadily after decompression became responsive in a few days. In one case elevated ICP was reduced but no clinical improvement ensued and the patient died in a few days. In the remaining two patients ICP continued to rise until death. 3 patients were not operated on: in one raised ICP began to decrease after repeated osmotic administration; from the clinical standpoint the patient improved very slowly. In 2 patients with huge infarction involving almost the entire perfusion area of the middle cerebral artery, ICP which was fairly elevated (about 30 mm Hg), and rose very steeply up to 70-80 mm Hg in the terminal stage. Besides a large area of encephalomalacia involving the perfusion area of nearly all the middle cerebral artery, post-mortem examination revealed a massive homolateral hippocampal herniation and widespread paramedian brainstem hemorrhages.

Discussion

Since intracranial hypertension which follows massive ischemic brain infarction is strictly related to increasing vasogenic edema, the ICP time course should be in parallel with developing and spreading edema into the white matter. Consequently, the peak of the intracranial hypertension should correspond to maximal edema. In animal experiments O'BRIEN et al. (8) found that increased water content in brain substance, and mainly in the infarcted hemisphere, was apparent 4 hours after occlusion of the middle cerebral artery and was maximal within two days. In 15 patients who came to the postmortem examination, SHAW et al. (11) plotted the shift of the midline in millimeters against the duration of the infarct, which was known in every case. Evidence of developing edema was found in the first 24 hours and the shift of the midline reached its maximum on the fifth day after the ictus. In YAMAGUCHI & UNEMURA's (12) angiographic study the peak of the "mass effect" was found to occur on the fourth day.

In the computer tomogram, AULICH et al. (1) observed hypodensities 6 to 10 hours after the ictus. In more of 50% of their patients the CT scan was positive within 48 hours, but definite parallelism between CT scan features and the clinical course was not found. KINKEL & JACOBS ($\underline{6}$) bear out AULICH's data and point out that in the first 48 hours a certain proportion of patients exhibit negative CT scan. HUK & SCHIEFER ($\underline{4}$) observed swelling of the necrotic area on the second day after the stroke.

In all reports on CAT (1, 4, 6) the "mass effect" lasted 2 or 3 weeks at most. On the whole, in our experience ICP time course seems to correspond closely to clinical, neuroradiological and pathological findings, where the maximum edema, midline shift and intracranial hypertension was attained 4-5 days after the ictus. By that time all patients had become deeply comatose. ICP behavior in massive ischemic brain infarction appears to be quite different from what we have observed in primary intracerebral hermorrhages (5, 10). As a matter of fact, in the vast majority of patients with large intracerebral hema-tomas, ICP is fairly elevated just after the stroke to decline in the following days irrespective of the treatment carried out. Increasing intracranial hypertension, except in cases with massive and deeply located clots, is fairly rare. In most cases of intracerebral hemorrhage, brain edema does not seem to play a relevant role, at least in the very early stage (5, 7). GRUMME et al. (3) and HUK & SCHIEFER (4)noticed only a thin hypodense area around the clot in the CT scans. In a small number of cases (14% in GRUMME's series) there is a large area of finger-shaped edema, which, however, takes several days to appear (7). To GRUMME (3) the mass effect is due to the hematoma itself and not to brain edema. The different role played by brain edema may account for the opposite ICP time course in massive brain infarction and primary intracerebral hemorrhage.

The next point to be stressed is that the patients with large ischemic lesions seem to be much less tolerant of intracranial hypertension than those suffering from intracerebral hemorrhages. We have noticed in our personal series (10) that several patients with huge intracerebral hematomas and a corresponding midline shift can bear high levels of intracranial hypertension, with a baseline ranging between 50 and 60 mm Hg, surprisingly well with only mild disturbances of consciousness. The same also holds true in intracerebral traumatic hematomas, when the lesion is strictly localised (9). Conversely, our patients with massive ischemia became comatose, at much lower levels of intracranial hypertension with a baseline ranging between 25 and 40 mm Hq. As mass effect and midline shift may be observed in the same way both in hematomas and in brain ischemia they cannot account for the different degree of tolerance to intracranial hypertension. It seems more likely that spreading edema and widespread disturbances in microcirculation leading to diffuse brain hypoxia and, consequently, giving rise to further edema, are responsible for the poor tolerance to raised ICP observed in ischemic patients.

In the neurosurgical literature several reports have recently dealt with the surgical treatment of cerebellar infarcts considered as acute expanding lesions. By contrast, little attention has been paid so far to the surgical treatment of acute cerebral ischemia with intracranial hypertension by means of direct decompressive procedures. Only GREEN-WOOD (2) reported on a small series of 10 patients submitted to decompression: of the 6 patients who survived 3 had moderate or acceptable neurological sequelae.

As regards the effectiveness of this procedure, our experience is still too limited to set up reliable criteria for making surgical decisions about such patients. Nevertheless, there is no doubt that after timely surgical decompression selected patients who otherwise would die, may recover with acceptable functional results.

- AULICH, A., WENDE, S., FENSKE, A., LANGE, S., STEINHOFF, H.: Diagnosis and follow-up studies incerebral infarcts. In: Computerized tomography. LANKSCH, W., KAZNER, E. (eds.), pp. 273-283. Berlin, Heidelberg, New York: Springer 1976
- GREENWOOD, J.: Acute brain infarction with high intracranial pressure: surgical indication. John Hopkins Med. J. <u>122</u>, 254-258 (1968)
- 3. GRUMME, Th., LANKSCH, W., WENDE, S.: Diagnosis of spontaneous intracerebral hemorrhage by computerised tomography. In: Computerised tomography. LANKSCH, W., KAZNER, E. (eds.), pp. 284-290. Berlin, Heidelberg, New York: Springer 1976
- 4. HUK, W., SCHIEFER, W.: Computerized tomography (Siretom) of acute cerebrovascular events. In: Computerised tomography. LANKSCH, W., KAZNER, E. (eds.); Berlin, Heidelberg, New York: Springer 1976
- 5. JANNY, P., COLNET, G., GEROGET, A.M., CHAZAL, J.: Intracranial pressure with intracerebral hemorrhages. Surg. Neurol. <u>10</u>, 371-375 (1978)
- 6. KINKEL, W: v., JACOBS, L.: Computerized axial tomography in cerebrovascular disease. Neurology (Minn.) <u>26</u>, 924-930 (1976)
- 7. LEGRE, J., DEBAENE, A., LAMOUREUX, J.M., TAPIAS, P.L.: Interest of control by CT of intracerebral spontaneous and traumatic

hemorrhage at different stages of evolution. Neuroradiology <u>15</u>, 51 (1978)

- O'BRIEN, M.D., WALTZ, A.G., JORDAN, M.A.: Ischemic cerebral edema. Arch. Neurol. (Chicago) <u>30</u>, 456-460 (1974)
- 9. PAPO, I., CARUSELLI, G., LUONGO, A., SCARPELLI, M.: Compotement de la pression intra-crânienne dans les foyers d'attrition et hématomes intracérébraux traumatiques. Neuro-Chirurgie. (In press)
- PAPO, I., JANNY, P., CARUSELLI, G., COLNET, G., LUONGO, A.: Intracranial pressure time course in primary intracerebral hemorrhage. Neurosurgery <u>4</u>, 504-511 (1979)
- 11. SHAW, C.M., ALVORD, E.C., BERRY, R.G.: Swelling of the brain following ischemic infarction with arterial occlusion. Arch. Neurol. <u>1</u>, 161-177 (1959)
- 12. YAMAGUCHI, K., UEMURA, K.: An angiographic study of brain swelling in cerebral infarction. Neuroradiology <u>16</u>, 150-151 (1978)

Non-Invasive rCBF Measurement for Pre- and Postoperative Evaluation of Patients with EC/IC Anastomoses G. MEINIG, A. FRENSKE, and K. SCHÜRMANN

Introduction

Knowledge of the mean CBF and rCBF pattern is of great value for the evaluation of cerebrovascular diseases, especially for establishing the indication for operations to improve circulation. Beyond this, criteria for assessing the postoperative course or for conservative therapy can be obtained. The technical improvement of non-invasive rCBF equipment has enabled us to employ the NOVO cerebrograph in Mainz for non-invasive rCBF measurement (nrCBF). This method is atraumatic, it is practically free of risks, and it permits follow-up examinations. A limitation for the frequency of examination is merely a certain radiation load. It can be carried out in out-patients, it enables assessments of total blood flow and the pattern of regional blood flow in the brain. In particular, it enables simultaneous recording of blood flow in both the affected and the healthy hemispheres.

Methods

Measurement Procedure and Equipment

We use a commercially available sixteen-channel system with an eightcrystal detector of the dimensions of $3/4 \ge 3/4$ inch with a 20 mm cylindrical lead collimator on each hemisphere¹. The patient was attached to the xenon administration system using a mouthpiece sealed by self-adhesive surgical drape in order to avoid loss of radioactivity from the respiration cycle. The end tidal values of the expired air were measured for xenon activity in a special air detector, and the concentration of CO₂ in a capnograph in the same air sample. Data accumulation and processing were done by an on-line computer, the calculations were done by biexponential analysis as described by OBRIST et al. (1975) (4) and the Initial Slope Index (ISI) as introduced by RISBERG (1975) (7).

Immediately after the study the following parameters were tabulated by the computer: flow grey (F_g) in ml/100 g/min), the ISI calculated between the 2nd and 3rd min, which is independent of the partition coefficient for xenon. In addition the relative weight of the grey matter compartment, the fractional flow in percent of the total flow, the decay constant K of the low-perfused (white) tissue and the interhemispheric differences of the corresponding areas were printed out (RIFD = Regional Interhemispheric Flow Differences).

¹ NOVO CEREBROGRAPH available from NOVO-Industrie GmbH Pharmazeutica, Kantstraße 2, D-6500 Mainz.

Using this system, a reliable reproducibility could be attained using a xenon concentration of 2.5 to 3 mCi/l in the inspired air during equilibration of one minute duration. No spectrum subtraction was performed for hemispheric X-rays.

Our values are similar to other published data for normal inhalation CBF values.

Case Material

From our patients with cerebrovascular disease in whom nrCBF was performed, we have selected two groups:

- 25 patients with ICA/MCA obstruction (angiographically demonstrated occlusions and high-grade stenoses with over 50% narrowing of the lumen)
- 2. 10 patients in whom we have performed nrCBF measurement pre- and postoperatively following EC/IC anastomoses.

20 healthy persons served as controls.

Results

Figure 1 shows the pattern of flow distribution in normal patients (n = 20). Hemispheric means of both sides and average flow pattern distribution as well as the distribution are given. Our results are similar to those obtained by other groups using nrCBF (1-5, 7, 8).

In a group of 25 patients with unilateral circumscribed obstruction in the area supplied by the carotid artery (occlusions and high-grade stenoses with over 50% narrowing of the lumen) there was a marked reduction in blood flow in about 90% of cases on the affected side compared to the non-affected side (Fig. 2). Comparison of the bloodflow pattern of the affected side with our normal values revealed a pronounced reduction in cerebral blood flow, especially in the area supplied by the middle cerebral artery. Comparison with the clinically non-effected hemisphere is less useful, since as a rule the "healthy" hemisphere has a reduced blood flow (steal syndrome? increased cerebrovascular resistance in diffuse vascular sclerosis?).

Figure 3 shows the nrCBF pattern of a 61-year-old man with a chronic ICA obstruction beyond the bifurcation on the right side. Mean ISI is significantly reduced on the affected right side as compared to our normal values. However, the ISI on the non-affected left side is also reduced. The flow pattern is reduced more or less in all areas which were measured on the affected side. As early as 12 days after the EC/IC anastomoses, mean ISI shows an increase of 2 ml/100 g and minute, and the comparison of nrCBF patterns shows an increase in most areas (Fig. 4).

Figure 5 shows the postoperative angiogram which was performed nearly 3 months later. The measurement of blood flow in the anastomosis by videodensitometry revealed a value of 73 ml/min.

The example of a left-sided ICA occlusion of a 38-year-old female (Figs. 6 and 7) shows an enormous improvement of the cerebral blood flow of both hemispheres three months after the EC/IC anastomosis (Fig. 7). This increase of nrCBF was not only the result of the by-pass, since the first nrCBF measurement was (unfortunately) performed

in the postacute stage of stroke with its diffuse reduction of flow throughout the entire brain (lasting about 1-3 weeks).

In the meantime, we have measured cerebral blood flow in 10 cases preoperatively and postoperatively (at an interval of 2.8 months). Table 1 shows that the anastomosis improves the postoperative blood flow not only on the affected side, but also in the "healthy" hemisphere, so that the interhemispheric difference in the blood flow is almost abolished postoperatively. In all cases, an open bypass could be demonstrated semiguantitatively by means of Doppler sonography. In addition, the function of the shunt was examined by means of angiography and quantitatively with videodensitometry.

	Affected hemisphere	"Non-affected" hemisphere
FG Preoperative (ml/100 g/min)	71.5 <u>+</u> 11.3	76.7 <u>+</u> 13.5
FG Postoperative (ml/100 g/min)	80.6 <u>+</u> 21.8	81.0 <u>+</u> 25.5
ISI Preoperative (ml/100 g/min)	46.8 <u>+</u> 9.0	50.3 <u>+</u> 9.7
ISI Postoperative (ml/100 g/min)	53.5 <u>+</u> 15.5	53.9 <u>+</u> 17.6

Table 1. Change of mean CBF following EC/IC anastomosis

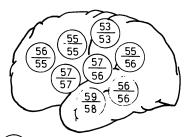
Discussion

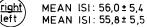
Besides the low burden on the patient through the application of this method, a special value of non-invasive cerebral blood flow determination is that an overall evaluation of the blood flow condition is possible. In general, it is shown that in the cases of unilocular vessel obstruction, a reduction in blood flow is displayed in the "healthy" hemisphere.

The non-invasive measurement of cerebral blood flow provides valuable parameters for establishing the indication for operations to improve circulation. It can be shown with this method that there is a postoperative improvement in blood flow after EC/IC anastomosis not only of the affected, but also of the healthy hemisphere, i.e. the overall hemodynamics are improved. The good agreement of the cerebral blood flow findings with the angiographic findings as well as with those of Doppler sonography indicates that in future (apart from problem cases), control angiography which is not entirely unharmful can be dispensed with for postoperative appraisal of patients with brain damage.

- BLAUENSTEIN, U.W., HALSEY, J.H., WILSON, E.M. et al.: 133 Xenon inhalation method: significance of indicator maldistribution for distinguishing brain areas with impaired perfusion, stroke <u>9</u>, 57-66 (1978)
- DESHMUSK, V.D., MEYER, I.S.: Noninvasive measurement of regional cerebral blood flow in man. New York, London: SP Medical and Scientific Books 1977

- MEYER, J.S., ISHIHARA, N., DESHMUKH, V.D. et al.: Improved method for noninvasive measurement of regional blood flow by 133 Xenon inhalation, Part I. Stroke 9, 195-205 (1978)
- MEYER, J.S.: Improved method for noninvasive measurement of regional blood flow by 133 Xenon inhalation, Part II. Stroke <u>9</u>, 205-210 (1978)
- 5. OBRIST, W.D., THOMPSON, H.K., WANG, H.S. et al.: Regional cerebral blood flow estimated by 133 Xenon inhalation. Stroke <u>6</u>, 246-256 (1975)
- PIROTH, H.D., MAGIN, E., GEORGI, M., MARBERGER, M.: Ergebnisse der Knieangiodensitometrischen Nierendurchblutungsbestimmung. Fortschr. Röntgenstr. <u>126</u>, 126-128 (1977)
- 7. REIVICH, M., OBRIST, W., SLATER, R. et al.: A comparison of the Xe¹³³ intracarotid injection and inhalation techniques for measuring regional cerebral blood flow 8.3-8.6 in: Blood metabolism in the brain. HARPER, A.M., JENNETT, W.B., MÜLLER, J.D., ROWAN, J.O (eds.). Edinburgh, London, New York: Churchill Livingstone 1975
- RISBERG, J., ALI, Z., WILSON, E.M. et al.: Regional cerebral blood flow by 133 Xenon inhalation. Stroke 6, 142-148 (1975)





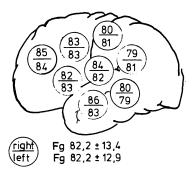


Fig. 1. Blood flow in normal subjects. Normal control values (n=20). MABP, 102,5 \pm 8,5 mm Hg; MPa CO $_2$, 38,7 \pm 2,3 mm Hg

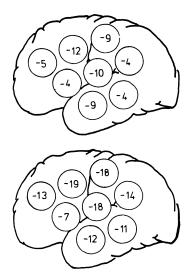


Fig. 2. Percent of change in CBF (Fg) compared with that of the non-affected hemisphere (*above*) as well as compared with the normal control values (*below*). MABP, 109,4 \pm 14,7 mm Hg; MPa CO₂, 38,0 \pm 2,74 mmHg

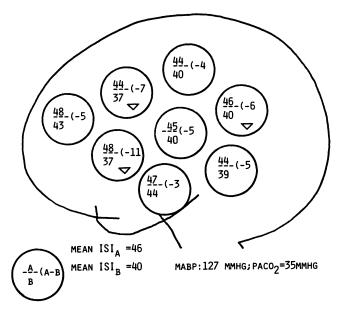


Fig. 3. nrCBF pattern of a 61-year-old man with an ICA occlusion 8 weeks ago on the right side beyond the bifurcation. <u>A</u>, non-affected hemisphere; <u>B</u>, affected hemisphere

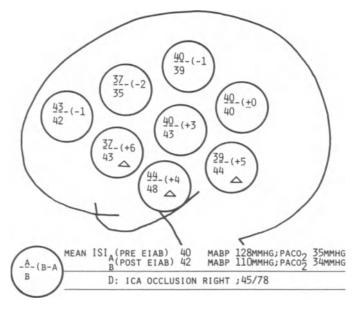


Fig. 4. nrCBF pattern 12 days after an EC/IC anastomosis (same patient as in Fig. 3)

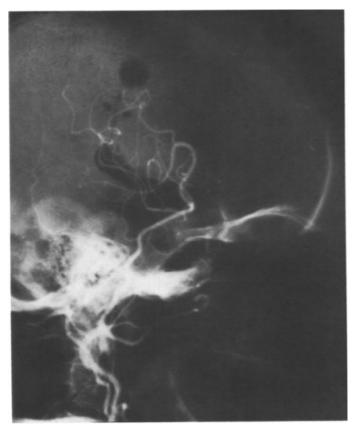
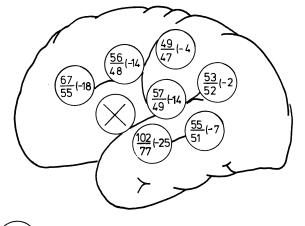


Fig. 5. Postoperative angiogram of same patient as in Figs. 3 and 4. The flow measured by videodensitometry in the anastomosis was 73 ml/min



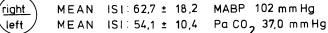
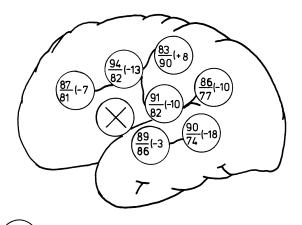


Fig. 6. nrCBF pattern of a 38-year-old female with ICA occlusion on the left side beyond the bifurcation. D, left ICA occlusion pre EC/IC anast.



<u>right</u> MEAN ISI: 88,6 ± 3,6 MABP: 113 mmHg Left MEAN ISI: 81,7 ± 5,3 Pa CO₂: 36,0 mmHg

Fig. 7. nrCBF pattern 3 months following EC/IC anastomosis (same patient as in Fig. 6). D, left ICA occlusion, 10 weeks post EC/IC anast.

Testing of Collateral Circulation of the Brain Before Surgical Treatment of Internal Carotid Artery Lesions

H. WASSMANN and K.-H. HOLBACH

Introduction

Surgical treatment of our patients suffering from cavernous sinus fistula, certain types of aneurysms of the internal carotid artery and internal carotid artery stenosis involved either temporary or permanent occlusion of the affected internal carotid artery. Before interrupting the blood flow through the affected internal carotid artery, it is important to find out whether the collateral cerebral circulation can tolerate this inherent reduction of blood flow. We attempted to find a solution to this problem by studying the effects of carotid compression of the affected carotid artery on the EEG. Carotid compression tests (CCT) under different arterial oxygen pressures (aPO₂) should clarify the question whether a positive clinical or EEG effect is an ischemic hypoxic reaction.

<u>Method</u>

We compress the affected carotid artery of a supine patient by pressing the second to fourth fingers against the cervical spine. In this way, the arriving pulse-wave can be registered and complete occlusion of the common carotid artery controlled. A continuous bipolar EEG was recorded from the affected parietal region and quantified by an interval-amplitude-analysis-system (3, 4). To achieve different arterial oxygen concentrations, the CCT was performed under hyperbaric conditions.

Results

An example of the effect of CCT on a 21-year-old patient with a left sided cavernous sinus fistula is shown in Fig. 1. During the first CCT under normobaric conditions a significant decrease of alpha-wave activity in the EEG was recorded in the first minute, which receded only insignificantly in the second minute. At the same time the patient noticed nausea without changes of heart rate, blood pressure or respiration, whereupon the test was interrupted. The same reactions were noticed at the subsequent performance of the CCT under hyperbaric conditions, breathing air at 1.5 atmospheres absolute (ATA) and an aPO₂ of 100 mm Hg. The repetition of CCT however while breathing oxygen at 1.5 ATA and an aPO₂ of 750 mm Hg caused no significant EEG changes or clinical symptoms. After 20 daily performances of the CCT under EEG control, it was tolerated for 15 minutes even under normobaric conditions without effecting EEG changes or clinical symptoms, so that the fistula and the corresponding internal carotid artery could be occluded by a Fogarty catheter. Postoperative EEG control two months later showed a distinct increase of alpha-wave activity. Altogether we examined 27 patients suffering from internal carotid stenosis, 1 patient with an infraclinoidal carotid aneurysm and 5 patients with cavernous sinus fistula. CCT performed in such a way did not provoke any irreversible neurological deficits in these patients. Six patients showed a distinct positive reaction at the first CCT during the first minute. In two patients this positive reaction continued in spite of vascular training, so that the operation could not be carried out. Operated patients however, showed no additional neurological deficits after surgery.

Discussion and Conclusion

These investigations showed, in accordance with other authors $(\underline{1}, \underline{2})$, that CCT is a simple test for the examination of collateral cerebral circulation. Controlling the positive reaction during the CCT under raised arterial aPO₂ provides evidence that clinical reactions and EEG changes are caused by ischemic hypoxia. A positive reaction to the CCT may be interpreted as an uncompensated disturbance of the cerebral blood supply. A diminishing reaction during vascular training is considered to be due to an improved collateral cerebral circulation. The applied EEG-analysis-system presents these dynamic changes as a distinct and clear picture of cerebral function.

<u>References</u>

- KIDRON, D.P.: The electroencephalographic effects of carotid compression. EEG Clin. Neurophysiol. <u>6</u>, 469-472 (1954)
- 2. MÜLLER, D.: Der Karotisdruckversuch als Provokationsmethode in der klinischen Elektroenzephalographie. Jena: Gustav Fischer 1972
- REETZ, H.: EEG-Analyse mit digitaler Intervall- und Amplituden-Klassierung. Z. EEG-EMG 2, 32-36 (1971)
- 4. WASSMANN, H., HOLBACH, K.-H., BERTSCH, P.: Die Anwendung der EEG-Intervall-Amplituden-Analyse bei der Behandlung von Hirnarterienverschlüssen mit der hyperbaren Oxygenation. In: Quantitative Analysis of the EEG. MATEJCEK, M., SCHENK, G.K. (eds.), pp. 395-406. Konstanz: AEG-Telefunken EDP-Division 1975





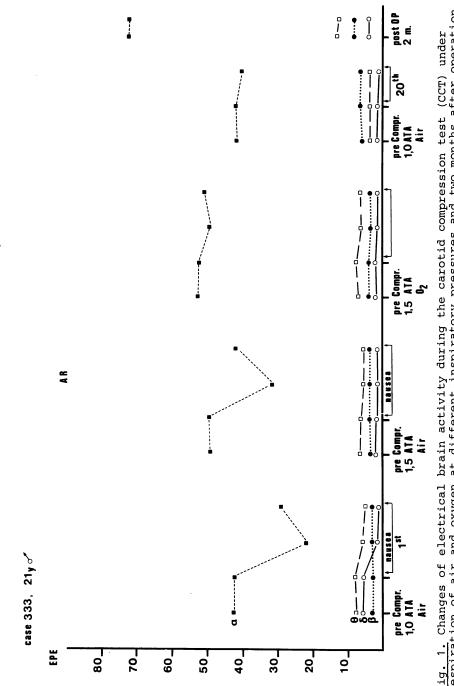


Fig. 1. Changes of electrical brain activity during the carotid compression test (CCT) under respiration of air and oxygen at different inspiratory pressures and two months after operation. AR, affected region; EPE, electrical power equivalent; ATA, atmospheres absolute; m., months

Treatment of Vascular Malformations of the Head with Detachable Balloon Catheters

W. Huk

In cases of intra- and extracranial vascular malformations microsurgical techniques have become the treatment of choice. Parallel to this, efforts have been made to utilize the endovascular approach $(\underline{1}, \underline{2}, \underline{3}, \underline{4}, \underline{5}, \underline{6}, \underline{7})$.

In this paper our first experiences with detachable balloon catheters in the treatment of vascular malformations of various kinds are presented. In the literature detachable balloons are mainly used for the occlusion of carotid-sinus cavernosus fistulas and of arterio-venous aneurysms.

Method

The balloon catheters are "custom-made" in a modification of the technique described by SERBINENKO in 1974 (7). They are inserted into the carotid artery directly. After the balloon has reached its desired position, it is filled with a silicon-polymer and detached from the catheter.

Report of Four Cases

<u>First Case</u>. 54-year-old male with a traumatic shunt between the superficial temporal artery and the external jugular vein. The fistula was occluded with a "waist-fitting" balloon; this special shape of the balloon was chosen in order to prevent it from gliding in the vessel or from passing onto the venous side. Three months later the patient was still without symptoms.

<u>Second Case</u>. 48-year-old female with a large saccular aneurysm of the internal carotid artery. Because of a kinking of the vessel the large balloon could not pass into the aneurysm. As the circulus arteriosus of WILLIS was functioning the internal carotid artery could be occluded with the balloon. Postoperatively no additional neurological deficits developed. On the contrary, the occulomotor palsy, which was complete before operation, slowly subsided.

<u>Third Case</u>. 30-year-old male with an inoperable, large angioma of the right temporal region. In a first session a hypertrophic branch of the middle cerebral artery feeding the angioma was occluded with a detachable balloon, after a trial transient occlusion of the vessel for about 30 minutes had produced no neurological symptoms. In a second attempt the hypertrophic pericallosal artery will be occluded.

Fourth Case. 38-year-old male with an aneurysm of the anterior communicating artery, which had been clipped at operation. On control angiography the aneurysm was filled again as both clips had slid off its neck. Because of the patient's poor condition, the endovascular approach was chosen. A non-detachable and a detachable balloon were introduced simultaneously; the non-detachable balloon was placed into the middle cerebral artery so that the main blood stream would carry the detachable balloon into the anterior cerebral artery and into the aneurysm. When the contrast medium was exchanged against the polymer, also the right anterior cerebral artery was occluded. On control angiography two months later the anterior cerebral artery was recanalized again and the aneurysm was bigger than before with the balloon inside.

Discussion

The endovascular approach represents a true alternative in the treatment of vascular malformations of the head; particularly in cases which are considered inoperable because of their size and location or because of the poor condition of the patient, which would not tolerate craniotomy. The endovascular approach is much less stressing for the patient and less time consuming.

With the double-balloon technique, the blood stream can be controlled at the bifurcation of the internal carotid artery. This method is also valuable in the evaluation of possible neurological deficits caused by vascular occlusion. For this purpose a trial transient occlusion of the desired vessel can be performed with a non-detachable balloon without the risk of a premature detachment of the balloon.

The last case demonstrated that in the absence of spasm, aneurysms of the anterior communicating artery can also be reached by a balloon. However, the permanent occlusion of the aneurysm sack still remains problematic; further investigation will be necessary in this field.

- DEBRUN, G., LACOUR, P., CARON, J.-P., HURTH, M., COMOY, J., KERAVEL, Y.: Detachable balloon and calibrated-leak balloon techniques in the treatment of cerebral vascular lesions. J. Neurosurg. <u>49</u>, 635-649 (1978)
- DEBRUN, G., LEGRE, J., KASBARIAN, M., TAPIAS, L., CARON, J.-P.: Endovascular occlusion of vertebral fistulae by detachable balloons with conservation of the vertebral blood flow. Radiology <u>130</u>, 141-147 (1979)
- KERBER, C.W., BANK, W.O., CROMWELL, L.D.: Calibrated leak balloon microcatheter: A device for arterial exploration and occlusive therapy. AJR <u>128</u>, 207-212 (1979)
- PEVSNER, H.P.: Micro-balloon catheter for superselective angiography and therapeutic occlusion. AJR <u>128</u>, 225-230 (1977)
- ROMODANOV, A.P., ZOZULIA, Y.A., SHCHEGLOV, V.I.: Balloon catheter occlusion of the feeding vessels of arteriovenous malformations of the brain. Zbl. Neurochirurgie 40, 21-28 (1978)
- SCHAPS, P.: Die Behandlung der Karotis-Kavernosus-Fistel mit dem Ballonkatheter nach Serbinenko. Zbl. Neurochirurgie <u>38</u>, 105-118 (1977)
- SERBINENKO, F.A.: Balloon catheterization and occlusion of major cerebral vessels. J. Neurosurg. <u>41</u>, 125-145 (1974)

Treatment of Intracerebral Hemorrhage Related to Anticoagulation Therapy Since Advent of Computer-Tomography: Report of Eight Cases

W. I. STEUDEL, I. SCHARRER, G. HOPP, and H. HACKER

The prognosis of intracerebral hemorrhage resulting from anticoagulant treatment is rated differently by different authors $(\underline{1}, \underline{2}, \underline{4}, \underline{6}, \underline{10}, \underline{14}, \underline{18})$. A review of the cases reported in the literature up to 1967 reveals that out of 61 hemorrhages only 5 survived ($\underline{1}$). Specific medical therapy of blood coagulation before the operation improved the prognosis appreciably (9, 10, 22). The use of computer-tomography (CT) provides a further possibility of improving treatment by means of follow-up examinations of the intracerebral hematomas ($\underline{5}, \underline{7}, \underline{9}, \underline{13}, \underline{19}, \underline{23-25}$).

Since CT became a routine diagnostic procedure in our department, 8 consecutive patients with anticoagulant intracerebral hemorrhages were treated. With reference to these cases, the advantages of CT in the treatment of this kind of intracerebral hematoma is shown.

Patients and Methods

From 1974-1978 seven men and one woman aged from 24-71 years were studied (Table 1). All CT-examinations were carried out with the SIRETOM I (128x128 matrix). The patients were examined by CT on admission and before discharge. In three cases (No. 1, 2 and 3) a scan was performed each week. The surviving patients were re-examined after six months.

Summary of Cases

A compilation of the CT-findings, clinical course and nature of the surgical intervention is presented in Table 1. The hemorrhages occurred in relation to anticoagulation therapy in eight cases. In addition, a minor trauma before the hemorrhage was reported three times (No. 1, 3 and 4). In one of these, diabetes was present. In another case (No. 2), the hemorrhage occurred one week after cessation of the Marcumar^R therapy.

<u>Time of Hemorrhage</u>. In five cases, the intracerebral hematoma appeared two to four years after the beginning of the anticoagulant therapy. However, the hemorrhage occurred within a shorter period three times (No. 3, 6 and 7). In these cases, a combination therapy with heparin and streptokinase was started: the hemorrhage occurred twice on the 7th day and once after four hours.

<u>Symptoms and Signs.</u> There was an interval from one to five days between the first symptoms and the disturbance of consciousness in seven cases. Two of these patients became comatose: one with an intracerebellar bleeding (No. 7). In the other case, somnolence was present

Table 1.	Sumr	Summary of		cases with anticoagulant intracerebral hemorrhage	lant intracerebr	al hemorrhage		
No.	Age	Sex	Сота	Reason for anticoagulant therapy (duration)	Anticoagulant drugs	CT-findings localisation	Intra- ventricular hemorrhage	Clinical course
-	71	M	NO	Myocardial infarction (4 years)	Marcumar	Left temporal lobe	Yes	No operation, survived
N	67	м	NO	Myocardial infarction (2 years)	Marcumar	Right occipital lobe, hydro- cephalus	Yes	Shunt-operation, survived
3 (Fig.1)	24	ſĿı	Yes	Venous thrombosis (7 days)	Heparin, Streptokinase	Left frontal lobe	Yes	Operation, survived
4	39	¥	NO	Myocardial infarction (4 years)	Marcumar	Left frontal lobe	Yes	Operation, survived
5 (Fig.2)	47	ъ	NO	Myocardial infarction (3 years)	Marcumar	Right occipital lobe	Yes	No operation, survived
9	49	×	Yes	Myocardial infarction (4 h)	Heparin, Streptokinase	Left fronto- temporo-medial region	NO	No operation, death after 2 days
2	38	¥	Yes	Myocardial infarction (7 days)	Heparin, Streptokinase	Intracerebellar, hydrocephalus	Yes	Operation, shunt, death after 6 days
ω	67	¥	NO	Myocardial infarction (3 years)	Marcumar	Left temporo- occipital lobe	No	Operation, death after 2 months, re-infarction?

ahral hamorrhaga ŝ 7 + \$ + 40 i i + + 2 4 ć r

on admission; this worsened into coma after 24 hours (No. 3). CT then showed a marked enlargement of a left frontal hematoma with intraventricular penetration (Fig. 1). The hemorrhage occurred acutely in one case (No. 6). In this patient, there was a large left frontotemporal hematoma in CT; this patient was deeply comatose on admission and died two days later.

 $\underline{CT-Findings}$. An intracerebral hematoma was present seven times: five times on the left side, twice on the right. In one case there was an intracerebellar hematoma. The hematoma were localised three times in the occipital lobe, twice in the frontal, once in the temporal and once in the fronto-temporo-medial region. In six cases there was intraventricular involvement. In four cases the ventricle contained a CSF-blood mixture and only twice was there clotted blood in the right lateral ventricle (No. 5) and in the fourth ventricle (No. 7).

CT control studies (six months to two years after the hemorrhage) revealed focal enlargement of the adjacent ventricular cavity (No. 1, 3 and 4).

<u>Treatment</u>. An operation was performed when the level of consciousness did not improve (No. 2, 3, 4, 7 and 8). In one of these, repeated CT scanning showed an increase of the intracerebral hematoma. An operation was not carried out in a patient with an acute course (No. 6) and in patients in whom the neurological signs were improving and who were not comatose (No. 1, 5). The intracerebral hematoma was removed by craniotomy. In two patients with an additional acute hydrocephalus, a shunt procedure was performed (No. 2, 7). The operation was started after normalisation of blood coagulation with PPSB (Prothrombin-Proconvertin-Stuart Prower Factor-Antihemophilia Factor B).

<u>Clinical Course</u>. Of the five patients who were operated on, three survived. The patient with an intracerebellar hematoma died. A further patient died from an unknown cause (an autopsy could not be carried out) two months after the operation, having improved. Of the three patients who were treated conservatively, the patient who was in deep coma died. The five surviving patients were all in good condition, three of them without any neurological deficit.

Discussion

<u>Computer-Tomography</u>. CT has special importance in the management of intracerebral hematomas. As a non-invasive method, it can determine precisely the size and localisation of the hematoma (5, 7, 11, 18, 23-25). With one examination, an acute hydrocephalus or rupture into the ventricle can be detected (Fig. 2). Since the introduction of CT, the long-held supposition could be confirmed that an intraventricular hemorrhage may sometimes have a favorable prognosis (16, 17, 21). It is surprising that there was intraventricular involvement in six of our eight cases. However, CT showed a CSF-blood mixture four times and clotted blood only twice. In the case with a clot in the fourth ventricle, the prognosis was poor. Repeated CT scans detected an enlargement of the hemorrhage in one case. The fact that intracerebral bleeding may recur is well-known in aneurysms and angiomas, but it is only rarely observed in hypertensive hematomas (12). In our case, the clinical deterioration corresponded to the re-bleed according to the CT-findings. The operation which was then performed resulted in rapid clinical improvement. A similar case of enlargement of a hematoma was reported eleven days after heparinisation for a suspected pulmonary embolus (3). CT has also proved effective in detection of a disturbed CSF circulation $(\underline{5}, \underline{7})$. A shunt procedure was performed twice in cases with acute hydrocephalus. The control scan showed a marked decrease of ventricular dilatation corresponding to a substantial improvement of the patient in one case. Sequential CT studies revealed a progressive decrease of the mass effect and resorption of the hematoma ($\underline{3}$). In our cases, the isodense phase was attained after six weeks. However, it cannot be concluded from this that complete resorption of the hematoma occurs ($\underline{11}$). It seems that the rate of resorption varies with the initial size of the clot ($\underline{3}$). However, it is striking that there are different rates of resorption in similar cases. This requires further investigation (5).

Etiological Factors and Cause of the Hemorrhage. Even though intracerebral hemorrhage occurred in connection with anticoagulant therapy, the etiology of the intracerebral hemorrhage cannot be attributed with certainty to this medication (20). In three of our cases, a minor trauma was reported which was related to the onset of symptoms (18). In addition, there was diabetes in one case. We are unable to confirm that intracerebral hematomas due to anticoagulants occur mainly in hypertensive patients (8). There was no hypertension in any of our patients. On the other hand, we were able to support the view that an anticoagulant hemorrhage occurs more frequently in patients who have taken this medication (Marcumar^R) for years (15). The etiological relation is more readily evident in cases with the combination therapy - heparin and streptokinase - since the interval between the beginning of therapy and occurrence of the hemorrhage is usually only a few days or even hours. The observation of an enlargement of the hemorrhage in one of these cases indicates that an additional unknown vascular factor must be present (12).

Conclusion

In cases of intracerebral hemorrhage related to anticoagulation therapy, computer-tomography offers special advantages: the patient may be examined before normalisation of blood coagulation; CT shows the precise localisation, size and extent of the hematoma. An intraventricular involvement and an acute hydrocephalus may be demonstrated simultaneously. In addition, CT sequential studies reveal the decrease of the mass effect and the rate of resorption. Patients with progressive impairment of the level of consciousness should be operated upon, whereas patients with improvement and/or decrease of mass effect in the CT can be treated conservatively.

- ANGSTWURM, H., FRICK, E.: Nil nocere! Neurologische Komplikationen der Antikoagulantientherapie. Münch. Med. Wochenschr. <u>109</u>, 1103-1109 (1967)
- BARRON, K.D., FERGUSSON, G.: Intracranial hemorrhage as a complication of anticoagulant therapy. Neurology 9, 447-455 (1959)
- 3. DOLINSKAS, C.A., BILANIUK, L.T., ZIMMERMANN, R.A., KUHL, D.E.: Computed tomography of intracerebral hematomas. I. Transmission CT observations on hematoma resolution. Am. J. Roentgenol. <u>129</u>, 681-688 (1977)
- 4. GROTE, E., GELETNEKY, C.L., ROMPEL, K., ZORBAS, K., MEYER, E., PRILL, A., HEENE, D.: Atypische intracerebrale Massenblutung. Dtsch. Z. Nervenheilk. 197, 66-76 (1970)

- GRUMME, Th., LANKSCH, W., KRETZSCHMAR, K.: Intrakranielle Blutungen im Computertomogramm. Dtsch. Ärzteblatt <u>76</u>, 1627-1634 (1979)
- JOHANSSON, S.H.: Hypertensive and normotensive intracerebral haemorrhage. Acta Psychiat. Scand. (Suppl.) <u>150</u>, 90-94 (1961)
- KAZNER, E.: Computertomographie. Ihre Bedeutung für die Diagnose und Differentialdiagnose zerebraler Durchblutungsstörungen. Neurol. Psychiat. <u>5</u>, 312-324 (1979)
- KOLLER, F.: Klinische Beurteilung der Anticoagulantien. Internist 10, 8-12 (1969)
- 9. LUYENDIJK, W.: Intracerebral haematoma. In: Handbook of clinical neurology Vol 11. VINKEN, P.J., BRUYN, G.W. (eds.), pp. 660-719. Amsterdam: North-Holland Publ. Comp. 1972
- 10. MAZARS, G., RIBADEAU-DUMAS, Ch., ROGE, R.: Accidents hémorragiques cérébraux au cours des traitements anticoagulants. Marseille Med. <u>104</u>, 27-30 (1967)
- 11. MESSINA, A.V., CHERNIK, N.L.: Computed tomography: the "resolving" intracerebral hemorrhage. Radiology <u>118</u>, 609-613 (1975)
- 12. MIZUKAMI, M., ARAKI, G., MIHARA, H., TOMITA, T., FUJINAGA, R.: Arteriographically visualized extravasation in hypertensive intracerebral hemorrhage. Stroke 3, 527-537 (1972)
- 13. MÜKE, R., HEIENBROK, W., KÜHNE, D.: Spontane intrazerebrale Hämatome. Zbl. Neurochirurgie <u>39</u>, 135-144 (1978)
- NICHOL, E.St.: Long-term anticoagulant therapy in coronary atherosclerosis. Am. Heart J. <u>55</u>, 142-152 (1958)
- PASTOR, B.H., RESNICK, M.E., RODMAN, Th.: Serious hemorrhagic complications of anticoagulant therapy. J.A.M.A. <u>180</u>, 747-751 (1962)
- 16. PIA, H.W.: The surgical treatment of intracerebral and intraventricular hematomas. Acta Neurochir. 27, 149-164 (1972)
- 17. PIA, H.W.: Die operative Behandlung der spontanen Massenblutungen des Gehirns. Dtsch. Ärzteblatt <u>72</u>, 423-430 (1975)
- 18. PIA, H.W.: Die operative Behandlung der spontanen intrazerebralen Blutungen. Neurol. Psychiat. <u>5</u>, 356-364 (1979)
- PINEDA, A.: Computed tomography in intracerebral hemorrhage. Surg. Neurol. <u>8</u>, 55-58 (1977)
- ROOS, J., VANJOOST, H.E.: The cause of bleeding during anticoagulant treatment. Acta Med. Scand. <u>178</u>, 129-131 (1965)
- 21. STEUDEL, W.I., SCHNEIDER, E., BECKER, H.: Management and prognosis of intraventricular hemorrhage. In: Advances in neurosurgery, Vol. 7. MARGUTH, F., BROCK, M., KAZNER, E., KLINGER, M., SCHMIEDEK, P. (eds.), pp. 81-88. Berlin, Heidelberg, New York: Springer 1979
- THALER, E., NIESSNER, H.: Physiologische und pharmakologische Grundlagen der Antikoagulantienbehandlung. Dtsch. Ärzteblatt <u>76</u>, 787-794 (1979)
- 23. WALSHE, Th.M., DAVIS, K.R., FISHER, C.M.: Thalamic hemorrhage: A computed tomographic-clinical correlation. Neurology <u>27</u>, 217-222 (1977)
- 24. WEISBERG, L.A., NICE, C.N.: Intracranial tumors simulating the presentation of cerebrovascular syndromes. Am. J. Med. <u>63</u>, 517-524 (1977)

25. WÜLLENWEBER, R., ZUMWINKEL, K., GRUMME, Th., LANGE, S., MEESE, W.: Differentialdiagnose des Schlaganfalles im Computer-Tomogramm. Neurochirurgia <u>19</u>, 1-9 (1976)

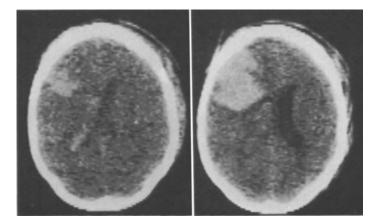


Fig. 1. Left: CT: Left frontal hematoma with CSF-blood mixture in the left lateral ventricle in a 25-year-old girl who was somnolent on admission. Right: CT 24 hours later of the same patient: Enlargement of the hematoma. The patient became comatose. Removal of the hematoma resulted in rapid improvement

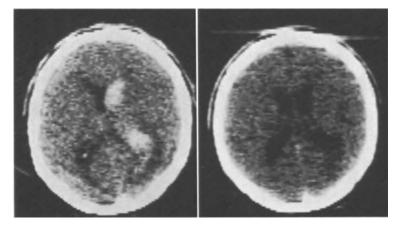


Fig. 2. Left: CT: Blood clots in the right lateral ventricle in a 47-year-old man. No operation. *Right:* CT of the same patient 18 days later: Resorption of the clot. The patient survived with hemianopia

Evaluation of Sleep EEG Alterations During Posttraumatic Coma in Order to Predict Cognitive Defects

A. ALEXANDRE, F. COLOMBO, L. RUBINI, and A. BENEDETTI

Introduction

Because of the large number of young people affected by severe cerebral traumatic lesions, the problem of predicting and treating residual neuropsychological deficits has recently been emphasized by several authors (2, 10, 16, 17, 23). Correlations with the entity of injury have been searched for, expressed by parameters such as the duration of unconsciousness (17), post-traumatic amnesia (PTA) (10, 16), the presence of skull fractures (23) and neurological deficits (10, 23). We (2) were able to demonstrate a significant correlation with the neurological syndrome of the level of the lesion in the acute phase.

In consideration of the role played by medial temporal structures both on sleep and cognitive activities, we have studied the possibility of predicting the neuropsychological outcome on the basis of sleep EEG changes in the acute phase.

Patients and Methods

This paper presents a series of 40 severe head injuries admitted and operated on for intra and/or extracerebral expanding lesions. In 24 cases the lesion was on the left, in 16 on the right. On admission, neurological situation was classified according to PLUM and POSNER (21) and BRICOLO et al. (8). Eleven patients showed the picture of a hemispheric syndrome, 17 a diencephalic picture, and 12 a mesencephalic syndrome. As soon as possible, daytime EEG recordings were performed, and tracings were classified following the proposal of BRICOLO et al. (9). Nocturnal electropolygraphic recordings were performed in the acute phase, and during post-traumatic amnesia. They were repeated every two months thereafter, together with the application of neuropsychological testing by means of the Wechsler Scale, Raven, Corsi, Kimura, and "token" tests. Those subjects whose performance was up to 20% below the control group were classified as having "mild deficits", "severe deficits" includes the patients with lower performances.

Results

Electroencephalographic recordings in the 11 patients with a hemispheric syndrome showed moderate and focal alterations. In the 17 patients with a diencephalic syndrome, diphasic tracings with spindles were recorded in 10 cases, diphasic without spindles in 2, and borderline in 5. The 12 patients with a mesencephalic syndrome showed a diphasic tracing with spindles in 6 cases, diphasic without spindles in 4, and monophasic in 2. Nocturnal electropolygraphic recordings in the acute phase revealed relevant sleep dysregulations (Table 1) in patients affected by most severe brain injuries, with abundant sleep phases 1 and 2, reduced phases 3 and 4, and severely reduced or absent paradoxical sleep.

The follow-up of sleep alterations is synthetized by the observations obtained at 12 months after injury: a progressive restoration of the various sleep phases is observed in each neurological syndrome (Table 2). The hemispheric syndrome allows an almost complete restructuration and particularly the slow-EEG sleep phase 4 and REM sleep are normal. One year later patients coming out of the diencephalic syndrome show various sleep EEG changes, consisting of persistently increased phases 1 and 2, a significant reduction of phase 3, and remarkable impairment of phase 4 and REM sleep. Patients recovering from the mesencephalic syndrome, the REM sleep and slowsleep phase 4 are severely impaired, and this parallels a marked reduction of phase 3 and an augmentation of phases 1 and 2.

The application of neuropsychological testing up to one year after injury (Table 3) shows that patients affected by mild cognitive deficits are usually those recovering from a diencephalic syndrome, while the great majority of subjects having severe deficits come from the mesencephalic group. No residual cognitive deficits are observed in patients recovering from the hemispheric syndrome as a rule.

A correlation has been attempted between neuropsychological and sleep EEG abnormalities at one year after injury (Table 4). Patients free from cognitive defects show minimal sleep dysregulations, and every phase is restored, paradoxical sleep always reappearing after complete restructuration of slow-EEG sleep. Patients with mild cognitive deficits show moderate sleep alterations, mostly concerning the sleep phases 4 and REM. Such phases never appear normal in these patients. Severe cognitive deficits parallel conspicuous alterations of all sleep phases, but phases 4 and REM are eminently involved, and in some cases may even be absent.

We have then looked back at the EEG sleep tracings of the acute phase, on the basis or our neuropsychological classification (Table 5). Apart from the initial neurological syndrome, a slight sleep alteration correlates with a good cognitive outcome, while a more severe sleep. dysregulation is found in patients who will show severe cognitive deficits. Only 12% of the patients with initial REM sleep abolition have a normal performance one year later and all patients with severe cognitive deficits have an acute abolition of REM sleep.

Discussion

In our series of patients, the changes in the sleep EEG, which match those observed by BERGAMASCO et al. $(\underline{4})$, by BRICOLO et al. $(\underline{5}, \underline{6})$ and by NAJENSON (<u>18</u>), seem to correlate with the neurological syndrome of the level of the lesion, and even more precisely with the neuropsychological outcome. Patients with a diencephalic syndrome for example may show none (35.2%) or mild (41.1%) or severe (23.5%) cognitive dysfunctions, but sleep analysis allows us to make a distinction between those who will be impaired mostly on the basis of the alterations of phases 4 and REM. 66% of patients affected by severe neuropsychological deficits recover from a mesencephalic syndrome, and the remainder from the diencephalic syndrome, but they all showed acutely a complete abolition of REM sleep and of sleep phase 4. In

Table 1												
Acute phases	slo	Slow sleep	eep				1			REM	REM sleep	d,
syndromes	Pha	ses	Phases 1 and 2	Pha	Phase 3		Phase	se 4				
No. patient	N	н	Ab	z	ч	A	z	ч	A	Z	ы	A
Hemispheric 11	m	œ		.9	ъ		و	ъ		4	7	
Diencephalic 17	-	4	12		17			7	10		5	12
Mesencephalic 12		7	10		6	e		-	11			12
<u>N</u> , normal; <u>I</u> , increase	d; <u>A</u>	þ, a	increased; <u>Ab</u> , abundant; <u>R</u> ,	<u></u> В, г	reduced; <u>A</u> ,	sd; ₽		absent.				
Table 2. Twelve months after injury	aft	er i	njury									
Previous neurological syndrome	Slo	Slow sleep phases 1 au	Slow sleep phases 1 and 2	Pha	Phase 3		Phase	se 4		REM	REM sleep	Q,
No. patient	z	н	Ab	z	ы	A	N	Я	A	N	R	A
Hemispheric	6	5		6	2		11			11		
Diencephalic	4	6	4	8	6		-	15	-		17	
Mesencephalic		8	4	-	1			7	ъ		8	4

Abbreviations see Table 1.

Mesencephalic

376

т	ab	le	3

Residual	Initial Neurol	ogical Syndrome		Total
cognitive deficits	Hemispheric	Diecenphalic	Mesencephalic	
None	9	6	1	16
Mild	2	7	3	12
Severe		4	8	12
Total	11	17	12	40

no case were the other sleep phases organized normally. Only 18.7% of the patients free from cognitive alterations revealed an acute abolition of REM sleep, and the slow-sleep phases were normal in about 37.5% of cases. The correlation is particularly valid for sleep phase 4.

Longitudinal electropolygraphic recording resulted in the observation that the sooner the EEG is restored, the earlier we find normalisation of the cognitive functions.

Temporo-basal and medial structures, among them the hippocampus, assume a specialized function during REM sleep: there is evidence of a special EEG activity (<u>15</u>, <u>6</u>), which parallels an increase in cerebral blood flow (<u>13</u>) in the structures bearing direct connections to the locus ceruleus. This locus is fundamental for inducing and maintaining REM sleep. Furthermore hippocampectomy influences sleep activity, reducing fast sleep, and increasing slow sleep (<u>19</u>, <u>14</u>). On the other hand these same structures have a significant role in cognitive functions: specific electrical activity persists in pyramidal hippocampal cells a long time after the end of a stimulus in the specific cortical areas (<u>24</u>), and several authors have observed amnesia following occlusion of the posterior cerebral artery (<u>3</u>) and following ablation of these structures (<u>22</u>, <u>20</u>). Furthermore in normal subjects REM sleep deprivation affects mnesic processes (<u>24</u>, <u>11</u>) and rats deprived of REM sleep have a reduced learning ability (<u>24</u>).

Cerebral traumatic lesions are polytopistic $(\underline{1}, \underline{7})$ and affect several neuronal systems, but the highly frequent involvement of medial temporal lobe structures and of mesencephalic nuclei, which are involved both in cognitive and sleep functions $(\underline{24}, \underline{12})$, is suggestive of the anatomical basis of the proposed correlation. This may offer a further contribution to the individual prognosis of the head injured patient during the acute phase and post-traumatic amnesia.

<u>Table 4</u> . Twelve months after injury	lve months	aft(i Sr	njury.									
Residual		slov	v s]	Slow sleep			1				REM	REM sleep	0,
cognitive deficits		Pha	Phase 1	and 2	Phas	Phase 3		Phase	se 4				
No. patient		N	н	Ab	z	Я	A	z	ы	A	N	Я	A
None	16	6	7		12	4		12	4		11	Ŋ	
Mild	12	7	9	4	4	ဆ			10	2		12	
Severe	12	7	9	4	2	10			8	4		ω	4
Residual cognitive deficits		Acu Slo	κe s]	lase eep	ep al	sleep alterations	ions				REM	REM sleep	_ ۵
מפדדכדרס		Pha	Phase 1	l and 2	Phase	se 3		Phase	se 4				
No. patient		z	н	Ab	N	Я	A	N	В	A	z	8	A
None	16	4	6	£	9	10		9	10		4	6	с
Mild	12		m	6		12			m	6		ε	6
Severe	12		7	10		6	3			12			12

Abbreviations see Table 4.

Severe

- ADAMS, J.H., GRAHAM, D.I.: The pathology of blunt head injuries. Scientific foundations of neurology. CRITCHLEY, M., O'LEARY, J.D., BENNETT, B. (eds.), p. 478. London: Heinemann 1972
- ALEXANDRE, A., NERTEMPI, P., FARINELLO, C. et al.: Test di Kimura, memoria visiva e capacità di apprendimento nei traumatizzati cranici. Riv. Ital. EEG 2, 153-158 (1979)
- BENSON, D.F., MARSDEN, C.D., MEADOWS, J.C.: The amnesic syndrome of posterior cerebral artery occlusion. Acta Neurol. Scandinav. 50, 133-145 (1974)
- BERGAMASCO, B., BERGAMINI, L., DORIGUZZI, T. et al.: EEG sleep patterns as a prognostic criterion in post-traumatic coma. Electroencephalogr. Clin. Neurophysiol. 24, 374-377 (1968)
- BRICOLO, A.: Prolonged posttraumatic coma. In: Handbook of clinical neurology. LINKEN, P.J., BRUYN, G.W. (eds.), pp. 699-755. North-Holland Publ. Co. 1976
- BRICOLO, A.: Neurosurgical exploration and neurological pathology as a means for investigating human sleep semiology and mechanisms. In: Experimental study of human sleep: Methodological problems. LAIRY, G.C., SALZARULO, P. (eds.), pp. 51-82, Amsterdam: Elsevier Scientific Publ. Co. 1975
- 7. BRICOLO, A., TURAZZI, S., ALEXANDRE, A. et al.: Decerebrate rigidity in acute head injury. J. Neurosurg. 47, 680-698 (1977)
- BRICOLO, A., BATTISTINI, N., BERGAMINI, L. et al.: A proposal for the classification of acute coma due to organic cerebral lesion. J. Neurosurg. Sci. <u>19</u>, 113-117 (1975)
- 9. BRICOLO, A., SIGNORINI, G.C., MAZZA, C. et al.: Clinical criteria for the prognosis of acute cerebral traumatic coma. In: Presents limits of neurosurgery. FUSEK, I., KUNC, Z. (eds.). Prague: Avicenum 1972
- BROOKS, D.N.: Wechsler memory scale performance and its relationship to brain damage after severe odsed head injury. J. Neurol. Psychiat. <u>39</u>, 593-600 (1976)
- 11. CAMBIER, J.: Personal communication (1978)
- 12. HOREL, J.A.: The neuroanatomy of amnesia: a critique to the hippocampal memory hypothesis. Brain <u>101</u>, 403-445 (1978)
- INGVAR, D.H.: Cerebral metabolism and circulation in wakefulness, sleep and coma. Astronautica Acta <u>17</u>, 171-178 (1972)
- 14. KIM, C., CHOI, H., KIM, C.C. et al.: Effect of hippocampectomy on sleep patterns in cat.s Electroencef. Clin. Neurophys. <u>38</u>, 235-243 (1975)
- 15. LENA, C., PARMEGGIANI, P.: Hippocampal theta rhythm and activated sleep. Helv. Physiol. Acta <u>22</u>, 120-135 (1964)
- MANDLEBERG, I.A.: Cognitive recovery after severe head injury.
 WAIS Verbal and performance IQs as a function of post-traumatic amnesia duration and time from injury. J. Neurol. Neurosurg. Psychiat. 39, 1001-1007 (1976)
- 17. MAZEAU, M., DUCARNE, B., HELD, J.P.: Le devenir neuropsychologique des traumatisés craniens sévères. 3rd World Congress of the IRMA. Basel, 2-8 July 1978

- 18. NAJENSON, T., GROSWASSER, Z., MENDELSON, N. et al.: Rehabilitation of the severe head injuried. 3rd World Congress of the IRMA. Basel, 2-8 July 1978
- 19. PARMEGGIANI, P., ZANOCCO, G.: A study of the bioelectrical rhythms of cortical and subcortical structures during activated sleep. Arch. Ital. Biol. <u>101</u>, 385-412 (1963)
- 20. PENFIELD, W., MATHIESON, G.: Memory. Autopsy findings and comment on the role of hippocampus in experimental recall. Arch. Neurol. <u>31</u>, 145-154 (1974)
- 21. PLUM, F., POSNER, J.B.: The diagnosis of stupor and coma. 2nd ed. Philadelphia: F.A. Davis 1972
- 22. SCOVILLE, W., MILNER, B.: Loss of recent memory after bilateral hippocampal lesions. J. Neurol. Neurosurg. psychiat. <u>20</u>, 11-21 (1957)
- 23. SMITH, E.: Influence of site of impact on cognitive impairment persisting long after severe closed head injury. J. Neurol. Neurosurg. Psychiat. <u>37</u>, 719-726 (1974)
- 24. SPINLER, H.B., STERZI, R., VALLAR, G.: Le amnesie. Milano: F. Angeli 1977

Changes of Lactate in Blood and Cerebro-Spinal Fluid After Osmotherapy with Sorbitol in Neurosurgical Patients

A. SPRING, G. SPRING, and H. DIETZ

Introduction

A rise the lactate levels in blood and cerebro-spinal fluid (CSF) may indicate an increase of anaerobic processes. Especially local hypoxia of brain, which does not change the oxygen content in the CSF and blood, may be recognized by a determination of the lactate level (10). There are many causes for CSF lactate acidosis such as meningitis, brain injuries, intracranial bleeding and metabolic disturbances (20) for example. Infusions of carbohydrate substances also produce an iatrogenic increase in the serum lactate level, if they are metabolised via fructose to lactate (22, 24). The question arises, whether these changes in serum lactate are reflected in the CSF after osmotic therapy with sorbitol.

Material and Approach

Our study was performed in 36 patients with brain tumors. In 15 patients the influence of sorbitol (1 g/kg b.w.) on the serum lactate level was tested (group I). In 9 patients with an external CSF drainage from the right lateral ventricle, the CSF- and serum-lactate concentration were examined at the same time (group II). Twelve patients who served as the control group had no osmotherapy (group III). During this study all patients were ventilated artificially with a range of pCO₂ between 32 and 36 mm Hg.

Results

After osmotherapy with sorbitol in all patients in group I, there was a significant increase of arterial serum lactate up to 4 hours compared with the control group. At the same time, the blood gas analysis shifted to acidotic (<u>16</u>). After 6 hours the serum lactate level showed no significant increase (Fig. 1). In group II serum lactate showed the typical significant elevation within the first hour. Later the CSF lactate concentration increased gradually reaching a maximum after 6 hours (Fig. 2). The degree of dependence of CSF lactate on serum lactate concentration correlated progressively and reached the maximum correlation after two hours (Fig. 3). This statistically proven correlation between blood and CSF lactate is maintained for 6 hours. It is remarkable, that the serum lactate concentration declines approximately to the initial values after 6 hours. On the other hand the CSF lactate does not have the trend to decrease after 6 hours. The cause is the delayed lactate clearance of CSF (<u>2</u>, <u>7</u>).

Discussion

Our results show that serum and CSF lactate rise after osmotherapy with sorbitol and that the passage of lactate from blood to CSF through the blood-brain-CSF-barrier is possible. These findings agree with animal experiments of DANIEL, LEUSEN and WEYNE, NEMOTO and OLDENDORF (5, 8, 11, 12, 13, 14) and are in contrast to previous studies, done by CRONE, POSNER and VALENCA (4, 17, 23). As we observed, the *delayed* passage of lactate from blood to CSF explains the false opinion, that there is no passage of lactate from blood to CSF.

It is obvious, that the increase of CSF lactate due to sorbitol is of importance, because there are close correlations between the severity of several diseases and the CSF lactate concentration (3, 9, 20, 21). Especially the prognosis of brain injury and stroke is unfavourable, if the lactate levels in CSF reach 35 mg% (9, 19, 20, 21).

Following a single sorbitol infusion we observed a rise in the CSF lactate level to 27 mg% on the average. In some cases values of more than 40 mg% were reached. Repeated infusions of sorbitol may potentiate this effect because of the low CSF lactate clearance (2, 7). A previously existing acidosis will be intensified. Unfavourable reactions of the edematous brain are to be expected. It is well-known that lactic acidosis causes deficiency in the cardio-vascular system (15), renal function (1, 6, 22) and carbohydrate metabolism (18, 22, 24). This induces progressive cerebral malfunction.

The indication for osmotherapy with sorbitol must be critically examined. The risk of possible side effects can be reduced, if one takes in account the effects of sorbitol on metabolism and acidbase balance.

- BERG, G., MATZKIES, F., HEID, H.: Zur Wirkung hochdosierter Sorbitinfusionen auf den Kohlenhydrat-, Fett- und Harnsäurestoffwechsel, den Säure-Basen-Haushalt und die Elektrolytkonzentrationen im Serum und Urin bei gesunden Männern. Dtsch. med. Wschr. <u>99</u>, 2352-2356 (1974)
- 2. CAUTHEN, J.C., NELSON, S.R., HUSTEAD, R.F., SAYLOR, C.R., OVERMANN, J.W.: Spinal fluid pyruvate and lactate levels. Arch. Neurol. (Chic.) <u>22</u>, 463-469 (1970)
- CROCKARD, H.A., TAYLOR, A.R.: Serial CSF lactate/pyruvate values as a guide to prognosis in head injury coma. Europ. Neurol. <u>8</u>, 151-157 (1972)
- 4. CRONE? C., SORENSEN, S.C.: The permeability of the blood-brain barrier to lactate and pyruvate. Acta physiol. scand. <u>80</u>, 47A (1970)
- 5. DANIEL, P.M., LOVE, E.R., MOORHOUSE, S.R., PRATT, O.E., WILSON, P.: The movement of ketone bodies, glucose, pyruvate and lactate between the blood and the brain of rats. J. Physiol. <u>221</u>, 22-23 (1972)
- 6. GOFFERJE, H., LANDGRAF, H., v. SCHWERIN, W., SCHULTIS, K.: Zur Osmo-Onkotherapie bei Hirnödem. Infusionstherapie <u>2</u>, 355-358 (1975)

- GRANHOLM, L.: The effect of blood in the CSF on the CSF-lactate, pyruvate and bicarbonate concentrations. Scand. J. clin. Lab. Invest. 23, 361-366 (1969)
- LEUSEN, I., WEYNE, J.: Metabolic processes in the brain during respiratory and non-respiratory alkalosis and acidosis. In: Acid base homeostasis of the brain. Extracellular fluid and the respiratory control system. LOESCHKE, H.H. (ed.), pp. 27-42, Stuttgart: Thieme 1976
- 9. MARX, P.: The clinical significance of CSF acid-base determination. In: Advances in neurosurgery, Vol. 3. PENZHOLZ, H., BROCK, M., HAMER, J., KLINGER, M., SPOERRI, O. (eds.), pp. 81-84. Berlin, Heidelberg, New York: Springer 1975
- 10. MÜLLER-PLATHE, O.: Säure-Basen-Haushalt und Blutgase. Pathophysiologie - Klinik - Methodik. In: Klinische Chemie in Einzeldarstellungen. Bd. I., BREUER, H., BÜTTNER, H., HILLMAN, G., STAMM, D. (eds.), pp. 47-51. Stuttgart: Thieme 1973
- 11. NEMOTO, E.M., HOFF, J.T., SEVERINGHAUS, J.W.: Lactate uptake and metabolism by brain during hyperlactatemia and hypoglycemia. Stroke 5, 48-53 (1974)
- 12. NEMOTO, E.M., SEVERINGHAUS, J.W.: Stereospecific permeability of rat blood-brain barrier to lactic acid. Stroke <u>5</u>, 81-84 (1974)
- OLDENDORF, W.H.: Blood brain barrier permeability to lactate. Europ. Neurol. <u>6</u>, 49-55 (1971)
- 14. OLDENDORF, W.H.: Carrier-mediated blood-brain barrier transport of short-chain monocarboxylic organic acids. Am. J. Physiol. <u>224</u>, 1350-1453 (1973)
- 15. OLIVA, Ph.B.: Lactic Acidosis. Am. J. Med. <u>48</u>, 209-225 (1970)
- 16. OTTEN, B., SPRING, G., OTTEN, G.: Veränderungen des Säure-Basen-Haushaltes durch intraoperative Osmotherapie bei Craniotomien. Vortrag DGAI Jahrestagung, Würzburg 1978
- 17. POSNER, J.B., PLUM, F.: Independence of blood and CSF lactate. Arch. Neurol. (Chicago) <u>16</u>, 492-496 (1967)
- 18. RENOLD, A.E., THORN, G.W.: Clinical usefulness of fructose. Am. J. Med. <u>19</u>, 163-167 (1955)
- 19. SCHNABERTH, G.: Aussagekraft von Verlaufsuntersuchungen des Säure-Basen-Haushaltes im Liquor cerebrospinalis beim cerebralen Insult. Dtsch. med. Wschr. <u>96</u>, 2018-2019 (1971)
- 20. SCHNABERT, G.: Säure-Basen-Haushalt und Atemgase im Liquor cerebrospinalis. Stuttgart: Thieme 1977
- 21. SEITZ, H.D.: Liquorveränderungen beim schweren Schädel-Hirn-Trauma und ihre therapeutische Beeinflussung. Fortschr. Med. <u>35</u>, 2088-2094 (1976)
- 22. TALKE, H., MAIER, K.P.: Zum Metabolismus von Glukose, Fruktose, Sorbit und Xylit beim Menschen. Infusionstherapie <u>1</u>, 49-56 (1973/ 74)
- 23. VALENCA, L.M., SHANNON, D.C., KAZEMI, H.: Clearance of lactate from the cerebrospinal fluid. Neurology (Minneap.) <u>21</u>, 615-620 (1971)
- 24. WOODS, H.F., ALBERTI, K.G.M.M.: Dangers of intravenous fructose. Lancet 23, 1354-1357 (1972)

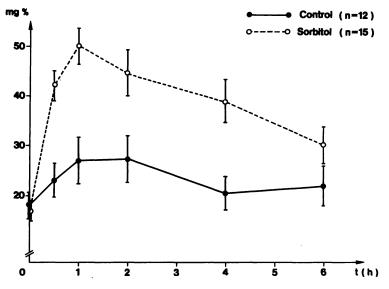


Fig. 1. Serum-lactate concentrations (mean \pm SEM) after infusion of sorbitol (1 g/kg) in relation to a control-group

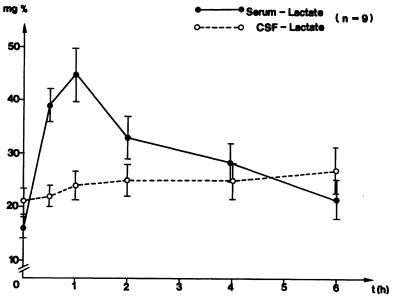


Fig. 2. Lactate concentrations of serum and CSF (mean \pm SEM) in sorbitol-treated patients at different periods

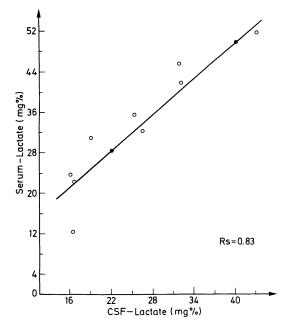


Fig. 3. Correlation of serum-lactate to CSF-lactate two hours after an infusion of sorbitol in 9 patients

Significance of VF-Lactate Analysis in Lesions with Increased Intracranial Pressure in Respect to the Prognosis

K. E. RICHARD and R. A. FROWEIN

Brain function disturbance of acute brain lesions such as brain trauma and space-occupying lesions are recognizable by neurologic and electroencephalographic alterations. The extent of the underlying cerebral hypoxia, can be estimated by the lactate concentration of the cerebrospinal fluid (2, 3, 5, 9).

How far can the estimation of the lactate concentration be helpful for the prognosis of an acute brain lesion?

Material

In 135 patients with space-occupying lesions, lactate concentration of the ventricular fluid (V-Lac), neurological state and mean ventricular fluid pressure (VFPx) were examined preoperatively and in the first postoperative week. VF-samples were obtained through a catheter in the right frontal horn. Blood-stained samples were excluded. In literature normal values of CSF-lactate are quoted to lie between 1,5-2,0 mmol/1 (Table 1).

References		N	CSF-lactate (mMol/l) X <u>+</u> SD
POSNER PLUM	1967	14	1.58 <u>+</u> 0.13
KAASIK ZUPPING	1969	?	1.62
ZUPPING	1970	?	2.03
METZEL ZIMMERMANN	1971	10	1.43 <u>+</u> 0.54
LAMISSE et al	1973	46	1.67 <u>+</u> 0.63
SCHNABERT	1977	10	1.62 <u>+</u> 0.91
RICHARD ELSNER	1977	13	1.54 ± 0.20

Table 1. Normal values of CSF-lactate

Results

Lactate concentration in millimoles per liter can be seen on the ordinate of the following figures.

As far as the outcome is concerned, the mean postoperative lactate values of the primary or the secondary dying patients are remarkably higher than those who survived (Fig. 1). In relation to the *mean VFP*, an increase of the mean lactate level can be determined only under very high postoperative pressures. The values show a considerable scattering (Fig. 2).

In relation to the *state of consciousness* the mean lactate concentration shows an increase preoperatively from 1.81 up to 3.33 mmol/l and postoperatively from 2.33 up to more than 4 mmol/l (Fig. 2).

In the group of normal VFP (Fig. 3), the VF-lactate of the unconscious patients was 13% higher than that of conscious patients, under slightly elevated pressure 53%, under severe elevated pressure 90%, finally under extremely elevated pressure it proved to be 143% higher (Fig. 3).

If we consider the significance of VF-lactate for the *prognosis* we must regard the neurological state, the intracranial pressure, the timing of the lactate estimation, and last but not least the patient's age.

Referring to the *neurological state*, 6 groups of increasing neurological deficit are differentiated: clear, clouded without or with slight neurological disorders, clouded with severe neurological disorders and the four well-known grades of coma (Fig. 4 a-c).

In the range of *normal* or *slightly increased pressures* high lactate levels up to 8 mmol/l were measured in the surviving patients (open symbols) as well as in the later dying patients (black symbols), even after the third post-operative day in the period of disturbed ventricular fluid circulation (Fig. 4a).

Unconscious patients survived only if the lactate levels did not exceed 3.5 mmol/l. On the other hand the follow-up was not always favourable, if the lactate concentration remained below this level.

In the range of *pressure* increases between 21-39 mm Hg the situation was similar: Large scattered values in the clear or clouded patients who usually survived. In the state of coma only a few children survived lactate levels up to 3.5 mmol/l (Fig. 4b).

With the increase of the mean *VFP over 40 mm Hg* favourable outcomes were only observed, when lactate levels did not exceed 3.0 mmol/l.

As a rule unconscious patients had an unfavourable prognosis except one child with a lactate level of 2.0 mmol/l on the day of operation (Fig. 4c).

Conclusion

From these findings the conclusion may be drawn, that the height of the VF-lactate concentration depends mainly on the extent of the acute brain lesion.

For patients with acute neurological diseases such as strokes, meningitis or alcohol delirium SCHNABERT (8) found, that CSF-lactate values above 3.0 mmol/l were absolutely unfavourable. As to brain injuries, other authors quoted higher critical values up to 6 mmol/l for the prognosis ($\underline{1}, \underline{4}, \underline{7}$). Our own findings, however, prove that the lactate concentration by itself does not allow any conclusion for the prognosis.

Clear or clouded patients survived VF-lactate levels up to 8 mmol/l. But as far as the number of the existing examinations permit, it can be said, that the prognosis of unconscious patients was always unfavourable, if the VF-lactate exceeded 3.5 mmol/l.

- CROCKARD, H.A., TAYLOR, A.R.: Serial CSF lactate/pyruvate values as a guide to prognosis in head injury coma. Europ. Neurol. <u>8</u>, 151-157 (1972)
- GRANHOLM, L., SIESJÖ, B.K.: Lactate and pyruvate levels in blood, cerebrospinal fluid and brain tissue. Acta Neurologica Scandinav. 43, Suppl. 31, 200 (1967)
- 3. KAASIK, A.E., NOLSSON, L., SIESJÖ, B.K.: The effect of asphyxia upon lactate, pyruvate and bicarbonate concentrations of phosphocreatine and adenine nucleotides in anesthetized rats. Acta physiol. scand. 78, 448-458 (1970)
- 4. METZEL, E., ZIMMERMANN, W.E.: Changes of oxygen pressure, acidbase balance, metabolites and electrolytes in CSF and blood after cerebral injury. Acta Neurochir. (Wien) 25, 177-188 (1971)
- 5. POSNER, J.B., PLUM, F.: Independence of blood and cerebrospinal fluid lactate. Arch. Neurol. <u>16</u>, 492-496 (1967)
- RICHARD, K.E., FROWEIN, R.A.: The relationship between intracranial pressure, disturbances of brain function and prognosis. Neurosurg. Rev. 1/2, 25-36 (1978)
- SCHLAG, G.: Kann die Bestimmung der Laktate im Liquor eine Aussage über einen irreparablen zerebralen Funktionsverlust geben? In: Die Bestimmung des Todeszeitpunktes. KRÖSL, W., SCHERZER, E. (eds.), Wien: Maudrich 1973
- SCHNABERTH, G.: Säure-Basen-Haushalt und Atemgase im Liquor cerebrospinalis. Stuttgart: Georg Thieme 1977
- ZUPPING, R.: Cerebral acid-base and gas metabolism in brain injury. J. Neurosurg. <u>33</u>, 498 (1970)

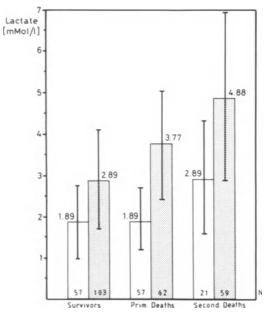


Fig. 1. Pre- () and postoperative () mean lactate values of ventricular fluids (VF) in survivors, primary or secondary dying patients

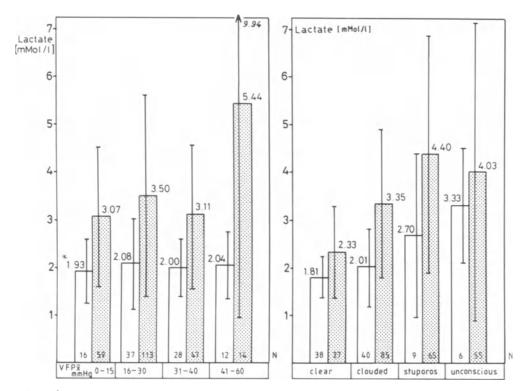


Fig. 2. Mean ventricular lactate in relation to mean ventricular fluid pressure (mm Hg) (left) and to the state of consciousness (right). \Box preoperative, \fbox postoperative

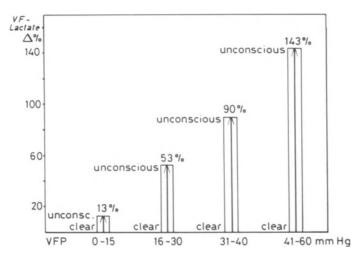
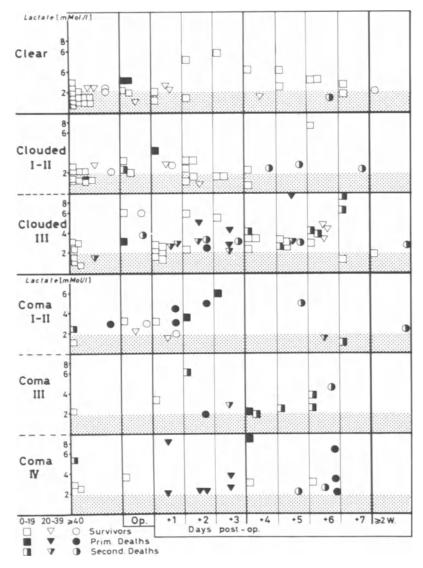


Fig. 3. Increase of ventricular lactate (%) in relation to ventricular fluid pressure and to state of consciousness (clear-unconscious)

390





<u>a</u> VF-lactate in the first week of treatment (N = 169), VFP, O-20 mm Hg

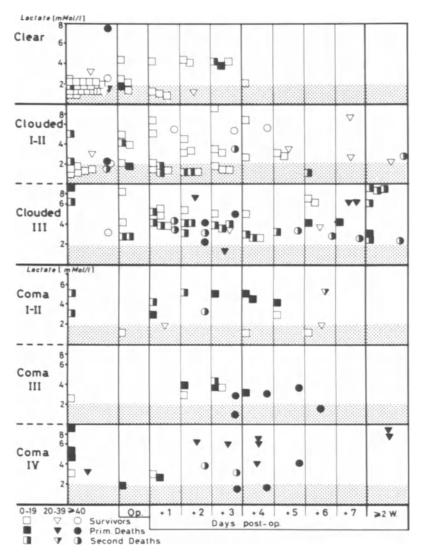


Fig. 4. b VF-lactate in the first week of treatment (N = 195), VFP, 21-39 mm Hg

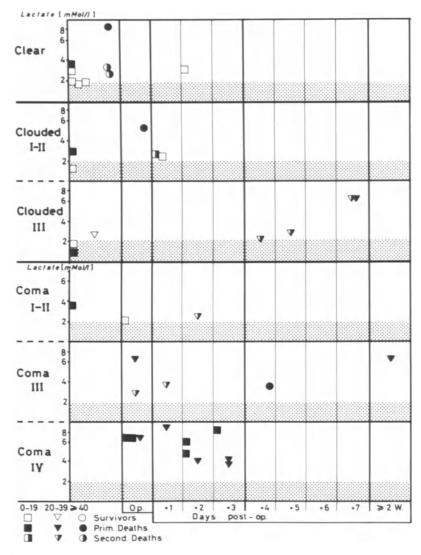


Fig. 4. c VF-lactate in the first week of treatment (N = 39), VFP, >40 mm Hg

Longitudinal Investigation on CSF Levels of Cyclic Nucleotides and Adrenergic Metabolites in Non-Neoplastic Hydrocephalus M. A. GEROSA, A. OLIVI, P. L. LONGATTI, L. MIETTO, S. TEOLATO, and A. CARTERI

Introduction

Metabolic reactions, control of ionic fluxes, neurosecretion and axonal transport have been reported to be some of the adenosine 3'-5' cyclic monophosphate (cAMP)-dependent cellular functions in the central nervous system (CNS) (6, 14). Several investigations indicate that cAMP may act as a neurotransmitter of the monoaminergic function (3, 9, 19). Catecholamine sensitive adenylcyclase in the brain is probably associated primarily with neurons, but to some extent also with glia $(\underline{6})$. Guanosine 3'-5' cyclic monophosphate (cGMP) has been suggested to be involved in the muscarinic actions of acetylcholine and cholinergic agonists have been described to enhance the accumulation of cGMP in brain slices from the cerebral cortex and cerebellum of mammals, via interactions with muscarinic receptors $(\underline{6})$. A correlation between these two cyclase systems and their blood brain barrier turnover has recently been proposed (1, 9, 12). Cerebrospinal fluid (CSF) concentrations of cyclic nucleotides and of the main monoaminergic metabolites (Homovanillic Acid, HVA; 5 Hydroxy-Indoleacetic Acid, 5 HIAA) were found to be severely altered in intracranial hypertension (12, 16, 17), as well as in non-neoplastic hydrocephalus (11, 13), before and after surgical treatment.

Materials and Methods

Three groups of children with post-meningitis hydrocephalus were investigated. The age range was from 7 days to 12 years, with a mean age of 5 years.

The first group (Group A, 43 cases) was composed of children with hypertensive post-meningitis hydrocephalus, never previously submitted to surgical treatment.

The second group (Group B, 34 cases) was composed of children with a "normal functioning" shunt. Samples were obtained during routine controls, 4-6 months after the operation.

The third group (Group C, 20 cases) included children with a malfunctioning shunt, hospitalized for surgical revision. Control values were obtained during ICP monitoring of children with suspected intracranial hypertension, with clinico-radiological findings and ICP recordings within normal limits. 4-7 ml of CSF were collected from each patient and 1 ml was treated with 1 ml of trichloro-acetic solution (12%), centrifuged and eventually frozen. The rest of the CSF was immediately frozen and stored at -25° for HVA and 5-HIAA assays. cAMP and cGMP were tested according to the Radio-Immuno-Assay Method of the Radio-Chemical Center (Amersham, Buckinghamshire, U.K.). HVA was evaluated according to KORF's method (21), 5-HIAA was determined with ASHCROFT and SHARMAN's technique (2), with minor modifications.

Results

Cerebrospinal fluid concentrations of cAMP (Fig. 1) turned out to be rather increased in the pre-operative findings. In normal functioning shunts cAMP values are slightly depressed, but very close to normal limits in shunt obstruction. A significant increase (P < 0.01) of cGMP levels (Fig. 2) was found in group A. This feature was even more pronounced in group B and C, with a complete inversion of the mean cAMP/cGMP ratio.

Pre-operative findings showed higher HVA concentrations (Fig. 3). A mild decrease was found in normal functioning shunts, in shunt obstruction HVA levels were within normal limits. A small increase of 5-HIAA values was observed in group A. No relevant changes in group B and C (Fig. 4).

Discussion

Most of our pre-operative findings are probably related to blood-brainbarrier lesions, or to the size and extent of destruction of ependymal and neuronal cells following meningitis and acute intracranial hypertension (7, 10, 17, 19, 20). Furthermore, the slight increase in cAMP, HVA and 5-HIAA levels, might also be associated with increased sympathetic activity (10).

cAMP concentrations were slightly depressed in the group B, but within normal limits in group C. The possible role of the cAMP system in intracranial hypertension is still a matter of debate. A mild decrease in cAMP levels was reported in patients with severe posttraumatic or post-hemorrhagic hypertension, either due to the decreased production of cAMP by brain adenylate cyclases or to accelerated active reabsorption of the cyclic nucleotide by means of the Probenecid-sensitive transport system (5, 18). On the contrary, no relevant changes were observed in patients with intracranial hypertension following mechanical stenosis of the cisternal pathways (20).

cGMP levels, in groups B and C turned out to be significantly increased. In a previous report (8), a very small group of children with shunt obstruction was described to have lower concentrations of cGMP: after a wider CSF monitoring, we would suggest that chronic or acute intracranial hypertension, particularly in shunt occlusion, is mostly associated with higher CSF levels of cGMP. A close correlation between ventricular cGMP concentration and ventricular pressure had recently been indicated (<u>17</u>): the tendency to accumulate in CSF should be proportional to the duration and magnitude of intracranial hypertension (<u>18</u>).

The decrease in the levels of HVA and 5-HIAA, fairly pronounced in the B group as regards the former, barely detectable in B and C group as regards the latter, might also be related to modified ICP. In fact the periventricular dopaminergic system might be directly involved in the functional and anatomical lesion due to raised ICP $(\underline{11}, \underline{12}, \underline{19})$.

Moreover, the altered CSF dynamics might influence the ventricular and choroid plexus absorption of HVA and 5-HIAA $(\underline{11})$.

An explanation for the lower levels of HVA should be provided by the low ventricular production and by the higher rate of turnover of this organic metabolite (1, 4, 11, 15).

Some of the above mentioned items (particularly the marked increase in cGMP, the decrease in cAMP and HVA concentrations) seem to suggest a very peculiar picture in the CSF of "normal" functioning shunts: metabolic patterns of a mild, chronic intracranial hypertension, even in absence of intracranial hypertension recordings.

- ANDERSSON, H., Von ESSEN, C., ROOS, B.E.: On the elimination of 5-Hydroxyindoleacetic acid and Homovanillic Acid from cerebrospinal fluid. Acta Pharmacol. et Toxicol. <u>32</u>, 129-138 (1973)
- ASHCROFT, G.W., SHARMAN, D.F.: 5-Hydroxyndoles in human cerebrospinal fluid. Nature (London) <u>186</u>, 1050-1051 (1960)
- 3. AXELROD, J.: Neurotransmitters. Sci. Amer. 230, 59-72 (1974)
- BULAT, M.,ZIVICOVIC, B.: Origin of 5-HIAA in the spinal fluid. Science 173, 738-740 (1971)
- 5. CRAMER, H., LARRY, K.Y., CHASE, T.N.: Adenosine 3'-5' Monophosphate in cerebrospinal fluid. Arch. Neurol. <u>29</u>, 197-199 (1973)
- DALY, J.W.: The formation, degradation and function of cyclic nucleotides in the nervous system. In: International review of neurobiology <u>22</u>, pp. 105-106. New York, S. Francisco: Academic Press 1977
- 7. EDVINSSON, L., OWMAN, C., ROSENGREN, E., WEST, K.A.: Brain concentration of dopmaine, noradrenaline, 5 hydroxytryptamine and homovanillic acid during intracranial hypertension following traumatic brain injury in rabbit. Acta Neurol. Scand. <u>47</u>, 458-462 (1971)
- GEROSA, M.A., CARTERI, A.: Cerebrospinal fluid of hydrocephalic children before and after ventricular shunting procedure. Child's brain <u>4</u>, 195-204 (1978)
- 9. GOLDBERG, N.D., HADDOX, M.K., NICOL, S.E., GLASS, D.B., SANFORD, C.H., KUHEL, F.A., Jr., ESTENSON, R.: CAMP and cGMP inhibitory interrelationship. Adv. Cyclic Nucelotide Res. <u>5</u>, 307-330 (1975)
- 10. HEIKINNEN, E.R., MYLLYLA, V.R., HOKKANEN, E., VAPAATALO, H.: CSF concentration of cAMP in cerebrovascular diseases. Euro. Neurol. 14, 129-137 (1976)
- 11. MAIRA, G., BAREGGI, S.R., DI ROCCO, C., CALDERINI, G., MORSELLI, P.L.: Monoamine acid metabolites and cerebrospinal fluid dynamics in normal pressure hydrocephalus: preliminary results. J. Neurol. Neurosurg. and Psychiat. 38, 123-128 (1975)
- 12. MOIR, A.T.B., ASHCROFT, G.W., CRAWFORD, T.B.B., ECCLESTON, D., GULDBERG, H.C.: Cerebral metabolites in CSF as a biochemical approach to the brain. Brain <u>93</u>, 357-368 (1970)
- 13. MASSAROTTI, M., MIGLIORE, A., ROCCELLA, P., TEGONS, S., TOFFANO, G.: 5-hydroxy-indoleacetic acid (5-HIAA) levels in the cerebrospinal fluid of hydrocephalic children before and after ventricular shunting procedure. Child's brain <u>4</u>, 195-204 (1978)

- 14. NATHANSON, J.A.: Cyclic nucleotides and nervous system function. Physiol. Rev. The A. Physiol. Soc. <u>57</u>, No. 2 (1977)
- 15. PERKINS, J.P., MOORE, M.M., KALISKER, A., SU, Y.F.: Regulation of cAMP content in normal and malignant brain cells. Adv. Cyclic Nucleotide Res. <u>5</u>, 641-660 (1975)
- 16. ROBINSON, A.G., BUTCHER, R.W., SUTHERLAND, E.W.: Cyclic AMP. New York: Academic Press 1961
- 17. RUDMAN, D., O'BRIEN, M.S., MCKINNEY, A.S., HOFFMAN, J.C. Jr., PATTERSON, J.H.: Observations on the cyclic nucleotide concentration in human cerebrospinal fluid. J. Clin. Endocrinol. Metab. <u>42</u>, 1088-1092 (1976)
- 18. RUDMAN, D., FLEISCHER, A., KÜTNER, M.N.: Concentration of 3'-5' adenosine monophosphate in ventricular CSF of patients with prolonged coma after head trauma or intracranial hemorrhages. N. Engl. J. Med. 295, 635-638 (1976)
- 19. TABBADOR, K., WOLFSON, L.I., SHARPLESS, N.: Ventricular fluid homovanillic acid and 5 Hydroxy-indoleacetic acid concentrations in patient with movement disorders. Neurology <u>28</u>, 1249-1253 (1978)
- 20. TSANG, D., LAL, S., SOURKES, T.L., FORD, R.M., ARONOFF, A.: Studies in cAMP in different compartments of cerebrospinal fluid. J. Neurol. Neurosurg. and Psychiatry <u>39</u>, 1186-1190 (1976)
- 21. WESTERINK, B.H.C., KORF, J.: Determination of nanogram amounts of HVA in the C.N.S. with a rapid semi-automated fluorimetric method. Biochemical Medicine <u>12</u>, 106-115 (1975)

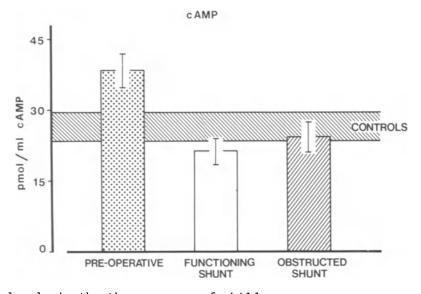


Fig. 1. cAMP levels in the three groups of children. Group A (43 cases): pre-operative findings. Group B (34 cases): "normal functioning shunt". Group C (20 cases): obstructed shunt. pmol/ml = picomoles/milliliter

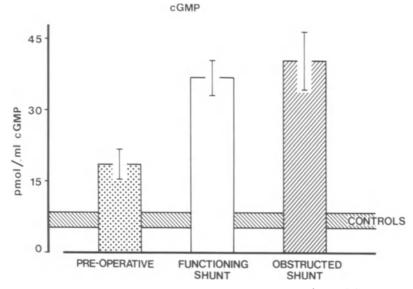


Fig. 2. cGMP levels in the three groups of children. Group A (43 cases): pre-operative findings. Group B (34 cases): "normal functioning shunt". Group C (20 cases): obstructed shunt. pmol/ml = picomoles/milliliter

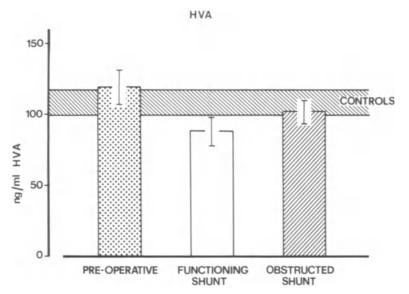


Fig. 3. HVA levels in the three groups of children. Group A (43 cases): pre-operative findings. Group B (34 cases): "normal functioning shunt". Group C (20 cases): obstructed shunt. ng/ml = nanograms/milliliter

398

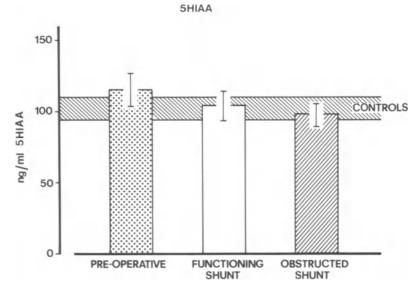


Fig. 4. 5-HIAA levels in the three groups of children. Group A (43 cases): pre-operative findings. Group B (34 cases): "normal functioning shunt". Group C (20 cases): obstructed shunt. ng/ml = nanograms/milliliter

Immunoelectrophoretic Studies on Human Intracranial Tumors D.-K. BÖKER

A prerequisite for immunotherapy in cases of tumor is the presence of tumor-specific antigens. The presence of at least tumor-associated antigens is necessary for the immunologic diagnosis of tumors. Therefore an attempt was made to find out whether there exists an antigen common to all glioblastomas and possibly specific for glioblastomas.

Material and Methods

Rabbits were immunized by s.c. or i.c. injection of homogenized glioblastoma. This was macroscopically free of necrosis as well as surrounding normal brain tissue and was suspended in complete FREUND's adjuvant. Immunization was carried out in weekly intervals. Rabbit sera were collected after 10 weeks, pooled, and stored until use at -20° C.

Tumor tissue was homogenized with an aliquot of physiological saline, and stored for extraction for 24 h at $+4^{\circ}$ C, being agitated occasionally. After centrifugation at 20.000 x g for 30 min the supernatant was considered to be a "soluble antigen", the protein concentration being adjusted to 10 mg/ml.

The rabbit sera were absorbed with pooled lyophilized human plasma, with liver or kidney homogenisate or normal brain immediately before use. The electrophoretic separation of the tumor extracts was performed in agar gel (1.5%) in MICHAELIS' buffer. After electrophoresis, grooves were cut into the gel, which were filled with the rabbit serum and left overnight. The experiment was terminated after the appearance of a precipitate in the positive control or after 16 hours at the latest.

Results

Immunoelectrophoresis with unabsorbed rabbit serum revealed several precipitation lines (Fig. 1). These lines disappeared except one after absorption of the rabbit serum with lyophilized pooled human plasma (Fig. 2).

The extracts of 165 human intracranial tumors were examined, and Table 1 shows the results. Positive reactions, i.e., precipitation lines in corresponding sites in the immunoelectrophoresis of extracts from the tumors and positive control were obtained in all neuroectodermal tumors, such as glioblastomas, astrocytomas, oligodendrogliomas, spongioblastomas, ependymomas, neurinoms, and plexus papillomas. Tumors of other than neuroectodermal origin (meningiomas, angioblastomas, metastases) gave negative results. The results in "medulloblastomas"

<u>Table 1</u>

Immunoelectrophoresis	Positive	Negative
Glioblastomas	53	0
Astrocytomas, oligodendro- gliomas, spongioblastomas	51	0
Ependymomas, neurinomas, plexus papillomas	12	0
Meningiomas	0	21
Metastases	0	9
Angioblastomas	0	4
"Medulloblastomas"	5	9

have been reported elsewhere $(\underline{1})$. Further absorption of the rabbit sera with liver or kidney homogenisate gave results identical to those after absorption with pooled human plasma alone: A single precipitation line still appeared.

Table 2 shows the results of some additional experiments: Immunoelectrophoresis was also done with extracts from normal brain. At a protein concentration of 10 mg/ml the result was negative. Results became positive after a 10-fold concentration of the extracts by dialysis. Extracts from the tumor vicinity which appeared macroscopically free of tumor gave positive results at a concentration of 10 mg protein/ml.

Table 2

Immunoelectrophoresis	Positive	Negative
Extract from glioma-vicinity 10 mg prot/ml	3	0
Extract from normal brain 10 mg prot/ml	0	1 1
30 mg prot/ml	õ	1
100 mg prot/ml	2	0

Discussion

In order to obtain an antiserum with activity against glioblastoma, five rabbits were immunized with a glioblastoma homogenisate, and immunoelectrophoretic investigations were performed in 165 cases of intracranial tumor. In all neuroectodermal tumors the results were positive, whereas all other tumors gave negative results. Positive results were also obtained by the examination of extracts from normal brain, but the protein contents had to be about 10-fold higher. Extracts from the macroscopically tumor-free vicinity of the tumors gave positive results at a protein concentration of 10 mg/ml corresponding to that of the tumor extracts. Because of its presence in normal brain tissue as well as in all neuroectodermal tumors, the antigen demonstrated by this study may be identical to the brainspecific antigen 14.3.2 on which among other HAGLID (2) has reported previously. It may be concluded that this antigen can be found in neuroectodermal tumors in a much higher concentration than in normal brain tissue. In the tumor vicinity it is found in concentrations similar to those within the tumor itself. This may indicate a pre-malignant/pretumoral state of this tissue.

- BÖKER, D.-K., ENTZIAN, W., GULLOTTA, F.: Immunoelectrophoretic evaluations in posterior fossa tumors. Advances in neurosurgery, Vol. 5. FROWEIN, R.A., WILCKE, O., KARIMI-NEJAD, A., BROCK, M., KLINGER, M. (eds.), pp. 236-238. Berlin, Heidelberg, New York: Springer 1978
- HAGLID, K., CARLSSON, C.A., STAVROU, D.: An immunological study of human brain tumors concerning the brain specific proteins S-100 and 14.3.2. Acta Neuropath. (Berl.) 24, 187-196 (1973)

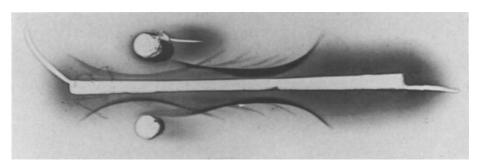


Fig. 1. Multiple precipitation lines, obtained with unabsorbed rabbit serum

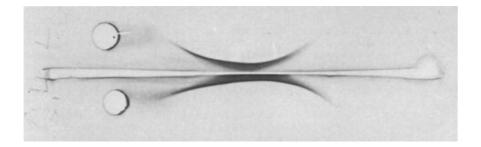


Fig. 2. Single precipitation line after absorption of the rabbit serum with pooled human plasma. Same results after additional absorption with liver and kidney homogenisate

Microsurgery of Brainstem Tumors in Childhood and Adolescence: A Review of Past Experience

G. PENDL and W. KOOS

A decade ago microneurosurgery was introduced into the operative management of brain tumors and has meanwhile produced encouraging results in many instances. This prompted us to review the experience acquired in the treatment of brainstem tumors, which BAILEY and associates (<u>1</u>) apostrophized as a "pessimistic chapter in the history of neurosurgery", no doubt with good reason.

Reports on the surgical management of brainstem tumors are rather scarce. In a noteworthy publication dating from 1971 LASSITER, ALEXANDER and coworkers (2) stressed the need for surgical exploration prior to conventional radiotherapy and, in fact, reported considerably longer survival times in cystic brainstem tumors thus managed.

At the Department of Neurosurgery, University of Vienna Medical School, tumors of the caudal brainstem (pons and medulla oblongata) accounted for 1.7% (176 cases) of all brain tumors seen since 1940. It is of interest to note the relative incidence of caudal brainstem tumors in children versus adults: in children 8% of all brain tumors affected the caudal brainstem, while only 0.9% were localized in this region in adults.

Prior to the introduction of the operative microscope (1940-1969) 47 of the 728 brain tumors in children involved the caudal brainstem (i.e., 8% of the total). Of these, 24 were surgically explored and biopsied. The operative mortality was approximately 60%, the ratio between adults and children 54% : 46% (considering surgical cases only). After the introduction of microsurgery, i.e., from 1970 -1978, caudal brainstem tumors accounted for 12% (42 cases) of the 350 childhood tumors undergoing microsurgery. In 22 of the 42 pontine and medullary tumors surgery was done with the help of the operative microscope. In terms of incidence, the ratio between adults and children was 48% : 52%.

Painstaking preoperative diagnostic studies including neurologic tests, ventriculography and cisternography with special emphasis on the 4th ventricle and the basal cisterns, computer tomography and technetium scans as well as past surgical experience served as a basis for classifying brainstem tumors in 4 groups by topography and morphology. Of these, 3 were found to be eligible for direct surgery. Figure 1 a-c list the groups and the associated operability.

The localization, morphology and prognosis of caudal brainstem tumors in 22 children undergoing microsurgery are shown in Fig. 2.

Group I, i.e., tumors exclusively growing in the brainstem proper, was predominantly composed of solid malignancies (1 spongioblastoma,

3 astrocytomas) and included 2 glioblastomas. It was in this group that the only intra-operative death was recorded. The patient lost was operated for a glioblastoma of the medulla oblongata and upper cervical spinal cord.

The symptomatology is primarily characterized by severe, rapidly progressing neurological brainstem symptoms. In some cases, even in the extensive tumors, neurologic deficits were, however, found to be relatively inconspicuous. This was probably referable to the infiltrative growth.

The 9 tumors in group II, i.e. medio-ventricular tumors, were most favorable both histologically and prognostically. Histologically, the material consisted of 1 spongioblastoma and 8 astrocytomas. In the majority of cases, single or multiple tumor cysts obstructed the lumen of the 4th ventricle and were thus responsible for an occluding internal hydrocephalus. For this reason, ventriculo-atrial shunting was done in 5 of the 9 children preoperatively with rapid improvement of the neurologic deficits.

Group III (3 cases) and particularly group IV (4 cases) were characterized by atypical cerebellopontine symptoms with associated severe cerebellar deficits in group III. Histologically, astrocytoma was present throughout.

Of the 22 patients who underwent microsurgery 8 are still alive, the survival time being 8 months to 9 years. Five of the survivors had group II tumors! 80% of the survivors are able to attend school or go to work. The operative mortality was 13%.

Aside from surgical exploration, the treatment plan followed in the cases reviewed here included radiotherapy as commonly practiced (telecobalt treatment) and, in the past few years, chemotherapy.

In summary, microsurgery in brainstem tumors should serve a number of purposes, i.e.:

- in all 4 groups to obtain biopsy material for establishing a histologic diagnosis and, as a consequence, planning the further course of treatment (radiotherapy, chemotherapy);
- in group II, to evacuate ventricle-blocking tumor cysts and resect the tumor walls as well as the cystically degenerated tumor mass, which serves to restore C.S.F. passage and decompress the brainstem;
- 3. in groups III and IV, to decompress the cerebellopontine cranial nerves and the brainstem itself by evacuating the cyst contents and partially resecting the tumor; and
- to extend the space available in the posterior fossa by suboccipital duraplasty (using lyophilized dura) with a view to accomodating potential radiation or chemotherapy - induced brain edema.

Summary

Before introduction of microsurgical techniques (1940-1969) 47 tumors of 728 brain tumors in children operated in the Neurosurgical University Clinic Vienna were located in the lower brainstem (i.e., 8% of the total number of brain tumors). In 24 cases of this group the tumor was surgically approached and the tumors histologically verified. In the second series of the microsurgical area 1970-1978 among 350 tumors in children 42 brainstem tumors were found (12%). On basis of exact preoperative diagnostic studies, four groups of tumors could be differentiated on the basis of topographic and morphological criteria. Three groups were eligible for direct surgical approach. Of 42 diagnosed brainstem tumors, 22 were explored by means of microsurgical techniques, with an intraoperative mortality of one case. To this day 9 patients are still alive 8 months to 9 years postoperatively. Useful life was achieved by 80% of the cases.

Besides direct surgical exploration (to evacuate cystic lesions and to perform a partial resection of the tumor), treatment included radiotherapy (tele-cobalt), chemotherapy, and in 5 cases with a concomitant hydrocephalus, a preoperative ventriculo-atrial shunting procedure was performed.

- BAILEY, P., BUCHANAN, D.N., BUCY, P.C.: Intracranial tumors of infancy and childhood. 3rd ed. Chicago, London: The University of Chicago Press 1964
- LASSITER, K.R.L., ALEXANDER, E. Jr., DAVIS, C.H. Jr., KELLY, D.L. Jr.: Surgical treatment of brainstem gliomas. J. Neurosurg. <u>34</u>, 719-725 (1971)

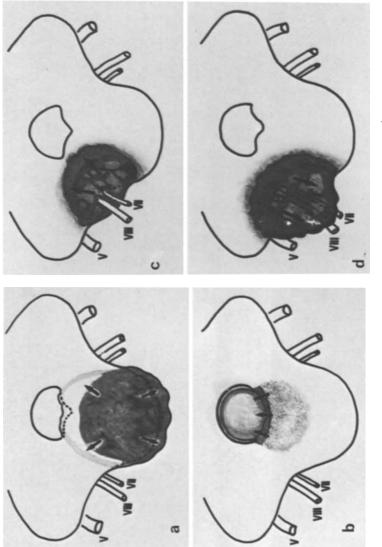
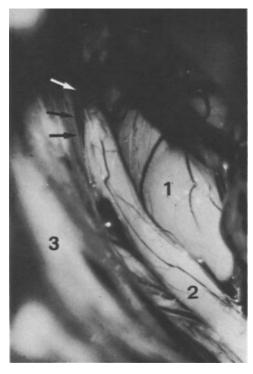


Fig. 1 a-d. Classification of caudal brainstem tumors into 4 groups ac-cording to topography and morphology. a Group I: medio-intratruncal; <u>b</u> Group II: medio-intraventricular; <u>c</u> Group III: latero-dorsal; <u>d</u> Group IV: latero-angular

Astrocytoma Spongioblastoma Glioblastoma	TUMOR-		L	ocalisatio	n
+ Postop. death * Living	GROUP		Pons	Med.obl.	Cerv.
S Va-Shunt		Solid			+
		Cyst.			
	6 Cases	Sol cyst.			*
		Solid	*	S	
	п	Cyst.			
	9 Cases	Sol cyst.	*	S *	S
		Solid			
	ш	Cyst.			
	3 Cases	Sol cyst.	*	*	
		Solid			
	IV	Cyst			
Fig. 2	4 Cases	Solcyst.			



<u>Fig. 3</u>. Dorsolateral astrocytoma of the pons compressing the cranial nerves VII and VIII. <u>1</u> tumor, <u>2</u> cranial nerves VII and VIII, <u>3</u> pyramid; *arrows* = internal auditory meatus

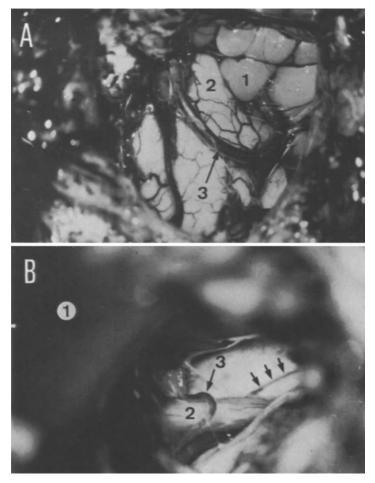


Fig. 4 A, B. Astrocytoma of the pons with extension into the cerebellopontine angle. Compression of the cranial nerves VII and VIII and the nerves of the vagus group. A Situation after right lateral suboccipital craniectomy and exposure of the tumor in the right cerebellopontine angle. 1 tumor, 2 pons, 3 PICA. B Situation after removal of the extrapontine portion of the tumor. 1 retracted cerebellum, 2 cranial nerves VII and VIII, 3 AICA; arrows = internal auditory meatus

Radiological and Therapeutic Aspects of Glomus Tumours E. LINS and H. J. BASEDOW

Seven cases of glomus tumors are reported. The diagnostic and therapeutic measures are described and discussed with the aid of X-ray pictures. The plain X-rays show the extension into the bone. The whole vascularisation of the tumour can only be demonstrated by the use of selective angiography, whereby the entire extension of the mass into the soft tissue can be estimated exactly. In cases of intracranial glomus tumours it was shown that the tumour can be vascularized by dural vessels coming from the internal and external carotid arteries as well as by collateral arteries from the vertebral system. Selective angiography also allows simultaneous therapy, because the blood supply of the tumour can be occluded by artificial embolization.

The glomus tomours are nonchromaffin parasympathic paragangliomas. Arteriography can be used with success when the tumor is situated at the level of the common carotid artery bifurcation, jugular foramen or at the level of the external auditory meature. Selective and superselective arteriography is of great value in demonstrating feeding arteries to the tumor. Consequently such arteriography is the method of choice in the diagnosis of these tumors. Selective arteriography has two advantages. First, by the careful placement of the catheter tip, one may be able to demonstrate feeding arteries. In addition one may be able to carry out therapeutic embolization at the same sitting. Because of the hypervascularity of these tumors, a biopsy is often associated with risk. In contrast, arteriography is associated with less risk and in addition will give information as to the operability of the tumor. In the cases reported, the transfemoral approach was used. By this method it was possible to demonstrate the external carotid artery and its tumor feeding branches. During arteriography it is important to demonstrate the posterior auricular artery and the stylomastoid artery since they often take part in tumor vascularization. Because these tumors are often found bilaterally, the contralateral carotid system should also be studied. A vertebral arteriogram should also be performed since these tumors are fed by branches of the vertebral and basilar arteries. A shift in the position of these arteries may indicate tumor extension into the posterior fossa. Arteriography of the internal carotid artery is not important in the diagnosis of glomus tumors. The internal carotid artery is involved only in very large tumors that receive their blood supply from branches of the internal carotid artery and middle cerebral artery.

In external carotid arteriograms the following are characteristic signs of a glomus tumor:

- an increase in the size and number of external carotid branches found in the temporal region;
- an increase in the amount of contrast medium in the temporal region;

- 3. an increase in the vascular supply of the temporal bone;
- 4. direct and indirect A.V. shunting;
- 5. occlusion of the lateral or sigmoid sinus and venous congestion;
- an increase in the angle of the bifurcation of the internal and external carotid arteries in carotid glomus tumors;
- 7. an anterior bowing of the external and internal carotid arteries as seen on the lateral projection especially by vagal glomus tumors.

In addition to the diagnostic value of selective arteriography is its therapeutic use in vascular embolization. Embolization was first used therapeutically for the treatment of glomus tumors by DJINDJIAN (1973/1974). Since the catheter is situated in the tumor feeding vessel, as demonstrated by arteriography, the opportunity presents itself to selectively embolize this vessel and consequently occlude it.

The advantages of embolization of glomus tumors are:

- because of the profuse vascularity of the tumors, operative management may be associated with a large blood loss;
- these tumors are often situated close to vital structures and associated with a high operative mortality rate (21,5%; WINSHIP 1952).

Embolization is performed immediately following arteriography. Nevertheless, revascularization of the tumor is inevitable. The combination of selective arteriography and embolization was used in seven cases. Figures 1, 2 and 3 demonstrate a case of a carotid glomus tumor. Figure 1 is an arteriogram of the common carotid artery showing a tumor blush at the level of the common carotid bifurcation. Figure 2 is a selective arteriogram of the occipital artery. The tumor blush indicates that branches of the occipital artery feed the tumor. Figure 3 is a selective injection of the ascending pharyngeal artery. As can be seen from this angiogram, it too is an important feeding artery. The tumor extends into the upper pharyngeal space and is thus inoperable.

Figure 4 is a case of metastatic glomus tumor to the frontal region as demonstrated via a brachial reflux study.

Conclusion

In conclusion selective arteriography is able to reveal most if not all feeding vessels to the tumor as well as the extent of tumor growth. At the same time, it presents the opportunity for a therapeutic embolization.

Embolization has the following advantages over pure operative and pure radiotherapeutic management:

- 1. It avoids repeated operative interventions.
- 2. During embolization, vital structures are not injured.
- 3. The procedure can be repeated several times without great risk to the patient.

References

- DJINDJIAN, R., COPHINGNON, J., THERON, J., MERLAND, J.J., HOUDART, R.: Embolisation by superselective arteriography from the femoral route in Neurora diology. Review of 60 cases. Technique, indications, complications. Neuroradiology <u>6</u>, 20-26 (1973)
- DJINDJIAN, R.: Indications, contre-indications, accidents, incidents dans l'embolisation de la carotide externe. J. Neuroradiol. <u>2</u>, 173-200 (1975)
- 3. DJINDJIAN, R., MERLAND, J.J.: Superselective arteriography of external carotid artery. Berlin, Heidelberg, New York: Springer 1978
- RITTER, K.: Glomustumoren in Hals-Nasen- und Ohrenbereich. HNO <u>22</u>, 6-9 (1974)



Fig. 1. Carotid common arteriogram of a carotid glomus tumor

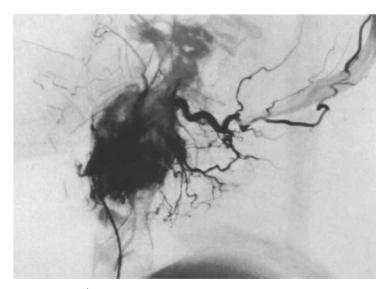


Fig. 2. Selective occipital arteriography



Fig. 3. Selective arteriography of the ascending pharyngeal artery

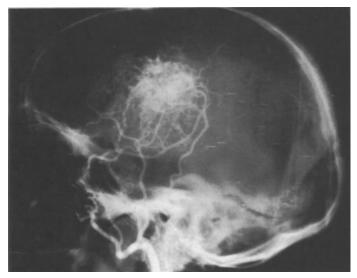


Fig. 4. Metastatic glomus tumor in the frontal region

Radiofrequency Percutaneous Trigeminal Thermal Rhizotomy Without General Anaesthesia

G. GRAZIUSSI and S. TERRACCIANO

Radiofrequency percutaneous trigeminal thermal rhizotomy described by SWEET and WEPSIC (3) was a great progress in the treatment of trigeminal neuralgia. This method is as effective as the traditional open surgery techniques but has the advantage of being very safe and of having a very low rate of complications.

Although greatly reduced, some complications still occur: these are unwanted trigeminal analgesia or anaesthesia and ocular paresis $(\underline{1}, \underline{2}, \underline{3}, \underline{5})$. Furthermore, repeated ultrashort anaesthesias may be an added risk to the procedure in patients with serious cardiac, renal or hepatic insufficiency. In order to minimize the abovementioned risks and complications we have adopted the technique of percutaneous rhizotomy in the awake patient attributed to ATKINSON $(\underline{3})$. There are two underlying principles to this method: (1) the size of the thermal lesion is temperature-dependent and time-independent using radiofrequency lesion generators; (2) coagulation of the trigeminal root is possible in the awake patient if very slow heating is employed.

Materials and Methods

The 115 procedures included in this study were performed on 112 patients from June 1977 to February 1979. All patients were suffering from idiopathic trigeminal neuralgia. Sixteen patients had been treated previously by retrogasserian root section or by alcohol injection into the Gasserian ganglion; 5 by percutaneous radiofrequency thermal rhizotomy; 19 by avulsion or alcohol injection of peripheral branches. In all these cases, sensory deficits following these procedures did not prevent a reliable evaluation of the operative results and complications. Three cases with considerable trigeminal sensory loss due to previous operations were excluded from this study. The standard technique was employed in 78 operations while the modified technique was employed in 37 operations.

Description of the Technique

Up to the moment of actual thermocoagulation, the technique is the same as that described by SWEET and WEPSIC $(\underline{3})$ and we omit its description for the sake of brevity.

We begin the radiofrequency coagulation raising the temperature quite quickly until the patient feels a burning sensation in the appropriate trigeminal areas; this usually occurs between 43° and 47° . From this moment on the temperature is raised very slowly, with 1/2-1 degree increments. After each increment the patient feels an increase of the

burning sensation in the ipsilateral side of the face, which usually lasts 30 seconds or less. As soon as this burning decreases, the patient himself gives the signal for the next temperature increment. During the entire coagulation procedure, the operator continuously tests trigeminal sensation, corneal reflex and ocular motility. The procedure is terminated as soon as the desired degree of analgesia has been obtained.

Results

Operative results are examined comparatively in the two different groups of procedures; they are summarized in Tables 1-4.

Table 1. Operative results

	Total relief	Partial relief	Total
Patients operated with general anaesthesia (78 cases)	71 (91.3%)	2 (2.2%)	73 (93.5%)
Patients operated with- out general anaesthesia (37 cases)	33 (88.2%)	2 (5.9%)	35 (94.1%)

Table 2. Operative complications

	Undesirable trigeminal analgesia	Undesirable trigeminal anaesthesia	Ocular paresis
Patients operated with general anaesthesia (78 cases)	14 (17.9%)	3 (3.9%)	1 (1.2%)
Patients operated with- out general anaesthesia (37 cases)	2 (5.4%)	0	0

Table 1 shows the percentage of short term successes: we obtained 93.5% successes in the first group (patients operated upon under intermittent general anaesthesia) and 94.1% in the second group (patients operated upon without general anaesthesia).

Table 2 deals with the complications: in the first group we had 3.9% trigeminal anaesthesia, 17.9% trigeminal analgesias in divisions not affected by the neuralgia and 1.2% ocular paresis. In the second group we had only 5.4% unwanted trigeminal analgesias and no cases of trigeminal anaesthesia or ocular paresis.

Table 3 shows operative results in first division neuralgias only. We must point out that in these cases our goal is to obtain hypalgesia and not complete analgesia in order to partially preserve the corneal reflex. In the first group of patients we had 53.8% complete corneal analgesias while in the second group we succeeded in obtaining only hypalgesia with preservation of the corneal reflex.

Table 3. Operative result	Operative results in first division neuralgias ^a	n neuralgias ^a			
	First division analgesia	First division hypalgesia	Ocular paresis	Total relief	Partial relief
Patient operated with general anaesthesia (13 cases)	7 (53.8%)	6 (46.2%)	1 (7%)	13 (100%)	0
Patient operated with- out general anaesthesia (6 cases)	0	6 (100%)	0	4 (33%)b	
a Five isolated and 14 cc b Atypical first divisior	ed and 14 combined with second rst division neuralgias.	second division neuralgia.	a.		
Table 4					
	Minimal	Maximal	Mean		
Temperatures reached					
Operation with general anaesthesia	540	006	75.8 ⁰		
Operation without general anaesthesia	50 ⁰	0 ⁰⁶	70.8 ⁰		
Duration of coagulation					
Operation with general anaesthesia	-	32'	- 9		
Operation without general anaesthesia	31	451	11.		

Table 4 shows temperatures reached and duration of coagulations in the two different groups of procedures. The mean coagulating temperatures were respectively 75.8° and 70.8° ; the mean durations of coagulation were respectively 6 and 11 minutes.

Finally it must be mentioned that we were not able to perform the modified operation because of patient's intolerance in 5 cases and we were therefore compelled to use intermittent ultrashort anaesthesia.

Discussion

The main advantages of percutaneous radiofrequency trigeminal thermocoagulation versus traditional "open" techniques are safety and selectivity. It is our opinion that every effort must be made to insure such a selectivity. Therefore we define as a complication not only the production of anaesthesia in the affected areas but also the appearance of severe analgesia in divisions not affected by the neuralgia. Ultrashort anaesthesias and staged increments of temperature of 5° each do not prevent these complications in every case (<u>3</u>).

On the one hand the technique of thermocoagulation in the awake patient enabled us to detect the appearance of hypalgesia in an inappropriate area and to promptly modify the electrode-tip position; on the other hand, it permited us to immediately perceive the production of the desired degree of analgesia, therefore avoiding undesirable anaesthesia. In first division neuralgias, it prevented production of too deep an analgesia with consequent loss of the corneal reflex. While complications were reduced in this way, the therapeutic results, in terms of short term success, were comparable to those achieved with the standard technique.

Conclusions

Percutaneous radiofrequency thermal rhizotomy without general anaesthesia appears to be a valuable procedure for the treatment of trigeminal neuralgia: the rate of operative complications is reduced while the therapeutic effectiveness is as good as with the standard technique; the greater discomfort associated with the procedure is accepted and tolerated by the vast majority of patients in view of the advantages.

We believe that is should be used at least for the treatment of first division neuralgias where production of anaesthesia or deep analgesia is particularly undesirable owing to the danger of keratitis.

- ONOFRIO, B.M.: Radiofrequency percutaneous Gasserian ganglion lesions. Results in 140 patients with trigeminal pain. J. Neurosurg. <u>42</u>, 132-139 (1975)
- SIEGFRIED, J.: 500 percutaneous thermocoagulations of the Gasserian ganglion for trigeminal pain. Surg. Neurol. 8, 126-130 (1977'
- SWEET, W.H., WEPSIC, J.G.: Controlled thermocoagulation of trigeminal ganglion and rootlets for differential destruction of pain fibers. Part 1: trigeminal neuralgia. J. Neurosurg. <u>40</u>, 143-156 (1974)

4. TEW, J.M. Jr., KELLER, J.T.: The treatment of trigeminal neuralgia by percutaneous radiofrequency technique. Clin. Neurosurg. <u>24</u>, 557-578 (1977)

.....

 TURNBULL, I.M.: Percutaneous rhizotomy for trigeminal neuralgia. Surg. Neurol. <u>2</u>, 385-389 (1974)

Stereotaxic Procedures Within the CT-Scanner with a New Targeting Device W. Huk

In order to arrive at a histological diagnosis in cases of spaceoccupying lesions, which cannot be established by other means, we have developed a new targeting device in close cooperation with the Siemens Corp. It enables us to perform stereotaxic procedures within the CT-scanner under CT-control. Several authors have used CT data for the localization of intracranial targets (1, 2, 3, 4).

Method

The apparatus consists of a) a head holder and b) a phantom. Both are provided with identical targeting rings.

The *head holder* is made out of a plexyglas tube which is adjustable in height and firmly attached to a stretcher.

The *phantom* carries three rings - a tumor ring, a burrhole ring and a targeting ring. These rings can be moved in relation to each other, in order to simulate the spatial relationship between the target, the burrhole and the targeting ring.

The unit is calibrated by measuring the biopsy needle in the zeroposition. This zero-position of the needle is the *reference point* for all measurements. It is stored in the computer or on the floppy disc. The images of the zero-position of both targeting rings must be identical.

Procedures

After the burrhole is performed in the operating room, the anaesthetised patient is placed inside the head holder. The patient's head is immobilized with firm plastic cushions. Now the section with the target point is scanned and superimposed on the image of the reference point. The coordinates of the target which are now seen, can be adjusted to the tumor ring of the phantom.

The same procedure is repeated with the burrhole. The distance between the level of the scan of the target point and the level of the scan of the burrhole is indicated on the millimeter scale of the patient's table on the scanner. The distance between the burrhole and the targeting ring can be measured directly. The straight line between the target and the burrhole now represents the position and the direction of the needle of the phantom.

The coordinates of this needle can be transferred to the needle of the biopsy device and the biopsy can be performed.

The coordinates of the needle can also be calculated by the computer with the help of a special program.

The present accuracy of the system is four millimeters in diameter.

Discussion

The main fields of indication for this unit are:

- 1. Biopsies of unknown lesions.
- Preoperative marking of small lesions in order to make their detection during operation easier and safer.
- 3. Therapeutic punctures of small cystic lesions.
- The implantation of radioactive substances into inoperable (midline) tumors.

The *disadvantage* of the unit is its restriction to supratentorial lesions. Access to the posterior fossa as well as to the frontal or subfrontal approach are not possible with the present version.

Its advantages are:

- The simple and accurate determination of the target point.
- The possibility of permanent CT-control during the operation.
- The simple and exact documentation of the origin of the biopsy specimen or of the position of radioactive implantations.
- The immediate detection of intraoperative complications.
- The short duration of the procedure.

Finnaly, the apparatus is independent of the brand of CT-scanners; without major difficulties it can be attached to all scanners with head openings of adequate size.

- BERGSTRÖM, M., GREITZ, T.: Stereotaxic computed tomography. Am. J. Roentgenol. 127, 167-170 (1976)
- BOETHIUS, J., COLLINS, V.P., EDNER, E., LEWANDER, E., ZAJICEK, I.: Stereotactic biopsies and computer tomography in gliomas. Acta Neurochir. 40, (3-4), 223-232 (1978)
- 3. CAILLE, J.M., COHADON, F., CONSTANT, P., CAMPAGNE, J.P.: CAT under stereotaxic conditions. In: Computerized acial tomography. DuBOULAY, G.H., MOSELEY, I.F. (eds.), pp. 43-45. Berlin, Heidelberg, New York: Springer 1977
- 4. MAROON, I.C., BANK, W.O., DRAYER, B.P., ROSENBAUM, A.E.: Intracranial biopsy assisted by computerized tomography. J. Neurosurg. <u>46</u>, 740-744 (1977)

Pathogenesis and Pathophysiology of Chronic Subdural Haematoma M. GAAB, U. BAUMGARTNER, and K. W. PFLUGHAUPT

Introduction

The chronic subdural hematoma is defined as a clearly encapsulated accumulation of fluid between the dura and arachnoid membrane $(\underline{11})$, generally occurring after slight brain trauma, often without apparent cause (2, 5, 11, 13, 18), particularly in elderly patients with brain atrophy. The slow development of clinical symptoms is a characteristic feature. Although a subdural *fibrinous clot*, e.g. following venous bleeding, is widely regarded as the *iniating factors* (8, 11, 12), the pathophysiology of the subsequent massive increase in size remains controversial. Table 1 shows proposed hypotheses.

Table 1. Proposed hypotheses for the growth and development of chronic subdural hematoma

Low intracranial pressure / aliquorrhoea (SCHALTENBRAND, 1938) Rebleeding originating from membrane (WEBER et al., 1964) Disturbed capillary permeability of inner dura sheet (WEPLER et al., 1954) Inflammatory effusions (VIRCHOW, 1857; GLOVER and LABADIE, 1976) Osmotic or oncotic water absorption (GARDNER, 1932; WEIR, 1971)

In our study, we compared the changes of various physical, chemical and morphological parameters in the chronic subdural hematoma.

Patients and Approach

Only patients with a typical, unilateral, chronic subdural hematoma with a trauma in the history were studied. The mean age was 60 years (range 47-75).

Morphological Investigations

In the preoperative CAT-scan, the maximal hematoma thickness and the midline shift were measured. During the operation, a part of the inner and outer hematoma membrane was fixed in glutaraldehyde/OsO₄ and investigated by light and electron microscopy.

Registration of Intracranial Pressure (ICP)

The lumbar CSF-pressure was measured 3 h before as well as throughout the operation (catheter, Statham Sp. 37). During the operation (13 patients in normoventilated neurolept analgesia; in 2 patients burr holes in local anesthesia), the epi- and subdural pressures above the hematoma were measured for 30 min by miniature force transducers $(\underline{3}, \underline{4})$, inserted into the burr hole before opening the hematoma. In 4 patients, the epi- and subdural pressures were also recorded over the opposite hemisphere, and in 7 patients the subdural pressure was registered for 3 days after removal of the hematoma and the surrounding membrane.

Physical and Chemical Examination

Hematoma fluid, lumbar CSF and blood were removed simultaneously during the operation. Osmotic (Knauer Osmometer) and oncotic (Wescor colloid osmometer) pressures were measured, and the albumin content quantitatively (Biuret) and electrophoretically determined. An autoanalyser was used to analyse Na⁺, K⁺, Ca⁺⁺, C1⁻, glucose, creatinine, urea-N, uric acid, triglycerides, cholesterol, bilirubin, anorganic phosphate, Fe, Cu, Mg, GOT, GPT, LDH and alkaline phosphatase.

Membrane Permeability

To study the membrane permeability, a 1 $\rm cm^2$ of inner hematoma membrane was used to separate 2 equal columns of fluid, one containing CSF and the other hematoma fluid. The temperature was maintained at 37° C and humidity at 100% (Fig. 2). The resulting levels were then recorded.

Results

Hematoma Size and Intracranial Pressure (ICP) (Fig. 1, 2)

Despite the *considerable size* of the hematomas and the clear midline shift, only *moderate* ICP pressure values were observed and epi-, subdural and lumbar pressures were equal. No difference was found between patients in local anesthesia and those in neurolept analgesia. Only the youngest patient (47 years) with the diagnosis of multiple sclerosis, showed marked elevations. In all cases no significant pressure gradients were observed between the two hemispheres.

After the removal of the hematoma however, the pressure remained lowered by some 10-15 mm Hg (1,5-2 kPa).

Physical and Chemical Values (Table 2)

Whereas the osmotic pressures of hematoma, CSF and plasma were more or less equal, the colloidal osmotic pressure in the hematoma was considerably higher than that of the CSF. The oncotic gradient from the hematoma to the CSF resulted from the difference in albumin content, which was also somewhat elevated in CSF, as compared to normal values. In the hematoma and peripheral blood, the albumin concentration varied; the globulins were found to be lower in the hematoma. Following hemolysis, the LDH was clearly increased, as were the bilirubin and Fe. GOT and GPT however, were not present in the hematoma and the alkaline phosphatase concentration was distinctly lowered. Triglyceride, glucose

in patié fluids a	in patients with chro fluids are underlined	inronic subduned	ural hematon	lata. Values	with marked	difference	in patients with chronic subdural hematomata. Values with marked differences between the individual fluids are underlined	individual
	Osm. pr. (mmol/kg)	Colloid osm. Tot. prot. pr. (mm Hg) (g/d1)	Tot. prot. (g/dl)	Albumine (g/dl)	Glucose (mmol/1)	Triglycer. (mmol/1)	Bilirubine (µmol/1)	Fe (µmol/1)
Blood Hematoma CSF	296 <u>+</u> 7,5 294 <u>+</u> 7,4 297 <u>+</u> 8,0	$20,9 \pm 3$ $21,3 \pm 5$ $0,5 \pm 0,5$	$\begin{array}{rrrr} 6,1 & \pm 0,5 \\ 5,7 & \pm 1,1 \\ 0,09 & \pm 0,08 \end{array}$	3,9 ± 0,3 4,3 ± 0,9 0,07 ± 0,06	$\frac{6,63 \pm 3,55}{3,73 \pm 1,32}$ $\frac{4,08 \pm 1,11}{2}$	1,08 <u>+</u> 0,2 0,50 <u>+</u> 0,2	$11,66 \pm 5,8 12,4 \pm 6$ $238,0 \pm 55 47,6 \pm 11$ $1,3 \pm 1$	$\frac{12,4 + 6}{47,6 + 11}$ $1,3 + 1$
	Na ⁺ (mmol/l)	К ⁺ (mmol/1)	ca ⁺ (mmol/1)	GOT (E/1)	GPT (E/1)	Alk. Phos. (E/1)	LDH (E/1)	Creat. (μmol/l)
Blood Hematoma CSF	$141 \pm 3,5$ $140 \pm 2,7$ $145 \pm 4,5$	4,2 <u>+</u> 0,7 4,3 <u>+</u> 0,5 3,0 <u>+</u> 0,3	$\frac{2,4 \pm 0,23}{1,9 \pm 0,45}$ $\frac{1,2 \pm 0,14}{1,2 \pm 0,14}$	9,0 <u>+</u> 3,5 0,0 <u>+</u> 0 8,0 <u>+</u> 2,7	$\frac{13,0 \pm 7,8}{2,3 \pm 4,0}$ 2,5 ± 0,7	$ \begin{array}{r} 185 \pm 73 \\ 69 \pm 20 \\ 2,8 \pm 0,8 \end{array} $	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	71 <u>+</u> 7 84 <u>+</u> 14 80 <u>+</u> 7

Table 2. Physical and chemical parameters of simultaneously determined hematoma content, CSF and blood in patients with chronic subdural hematomata. Values with marked differences between the individual

and Ca⁺⁺ were also *lower* than serum levels, and were comparable to CSF concentrations.

Morphological Membrane Characteristics (Fig. 3)

Examination of the inner hematoma membrane revealed a *collagenous neomembrane* in which fibrin fibres with capillaries and a clear macrophage activity with erythro-phagozytes were evident. Inflammatory infiltrative changes were not found in either the inner or outer hematoma membrane. No epithelial or fibroid layer existed.

Membrane Permeability (Fig. 2d)

By using a thin section of inner membrane, clear signs of *semiperme-ability* were demonstrated. Due to the elevated colloidal osmotic pressure in the hematoma, a difference of the height of the fluid columns of more than 14 cm $H_2O/10$ mm Hg/1,5 kPa was established between the hematoma and CSF fluids.

Discussion -

Despite large space-occupation, the relatively low ICP values observed in the chronic subdural hematoma were in good agreement with previous ventricular pressure values (4, 6). The considerable size of the hematoma and midline shift without substantial increase in the ICP values is only possible by *slow*, *continual volume increase*, which corresponds to the observed chronic progressive clinical development. This would appear to exclude repetitive acute bleeding (3, 16) as a cause of hematoma expansion. The lower concentrations of enzymes, glucose, triglyceride and Ca⁺⁺ in the hematoma would seem to rule out a hematoma growth through increased capillary permeability (18) of the inner dura sheet. These values cannot be reconciled with an "inflammatory effusion" (7) as there is no indication of actual inflammatory infiltration in the membrane as seen by electron microscopy, or decreased globulin in the hematoma.

A low molecular osmotic expansion can also be excluded $(\underline{11}, \underline{17})$ as there is no osmotic gradient between the hematoma and the CSF, and the absence of epithelial and basal membrane would suggest that the free diffusion of small molecules is possible.

In contrast, the results are compatible with the original suggestion by GARDNER (5), of "oncotic growth". There exists a constant colloidal osmotic gradient, resulting from differences in protein concentration, between the hematoma and CSF, and thus to the cerebral extracellular space. Our permeability studies show that water absorption from the CSF, through the membrane, is possible, thus indicating the membrane's semipermeability. This further supported by the apparent collagenous membrane ultrastructure, which would be expected to convey semipermeability. The lower concentrations of Ca⁺⁺, glucose and triglyceride, as compared with serum values, may be explained by dilution, resulting from water absorption. Still further support for "oncotic growth" is the relatively small ICP increase, which actually corresponds to the colloidal osmotic pressure difference. When the patient is in an upright position, there is a fall in CSF pressure and consequently, the water flow may be reversed hydrostatically. The absence of this reversal in immobilized patients would explain their faster deterioration (Fig. 4). In Fig. 4 is shown schematically our model for pathogenesis of chronic subdural hematoma. *Initially* a *fibrinous coagulum* is established be-tween the dura and arachnoidea.

Its growth in brain atrophy is promoted by the presence of a large subarachnoidal space. After bleeding, fibrinolysis sets in and fibrous tissue from the inner dura layer (10, 15) attempts to encapsulate the hematoma (Fig. 4b: 10). Both neomembranes (i.e. inner and outer hematoma membrane) are then separated by an albumin-rich hemolysate, the volume of which slowly increases through colloidal osmotic absorption of water from the subarachnoid space (Fig. 4c) and gives rise to the clinical symptoms. In addition, the obstruction of CSF absorption pathways by extended membranes over the brain convexity must be considered. This is also an argument against aliquorrhea (1, 14). This would also indicate that a mere bore hole trepanation (6) is not the method of choice but instead an operative removal of the outer membrane is preferable.

Conclusion

Despite considerable space occupation, only a moderate elevation of ICP was observed in cases of chronic subdural hematoma. This elevation and subsequent postoperative fall in pressure results from the colloidal osmotic pressure difference between the hematoma and CSF (cerebral extracellular space). No osmotic pressure differences exist. Substrate and enzyme concentrations in the hematoma are lower than in blood.

The ultrastructure of the hematoma as observed by electron microscopy and permeability studies, appears to be of a semipermeable nature. In conclusion therefore, it is considered that the progression of an initial clot into a chronic subdural hematoma, depends upon the "oncotic growth" through water absorption from the CSF.

References

- 1. BELL, W.E., JOYNT, R.J., SAHS, A.L.: Low spinal fluid pressure syndromes. Neurology 10, 512-521 (1960)
- DRESSLER, W., ALBRECHT, K.: Klinische Betrachtungen zur Pathogenese des subduralen Hämatoms. Acta Neurochir. 5, 46-81 (1957)
- GAAB, M., KNOBLICH, O.E., DIETRICH, K., GRUSS, P.: Miniaturized methods for monitoring intracranial pressure in craniocerebral trauma before and after operation. In: Advances in neurosurgery, Vol. 5. FROWEIN, R.A., WILCKE, O., KARIMI-NEJAD, A., BROCK, M., KLINGER, M. (eds.), pp. 5-11. Berlin, Heidelberg, New York: Springer 1978
- 4. GAAB, M., KNOBLICH, O.E., DIETRICH, K.: Miniaturisierte Methoden zur Überwachung des intrakraniellen Druckes. Techniken und klinische Ergebnisse. Langenbecks Arch. Chir. (in press)
- 5. GARDNER, W.J.: Traumatic subdural hematoma. Arch.Neurol. Psychiat. <u>27</u>, 847-858 (1932)
- GJERRIS, F.: Intraventricular pressure in patients with chronic subdural hematomas before and after evacuation of the hematoma. In: Intracranial pressure III. BEKS, J.W.F., BOSCH, D.A., BROCK, M. (eds.), pp. 85-87. Berlin, Heidelberg, New York: Springer 1976

- GLOVER, D., LABADIE, E.L.: Physiopathogenesis of subdural hematomas. Part 2: Inhibition to growth of experimental hematomas with dexamethasone. J. Neurosurg. 45, 393-397 (1976)
- KRAULAND, W.: Über die Quellen des akuten und chronischen subduralen Hämatoms. Stuttgart: Thieme 1961
- 9. KRAYENBÜHL, H., NOTO, G.G.: Das intrakranielle subdurale Hämatom. Bern: H. Huber 1949
- 10. LINK, K., SCHLEUSSING, H.: Die offenen Verletzungen der dura mater cerebralis und spinalis sowie der Blutleiter. In: Handbuch der speziellen pathologischen Anatomie und Histologie, Bd. XIII/3. LUBARSCH, O., HENKE, F., RÖSSLE, R., SCHOLZ, W. (eds.), pp. 1-20. Berlin: Springer 1955
- 11. LOEW, F., KIVELITZ, R.: Chronic subdural haematomas. In: Handbook of Clinical Neurology, Vol. 24, Part II. VINKEN, P.J., BRUYN, G.W., (eds.), pp. 297-327. Amsterdam, Oxford: North Holland publ. Comp. 1976
- PETERS, G.: Die Pachymeningiosis haemorrhagica interna, das intradurale Hämatom und das chronische subdurale Hämatom. Fortschr. Neurol. Psychiat. <u>19</u>, 485-542 (1951)
- REISNER, H.: Das chronische subdurale Hämatom-Pachymeningiosis haemorrhagica interna. Nervenarzt <u>50</u>, 74-78 (1979)
- SCHALTENBRAND, G.: Neuere Anschauungen zur Pathophysiologie der Liquorzirkulation. Zbl. Neurochir. 3, 290-300 (1938)
- 15. WATANABE, S., SHIMADA, H., ISHII, S.: Production of clinical form of chronic subdural hematoma in experimental animals. J. Neurosurg. <u>37</u>, 552-561 (1972)
- 16. WEBER, G., ROSEMUND, H., DUCKERT, F.: Der Inhalt chronischer Subduralhämatome von Erwachsenen und subduraler Hygrome und Ergüsse von Kindern. Schweiz. Arch. Neurol. Psychiat. 94, 348-373 (1964)
- 17. WEIR, B.: The osmolality of Subdural Hematoma Fluid. J. Neurosurg. <u>34</u>, 528-533 (1971)
- WEPLER, W.: Zur Pathogenese und Begutachtung des chronischen Hämatoms der Dura mater. Zbl. allg. Pathol. <u>91</u>, 406-412 (1954)

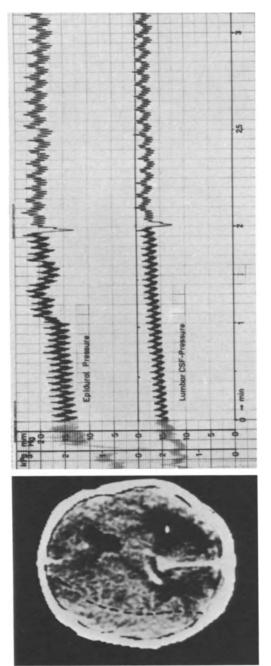


Fig. 1. Morphological size and ICP in a typical unilateral chronic subdural hematoma. Despite considerable space occupation in CAT scan (left), only moderately elevated ICP with normal pulse and breath amplitudes (right)

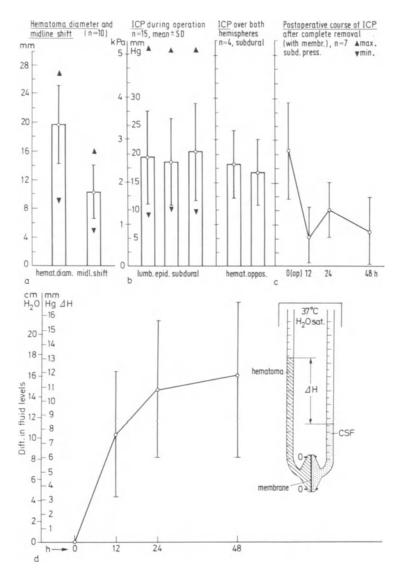
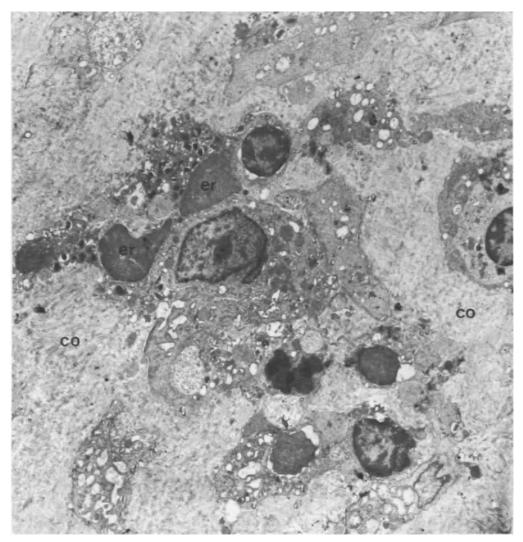
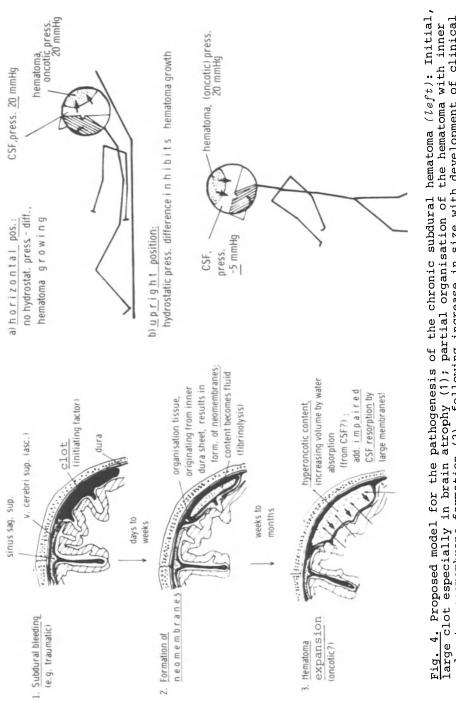


Fig. 2 a-d. Maximum hematoma thickness and midline shift (a), ICP values during operation (b), postoperative course of ICP (c) and examination of the permeability of the inner hematoma membrane (d). d indicates semipermeability of membrane by dividing 2 equal columns of CSF and hematoma fluid



<u>Fig. 3.</u> Electron micrograph of a thicker part of an inner subdural hematoma membrane. Collagenous network (co), fibrin, and macrophages (with erythro-phagocytosis, er). Magn. ca. 4600 x



and outer neomembrane formation (2), following increase in size with development of clinical symptoms by "oncotic fluid absorption" from the CSF (3), simultaneous obstruction of CSF reabsorption pathways. In upright position, the hematoma growth is inhibited by the hydrostatic pressure difference between hematoma and $\mathrm{CSF}~(right)$

Restitution of Skeletal Muscle After Experimental Denervation and Microsurgical Secondary Suture of the Peroneal Nerve in the Rabbit¹

H.-P. RICHTER, U. P. KETELSEN, and D. FRÖSCH

Introduction

From clinical practice it is well known that adequate motor recovery can be achieved by a secondary nerve suture even after prolonged denervation $(\underline{7}, \underline{8})$. In man, however, it is impossible to study this muscular restitution systematically by clinical, physiological and morphological methods. This can be done only in an animal experiment.

Material and Methods

In rabbits, the peroneal nerve was sectioned bilaterally. After various periods of denervation - from 1-12 months - the animals were reoperated, the neuroma was removed and the nerve re-anastomosed by microsurgical technique. In a control group, primary suture was performed. Six months later, clinical examination and electromyography were performed. Nerve and muscle tissue removed for histological, histochemical, and ultrastructural examination.

The tissue specimens were obtained from 47 animals and 75 nervemuscle preparations.

Results

Spreading of the toes $(\underline{4})$ depends on adequate function of the peroneal nerve and its muscles and was rarely seen beyond a denervation period of 10 months prior to the secondary suture.

When stimulating the peroneal nerve proximal to the suture site by electrical square impulses (0, 2 msec) an evoked potential can be recorded from the muscles innervated by this nerve. The latency of the fastest conducting nerve fibers (n=68) and the duration (n=58) of the potentials did not differ significantly from prolonged denervation intervals, although there was a tendency towards longer latencies and durations. A significant decrease (< 0,01) of the amplitude (n=66) and an increasing polyphasic shape of the electrically evoked potential were the most remarkable findings at electromyography (Fig. 1).

Although we were unable to show a clear time limit, it seems that beyond a denervation period of 8-10 months prior to the nerve suture, the amplitude becomes smaller than after shorter periods of denervation.

1 Supported by the Deutsche Forschungsgemeinschaft (Ri 338, Ke 231/6).

The histological examination of the nerves showed adequate neurotization distal to the suture site even if it was preceded by a denervation of 10-12 months. The diameters of the myelinated nerve fibers proximal and distal to the suture site were analysed by morphometry in several animals (7 nerves proximal, 19 nerves distal to the suture site). Proximal to the suture, there is a high proportion of large myelinated nerve fibers, whereas distal to the suture almost no fibers exceeding 10,3 μ in diameter were found even after primary suture.

The classification of the histological and histochemical muscular findings (n=66, peroneal muscle) (Table 1) was based mainly on the amount of atrophy. In the late stages of denervation, it was also dependant on the "secondary myopathic changes", as well as on the fiber type grouping, especially in the LDH (lactate dehydrogenase) reaction. The normal peroneal muscle shows a mosaic pattern of its fiber types with more type 2- than type 1-fibers (Fig. 2 a, b). In normal peroneal muscle, we never observed fiber type grouping. Fiber type grouping refers to a phenomenon typical for a previously denervated and subsequently reinnervated muscle (2, 3). Denervation and reinnervation of the muscle leads to two phenomena:

- a) fiber type grouping and
- b) a relative increase of type 1-fibers (type 2-fiber transformation by a collateral reinnervation of a *type 1-motoneuron?*).

Table 1. Classification of histological and histochemical muscular changes

- 1. Massive atrophy with secondary myopathic changs Muscle replaced by connective and fat tissue
- Marked atrophy Increase in connective and fat tissue Fiber type grouping incomplete (Bad restitution)
- Atrophy visible, partially in groups Variation of muscle fiber diameter Central location of nuclei frequent Fiber type grouping (Moderate restitution)
- 4. Almost no atrophy No fibrosis Central location of nuclei rare Fiber type grouping (Good restitution)

If reinnervation takes place 2 or 3 months after denervation, small groups of atrophic fibers of type 2 can be found besides groups of type 1- and type 2-fibers with a normal diameter (Fig. 2, see Discussion). In analogy to the decrease and disappearance of large myelinated nerve fibers distal to the suture site, we observed this muscle fiber type grouping after primary suture of the peroneal nerve already.

If the denervation period prior to the nerve suture exceeds seven months, muscular atrophy becomes moderate in most cases (Fig. 2).

If the period of denervation exceeds 10 months, then muscular atrophy is massive.

The motor endplates were localized by a cholinesterase staining method (LEWIS) and examined by electron microscopy. The normal endplate and the endplate of a well-regenerated muscle fiber are characterized by deep secondary synaptic clefts (Fig. 3) and a strong reaction during the staining process, whereas insufficiently reinnervated motor endplates (Fig. 4) reveal small secondary clefts and/or a weak reaction. The findings concerning the endplates have not been examined statistically.

Discussion

From our investigation it may be concluded that a denervation period of 8-10 months prior to the secondary nerve suture seems to be a critical interval in the rabbit. Denervation lasting longer than this interval is followed by only a highly insufficient muscular restitution, in spite of a fairly good degree of neurotization of the nerve distal to the suture site and in spite of the not significantly increased latencies of the electrically evoked muscle potential.

GUTMANN (5, 6) has already pointed out, that the quality of reinnervation after prolonged denervation and following nerve suture depends largely on the motor endplate and the muscle and not mainly on the nerve, as was stressed by SUNDERLAND (7). But even if a perfect functional and myographic recovery has been achieved, a primary nerve suture will never be able to restore the original pattern of innervation to this muscle. This is proved by the described type grouping of muscle fibers described above. A phenomenon observed already after crush injury of a motor nerve (1, 9) and a sign of muscle fiber transformation. Besides the type grouping the increase in type 1-fibers after denervation and subsequent reinnervation might be explained by a quicker regeneration rate for type 1- than type 2-motoneurons, and by reinnervation of initially type 2-muscle fibers by axons belonging to type 1-motoneurons.

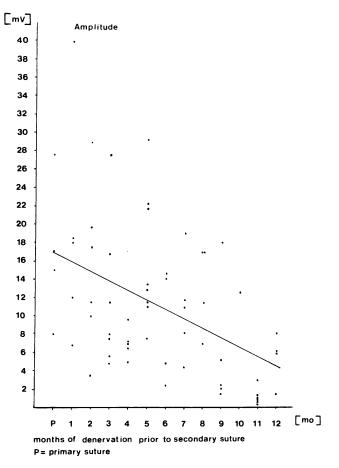
Conclusion

As far as the above mentioned critical interval of 8-10 months is concerned, our results cannot be transferred to man. But it seems at least possible that the underlying mechanisms are similar in other mammals and in man. From these experiments, it may be concluded that the critical organ for the restitution of the motor unit after prolonged denervation and following nerve suture is not the nerve, but the muscle.

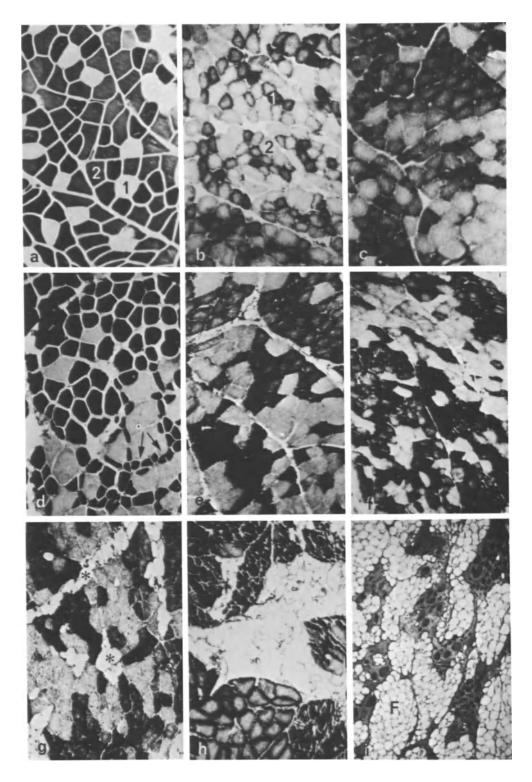
References

- BEERMAN, D.H., CASSENS, R.G., COUGH, C.C., NAGLE, F.J.: The effects of experimental denervation and reinnervation on skeletal muscle fiber type and intramuscular innervation. J. Neurol. Sci. <u>31</u>, 207-221 (1977)
- DORMAN, J.D.: The histopathology of neurogenic muscular atrophy. In: The striated muscle. PEARSON, C.M., MOSTOFI, F.K. (eds.), pp. 249-262. Baltimore: Williams and Wilkins 1973

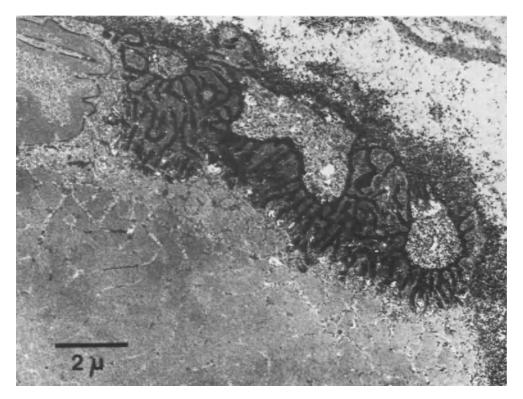
- DUBOWITZ, V., BROOKE, M.H.: Muscle biopsy: a modern approach. London, Philadelphia, Toronto: Saunders 1973
- 4. GUTMANN, E., GUTTMANN, L., MEDAWAR, P.B., YOUNG, J.Z.: The rate of regeneration of nerve. J. Exp. Biol. <u>19</u>, 14-44 (1942)
- GUTMANN, E., YOUNG, J.Z.: The reinnervation of muscle after various periods of atrophy. J. Anat. (London) <u>78</u>, 15-43 (1944)
- GUTMANN, E.: Die funktionelle Regeneration der peripheren Nerven. Berlin: Akademie-Verlag 1958
- 7. SUNDERLAND, S., BRADLEY, K.C.: Degeneration atrophy of the distal stump of a severed nerve. J. Comp. Neurol. 93, 401-409 (1950)
- SUNDERLAND, S.: Nerves and nerve injuries. Edinburgh, London: Churchill Livingstone 1972
- 9. WUERKER, R.B.: Changes in nerve and muscle produced by long-term nerve ligation. J. Neuropathol. Exp. Neurol. <u>36</u>, 821-834 (1977)



<u>Fig. 1</u>. Amplitudes and their regression line (p < 0,01) of the electrically evoked muscle potentials. Stimulation of the peroneal nerve proximal to the suture site 6 months after nerve suture. Recording from the anterior tibial muscle



- \triangleleft Fig 2 a-i. Transverse sections of the peroneal muscle under normal and pathological conditions.
 - <u>a</u> Normal peroneal muscle (ATPase preincubated at pH 10,5). Mosaic pattern of fiber types. Type 1-fibers with weak enzyme reaction $(\underline{1})$, type 2-fibers with strong enzyme reaction (2). X 106
 - b Normal peroneal muscle (lactate dehydrogenase reaction: LDH). Type 1-fibers (1, strong enzyme reaction), type 2-fibers (2, weak enzyme reaction). X 53
 - <u>c</u> Section of the peroneal muscle six months after *primary suture* of the peroneal nerve. Fiber type grouping. X 106
 - d Peroneal muscle denervated for 2 months, six months after nerve suture. ATPase preincubated at pH 10,5. Small groups of type 2fibers (→, dark) besides type 1- and type 2-fibers with normal diameter. X 106
 - <u>e</u> Peroneal muscle denervated for two months, six months after nerve suture. LDH. Fiber type grouping with relative increase of type 1fibers (dark). X 106
 - \underline{f} Peroneal muscle denervated for five months, six months after nerve suture. LDH. Fiber type grouping is very striking. X 53
 - g Peroneal muscle denervated for seven months, six months after nerve suture. LDH. Fiber type grouping and increase of connective and fat tissue (asterisk). X 106
 - <u>h</u> Peroneal muscle denervated for eleven months, six months after nerve suture. LDH. Large group atrophy. The majority of the atrophic fibers belongs to type 1 (dark). Increase of connective and fat tissue. X 53
 - <u>i</u> Peroneal muscle denervated for nine months without reinnervation. Trichrome. Massive atrophy of muscle parenchyma with secondary myopathic changes. The atrophic and degenerated muscle cells are replaced by fat and connective tissue (F). X 53



<u>Fig. 3</u>. Motor endplate (cholinesterase stain) from the anterior tibial muscle with excellent motor recovery. 6 months after secondary suture of the peroneal nerve; pre-suture interval 1 month. Deep secondary synaptic clefts, strong reaction (K 0343 L)

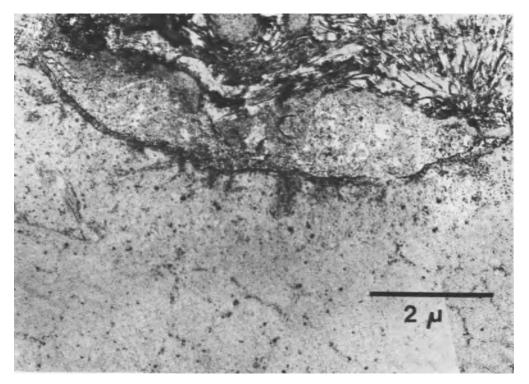


Fig. 4. Motor endplate (cholinesterase stain) from the anterior tibial muscle with bad muscular restitution. 6 months after secondary suture of the peroneal nerve; pre-suture interval 12 months. Irregular and small secondary synaptic clefts, weak reaction (K313 R)

Results After Anterior Transposition of the Ulnar Nerve for Tardy Ulnar Palsy

D. STOLKE, B.-U. SEIDEL, and H. MÜLLER

The most common factors predisposing to the compression of the ulnar nerve at the elbow are fractures, arthrosis, abnormal strain of the elbow joint and recurrent dislocation or subluxation of the ulnar nerve from its groove of the humeral epicondyle (5, 7, 8). These disorders may result in a dysfunction termed "tardy ulnar palsy". Fifty-one patients were treated for tardy ulnar palsy by anterior transposition at the Medical School of Hannover from 1971 to 1978. Thirty-one patients were followed from 4 months to 7 years. Particular attention was devoted to the age, sex, duration of symptoms, preoperative deficits, surgical findings, etiology, and EMG-findings. We classified our patients into a group of complete recovery, good improvement (= relief of pain, distinct improvement of motor and sensory function) and another group of no changes except relief of pain. There were no operative complications, no deteriorations.

Results

Table 1 shows the results in relation to the preoperative neurological deficits and the degree of pain. A complete recovery was achieved in 13 patients (= 37%), a good improvement in 11 (= 31%). The distribution of age ranged from 26-71 years of age (Table 1). 80% were between 40 and 70 years of age. There were 21 (= 60%) right ulnar nerves affected and 14 (= 40%) left ones. The duration of symptoms ranged from 3 months to 2 years, once even 7 years. At operation all the nerves were tightly bound by fibrous tissue at the cubital tunnel. They were thickened before the entrance into the flexor carpi ulnaris. The thickening concerned only the epineural tissue and not the fascicles themselves.

In 48% (= 17 patients), only, we found a cause for the late palsy: 11 fractures of the affected elbow, 4 cases of severe arthrosis and constriction of the nerve by scarry tissue after osteom surgery at the elbow in one case (Table 2). All the follow-ups underwent an EMG-study. The postoperative findings correlated very closely to the clinical results (12).

Discussion

A clear improvement indicating a complete recovery was found in 24 out of 35 patients (= 68%). Our results do not differ from those of other authors ($\underline{1}, \underline{2}, \underline{3}, \underline{4}, \underline{5}, \underline{11}, \underline{13}$). Males were affected more than females (33 out of 33) in contrast to the findings of NIGST (10) who did not see any difference between the sexes. Less than one half of the cases reveal a definite cause such as fractures, trauma and arthrosis. Sleeping habits, elbow leaning ($\underline{5}, \underline{6}, \underline{7}, \underline{8}, \underline{9}$) may lead to a kind of

		Postopera	tive su	ccess
	Preoperative	Recovery Complete	Good	No change
Grade of palsy	4 - 5	7	-	_
purby	2 - 3	6	6	3
	0 - 1	-	5	8
Sensory deficits	Light	8	2	-
	Distinct Severe	5 -	6 3	8 3
Pain	Light	8	10	11
	Distinct	5	1	-
	Severe	-	-	-
	n =	13	11	11

<u>Table 1</u>. Preoperative neurological deficits and pain in correlation to postoperative success

microtraumatisation that may be the cause for later ulnar palsy. On the other hand we found a large number of alcoholics and diabetics in our cases. The question is whether these agents (such as alcohol) or these diseases (such as diabetes) may lead to an overly vulnerable ulnar nerve. Subluxation may be regarded as a predisposing factor, in spite of patients who are seen for years without symptoms (5, 6, 7, 8, 9). Looking for signs which could predict a favourable recovery or a good improvement, we found that only light or less severe motor deficits (grade 4-5 and 2-3; grade 5 indicates normal muscle strength, grade 0 complete palsy) return to normal muscle strength after transposition. Sensory deficits are to be regarded likewise: only light or medium grade sensory deficits improve completely after surgery. Pain does not predict the grade of improvement.

Table	2.	Factors	predisp	posing	to	compression	of	the	ulnar	
nerve	and	l postope	erative	result	s					

Causes	Recovery			
	Complete	Good	No change	Total
Fractures	2	5	4	11
Arthrosis	-	3	3	6
Unknown	11	3	4	18
n =	13	11	11	35
	68%			

The duration of symptoms plays only a small part, the longest histories are to be found in the group with complete recovery. It is not the duration but the severity of the symptoms that predicts the outcome.

There are different surgical methods, namely the subcutaneous, the subfascial-intramuscular and the submuscular anterior transposition. Some authors (<u>14</u>) perform a lysis and a decompression of the nerve out of the surrounding tissue in the cubital tunnel and succeed. Others (<u>9</u>) report on successful medial epincondylectomy. The rate of success does not differ (<u>3</u>, <u>5</u>), even if some authors (<u>1</u>, <u>4</u>, <u>7</u>) claim to have a better rate of improvement (ca. 80%) after submuscular anterior transposition. When the elbow joint is bent, the nerve is expanded up to 2-3 cm (<u>1</u>) in the cubital tunnel and all the daily movements of the joint including industrial working result in recurrent microtraumatisation. In view of this fact, the criteria of the surgical treatment must be to get out the nerve of this area in order to avoid recurrent expanding or even overexpanding of the ulnar nerve. In general, this can be achieved only by anterior transposition.

We prefer the subfascial intramuscular transposition, taking particular care to provide a smooth new pathway for the ulnar nerve after transposition. To achieve this we perform a good resection of the ligamentum intermusculare on the upper arm and a generous incision of the flexor carpi ulnaris, especially in big-muscled individuals. A pathway performed like this will avoid further traumatisation and overexpansion of the nerve.

Conclusions

The less motor and sensory defects are present before the operation, the better is the prognosis.

Severe deficits do not return to normal. The grade of pain does not predict the grade of improvement. The duration of symptoms is of subordinate importance. By using the subfascial intramuscular anterior transposition with particular care to provide a smooth new pathway of the ulnar nerve after surgery, we achieve complete or good recovery in over two thirds of our patients.

References

- GERL, A., THORWIRTH, V.: Ergebnisse der Ulnarisverlagerung. Acta Neurochirurgica 30, 227-246 (1974)
- HARRISON, M.J.G., NURICK, S.: Results after anterior transposition of the ulnar nerve for ulnar neuritis. British Med. J. <u>1</u>, 27-29 (1970)
- HÄRTEL, P., RUPRECHT, E.-O.: Die chirurgische Behandlung des Sulcus-ulnaris-Syndroms. Münch. Med. Wschr. 47, 1580-1582 (1971)
- McGOWAN, A.J.: The results of transposition of the ulnar nerve neuritis. J. Bone Joint Surg. <u>32 B</u>, 293-301 (1950)
- 5. GOLDWARE, S., MAXWELL, J.A.: Tardy ulnar palsy. J. Kans. med. Soc. <u>73</u>, 51-53 (1972)
- 6. MUMMENTHALER, M.: Die Ulnarisparesen. Stuttgart: Thieme 1961
- MUMMENTHALER, M.: Die Ulnarislähmungen bei Bettlägerigen. Ihre klinische Bedeutung anhand von 35 eigenen Beobachtungen. Schweiz. med. Wschr. 88, 591 (1958)

- MUMMENTHALER, M., SCHLIACK, H.: Läsion peripherer Nerven. 3. Aufl. S. 238-252. Stuttgart: Thieme 1977
- 9. NEBLETT, Ch., EHNI, G.: Medial epicondylectomy for ulnar palsy. J. Neurosurg. 32, 55-62 (1970)
- NIGST, H.: Die traumatische Neuritis des Nervus ulnaris. Helvetia Chirurgica Acta <u>1</u>, 37-51 (1953)
- 11. PAINE, K.W.E.: Tardy ulnar palsy. The Canadian Journal of Surgery <u>13</u>, 255-261 (1970)
- 12. PAYAN, J.: Anterior transposition of the ulnar nerve: an electrophysiological study. J. Neurol. Neurosurg. Psychiat. <u>33</u>, 157-165 (1970)
- 13. USBECK, W.: Spätschäden des Nervus ulnaris bei Prozeß im Bereich des Ellenbogengelenkes. Handchirurgie 2, S. 109 (1970)
- 14. WILSON, D.H., KROUT, R.: Surgery of ulnar neuropathy at the elbow: 16 cases treated by decompression without transposition. J. Neurosurg. <u>38</u>, 780-785 (1973)

Hemodynamic Changes During Controlled Hypotension with Sodiumnitrousprusside in Thiopental-Anesthesia

K. HUSE

Introduction

Cerebral circulation can be severely impaired during vascular surgery of cerebral vessels. The extent of the postischemic brain swelling and cerebral infarction after temporary occlusion of cerebral vessels is also determined by the anesthetic managements during the surgical procedures. This was impressively demonstrated by the studies of SMITH et al. (35). In animal studies SMITH (35) observed that 28%-34% of the cerebral hemisphere was infarcted after temporary ligation of the middle cerebral artery and internal carotid artery in deep halothane anesthesia. Barbiturate therapy reduced the incidence of hemiplegia. This result in animal studies and the clinical observations of patients with head injuries stressed the special indication of barbiturate anesthesia as the method of choice in neuroanesthesia (14). The reduction of the vulnerability of the brain and the prolongation of the revival time of the brain tissue is the special indication for the use of Thiopental and Pentobarbital (1, 3). The mechanism of this protective effect of the barbiturates is not quite clear. After barbiturate loading the reduction of the oxygen (7, 13, 37) utilisation is in the range between 36%-56%, but again in deep halothane anesthesia the reduction in the brain metabolism is in the same range. The protective effects of the barbiturates obviously depends on different factors (1, 3, 24, 35). The increase of cerebral vascular resistance ameliorates the recirculation of the ischemic brain tissue and diminishes the steal phenomenon and the luxury perfusion syndrome which is typical for the central vascular effects of the Halothan (1, 3). In addition to this BLEYAERT (3) and NEMOTO (34) stressed the stabilizing effects of certain barbiturates on the blood/ brain barrier, reducing the brain swelling and the increased intracranial pressure (22). It is the special aim of this presentation to demonstrate that the anesthetic technique of thiopental-nitrous-oxideoxygen anesthesia and controlled hypotension with sodium nitrous prusside in thiopental anesthesia is a valuable method for neurosurgical patients with cerebral aneurysm.

Methods

10 neurosurgical patients (3 q, 7 d) were studied. All patients had cerebral aneurysms. Cardiac output was determined in duplicate within 1 min of each by the thermodilution method. The brachial or radial artery was cannulated for continuous monitoring of arterial blood pressure and for sampling of arterial blood. All subjects received 2 mg/kg pentobarbital (Nembutal) (R) and 0,5 mg atropine, 1 h before anesthesia. Anesthesia was induced with 7 ± 1,2 mg/kg thiopental. After injection of 100 mg succinylcholine the patients were intubated with a Woodbrige-tube and ventilation was controlled with a Dräger

as compared to	controlled h	Npotension	with sodium	are interested to controlled hypotension with sodium nitrous prusside			
Explanations	Heart rate	Systolic pressure	Diastolic pressure	Mean pressure	Cardiac index	Stroke index	Total peripheral resistance
	Heart rate min ⁻¹	gH mm	gH mm	pH mm	1.min ⁻¹ .m-2	ml•Heart rate ⁻ 1.m ⁻ 2	dyn, sec. cm ⁻⁵
Preoperative	94 ± 15	134 ± 19	75 ± 11	89 ± 15	3,19 ± 0,58	34,7 ± 8,3	1178 ± 224
Intraoperative 102 <u>+</u> 10	102 ± 16	112 ± 13	71 ± 9	85 ± 9	2,43 ± 0,62	24,3 ± 7	1588 <u>+</u> % (&
	P > 0,05	P < 0,01	P > 0,05	P > 0,05	P < 0,1	P < 0,001	P < 0,05
Controlled hypotension	117 ± 17	65 <u>+</u> 14	45 + 9	53 + 9	2,13 ± 0,56	18,68 <u>+</u> 6,04 1030 <u>+</u> 473	1030 ± 473
	P < 0,01	P < 0,001	P < 0,001 P < 0,001	P < 0,001	P < 0,001	P < 0,001	P > 0,05

 Table 1. Mean value and standard deviations of circulatory studies during Thiopental -N20-02-anesthesia

 as compared to controlled hypotension with codium nitroir action

Respirator. Anesthesia was maintained by intermittent doses of thiopental, the average maintenance dose being 7,14 \pm 4,2 mg/kg/h (Table 2).

<u>Table 2</u>. Average dosis of thiopental (Trapanal R) in 10 neurosurgical patients

	mg/m ²	mg/kg	mg/kg/h	mg/m ² /h
Initial dose	265 <u>+</u> 40	7 <u>+</u> 1,16		
Maintenance dose			7,14 <u>+</u> 4,2	227,5 <u>+</u> 154
Total dose	1512 <u>+</u> 550	39,75 <u>+</u> 14,5	8,94 + 4,52	337 <u>+</u> 167

Results

In 10 neurosurgical patients with intracerebral aneurysm, circulatory studies were performed during Thiopental anesthesia and controlled hypotension with sodium nitroprusside. The following parameters were measured or calculated: from recorded data: heart rate (HR), arterial pressure (P_{mean} , P_{syst} , P_{diast}) cardiac output (CO), cardiac index (CI), stroke volume (SV), stroke index (SI), total peripheral resistance (TPR).

For induction and maintenance of anesthesia, the patients received $8,9 \pm 4,5 \text{ mg/kg/h} (337 \pm 167 \text{ mg/m}^2/\text{h})$ thiopental (Trapanal R) under EEG control (Table 2). The circulatory studies revealed an increase of the heart rate by 8,5% under Thiopental anesthesia, at the same time the stroke index decreased by 29,9%, and the cardiac index by 23,8%. The stability of the blood pressure was maintained due to the increase of the total peripheral resistance by 34,6%. After the medication of nitrous prusside by continous infusion, the arterial blood pressure was adjusted to an average mean level of 53 ± 9 mm Hg. Under controlled hypotension the pulse rate increased by 14,7% while the stroke index decreased by 23% and the cardiac index by 12,4% (Table 1). As a result of the peripheral resistance decreased by 35%. The results of this study indicate that control of hypotension with Sodium nitroprusside under Thiopental-anesthesia is a valuable procedure for neurosurgical patients with normal circulatory functions.

Discussion

After induction and maintenance of anesthesia with thiopental, the heart rate increased (+ 14,7%). In the literature cardioaccelerations between 12% and 33% were reported (7, 8, 9, 10, 11, 17, 20, 27, 28, 37).

Different causes of this cardiac acceleration were discussed:

- 1. Reflex compensatory mechanism of the circulation (PRICE, 1960) (17)
- 2. Increase of the catecholamin secretion (BURN, 1959) ($\underline{18}$)
- 3. Parasympathicolytic effects of thiopental (GRUBER et al., 1952)(20)
- 4. Direct central nervous effects of thiopental (PRICE, 1960) $(\underline{17})$

The induction of controlled hypotension to 53 + 9 mm Hg was accompanied by an additional increase in the heart rate by 14%. The cardiac acceleratory effect of sodium nitrous prusside is well documented in the literature (16). The decrease in mean blood pressure (- 16,4%) after medication with thiopental was reported by many authors (7, 9, 10, 11, 12, 37). PRICE especially demonstrated the circulatory effects of thiopental during controlled respiration with increased intrathoracic airway pressure, because the depression of the overshoot phenomenon decreases the return of the arterial pressure to normal levels (28). The most important circulatory effect of thiopental is the reduction of the stroke index (- 29,9%) and the cardiac index (- 23,8%). These circulatory changes were also reported in other publications (7, 8, 9, 10, 11, 17, 20, 27, 37). Not the decrease in the myocardiac contractility, but the diminished afterload and the redistribution of the intrathoracic blood volume (venous pooling) is the most important reason for the reduced cardiac output. During the maintenance phase of thiopental anesthesia, the calculated total peripheral resistance increased by 35%. Other studies reported an increase between 9% - 46% ($\underline{8}$, $\underline{9}$, $\underline{11}$, $\underline{37}$). After induction of controlled hypotension with sodium nitrous prusside, the total peripheral resistance decreased by 35%. This is in accordance with the peripheral circulatory effects of sodium nitrous prusside (16). The result of this study indicates that controlled hypotension with sodium nitrous prusside in thiopental anesthesia is a valuable method for neurosurgical patients with normal circulatory functions.

Summary

Using an anesthetic technique of thiopental - nitrous - oxide - oxygen anesthesia for neurosurgical patients with cerebral aneurysms, a comparison was made between the circulatory changes under thiopental - nitrous - oxide - anesthesia and controlled hypotension with sodium nitrous prusside in thiopental anesthesia. The results of this study indicate that *controlled hypotension* with sodium nitrous prusside in thiopental anesthesia is a valuable anesthetic technique for neurosurgical patients with normal circulatory functions.

References

- 1. AMES, A., WRIGHT, R.L., KOWADA, M.: Cerebral ischemia: II. The no-reflow phenomenon. Am. J. Pathol. 52, 437 (1968)
- 2. BENDIXEN, H.H., LAVER, M.B.: Circulatory effects of thiopental sodium in dogs. Anesthesia and Analgesia 41, 6, 674 (1962)
- 3. BLEYAERT, A.L., NEMOTO, E.M., STEZASKI, S.W., ALEXANDER, H., SAFAR, P.: Amelioration of postischemia enecephalopathie by sodium thiopental after 16 minutes of global brain ischemia in monkeys. The Physiologist <u>18/3</u>, 145 (1975)
- 4. BRISTOW, J.D., PRYS-ROBERTS, C., FISHER, A., PICKERING, T.G., SLEIGH, P.: Effects of anesthesia on baroreflex control of heart rate in man. Anesth. 31, 423 (1969)
- BURN, J.H., HOBBS, R.: Mechanism of arterial spasm following infra-arterial injection of thiopentone. Lancet <u>1</u>, 1112 (1959)
- CUSHING, H.: Some experimental and clinical observations concerning states if increased intracranial tension. Am. J. med. Sci. <u>124</u>, 375 (1902)

- DOBKIN, A.B., WYANT, G.M.: The physiological effects if intravenous anesthesia on man. Cand. Anaesth. Soc. J. <u>4</u>, 295 (1957)
- ETSTEN, B., LI, T.H.: Hemodynamics changes during thiopental anesthesia in humans: cardiac output, stroke volume, total peripheral resistance, and intrathoracic blood volume. I. Clin. Invest. 34, 500 (1955)
- 9. ELDER, J.D., NAGANE, S.M., EASTWOOD, D.W., HARNAGEL, D.: Circulatory changes associated with thiopental anesthesia in man. Anaesthesiol. <u>16</u>, 394 (1955)
- EXLEY, K.A.: Depression of autonomic ganglia by barbiturates. Br. J. Pharmac. Chemoth. 9, 170 (1954)
- 11. FIELDMAN, E., RIDLEY, R., WOOD, E.: Hemodynamic studies during thiopental sodium and nitrous oxide anesthesia in humans. Anesth. <u>16</u>, 473 (1955)
- 12. GRUBER, C.M., LEE, K.M.: A study of the effect of thiopbarbiturates on the cardiac-vascular system. Arch. internat. pharmacodyn. <u>91</u>, 461 (1952)
- 13. HIMWICH, W.A., HOMBURGER, E., MARESCA, R., HIMWICH, H.E.: Brain metabolism in man: unanesthetized and in pentothal narcosis. Amer. J. Psych. 103, 689 (1946)
- 14. HUNTER, A.R.: Thiopentone supplemented anaesthesia for neurosurgery. Brit. J. Anaesth. 44, 506 (1972)
- 15. HUSE, K.: Die Veränderungen des Serumlactatspiegels in Neuroleptanästhesie im Vergleich zu Trapanal-Lachgas-Sauerstoffnarkose nach der kontrollierten Hypotension mit Nitroprussidnatrium. (Unveröffentlichte Untersuchung)
- 16. HUSE, K.: Die kontrollierte Hypotension mit Nitroprussidnatrium in der Neuroanästhesie. (Anaesthesiologie und Wiederbelebung, Bd. 107. FREY, R., KERN, F., MAYRHOFER, O. (eds.). Berlin, Heidelberg, New York: Springer 1977
- IBBLER, M.: Hemodynamic changes due to induction of anesthesia using thiopentone. V. European Congress of Anesthesiology. Excerpta Medica, 452, Session 52, Abstract 428
- 18. KETY, S.S., WOODFORD, R.B., HARMEL, N.H., FREYHAN, F.A., APPEL, K.E., SCHMIDT, C.F.: Cerebral blood flow and metabolism in schizophrenia. The effects of barbiturates, semi-narcosis, insulin coma and electrochock. Am. J. Psych. 104, 765 (1947)
- 19. KIERSEY, D.K., BICKFORD, R.G., FAULCONER, A.: Electro-encephalographie patterns produced by thiopental sodium during surgical operations: Description and classification. Brit. J. Anaesth. <u>23</u>, 141 (1951)
- LI, T.H., REYNOLDS, R.N., RHEINLÄNDER, H.F., ETSTEN, B.E.: Cardiocirculatory dynamics during thiopental anesthesia in humans. Fed. Proc. <u>13</u>, 380 (1954)
- LIND, B., SNYDER, J., KAMPSHULTE, S., SAFAR, P.: A review of total brain ischemia models in dogs and original experiments on clamping the aorta. Resuscitation <u>4</u>, 19 (1975)
- 22. MARSHALL, L.F., SMITH, R.W., SHAPIRO, H.M.: The outcome with aggressive treatment in severe head injuries. Part II. Acute and chronic barbiturate administration in the management of head injury. J. Neurosurg. <u>50</u>, 26 (1979)
- MICHENFELDER, J.D., THEYE, R.A.: Cerebral protection by thiopental during hypoxia. Anesth. <u>39</u>, 510 (1973)

- 24. NEMOTO, E.M., SNYDER, J.V., CAROLL, R.G.: Global ischemia in dogs: cerebrovascular CO₂ reactivity and autoregulation. Stroke <u>6</u>, 425 (1975)
- 25. PAGE, J.H., CORCORAN, A.C., DUSTAN, H.D., KOPPANY, T.: Cardiovascular actions of sodium nitroprusside in animals and hypotensive patients. Circulation 11, 188 (1955)
- 26. PIERCE, E.C., LAMBERTSEN, C.J., DEUTSCH, S., CHASE, P.E., LINDE, H.W., DRIPPS, R.D., PRICE, H.L.: Cerebral circulation and metabolism during thiopental anesthesia and hyperventilation in man. J. Clin. Invest. 41, 1164 (1962)
- 27. POLLOCK, P., HARMEL, M.H., CLARK, R.E.: Estimation of cardiac output by the ballistocardiograph during thiopental nitrous oxide oxygen anesthesia. Anesth. <u>16</u>, 970 (1955)
- 28. PRICE, H.L., CONNER, E.H., ELDER, J.D., DRIPPS, R.D.: Effect of sodium thiopental on circulatory response to positive pressure inflation of lung. J. Applied Physiol. <u>4</u>, 629 (1952)
- 29. PRICE, H.L.: General anesthesia and circulatory homeostasis physiological. Reviews 40, 187 (1960)
- 30. PRIME, F.J., GRAY, T.C.: The effect of certain anesthetic and relaxant agents on circulatory dynamics. Brit. J. Anesthesia <u>24</u>, 101 (1952)
- 31. REDGATE, E.S., GELLHORN, E.: The tonic effect of posterior hypothalamus on blood pressure and pulse rate as disclosed by the action of intrahypothalamical injected drugs. Arch. Int. Pharmacodyn. Ther. 1, 193 (1956)
- 32. SAFAR, P., STEZOSKI, W., NEMOTO, E.M.: Ameliovation of brain damage after 12 minutes cardiac arrest in dogs. Arch. Neurol. <u>33</u>, 91 (1976)
- 33. SCHIEVE, J.F., WILSON, W.P.: The influence of age, anesthesia and cerebral arteriosclerosis on cerebral vascular activity to CO₂. Am. J. Med. <u>15</u>, 171 (1953)
- 34. SKOVSTED, P., PRICE, M.L., PRICE, H.L.: The effects of shortacting barbiturates on arterial pressure proganglion sympathetic activity and barostatic reflexes. Anesth. <u>33</u>, 10 (1970)
- 35. SMITH, A.L., HOFF, J.T., NIELSEN, S.L., LARSON, C.P.: Barbiturate protection in acute focal cerebral ischemia. Stroke <u>5</u>, 1 (1974)
- 36. SOGA, D.: Die Beeinflussung der Myokardkontraktilität durch Propanidid, Methohexital und Halothane: Vergleichende Untersuchungen am isolierten Papillarmuskel, Ganztier und Mensch. Habil.-Schrift Medizinische Fakultät der Ludwig-Maximilian-Universität München (1970)
- 37. SONNTAG, H., HOLLBERG, K., SCHENK, H.D., DONATH, U., REGENSBURGER, D., KETTLER, D., DUCHANOVA, H., LARSEN, R.: Effects of thiopental (trapanal) (R) on coronary blood flow and myocardial metabolism in man. Acta Anesth. Scand. <u>19</u>, 69 (1975)
- 38. WECHSLER, R.L., DRIPPS, R.D., KETY, S.S.: Blood flow and oxygen consumption of the human brain during anesthesia produced by thiopental. Anesthesiology <u>12</u>, 308 (1951)

Subject Index

abdominal pressure 278 abscess 196,321 -, acute 325 - in cats 321 -, development 321 -, experimental 321 -, prognosis 321 -, treatment 321 active absorption 174 ADH secretion 340 adult hydrocephalus 174 age of patients 96 - at surgery 212 air myelography 95 alloplastic interponate 288 amipaque 8 AMMON's horn 319 amyotrophic lateral sclerosis 24, 41, 49, 305 aneurysms of the vertebro-basilar junction 275 anterior approach 65, 71, 80, 88, 104, 106, 112, 120 - disc removal 50 50 - discectomy - reposition 144 - spinal artery 88 anticoagulant intracerebral hemorrhage 369, 371 anticoagulation therapy 368 aortography 122, 124 apprenticeship 158, 159 arachnoid space 120 arachnoiditis 54 366 arterio-venous aneurysms artery, anterior inferior cerebellar 275 275 -, basilar arthrotic changes 101 aseptic 195 ataxia 62, 280 atheroma 123 avoidable complications 159 bacterial colonization 228 balloon catheters 366

behavior of hydrocephalus 232, 234 best location for a trepanation 257 biomechanical irritations 95 - pathogenesis 3 birth injury 280 bladder disorders 83 bone chip 101 - dowel 122 bradycardia 278 brain abscess 321, 329 - -, ultrastructure 331 - damage 337, 338 - edema 321, 322, 332 - -, cold induced 336 - -, experimental 332 - -, ischemic 335 - -, models of 332, 334 - herniation 279 - ischemic disorders 347 - malformations 213 BROWN SEQUARD syndrom 54, 62, 78, 79, 90 calf bone 104 capsule formation 321 cardiac catheter obstruction 228 - failure 190, 199 carotid angiography 318 - compression test 363, 364, 365 - -sinus cavernosus fistulas 366 catheter, cardiac 220, 223 - insertion 257, 261 -, peritoneal 223 -, ventricular 220, 223 causal factors 5 causal factors cause of death 229, 235
- of hemorrhage 371
- of postoperative death 190
- of shunt revision 155 - of ventricular enlargement 165

```
cefazedon 321, 322
central regulatory disturban-
       337
  ces
cerebral anoxia 277
- hypoxia 386
-atrophy 209, 210
- mantle 210
- mantle
cerebrograph 356
cerebrospinal fluid 381
- - leakage 303
cerebrovascular diseases
                              356
cervical brachialgia 78
- cordotomy
- disc 115
              309
- - excision
              66
- - hernitation 50, 59
- - protrusion 26, 86
               302
- discopathy
- intervertebral disc 53
- laminectomy
                302
- myelopathy 8, 22, 24, 26, 33,
38, 47, 50, 55, 59, 61, 64, 69,
86, 88, 96, 97, 100, 112, 115,
  120, 291

-, spondylogenic 3, 78, 95
radiculopathies 26, 44

- root compression
                       49
- spinal stenosis
                     95
                26, 33, 54, 64,
- spondylosis
  104, 112, 122
          78
- trauma
changes of water content
                             326
children, shunted hydrocephalic
  210
choroidal-ependymal reactions
  196
chronic cervical myelopathy
                                 38,
  41, 44, 116
- spondylotic myelopathy 4
clamping of carotid artery
                                332
clivus 275
         284
CLOWARD
- fusion 124
- technique 87, 100, 104, 130,
  291
CLOWARDS procedure 80
- route 78
cognitive defects 374
- deficits 375, 377, 378
- and sleep functions
                         377
collateral circulation 363
complete migration 193
complication rate
                     220
- -, total 226
complications, flushing device
  224
-, shunt operations in children
  222
-, - procedures 267
- of ventriculo-cardiac shunt
  material 252
compression of spinal cord 95
```

computed tomography 13, 138, 164 configuration 174 congenital hydrocephalus 217 contrast ring enhancement 323 cortex-thickness 212 coughing 278 cranio cervical tumor 29 craniovertebral decompression 277, 279 CRUTCHFIELD extension 144 337 cryoscopy 347 CS CSF fistula 275 CSF lactate 386 - - acidosis 381 CF 208, 210, 211, 248, 322, 323, 325, 368, 370, 371 CT-controls 183 cyst _210 cytotoxic type 334 death rate 162 decompression 59 -, overrapid 199 - of vertebral artery 125 decompressive laminectomy 64. 98, 121, 314 280 - operations - procedure 354 degenerative process 68 demaged organs and tissues 179 dentate ligaments 90, 104, 106, 120 development of hydrocephalic children 212 dexamethasone 321, 322 diencephalic hypothalamic function 337 diffusion of CSF 174 disc degeneration 78 - excision 70 -, hard 284 - herniation 8 - prolapse 120 - protrusion 38, 86 -, soft 284 discectomy, anterior 302 -, cervical 284 discitis 297 discography 23, 116, 284, 297 disconnection 191, 193 dislocation 191, 193 - of catheter 156 - of the valve 252 disorders od micturation 44 disturbances of gait 100, 116 dorsal approach 63 dorsolateral exposure 132 drainage 199, 201

drainage, external 201 -, incongruous 199 dural enlargement 120 - graft 66 - plasty 98 - sac 9 duration of symptoms 96 dysaesthesia 96 dysphagia 288 early surgery 233 EC/IC anastomoses 257, 356, 358 echoencephalography 208 edema 322, 325 -, cytotoxic 333 -, ischemic 333 -, vasogenic 334, 353 EEG findings encephaloceles 248 - - myelomeningoceles 248 EEG long-term follow-up 247 electrical impedance techni-332 que electromyography 22, 287 electron microscope 332 encapsulation 321, 322, 327 encephaloceles 247 - CT findings 248 - EEG findings 248 endocarditis 196 enhancement 13 ependymitis 196 epidural bleeding 303 - space 14 epilepsy incidence 205 epileptic seizures 9, 200, 205 - - in children 247 etiology 212 evoked potentials 26 - spinal electrogram 29 exostoses 66, 293 experimental 321 experimental extracerebral fluid collection 210 extruded disc 115 fasciculation potentials 23 fibrillation 23 focal spinal involvement 28 follow-up 215, 227 - period 228 foramen magnum 14, 124, 277, 278 foraminal osteophytes 96 foraminotomy 47, 49, 60, 66, 87, 105, 106, 120, 287 formation of capsules 322 four-field correlation co-efficient 214 fracture dislocation 53, 54

fractures of nerval arch 14 frontal horn 269 gastrointestinal hemorrhage 90 giant cell tumor 14 Glasgow-Coma-Scale 337, 338, 352 HAKIM shunt 231 head injuries 374 headache, low pressure 199, 200 hematoma 199, 373 -, acute - CT 373 199 -, epidural 199 -, subdural 199, 200 hemianopia 123 hemiparesis 22 hemmer-sleeve 160 hernitated disc 9 histological investigations 267 HOLTER shunt 231 - valve 227 hydrocele 197 hydrocephalus 167, 173, 174, 267, 277, 371 -, cause of 227, 231 -, congenital 231 -, idiopathic 227 -, progressive 278 - after shunt procedures 231 -, surgical treatment of 227 hygroma, bifrontal subdural 233 hypaesthesia 28, 47 hyperosmolar disturbances 339, 345 - values 340 hypertension 371 hypo-osmolality 340 hypophysis 275 hypotension 88 hypoxidosis 100 iatrogenic factors 242 idiopathic normal pressure hydrocephalus 180 iliac crest 53 immediate visceral perforation 192, 193 immune complex disease 263, 264, 265 impotentia coeundi 62 incidence of mechanical shunt complications 193 complications - of periventricular lucency (PVL) 172 increase tone 105 - of tonus 100

infantile communicating hydrocephalus 177 infection 155, 188, 190, 196, 224, 225, 226, 239, 241, 242, 263 intellectual development 204, 230 - functions 210 - performance 229 - status 208, 209 intelligence 208 - quotient 163 interbody fusion 22, 66, 125, 305 interference pattern 25 intermittent shunt malfunction 239 internal carotid artery lesion 363 - - - -, surgical treatment 363 interrelations of causes of death 240 intervertebral foramen 287 - foramina 9 intracerebral hemorrhage 368 - -, prognosis 368 354 intracranial hypertension - -, tolerance to 354 - pressure 321, 337 - -, recording 352 - -, time course 352, 353 intractable pain 308 intramedullary ependymomas 313 - tumor 9 intraoperative bleeding 288 intraspinal tumors 138 intraventricular fluid pressure 327 intravertebral space 9, 59 involuntary closure (MPV) 193 ischemic damage 348 - hypoxia 364 KIEL bone 104 kinking 155, 192, 193 kyphosis 131, 285 lactate 381, 387 - in blood 381 - concentrations 384 - - of serum and CSF 384, 385 - values 389 laminectomy 22, 50, 62, 70, 71, 78, 87, 90, 91, 95, 104, 105, 106, 115, 120, 130, 132 laryngeal palsy 304 late motality 190 - results 218 - visceral perforations 197 length of history 115

lesions simulating PVL 182 LHERMITTE's sign 42, 61, 78, 79, 100 life expectancy 228 ligamenta flava 101 localisation of catheter 156 of ventricular shunt 255 localized cold 332 - induction of cold 334 long spinal tracts 96 long-term follow-up studies of hydrocephalus 247 - results 227 - - of shunt operations 217 loss of conciousness 122 low attenuation 173 lues cerebrospinalis 62 lumbar disc disease - puncture 33, 50 33 lyophilized dura 120 malformations of the brain 215 mass effect 353 massive ischemic brain infarction 352, 353 mean CBF 358 mechanical shunt complications 191 mechanisms of obstruction 268 medullary compression 50, 78, 113 meningioma 62 meningitis or meningoencephalitis 195, 226 mental development 157, 159, 210, 213, 214 impairment 209 26 metacrylate metal plate 54 metastāses 138 metrizamide 13, 173 microtraumatisation migraine cervicale 62 migration of system 191 monoradicular disturbance 130 - syndrome 132 MORQUIO syndrome 60 mortality 157, 159, 188, 226, 228 -, early 235, 238, 240 -, late 235, 238, 240 - rate 241 multiple sclerosis 26, 305 multisegmental protrusions 98 myelography 38, 47, 54, 65, 84, 116, 138, 284 -, cervical 287 myelomeningoceles 159, 168, 217, 247, 267 - CT findings 248 - EEG findings 248

myelopathy 23, 29, 83, 88, 90, 104, 122, 130, 133, 302 narrow intervertebral foramen spinal canal 8, 14, 22, 33, 38, PBS 349 49, 70, 78, 92, 101, 112 percutan needle electrodes 22 neonatal intraventricular hemorrhage 178 nephritis 263, 264, 265 nerve root compression 24 - roots 130 neurofibromatosis 144 neurogenic shock 239, 241 neurological deficits 205, 256 - impairment 2 255 -, diencephalic 377
-, hemispheric 377 - -, mesencephalic 377 neuropsychologic results 204 non-compensation of intracranial pressure rises 239 non-invasive r CBF measurement 356 non-survivors 344 n r CBF 360 - pattern 361 nuclear atrophies 90 obstruction 268 - of catheter 155, 159 occlusive hydrocephalus 179, 181 oedema 165 operative death 188 organic causes with shunt failure 239 osmolality 343 - and prognosis 339 osmoregulation 337 osmotherapy 381 29, 71, 293 osteochondrosis osteochondrotic spurs 287 osteolytic cervical dislocations 144 osteophytes 14, 39, 60, 122, 291, 302 osteophytic bars 95 - protrusion 61 - spurs 70, 88, 115 osteoporosis 288 overdrainage 199, 200 -, acute 199 -, chronic 199 PANCOAST's syndrome 305 paraparesis 22, 27, 62, 78, 83, 90 paraplegia 83

parent-physician-patientresignation 239, 242 paraesthesia 105 passive diffusion 174 132 pathological mechanism 174 percutaneous rhizotomy 308 perforated disc 115 perfusional brain scanning 347, 348 peripheral neuron 24 peritoneal catheter obstruction 228 - malabsorption 197 peritonitis 195, 196 periventricular lucency 165, 17 174 - oedema 165**,** 171 peroneal nerve 27 phlebothrombosis 197 physiotherapy 121 pincer mechanism 9, 33, 101 polycystic transformation of hydrocephalus 244, 245, 246 polyphasic waves 24 polyneuropathia 24, 28 porencephaly 210 posterior approach 65, 87, 104 - compression 115 - foraminotomy 38 - myelotomy 316 - spondylosis 78 postherpetic pain 309 postoperative course 231, 232 post-shunting subdural collections 164 posttraumatic coma 374 - -, sleep EEG alterations 374 preventive lengthening 156 - re-implantation 156 primary defects 236 - with infection and shunt failure 239 prognosis 228, 243 prognostic assessment 7 progressive deterioration 121 prolapse of intervertebral disc 3 prolapsed cervical discs 90 - discs 91 protruded disc 22 protrusion 130 proximal abscesses 195 pseudocraniostenosis 200, 201 pseudocysts 197 psychological results 204 - test 213 PUDENZ-HEYER shunt 210 PUDENZ pump 228 - valve 227 pulmonary arteritis 265 - embolism 90

PVL 173, 175, 180, 181, 182 quadriparesis 83 quadriplegia 50 QUECKENSTEDT's manoeuvre 278 **- -,** test 10 radicular disturbances 96 - pain 50, 117, 284, 304 - symptoms 83 - syndrome 54 - vessels 48, 66 radiculo-medullary arteries 104 radiculopathy 64, 122, 130, 133, 302 radiofrequency thermic lesion 308 recording of CSF pressure 184 recruitment 23 relative valve insufficiency 156 REM sleep 377, 378 retardation 204, 209 retrospondylosis revision 179, 187 - of atrial catheters 225 - of peritoneal catheters 225 - of ventricular catheters 223 - of ventriculo-atrial shunts 221 root compression 59, 287 rupture of reservoir 192, 193 27 scalp electrodes school situation 157 schooling 229 segmental evoked potential 30 selective catheterisation 124 sensory threshold 27 sepsis 54, 156, 195, 226 septicaemia 90, 195, 196 sequential CT studies 371 sequester perforation 130 serous meningitis 318 serum-lactate concentrations 384 severe brain injury 343 sharp waves 23 shortening of distal catheter 192, 193 shoulder arm syndrome 9 - pain 117, 122 shunt 200 -, clinical management 210 - complications 251, 267 - dependence 157 - dysfunctions 267, 269 - failure 241, 242

- induced immune complex disease 265, 266 intrathecal 201 - long-term results 217 - malfunction 183, 186 - no functioning 215 - non-invasive instrumental follow-up studies 208
- obstruction 184, 201
- operations 204, 215 - -, cause for 222 - -, survival time 236 - removal 201 - revision 188, 223 - -, cause for 228 -, ventricular size and mental development 209 silicon polymer 366 sitting position 120 sleep alterations 375 - analysis 375 - EEG alterations 374 - phase 376 slipped disc 51, 101 slit ventricle 165, 169, 174, 184 - - syndrome 199, 200, 201, 202 - - -, radiological findings 202 - - -, clinical findings 202 - - -, surgical treatment 202 sorbitol 381 spastic paraparesis 95, 96 - tetraparesis 70 spasticity 27, 41, 44, 101, 105 spina bifida 247, 277 spinal compression 44 - cord 9, 138 - - compression 47, 65, 115 - dysraphism 138 - elastance 33 - irritation 9 - lesions 138 - neoplasms 78 - pressure 278 - stenosis 96 - subarachnoid space 33 - tumor 70 sphincter disturbances 71 spondylarthrosis 71 spondylolisthesis 304 spondylosis 8, 38, 65, 78, 86, 123, 124, 284 spondylotic bars 91 - myelopathy 55, 67, 69, 70, 112, 125 - spurs 47, 285 spontaneous course 7 staphylococcus albus 264 state of consciousness 387, 390

- of development 229 steel plate spondylodesis 144 stenosis of the cervical canal 65, 80, 164, 306 - of spinal canal 13, 78 subarachnoid bleeding 277 - cysts 165, 169 - space 9, 13, 95 subdural fluid collection 199 - - -, chronic 200, 201 subependymal hypodense zone subluxation 23 173 supratentorial extradural compression 332, 335 surgical displacement 192, 193 - procedures 190 - revision procedures 189 therapy 4 survival time 236 survivors 344 SVS 183 syringobulbia 282 syringomyelia 277, 278, 305 TC99 347 tetraparesis 22, 53, 54, 90, 116 tetraspasticity 62 thermocoagulation 308 thermorhizotomy 308 thromboembolic stenosis 349 thrombosis 156, 161, 268 TIA 347 tibial bone 53 tick-borne encephalitis 318 time of hemorrhage 368 - of initial shunt operation 223 - interval 187 tinnitus 122 TORKILDSEN shunt 218, 219, 227 toxoplasmosis 228 transoral approach 275 transverse cord lesion 117 trigeminal neuralgia 308 tumors of posterior fossa 173 types of obstructions 269 umbilical fistulas 197

underdrainage 199, 200

VALSALVA manoeuvre 278 valve insufficiency 159, 199, 220 - - antisiphon 201 valvular effect 278 338 variations in osmolality vascular impairment 88 malformations 366 - myelopathy 26, 84 V-A-shunts 195 vasogenic type 334 ventral approach 60, 63, 90, 92, 130, 132 - discectomy 287 - fusion 70, 83, 116 - resection 53 - route 100 ventricular catheter 155 - -, obstruction 228 - -, placement 256 deformation 167 lactate 390 - shunt insertion 255 size after shunting 165 ventriculitis 195, 196 ventriculo-atrial shunts 217 ventriculo-cisternal shunts 217, ventriculo-peritoneal (VP) shunts 187, 195 vertebral artery 122 288 infractions - vertigo 62 vertebrobasilar ischaemia 122 - symptoms 47 vertigo 123 ventrolateral approach 132 vestibular disturbances 47 VF-lactate 387, 391 - analysis 386 - and prognosis 387 videodensitometry 358 visceral pain 310

width of external CSF spaces 210 wire fixation 54 wound infection 228

xenon activity 356

Advances in Neurosurgery

Distribution rights for Japan: Nankodo Co. Ltd., Tokyo



Springer-Verlag Berlin Heidelberg New York Volume 7

Neurovascular Surgery Specialized Neurosurgical Techniques

Editors: F. Marguth, M. Brock, E. Kazner, M. Klinger, P. Schmiedek

1979. 202 figures in 249 separate illustrations, 85 tables. XXII, 394 pages ISBN 3-540-09675-2 Distribution rights for Japan: Nankodo Co Ltd., Tokyo

Contents: Neurovascular Surgery. – Specialized Neurosurgical Techniques. – Free Topics. – Subject Index.

This volume is the product of an exchange of ideas and experience between the American Academy of Neurological Surgery and the Deutsche Gesellschaft für Neurochirurgie. It contains the papers delivered at their joint meeting, covering a number of different topics in neurosurgery. Special emphasis is placed on intracranial vascular surgery and specialized neurosurgical techniques for operative approaches to the skull, brain, pituirary gland, and peripheral nerves.

Also included are contributions on tomography, traumatology, functional and experimental neurosurgery and chemotherapy. The broad range of topics discussed provides an insight into the present state of neurosurgery. It points out those areas in which progress has been made and those where further research is indicated.

Advances in Neurosurgery

Distribution rights for Japan: Nankodo Co. Ltd., Tokyo

Volume 1

Brain Edema

Pathophysiology and Therapy

Cerebello Pontine Angle Tumors

Diagnosis and Surgery Editors: K. Schürmann, M. Brock, H. J. Reulen, D. Voth 1973. 187 figures. XVII, 385 pages ISBN 3-540-06486-9

Volume 2

Meningiomas

Diagnostic and Therapeutic Problems

Multiple Sclerosis

Misdiagnosis

Forensic Problems in Neurosurgery

Editors: W. Klug, M. Brock, M. Klinger, O. Spoerri 1975. 200 figures, 86 tables. XXI, 444 pages ISBN 3-540-07237-3

Volume 3

Brain Hypoxia Pain

Editors: H. Penzholz, M. Brock, J. Hamer, M. Klinger, O. Spoerri 1975. 160 figures, 110 tables. XIX, 460 pages ISBN 3-540-07466-X

Volume 4

Lumbar Disc Adult Hydrocephalus

Editors: R. Wüllenweber, M. Brock, J. Hamer, M. Klinger, O. Spoerri 1977. 154 figures, 67 tables. XXII, 338 pages (8 pages in German) ISBN 3-540-08100-3

Volume 5

Head Injuries Tumors of the Cerebellar Region

Editors: R. A. Frowein, O. Wilcke, A. Karimi-Nejad, M. Brock, M. Klinger 1978. 205 figures, 74 tables. XXI, 396 pages ISBN 3-540-08964-0

Volume 6

Treatment of Hydrocephalus Computer Tomography

Editors: R. Wüllenweber, H. Wenker, M. Brock, M. Klinger 1978. 111 figures, 86 tables. XXXI, 230 pages ISBN 3-540-09031-2



Springer-Verlag Berlin Heidelberg New York