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# Treatment of Hydrocephalus

# Computer Tomography

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# Preface

More than 40 years ago British and German neurosurgeons met in Berlin and Breslau to exchange their experiences, to strengthen their friendly bonds, and to enjoy the attractions of both cities and their surroundings.

In 1960 a joint meeting of the Dutch and German societies took place in Rotterdam by invitation of the **Nederlandse Vereniging van Neurochirurgen.** All who attended this meeting thankfully remember the great hospitality during these unforgettable days.

In 1970, by courtesy of the **Society of British Neurological Surgeons,** German neurosurgeons had the pleasure to become acquainted with the great tradition of British sciences at one of the most famous places during the meeting in Cambridge. These impressions were deepened by visits to other famous sources of British scientific tradition during the European Congress in Oxford in 1975.

The critical distance sometimes necessary towards our own discipline is implicit in the major themes of this meeting. Complications following shunting procedures for hydrocephalus have been discussed on the basis of the results of a cooperative study of some German neurosurgical departments.
The second main topic was problems and diagnostic errors in computer tomography. The papers presented contain critical considerations about the findings obtained by this method, as well as on its possibilities and limitations.

German neurosurgeons felt deeply indebted to their British and Dutch colleagues and wanted to return their kindness at this joint meeting in Berlin. We hope that all participants will retain pleasant memories of the days in this city.

In the name of the German Society for Neurosurgery, the organizers of the Joint Meeting in Berlin express their gratitude to Professor G. Finger of Sharp & Dohme GmbH München for generously supporting the publication and distribution of Advances in Neurosurgery 6.

Horst Wenker
Rolf Wüllenweber

# Reminiscences of the Meeting of 1937 and of Otfrid Foerster[1]

C. GUTIÉRREZ[2]

There are two steps to be taken by those who wish to advance a medical specialty: the formation of a society and the publication of a journal. The birth of the first neurosurgical society was reported by Ernest SACHS in his autobiography [27]: Harvey CUSHING delivered a memorable address on his brain tumor statistics in 1919 before the American College of Surgeons with William MAYO in the chair. At the conclusion Dr. MAYO announced solemnly: "Gentlemen, we have this day witnessed the birth of a new specialty – neurological surgery". After the meeting CUSHING was congratulated by many and he said enthusiastically: "Wouldn't it be a good idea to get the fellows interested in this work together? Why not form a society and hold regular meetings in which we could discuss our problems and compare results? In this way we could make much more rapid progress." This suggestion was followed and the first meeting was held in Boston in 1920. CUSHING was elected president, SACHS secretary, and the first neurosurgical society was founded. The first neurosurgical journal, the Zentralblatt für Neurochirurgie, was started in Germany in 1936 by TÖNNIS [15] but not until 1948 was the Deutsche Gesellschaft für Neurochirurgie founded [8]. The societies mentioned above were established by surgeons. It was different in the Netherlands. The Dutch Study Club for Neurosurgery was formed in 1936 [29], the membership consisting of four neurologists and four neurosurgeons. The initiative came from a neurosurgeon, VERBECK, but it was the neurologist BROUWER, who was the driving force. He was elected the first president and remained so until his death in 1949. The Nederlandse Vereniging van Neurochirurgen was founded in 1952 [29].

When the Society of British Neurological Surgeons (SBNS) was created by Geoffrey JEFFERSON in 1926, it intended to hold two meetings each year, at home in winter and abroad in summer. The first meeting abroad was held in Paris in 1930 and the next in Amsterdam in 1932 which I attended and found wonderfully rich in culture and hospitality, but not very stimulating neurosurgically. I remember how disappointed BROUWER was when OLJENICK, the neurosurgeon in his clinic, outdid CUSHING as regards the most minute operating details, performed a ventricular estimation (which was in fashion at that time), but when he opened the skull he did not find the tumor. The specialty hat not yet gotten on its feet in Holland, but soon thereafter de VET, LENSHOEK, VERBECK, and VERBRIEST brought it to a proper high standard.

The summer meeting of 1937 [16, 31] was held in Berlin and Breslau, and again I was a guest of the SBNS. The three days in Berlin included visits to the Neurosurgical Clinic of TÖNNIS, the Kaiser-Wilhelm-Institut für Hirnforschung and SAUERBRUCH's Clinic. A joint meeting was held with the Berlin Medical Society, where President MCCONNELL of

1 Society of British Neurological Surgeons, 29 June to 3 July 1937 at Berlin and Breslau.

2 Institute of the History of Medicine and Neurosurgical Clinic of the University of Göttingen.

the SBNS lectured on the Chiasmal Syndrome [20, 21]. The principal topic of the meeting [32] was intracranial tumors, their nature, and their diagnosis with ventriculography, arteriography and EEG. The distinction from pseudotumor cerebri was discussed by NONNE who had coined the term in 1904 [23]. Other matters considered were subdural hematomas, spasmodic torticollis, and the importance of angiography for the diagnosis and treatment of aneurysms. Among those who read papers were BUSCH (Copenhagen), BERGSTRAND, OLIVECRONA, RINGERTZ and SJÖQVIST (Stockholm), TORKILDSEN (Oslo), NONNE (Hamburg), SCHALTENBRAND (Würzburg), and OSTERTAG, SPATZ and ZÜLCH (Berlin).

The trip to Breslau to honor FOERSTER and to visit his institute was especially interesting and pleasant. FOERSTER had close neurological connections with England, having been a devoted disciple of HUGHLINGS JACKSON and of SHERRINGTON. He had given three lectures under the auspices of London University in 1931 [10], the SCHORSTEIN Lecture at the London Hospital in 1932 [11], and the HUGHLINGS JACKSON Centennial Memorial Lecture of the Royal Society of Medicine in 1935 [14]. He was made Emeritus Member of the SBNS at the Breslau meeting where he entertained and instructed us royally with three lectures. These formed a report of the 552 verified tumors of the nervous system which he had collected in 17 years from 12000 admissions to his Neurological Department at the Wenzel-Hancke-Krankenhaus. The social activities included a supper for the entire company at FOERSTER's villa in Scheitniger Park to which the visitors were transported in a specially provided tram, FOERSTER being of the opinion that the town could be seen better and more comfortably from a tram than from a taxi. FOERSTER restricted his work to Neurology and dedicated all his efforts to establishing neurology as an independent specialty [24]. Neurology had been a stepchild in Germany, at first of internal medicine, later of Psychiatry. ROMBERG, Professor of Therapeutics in Berlin, wrote the first textbook of Neurology in 1840 [26], having been influenced by the writings of Charles BELL, which he translated in 1832 [1]. Soon afterwaards GRIESINGER wrote the first German textbook of psychiatry in 1845 [19], and, declaring that mental illness was due to disease of the brain, brought psychiatry and neurology together for treatment and teaching. This set some psychiatrists to the very productive anatomical study of the brain, among whom were MEYNERT, FOREL, WERNICKE, NISSL and ALZHEIMER. But FOERSTER felt strongly that the field was too wide for one man to straddle and do justice to both specialties, thereby impeding the development of neurology [12]. But in spite of his constant efforts for 40 years, the German regulations for medical education and examination in 1966 [25] still lumped psychiatry and neurology together and stated that the examination in neurology might be conducted by an examiner of internal medicine.

When FOERSTER qualified as a physician in 1897 [5], he went to Heiden in Switzerland to study with H. S. FRENKEL and to the DEJERINES in Paris. Straight away he started physiological studies on the sensation and gait of patients with tabes dorsalis [6], which formed the basis for his great success with the treatment of pain, the relief of spastic paralysis, and exercise therapy. Within ten years on 3 May 1907, he directed the performance by TIETZE of the FOERSTER operation, the division of posterior roots for the treatment of spastic paralysis [9]. This was the beginning of physiological neurosurgery. At the same time he continued the study of movement and further developed the field of exercise therapy [7], now known as rehabilitation, to which he contributed consecutively

for 40 years [13]. FOERSTER prepared Ludwig GUTTMANN, who was his assistant for several years, to extend the field of rehabilitation. GUTTMANN did this with great zeal and success, but without giving FOERSTER any sign of recognition or of gratitude for the great debt he owed his master. Sadly, GUTTMANN had suffered for political reasons and had to leave his position in the hospital with FOERSTER in 1933. He complained unjustly that FOERSTER had not protected him, although he secured an appointment for GUTTMANN in another hospital in Breslau. But who was able to oppose the will of HITLER at that time? Nevertheless, Germany's loss was England's gain, and thousands of neurologically disabled throughout the world have benefitted, through GUTTMANN, from FOERSTER's neurophysiological research and teaching.

FOERSTER encountered many obstructions to his efforts for the liberation of Neurology. The greatest opposition came from psychiatrists such as BONHOEFFER, who considered themselves competent as neurologists and maintained that they should continue to direct both psychiatry and neurology [2, 34]. The fallacy of their opinion has been proven by the relatively few contributions made by them to the progress of Neurology. The field is too great for one man to be productive in both specialties.

BONHOEFFER was assistant to WERNICKE in Breslau for 10 years, from January 1893 until October 1903, and succeeded him in October 1904 as Director of the Psychiatric and Nervenklinik. He left Breslau for Berlin in 1912 [4]. FOERSTER was an assistent in the clinic and laboratory of WERNICKE from 1899 until October 1904. Thereafter for several years FOERSTER's papers were published from the surgical separtment of Prof. TIETZE in the Allerheiligen Hospital. Little is known about the relationship between BONHOEFFER and FOERSTER but it is striking, that in his autobiography [4] BONHOEFFER did not once mention the name of FOERSTER, although he wrote interestingly about his colleagues and life in Breslau for about 20 years. During 12 of these FOERSTER was there, and for 4 years they were both in WERNICKE's department. BONHOEFFER was in Breslau to live through FOERSTER's becoming Privatdocent in 1903 [8], titular professor in 1909, and head of an independent Neurological Department at the Allerheiligen Hospital in 1911. FOERSTER must have been very difficult for BONHOEFFER to overlook and, much more so, to swallow.

BONHOEFFER was just as determined to keep Neurology under the control of Psychiatry as FOERSTER was opposed. The battle smouldered until it burst into flame at the time of the First International Neurological Congress at Bern in 1931 [17, 18]. MINKOWSKI, the neurologist in Zürich, who had been trying to make Neurology independent in Switzerland, published data [22] to show that the development of Neurology in Germany was far behind that in a number of less important countries. BONHOEFFER [3] reacted vigorously to the report and the recommendations of MINKOWSKI. FOERSTER replied with an eloquent rebuttal of BONHOEFFER's arguments [12]. FOERSTER's forthright comments on BONHOEFFER's attitude at this time may be attributed to a confidence he acquired that Neurology was gaining ground, after the discussion he had in October 1930 about "Neurology in Germany" with Dr. Alan GREGG, who was head of the Medical Sciences Division of the Rockefeller Foundation. They developed a plan to make Neurology an independent specialty in Germany which appeared to lead to a certain success. It consisted in a promise that the Rockefeller Foundation would build a Neurological Research Institute in Breslau, to be controlled by the University, if the City

of Breslau, the Province of Silesia, and the State of Prussia would supply funds for the upkeep of the institute, and the Prussian Ministry of Science, Art and National Education would establish a Chair of Neurology in the University. After 2 years of tedious and strenuous work FOERSTER was able to assure the upkeep of the institute, but not the keystone of the plan, since the Ministry found it impossible to establish even one new chair in any university, because of the grievious state of the economy at that time. FOERSTER was almost in despair, but the Ministry did promise to make his professorship permanent and to continue it for his successors. This satisfied the Rockefeller Foundation, so the institute was built and it was opened in 1934. It was there that the Breslau meeting of the SBNS was held in 1937. After FOERSTER's death in 1941 it was named the Otfrid FOERSTER Institute [30].

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# The Development of Neurosurgery in Berlin

R. WÜLLENWEBER

Until the end of the Second World War, the street which lies between Brandenburg Gate and the Victory Column was named "Siegesallee" (Victory Avenue). As it was embellished with a collection of esthetically unremarkable statues, it was more pertinently described in the Berlin vernacular as "Puppenallee" (Dummy Avenue). At the midpoint of the street stood a statue of Markgraf Otto the Fourth „with the arrow". This margrave, head of the older Askanian line at the end of the 13th century, had suffered a head wound by an arrow in the course of one of his many battles, but this arrow was not removed for more than one year thereafter. The fact so impressed his posterity that he was henceforth referred to as "Otto with the arrow", but the phenomenon casts a shadow on 13th century neurosurgery in Berlin, since apparently no one had dared to remove the arrow.

We have no evidence of neurosurgical activity in Berlin in the late middle ages. This changed in the 18th century with the foundation of the Charité, a very progressive hospital for its time and a center for the training of military physicians. The spirit of medical practice at the Charité in the early years was influenced by the Dutchman BOERHAAVE, whose clinic at Leyden was known to the entire medical world. With all due regard to current knowledge in anatomy, physiology and chemistry, the guiding principle of BOERHAAVE's practice was his experience in practical medicine. Most physicians at the Charité were, directly or indirectly, pupils of BOERHAAVE, and his influence was extraordinary, as is evident in Frederick the Great's remark: "In medicine the professors must adhere to Boerhaave's method."

As described by DIEPGEN and HEISCHKEL, trepanation was one of the major operations that were current at the Charité:

> "The primary indications are: removal of bone splinters, foreign bodies, hemorrhages and other effusions; depression of the skull and compression of the brain after injury to the head, the symptoms of which have been well described; more rarley, carious and purulent processes of the inner layer of the calvarium; and refractory headache. In hemiplegia, one seeks the lesion on the opposite side of the cranium, and if it is not found there, one may still apply the trepan to the other side. It seemed to be especially important to avoid cooling of the brain, for which reason the instruments were kept near a brazier, the room was kept warm and rinsing solutions and medications were heated before application. The drill-like crown trepan was used with great care, layer for layer. The dura mater was opened only if it was tense and showed fluctuation. Otherwise, it was treated with alcohol, in order to protect it from 'corruption'. If it was inflamed, bloodletting was performed. Prolapse of the brain was prevented by applying a lead or silver cap."

Trepanation was controversial even at the time, and rancor among colleagues was not uncommon, as is evident in the comment that Dr. PALLAS, of the Charité, "had placed the trepan on the suture and near the sinuses with the greatest audacity." In fact, PALLAS had warned against this and allowed trepanation at these two locations only in emergencies.

After the foundation of the University of Berlin in 1811 under the influence of Wilhelm von Humboldt and after establishment of the Royal Surgical Clinic in the Ziegelstraße, surgery was well represented at the university, with two clinics, but the university itself stood entirely under the influence of the enlightenment, idealism and natural philosophy.

Fig. 1. Johann Friedrich Dieffenbach 1792–1847

In a speech at the 164th anniversary meeting of the "Gesellschaft für Natur- und Heilkunde zu Berlin", Ewald HARNDT reported that the faculty of philosophy, guided by the spirits of FICHTE, SCHLEIERMACHER, HEGEL and SCHELLING, was dominant for decades and the "science" was understood exclusively as humane arts and sciences, not at all as natural science. So the "sciences of nature" were ranked as an adjunct to the philosophical faculty at the University of Berlin. In succeeding generations, the battle raged between proponents and opponents of trepanation. The second physician to occupy the chair for surgery in Berlin, Johann Friedrich DIEFFENBACH (Fig. 1), belonged to the latter group. DIEFFENBACH was an exceptionally versatile person, and he has been called the father of plastic surgery with some justification. Among his many publications is an article "On division of the sternocleidomastoid muscle in the treatment of wry neck", which appeared in 1838. In 1828 he reported his experience with blood transfusion, and in 1845, in his book "Operative Chirurgie", he noted the influence of English surgeons on his work. In the same book, DIEFFENBACH reports that after unsuccessful division of the infraorbital nerve for treatment of trigeminal neuralgia, intracranial division of the V. nerve was considered but rejected as too risky. He recognized the advantages of ether anesthesia and published "An introduction to patient care" in order to improve nursing, which was apparently in a dismal state. The following lines are quoted from the "Introduction":

> "What is the situation in the wards, for young men, especially for students? There is no cake and pastry there, as there is in the women's wards, but there is certainly beer and tobacco. Thick smoke fills the room. Men with mustaches and long pipes lie about on the sofas and chairs, and it is only with effort that one discovers the patient in bed as though on a palanquin. And among all these one finds a busy person

running back and forth, carrying beer, filling pipes and so on. That is the nurse! He often runs out of the room, as though to carry out an empty bottle, but he puts it to his mouth and takes a long draught. And then he smokes a few draughts of tobacco ..."

DIEFFENBACH died while presenting a patient during a lecture. His successor, Bernhard von LANGENBECK (Fig. 2), took his chair for surgery in 1848. LANGENBECK, who had become lecturer in physiology and pathology in Göttingen in 1838, was decisively influenced by England in his development as a surgeon. He felt strongly attracted to Astley COOPER, who was the dean of British surgery, though almost 70 years of age, and who opened the doors of the Royal Medico-Chirurgical Society to LANGENBECK. His close ties to British surgery over several decades resulted in the nomination of PAGET, LISTER and SPENCER WELLS as the first honorary members of the Deutsche Gesellschaft für Chirurgie, of which LANGENBECK was a co-founder.

Fig. 2. Bernhard von Langenbeck, 1810–1887

Gunshot wounds of the skull dominated LANGENBECK's neurosurgical practice, since he was surgeon-general and entrusted with the education of Prussian military physicians (as was his successor, Ernst von BERGMANN). In addition, he was interested in the treatment of hydrocephalus. In this regard, there is the following reference on ventricular puncture in the "Handbuch der speziellen Pathologie und Therapie", edited by Rudolf VIRCHOW (1869): "Langenbecks method – which is to enter the anterior horn of the lateral ventricle from below, by introducing the trocar behind the upper eyelid and piercing the top of the orbital cavity – deserves special attention." He reported "On hypodermatic ergot injections in aneurysms" before the "Berliner Medizinische Gesellschaft" in 1869.

In 1880 he presented a patient before the same society "On nerve repair with presentation of a case of secondary suture of the radial nerve". LANGENBECK was also able to report success in the surgical treatment of sarcomas of the skull and the dura mater with a

procedure in which he removed part of the dura with the tumor. We today might suspect a number of meningeomas among these tumors.

Ernst von BERGMANN (Fig. 3), LANGENBECK's successor, came from Würzburg to Berlin, as has DIEFFENBACH more than fifty years before and as would TÖNNIS more than 50 years later. Neurosurgery was his special interest, and his experimental work on intracranial pressure, begun in Dorpat and Würzburg, established basic principles of the pathology of intracranial pressure which are still valid today. His observations on the symptoms of increased intracranial pressure, published in "Die Lehre von den Kopfverletzungen" in 1880, and his articles "Über den Hirndruck" from 1885 and 1886 contain, together with the work of NAUNYN and SCHREIBER, most of the knowledge that had been rediscovered in this area in the past ten years.

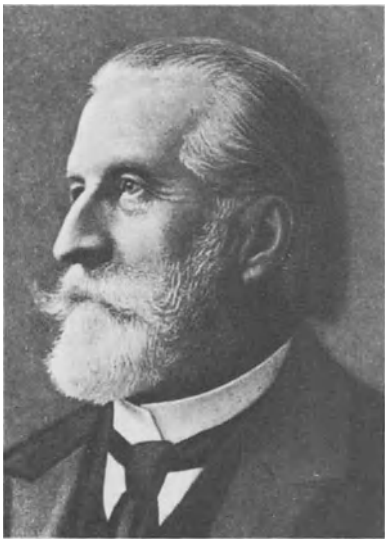

Fig. 3. Ernst von Bergmann, 1836–1907

In the second edition of his "Chirurgische Behandlung der Hirnkrankheiten" (1888–1889), von BERGMANN took an extremely critical position on attempts at surgical treatment of brain tumors, epilepsy and hemorrhage. The lists of papers, delivered before the "Gesellschaft für Natur- und Heilkunde", the "Berliner Medizinische Gesellschaft" and the "Berliner Chirurgische Gesellschaft" contain about ten lectures per year on neurosurgical topics for the period of the 1890's and at the turn of the century (including addresses by BERGMANN's pupils BORCHARD, GULEKE, KÖNIG, LEXER and SCHMIEDEN), ample evidence of the enormous interest in the newly developed field of neurosurgery.

In addition to the work at the university centers, surgeons in municipal and denominational hospitals performed neurosurgical operations. Eugen HAHN, a pupil of WILMS removed a brain tumor at the Bethanien Krankenhaus in 1882, making use of the diagnosis by WERNICKE. The Augusta-Krankenhaus, where Fedor KRAUSE (Fig. 4) was appointed head of a surgical department in 1900, was also a non-academic institution. KRAUSE had been trained as an ophthalmologist and then became director of the

municipal hospital in Altona. His inaugural dissertation from the year 1887 dealt with malignant neuromas, and a monograph on trigeminal neuralgia appeared during his period in Altona. In 1892 HARTLEY and KRAUSE, independently of each other, described the extirpation of the Gasserian ganglion via the extradural approach from the floor of the middle fossa. As a result of the training in ophthalmology, KRAUSE was especially interested in the question of keratitis in relation to the ganglion extirpation. He did not limit himself to neurosurgery, as lectures on gastric surgery, reconstructive surgery of the ureter, of the mandible and free transplantation of large areas of skin, as well as major works in the fields of ophthalmology and bacteriology, a monograph on tuberculosis of the bones and joints and his textbook of surgical operations clearly show. He was enormously productive in the field of neurosurgery and described surgical approaches to almost all areas of the brain cavity. He was the first to employ the transfrontal intradural approach to the pituitary gland, and he performed the first exposure of the cerebellopontine angle and the first operative removal of a tumor in the lamina quadrigemina. In 1909 he reported his experience in 28 operations on the spinal cord, and in 1908 and 1911 he published his "Chirurgie des Gehirns und Rückenmarks", in which he not only described and illustrated surgical technique of the highest order, but also furnished exact statistics on a patient group that was exceptionally large for that period.

Fig. 4. Fedor Krause, 1857–1937

A surgeon such as KRAUSE, with interests in all fields of surgery, depended on the co-operation of an outstanding neurologist in order to succeed in neurosurgery. This neurologist was Hermann OPPENHEIM, who had published the first edition of his "Lehrbuch der Nervenkrankheiten" in 1894. OPPENHEIM, a pupil of WESTPHAL, had mastered neurology as no other at the time and pursued his medical and scientific activities with untiring effort despite a difficult professional and personal situation.

The activity of Fedor KRAUSE and of his pupil and successor HEYMANN at the Augusta Krankenhaus made this house a center of neurosurgery, which was also cultivated by

SAUERBRUCH and others in the university hospitals in the 1920's and 1930's. The Surgical Congress of 1935 produced a break – in that neurosurgery was recognized as a separate speciality – and this lead to the appointment of TÖNNIS in Berlin 1936. TÖNNIS writes that he was able to begin work in the clinic at Hansaplatz on May 1, 1937 and that the British Society of Neurological Surgeons held its congress in Berlin and Breslau in June of the same year, in order to provide a fovorable start for TÖNNIS and his co-workers and to emphasize the significane of Berlin for european neurosurgery. The first efforts were, in fact, so successful that many foreign physicians came to Berlin for training in the short period before the outbreak of World War II. A large number of patients were treated also during the war in the first academic neurosurgical clinic in Germany, and many publications appeared, especially in the "Zentralblatt für Neurochirurgie" which was founded by TÖNNIS.

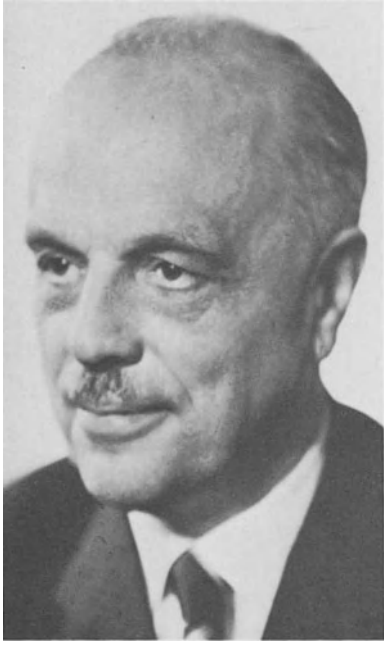

Fig. 5. Arist Stender, 1903–1975

At the end of the Second World War, in which the neurosurgical clinics were destroyed, Arist STENDER (Fig. 5), who had been Otfried FOERSTER's successor as director of the clinic in Breslau, began his work at the Augusta-Krankenhaus, where F. KRAUSE had worked. A few weeks later, he moved to the municipal hospital in Westend to develop a neurosurgical-neurological clinic where none had existed before. Shortly after the war, he successfully reestablished his earlier relations with the United States, and with his revered neurosurgical mentor Percival BAILEY. The basis of his practice, which encompassed the whole of neurosurgery, was neurological diagnosis. He was a master of this field and felt himself bound by a debt of gratitude to his teacher Max NONNE. Gangliolysis of the Gasserian ganglion carries his name. Trigeminal neuralgia was also a topic of major neurosurgical interest for Willy FELIX, SAUERBRUCH's successor at the Charité. The

quality of neurological and neurosurgical training in STENDER's clinic is most evident in the fact that a large number of his pupils became successful physicians and scientists not only in the field of neurosurgery, but in neurology and neurophysiology as well.

Berlin gained a second academic neurosurgical clinic with the construction of Klinikum Steglitz, where Wilhelm UMBACH became director until his untimely death. It was certainly not easy for Artist STENDER to accept a successor in his Westend clinic, who had been educated in an entirely different school and whose training was primarily in surgery rather than in neurology. Nevertheless, STENDER did so without prejudice and smoothed the way for him in Berlin. STENDER's memory lives on in many hospitals in this city, as one can hear when his baltic cadence is parodied in jovial company, for anecdotes spring up only around personalities.

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# Fedor Krause Memorial Lecture

Held on the occasion of the conferring of the Fedor Krause Medal on Professor Peter Röttgen by the German Society for Neurosurgery on May 5th, 1978, in Berlin.

## Reflections of a Neurosurgeon: Splendor and Trials of a Dedicated Neurosurgeon

P. RÖTTGEN

You have conferred the Fedor Krause medal on me, the highest distinction of our Society. That it is the Fedor Krause medal has pleased most the surgeon in me, and I wish to thank you very much indeed.
When a man has discarded the vanity of the first decades of life, as things have assumed other dimensions, his pleasure need not thereby become less. But one asks one's self with more self-criticism, how one has deserved this honor. After all, one cannot simply compare the high standard of German neurosurgery today, to which one is proud of having contributed, with that of the man depicted on the medal.

This man can only be seen in relation to his own time, in which he did pioneer work for the whole surgical world and in which he did so much so excellently for the first time. Sometimes his achievements were important enough to be covered by two names, as is the case for the Krause-Dandy-flap. I should like to recall the well-known phrase of Schiller, whose name, as well as Goethe's, is always mentioned on such occasions: "Whoever has satisfied the best people of his time" ... that is you, who confer medal ... "has lived for all times". This is too flattering as regards me, I must admit, and I do hope, all my friends agree with me in this respect.

For my lecture I have chosen a general theme, as fits a man of my age. This does not mean, though, that there is nothing more to say about the present problems of neurosurgery, mainly if we consider that especially in the last few years, reliability of diagnosis and therapy has made extraordinary progress, which, in my opinion, can only be evaluated properly by those who have witnessed the beginnings.

When a devoted neurosurgeon is about to leave the operation-theatre, which until then has represented the essential part of his professional life, and, tired and happy, is to retire to that proverbial bench under the beautiful tree, then he is asked by his friends and pupils, even with a certain right, to look into the mirror of his life. With age, such a mirror becomes agreeably dim. The contours are somewhat blurred and man's faculty to see things as they should be grows stronger. Well, I have always been known for naming disagreeable things by their name. Thus, I should not, I suppose, make exception to myself. After having tried to give the mirror a polish, I began to realize that also its owner had, himself, changed in his evaluation of things. "The clock calling me to duty" became more and more unbearable. Administrating and running even such a magnificent clinic

became rather burdensome. The classification of the gliomas became less important than that of, say, the conifers.

More and more resignation crept into the battle for the life and the health of the patient. Even operating often turned out to be more of a burden than an exciting life elixir. I think it is not true to say that parting with beloved things makes the heart grow fonder; that is, I suppose, only true with regard to people, as e.g. our colleagues.

First question of the mirror's owner to the mirrored picture: Do you really leave your place with pleasure? Answer: Yes, definitely! A few years ago he would still have answered with the ambiguity of the Rhenish peasant – with the greatest pleasure, if I must. To be able to free oneself is, I presume, in the whole nature, sign of increasing maturity. He who has not worked enough by the age of 68 is not likely to add anything essential through his later achievements.

Second question to the picture in the mirror: Do you really believe that you are irreplaceable? Grin of relief and the firm and earnest answer: my dear friend, if the youngsters do not become better than wie older ones, we shall have failed pitifully. And I am glad to be able to attest that there are so many pupils who more and more surpass their master. After these silly questions the mirror's owner turned away from his likeness and thought on his life's dearest hobby: Neurosurgery.

I have seen the time when the neurologist alone diagnosed the seat of a brain tumour, or perhaps not, and when he showed the general surgeon the place where the tumour was, or was not. The latter then tried to peel it out of the brain with his finger as he would have done with a prostatic adenoma. As I became more and more acquainted with contrast-medium diagnosis I was unorthodox enough towards my love of neurology so as to consider the following statement, attributed to the Prague neurologist Gamper, as an expression of the resignation of the neurologists: "Tumours are much too coarse formations to be detected by the refined neurological methods." Soon I learned to reject as a prejudice, the demand of neurologists for "more neurology and less Iodipin" in spinal diagnosis. A completely spiritless machine which allows a quick, reliable, safe and painless diagnosis, such as the computerized tomograph, transforms clinical pathology into an epitheton ornans (i.e., a decorative extra) or, to put it bluntly, the reflex hammer is an archaic relic. A diagnostic method, however interesting it may be, must not be an end in itself. We all were enthusiastic about Kroll's "Syndromdoktrin" and believed in its validity. But I shall never forget the blow that this doctrine received when one of the authorities in clinical brain pathology refused to admit a tumor of the corpus callosum that was shown to him by Tönnis at surgery: "It cannot be there, it does not fit into the symptomatology".

What a blessing to have grown up in a school in which the aim of therapy is the healing of the patient, irrespective of the method used. Our surgical technique must be as sparing, as quick, and as radical as possible. In this respect neurologists performing neurosurgical operation differ from neurological surgeons. Sparing means cautious and not hesitating or without decision. Nowadays the surgeon is made cautious by the microscope through which he sees much better what can be destroyed by him and what broad movements he makes. Today speed has no longer the importance it used to have in procedures performed under local anesthesia. But slowness is not synonimous of precaution. It is

usually due to lack of decision and aim against which the surgeon must firmly fight. Radical surgery has its limits where the patient's life begins to be in danger and additionally in the case of brain surgery where a radical operation would destroy the essential, i.e. the mental life of the patient.

Let me refer again to the subtitle of my lecture: Splendour and trials of a neurosurgeon. I would like to deal with splendour first.

*Iatros gar aner pollon antaxios allon,* Homer already said: "Doctor is a man that counts for more than many others". This is, of course, valid, above all for the best sort among the medical species: the neurosurgeon, who works at what is noblest in man, his brain. Let us be sincere: Whom of us does not delight himself in this bliss of having succesfully accomplished a major or minor operation, and of being able to say to himself: You have saved this patient's life or at least many years of it. Here I cannot help recalling a dictum of the great Billroth: "Only the simpletons are modest". That may sound hard to us today, but a surgeon who is not convinced of his worth cannot endure the daily toil. And why should he not be proud of it when he has done something particulary well or has done it for the first time, or, even, has done something new. Only, he should not think that his progress corresponds to the progress of all neurosurgeons in the world, and that all this has an eternal value. He even must wish that this progress may soon be surpassed. Thus, the most valuable results of his work are his pupils, of whom he therefore may be particulary proud, mainly when they belong to the elite of the neurosurgeons of this country.

Having said this, we may leave the "splendours" and turn to the larger and less agreeable chapter of "trials" of a surgeon's life. Defeats are always painful, even when expected, and when the best has been done. "Ultra posse nemo obligatur" – nobody can be challenged beyond his faculties – the Romans said, rightly and with insight into human nature. Only, all such maxims are equivocal. Before a neurosurgeon can appeal to this dictum he must ask himself whether he has done enough, and that always anew, to be at the height of his abilities, or whether he has undertaken too little both intellectually and technically prior to starting a procedure. He must also ask himself whether he kept enough self-command during it, whether he had sufficient control of his emotions, whether he made false decisions, whether he was sufficiently prepared, also physically, for instance due to those prolonged festivities the night before, etc. I do not want to enlarge on this. Whoever has operated on more than one brain tumour has probed his conscience at least occasionally, and is not completely possessed by the greatest vice of all, spiritual pride. He will not be able to acquit himself altogether and he will be well advised to keep this in mind when remembering the dead. The sentence about this will have to be pronounced to oneself privately. I am sure that such a probing of one's professional conscience is practised by the surgeons, those among all doctors who certainly need it most, more often than the layman would expect. In my youth it was an admired feature of the great surgeon that he gave way to his emotions at the operation table. The young assistant accepted this, as all weaknesses, with special pleasure. Today silence reigns in the modern operating theatres, among the real masters of the scapel. The reasons are various: The great demand for sterility, the concentrated work, especially in the teams where members have equal rights and cannot be treated as the little surgical nurse or the young assistant used to be. The reasons for controlled behaviour lie in simple humane education. But, who still believes in

the pedagogic value of self-discipline? Who realizes, for example, that showing one's feelings stirs the emotions whereas self-discipline suppresses their rise? To exemplify this should also be part of the training in the operating room, even if the temperamental master does not always succeed in it, just as he also surely does not work without making mistakes. That one makes mistakes should not lead the greatest "demi-god in white" into disgrace. That has always been so and will remain so.

That we are confronted with a wave of legal proceedings for liabilities has many reasons. We should certainly not gloss over real mistakes that occur, also in neurosurgery. It is, however, more and more obvious that the strongest reason for such law-suits is the artificially fostered envy in our present-day society, here as well as everywhere. One has given it a polite name ("Sozialkritik") as a palliative for the naive and stupid. I have no desire, however, to dwell further on that.

Every surgeon is given a comparatively wide scope for his indications. One has tried again and again to pose general rules.

In spite of this, the individual limitation remain large. It also depends on the personality of the surgeon, his character, his temperament, his training, his sense of responsibility and, as some believe, also on his smartness, which, in my view, is a bad counsellor. It is easy to be smart if one endangers other people. Indications that are influenced by non-medical motives, e. g., money, can certainly be a great temptation, especially today, in a world that is growing more and more materialistic. The neurosurgeons, with theire extensive and dangerous operations are, I suppose, less endangered in this respect. But does this ethos not waver easily when medical renown is at stake? Must we not now and then recall to our minds that medical curiosity alone is not sufficient for an indication? Neither true research nor the longing for scientific fame can be a justification for degrading man to a guinea-pig. My friends, we must not belittle this problem; with every implanted probe we must keep in mind not only the principle of "nil nocere" but also the question to whom it is of use.

The decision to operate is particularly difficult with tumours of the brain that have already been diagnosed as malignant. One often hears the opinion that one should not operate on such cases. I think one cannot always maintain this view. One is bound to take into consideration the patient's chance of a change for the better in the short or the long run. Nor can one in general exclude all metastases from an operation. Of course it does not make sense to operate on the brain in the case of an inoperable primary cancer and numerous metastases. But a suspected monocular metastasis, which is not at all rare, can indeed be worth operating on following the extirpation of the primary tumor. I shall never forget a young lady doctor who reported to me that a choreo-epithelioma had been apparently radically extirpated some weeks prior, and that she now had a metastasis in the left occipital lobe. She had brought with her the arteriogram and a neurosurgical report saying the tumor was inoperable. I was really of the same opinion, but I did not have the heart to confirm to the colleague sitting in front of me, that she was doomed. I told her, I did not agree, and that one should, for God's sake, have a try. As it happened, it was the only metastasis I was able to extirpate in toto in 1950. A few days ago, after 28 years, I received the latest confirmation of this in a letter, reporting that she was still well-off. Such a case compensates for many others in which one has tried in vain. This also applies to metastases of the spinal cord operated on at an early stage, in which a patient who is

doomed to being bedridden and badly paralysed for many months by a transverse lesion of the cord can be saved.

Thus, one should not say that one should never try. In neurosurgery also it is, in the last instance, the personal fate of the individual that counts. This also applies in particular to the heavy cross all neurosurgeons have to bear, the glioblastoma, and to similar tumors. Apart from the case of centrally seated tumours, the decision to operate can only depend on the question of whether eventually there will remain a patient who can still be considered a human being, or one that merely vegetates. A patient can live with a paralysis, but not with a grave psychic brain damage, with aphasia, agnosia, and pronounced changes in his intrinisic nature. To live six or eight months, or a year longer may be both subjectively desirable and objectively valuable. May not subspecies aeternitatis, a man who is facing death, attain his spiritual fulfilment only then? Of course, the spiritual strength of the patient as well as that of his helpers and friends are of need for this. What we should beware of, however, in such cases, is to try and attain medical perfection which, in the last instance, does not achieve anything, at least at present. That means we should beware of instant X-ray treatment, cytostatic drugs, etc., with which we can spoil the patient's happy month after surgery. We therefore irradiate only recurrencies. But to prolong a small child's life by a few months usually only amounts to prolonging its suffering. Our efforts to operate on all tumors as radically a possible, end where the human being as such reaches its limits, not to mention the fact that every effort of forced radicality precludes success. The neurosurgeon had better rest content with less in this case.

It is a blessing that the neurosurgeons also have to deal with less difficult cases, especially for the young colleagues who, thus, can be trained more easily. The young surgeon, of course, has the same feeling that we all had when we began: one is not allowed to do anything oneself early enough. Only the trouble is that once he has the scalpel in his hand by himself, nobody can help him, at least not in the critical situation. It is a heavy burden for the senior surgeon responsible having to tell himself: if only you had done it yourself. One does not fully realize this until one has the ultimate responsibility of a senior surgeon oneself. Youthful recklessness is a feature of the grand old surgeon only in the movies, where he then makes a good figure. His surgeon's soul grows, however, more vulnerable the older he becomes. Contrary to popular opinion, losses and defeats on the operation table hurt more the older one gets. However, to be quite frank – as promised – I am not sure wheter and to what extent it is hurt pride, "that such a thing could have happened to him", the old experienced silly ass.

May I add a word on the often inhuman way people have to die in clinics. Gottfried Benn, who was a doctor, once said: "Life in a hospital is bitter and one dies there without a vine-wreath in one's hair". What the poet wants to express by this metaphor is what relatives and friends often observe and consider undignifying. This applies in particular to our modern intensive care unit. Neurosurgery can be proud of having been the first operative discipline to install intensive care wards long before there were anesthetists. Certainly visitors must be kept out of the intensive care units for good reasons, such as to inadequate asepsis, a restful atmosphere, and undisturbed work. But this isolation only makes sense as long as there is hope for the patient to be cured. When it becomes obvious that all medical activity has become in vain, either the patient should be transferred or the

room should be opened to visitors, so that the dying man does not spend his last hours alone without family and friends. Medical equipment of the most modern perfection should, of course, be part of the outfit of an intensive care unit. But the extent to what it must be used in a particular case, i.e. a perspective of success, is a serious decision, which the surgeon must make. Scientific medicine is only one aspect of our therapy, and it does not take into account the entire patient. When death approaches, the human aspect of medicine must take the lead. There is no doubt that here much is left to be desired in our clinics. Thank God that death is merciful with most neurosurgical patients. Often kind nature protects them with the cloak of unconsciousness. But these patients must not simply perish in a lonely room either. The next of kin have, in my view, not only the right but also the duty to be present, and it is only to the good of the visitor to stand by someone who is dying. In this context allowed me to say also a few words about euthanasia. Euthanasia is no serious medical problem. It is the doctor's duty to help. He is allowed to stop treatment when he cannot help any longer, he even ought to do so. This is not, as people sometimes say mistakenly, passive euthanasia. Man has the right to a natural death. A doctor cannot be called upon to kill old people whose lives have become useless, or to eradicate people unfit for life. What is usually asked for, by the way, is not that the patient have a merciful death, but that the relatives may get rid of the dying patient. In my long career it happened once that a patient, who was suffering from a recurrent tumour, genuinely asked me to relieve him from his sufferings. On the other hand, many relatives have approached me to say that the sufferings of the patient were so great that one ought to put an end to them.

Ladies and gentleman, I would very much have liked to say something about what attracted me to neurology and later to neurosurgery fourty-five years ago. It was the relationship between the brain and the soul. This might have been quite an appropriate subject here in Berlin, where the Frenchman Lamettrie, who was in the service of Frederic the Great, died in 1751. He declared, as you know, that the brain produces the thoughts as the gland the saps. The modern equation "brain = computer" does not appear to me much better either. Contemporary quantum physicist have proven, for the first time, through the radioactive decay, that a true indetermination and no causality prevails in this process. Max PLANCK said: "An event is causal, if it can be predicted with certainty. The lack of the principle of causality must also be postulated for the other end of the cosmos, i.e. the mind. Our thoughts can be predicted as little as the courses of the ions." Thus, to me, the best, perhaps too metaphysical, notion of the relationship between the brain and the mind is that of my neurology teacher and friend LAUBENTHAL, who follows BUMKE's notion that we are still moving in the forecourt of the soul with our psychopathological ideas about the function of the brain. According to him, the human mind plays on the brain like the organ-player on his instrument. Each year we become more acquainted with the instrument, i.e. the pipes, and of course, also with the air producing the sounds there in. Of the player, however, we do not know more than Plato or other thinkers, not to mention the composer of sonorous music who has surely composed many more cantatas, sonatas, and even entire symphonies, which have never yet come out of the organ-pipes. As I said, this is only a metaphor. The neurosurgeon, who is for the greater part an artisan, lacks the philosophical training which, perhaps, he ought to have. But, to come to an end: what in the world should the ideal neurosurgeon be: an impeccable diagnostician, an infallible operator, a good-natured teacher who never explodes and always thinks of the well-being of his pupil, an excellent scientist, and what

all, in order to be a good doctor? Although you have conferred the medal to me, all who know me will now think that also as far as I am concerned there are some spots here and there in this ideal picture. My answer: the ideal picture always hangs so high that one does not reach it. It should be looked at as an indication of one's aim as long as one is young; it should be talked about as I have done just now when one has grown old and realizes that one has not succeeded in attaining it.

I thank you for having listened to me so patiently.

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# Treatment of Hydrocephalus

# Complications Following Ventriculo-Atrial Shunts in Hydrocephalus

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The following study was initiated by Prof. WÜLLENWEBER (Berlin). Nine West German neurosurgical clinics participated in this collective study. We would like to thank all collegues who helped gather the necessary data for this study. Assuming that surgical techniques and shunt systems have reached a certain standard, we concentrated on the most essential problems and limited our study to those patients who underwent their first shunt operation between 1970 and 1976.

We had the oppurtunity to examine the complications of a total of 1,612 patients provided with ventriculoatrial or ventriculoperitoneal shunt systems, regardless of their basic disease (Table 1).

Table 2 demonstrates the number of patients needing revision of their shunt systems. Forty percent of the total group examined (643 patients), underwent revision surgery. The total of 1,019 revisions were subdivided as follows:
ventricular catheter 37%, flushing device or valve, 11%, atrial catheter, 42%, and peritoneal catheter, 10%. In other words, the outflow of the shunt system caused 52% of all complications. It should be noted that revision rates at different clinics ranged from 28% to 56%. In most cases (56%) only one revision was necessary. Two revisions were needed by 28%, 10% needed three, and 6% needed four or more revisions. The latter group included individual cases known to have an extremely high rate of complications (Table 3).

Table 1. Total number of patients treated with ventriculoatrial shunt (1970 - 1976), N = 1612

| From neurosurgical clinics in: | |
|---|---|
| - Düsseldorf | - Hannover |
| - Erlangen | - Heidelberg |
| - Essen | - Köln |
| - Giessen | - Würzburg |
| - Günsburg | |

Table 2. Number of patients with shunt revisions: 643 (40% of 1612)

| Number of shunt revisions: | | |
|---|---|---|
| Ventricular catheter | 380 | (37%) |
| Flushing device or valve | 111 | (11%) |
| Atrial catheter (AC) | 430 | (42%) |
| Peritoneal catheter (PC) | 98 | (10%) |
| Total | 1019 | (100%) |

Table 3. Number of revisions per patient

| No. of revisions | Patients | |
|---|---|---|
| 1 | 360 | (56%) |
| 2 | 180 | (28%) |
| 3 | 63 | (10%) |
| 4 or more | 40 | (6%) |
| Total | 643 | (100%) |

Our study included 1,455 patients, who had been provided with an atrial catheter at their first shunt operation. Thirteen had their initial peritoneal catheter replaced by an atrial catheter. Thus, a total of 1,468 patients were provided with atrial catheters. Of these patients, 24% needed a total of 430 revision operations, resulting in an average of 1.24 revisions per patient (Table 4).

Table 5 shows the time interval between surgery and the occurence of the first complications. It is interesting to note the wide distribution of complication-free intervals ranging from 1 day to 6 years and probably more. The greatest number of complications, namely 48%, occur within the first year after operation, while a steady decrease of the complication rate is observed after the first year. The high rate of complications towards the end of the first year after operation indicates that revision operations caused by the atrial catheter being too short naturally depend on the age of the patient. Other correlations between the age of our patients and the onset or type of complication were not evident (Table 6).

Table 4. Number of patients with atrial catheter (AC)

| | |
|---|---|
| Primary insertion of AC | 1455 |
| Replacement of peritoneal catheter by AC | 13 |
| Total | 1468 |

Of these patients 348 (24%) needed 430 AC-revisions (I.E. 1.24 REV/PT)

Table 5. Atrial catheters. Time interval until occurrence of complications

| | | |
|---|---|---|
| Up to 2 weeks | 37 | |
| Up to 1 month | 29 | |
| Up to 6 months | 74 | (48%) |
| Up to 1 year | 67 | |
| Up to 2 years | 87 | (20%) |
| Up to 4 years | 77 | (18%) |
| Up to 6 years | 43 | (10%) |
| More than 6 years | 16 | (4%) |
| Total | 430 | |

Table 6. Time of initial shunt operation

| Age (years) | No. of patients | % |
|---|---|---|
| 0- 1 | 534 | 83 |
| 1- 2 | 26 | 4 |
| 3- 5 | 20 | 3 |
| 6-10 | 18 | 3 |
| 11-20 | 13 | 2 |
| Over 20 | 32 | 5 |
| Total | 643 | 100 |

The most common cause observed in 49% of the cases was that the catheter was relatively too short due to growth of the patient. At this point it should be mentioned that quite often, when x-ray findings showed an extra-atrial position of the properly functioning catheter, the catheter was replaced by a longer one. Thrombotic occlusion was the second most common cause, in 22% of the cases, which was often combined with the complication just mentioned. Infection occurred in only 9% of the patients. These 42 infections included 27 cases of septicaemia, 5 cases of meningitis and 10 cases of local wound infection.

Twenty-two atrial catheters had not been inserted far enough into the right atrium, so that early revision became necessary; 19 catheters disconnected. Of these, two slipped into the heart and had to be removed by thoracotomy.

Other common causes for revision were: wrong pressure range of shunt system, kinking of the catheter, and CSF-fistula. We found no connection between the different basic diseases of the patients and the type of complication. Only 72 of the 430 patients had been provided with a Spitz-Holter and only 18 with a Hakim system. Insofar as these data allow any conclusion, it can be said that the different types of complications are not favored by certain catheter models (Table 7).

Table 8 illustrates the outcome of 430 operations performed on 348 patients who developed complications after their first operation. No further surgical intervention was needed in 67%, whereas 17% needed further revision. In 13 cases the patient's condition deteriorated even though the shunt system functioned properly, according to clinical aspects. Of the patients, 22 died, 9 of them due to complications with their shunt system.

Table 7. Atrial catheters (AC) cause of revision

| | | |
|---|---|---|
| AC too short due to growth | 225 | (49%) |
| Thrombotic occlusion | 102 | (22%) |
| Infection | 42 | (9%) |
| Initial AC too short | 22 | (5%) |
| Disconnection of AC | 19 | (4%) |
| Opening pressure too high | 12 | (3%) |
| Kinking of AC | 10 | (2%) |
| Opening pressure too low | 6 | (1%) |
| CSF-fistula | 5 | (1%) |
| Other | 19 | (4%) |
| Total | 462 | |

Table 8. Atrial catheters
Treatment results of revision operation

| | | |
|---|---|---|
| Satisfactory | 287 | (67%) |
| Further revision necessary | 72 | (17%) |
| Deterioration of patients condition despite intact shunt | 13 | (3%) |
| Death due to complication | 9 | (2%) |
| Death due to other causes | 13 | (3%) |
| No information | 36 | (8%) |
| Total | 430 | |


## Summary

In conclusion it may be stated that the atrial catheter complication rate of 24%, observed in this study, coincides with values published by other authors. The extremely low infection rate of only 3% in connection with atrial catheters is quite astonishing, even though neither of the clinics in Düsseldorf or Essen, providing the majority of patients for this study, give antibiotics prophylactically. Finally I would like to thank Mr. LANGE from the Essen department of Biomathematics.


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# Complications Following Ventriculo-Peritoneal Shunts

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While evaluating the data obtained from the neurosurgical clinics in Düsseldorf, Erlangen, Essen, Giessen, Günzburg, Hannover, Heidelberg, Köln and Würzburg we were a little astonished to find that of the 1,612 patients in this study only 230 had been provided with a peritoneal catheter. With a few exceptions these catheters were used only in three participating clinics. Perhaps one could conclude a certain reservation about using peritoneal shunt systems.

Of the 230 patients, 157 had been initially provided with a peritoneal catheter, were as the remaining 73 received a peritoneal catheter as a replacement for their atrial one. Of these 230 patients, 71 (31%) needed a revision of their catheter, 7% more revisions in comparison with atrial catheters. Altogether, 98 revisions were performed on 71 of these patients, an average of 1.38 revisions per patient (Table 1).

In most cases (41%) there was no special indication for the insertion of the peritoneal catheter. Often the lack of access to a vein leading to the heart was the reason for using a peritoneal instead of an atrial catheter, mainly during atrial catheter revisions. Furthermore, cardiac disease, septicaemia, and superficial skin disease of the neck are listed. Other reasons included patients with tracheostoma or patients from whom no detailed information was available (Table 2).

Table 3 illustrates the time interval until occurrence of complications with perioneal catheters. Most of the complications occurred within the first year.

Table 1. Number of patients with peritoneal catheter (PC)

| | |
|---|---|
| Primary insertion of PC | 157 |
| Replacement of atrial catheter by PC | 73 |
| Total | 230 |

Of these patients 71 (31%) needed 98 PC revisions (I.E. 1.38 REV/PT).

Table 2. Indication for use of peritoneal catheter

| | | |
|---|---|---|
| No special indication | 29 | (41%) |
| No venous access | 18 | (25%) |
| Cardiac disease | 4 | (6%) |
| Septicaemia | 3 | (4%) |
| Superficial skin disease of neck | 3 | (4%) |
| Other | 14 | (20%) |
| Total | 71 | (100%) |

1 For evaluation of the date we are indebted to S. LANGE (Institut für Medizinische Informatik und Biomathematik der Gesamthochschule Essen).

Table 3. Peritoneal catheters. Time interval until occurrence of complications

| | | |
|---|---|---|
| Up to 2 weeks | 22 | |
| Up to 1 month | 14 | (77%) |
| Up to 6 months | 23 | |
| Up to 1 year | 16 | |
| Up to 2 years | 12 | (12%) |
| Up to 4 years | 11 | (11%) |
| More than 4 years | 0 | |
| Total | 98 | |

In the same time interval only 48% of the patients with an atrial catheter showed complications and nearly 60% of the complications occur within the first six months. This means that complications evidently occur earlier with peritoneal than with atrial catheters.

It is not possible to make any precise statements about the complication-free period in comparison with the age of the patients, because most of them (89%) were not older than one year at the time of their first shunt operation.

Table 4 shows the reasons for revision of peritoneal catheters. The most common cause for such a revision was infection, which occurred in 20% of the cases.

These 23 infections included 7 cases of meningitis, 6 cases of septicaemia, 5 cases of local wound infection, 3 infections persisting in the shunt system itself, and 2 cases of peritonitis. Disconnections of the peritoneal catheter follow with 19%. Six of these 21 patients required a laporotomy in order to remove the detached catheter which had slipped into the peritoneal cavity. Other causes of revision were peritoneal adhesions and malabsorption of the CSF (12%), obstruction of the catheter (11%), subcutaneous accumulation of CSF (7%), and kinking of the catheter (6%). It should be emphasized that only 6% of the patients had a shortening of the catheter due to growth of the patient which necessitated a revision. This complication, on the other hand, was the most frequent cause for atrial catheter revision. With few exceptions, all the shunt systems used in the clinics have an opening pressure of 5-10 cm $H_2O$. While in 5% of the patients with complications the opening pressure of the shunt system was too high, thus not providing sufficient drainage, no cases of low-pressure syndrome or subdural hematoma were reported. Therefore, the hydrostatic pressure in the longer tube of the

Table 4. Peritoneal catheters (PC) - cause of revision

| | | |
|---|---|---|
| Infection | 23 | (20%) |
| Disconnection of PC | 21 | (19%) |
| Peritoneal adhesion and malabsorption of CSF | 14 | (12%) |
| Obstruction of PC | 12 | (11%) |
| Subcutaneous accumulation of CSF | 8 | (7%) |
| Kinking of PC | 7 | (6%) |
| PC too short | 7 | (6%) |
| CSF fistula | 5 | (4%) |
| Opening pressure too high | 5 | (4%) |
| Other | 11 | (10%) |
| Total | 113 | |

peritoneal catheter does not seem to be so important. It was not possible to compare peritoneal catheters of different shunt systems because the clinics participating in this study primarily used Pudenz-Heyer systems, whereas only 6 Hakim and 2 Holter catheters were inserted. When comparing the different types of catheters marketed together with the Pudenz system, obvious differences were apparent with regard to one type of complication.

Table 5 shows that a detachment of the catheter is more common with the Raimondi spinal type than with the Mathews and Pudenz catheters which had a disconnection rate of 6%. Conversely with the Raimondi spiral catheters, kinking occurred in only 4% while the other types showed 11% such complications.

In our experience with the silicone rubber of the Raimondi catheter, we found that it is not as elastic as that of the other types and tends to tear more easily. For this reason, we stopped using Raimondi catheters and since then have not observed such complications.

Different placement of the catheter in the abdomen had no influence on the type of complication nor on the time of onset of complications. We also found no connection between basic disease and type of complication in this patient group.

According to Table 6, 66% of the patients needed no further surgical intervention, whereas further revisions were necessary in 27 cases. Most of these patients had revisions due to infections, peritoneal adhesions and malabsorption of CSF. Two patients died of these complications, while at the death of one patient was caused by reasons other than the shunt operation.

We can conclude that the complication rate of 40% observed in this study coincides with values published by other authors. The somewhat higher frequency of peritoneal catheter complications in comparison with atrial catheters (in our study 31%-24%) has also been described by others. The higher rate of complications with peritoneal catheters, especially frequent infections, seems to confirm the reservation towards use of peritoneal catheters. We must mention, however, that the

Table 5

| | Raimondi spiral (No. = 68) | Mathews and Pudenz (No. = 36) |
|---|---|---|
| Detachment of PC | 17 (25%) | 2 (6%) |
| Kinking of PC | 3 (4%) | 4 (11%) |

Table 6. Peritoneal catheters. Treatment results of revision operation

| | |
|---|---|
| Satisfactory | 66 |
| Further revision necessary | 27 |
| Death due to complication | 2 |
| Death due to other causes | 1 |
| No information | 2 |
| Total | 98 |

peritoneal catheters, which this data refers to, have been in use for only a short time and we believe from our own experience that many complications can be attributed to the initial lack of experience in surgical technique. Probably the number of complications will decrease with increasing experience with this type of catheter.

In our opinion, shortening of the catheter due to growth of the patient will also lead to far fewer revisions than are necessary with atrial catheters. Therefore, despite the impression of a higher rate of severe complications found in this study, we believe that the drainage of CSF into the peritoneal cavity is an acceptable alternative to cardiac drainage.

# Complications Following Ventriculo-Cisternal Shunts

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Ventriculo-cisternostomy, introduced by TORKILDSEN in 1939 (8) for the treatment of occlusive hydrocephalus, has been used as a final treatment in patients with nontumorous occlusive hydrocephalus and as a palliative operation in cases with surgically inaccessible deep-seated tumors producing occlusion of CSF pathways within the ventricular system. Since the introduction of ventriculo-atrial and ventriculo-peritoneal shunts, particularly since the beginning of the 1960's, the number of Torkildsen operations performed in neurosurgical centers has diminished and some neurosurgeons have almost completely abandoned the procedure. Major reviews dealing with indications and results were reported by GERLACH, who analysed 931 patients operated on in the Departments of Neurosurgery in Germany till 1960 (1), GRÖSCHEL and MARGUTH, who reported on 155 patients operated on between 1951 and 1959 (2), and LANG and PIA (3). Complications of the procedure were reviewed by LORENZ in 1963 (4).

The aim of this paper is to present an analysis of the complications of ventriculo-cisternostomy.

## Material and Method

Two hundred and thirty nine cases were collected from the Department of Neurosurgery in Giessen, Erlangen and Hannover. The type of hydrocephalus, preoperative diagnostic measures, and technical aspects of the procedure were correlated with the incidence of intra- and postoperative complications and function of the shunt. Patients operated on up to 1961 were listed separately.

## Results

As shown in Table 1, 37.2% of the patients with Torkildsen shunts had occlusive hydrocephalus due to tumors in the anterior or posterior part of the third ventricle; 12.1% suffered from tumors of the upper brain-stem; 18.8% had tumors of the fourth ventricle; and 19.1% were operated upon because of nontumorous aqueduct stenosis or occlusion. Over the last 15 years hydrocephalus produced by tumors of the basal ganglia and lateral ventricles became a less frequent indication for this procedure (a decrease from 16% to 2.5%), whereas the percentage of patients with tumors occluding the third ventricle, who underwent Torkildsen operations increased from 28.3% to 41.7%).

A Torkildsen shunt was performed in 78.8% of the patients as the only operative procedure and in 21.2% of patients as an additional procedure supplementing the removal of tumor. The shunt was implanted on one side in 58.2% and bilaterally in 41.8% of the cases. Bilateral insertion of the shunt was performed in patients with tumors involving the anterior part of the third ventricle or basal ganglia leading to obstruction of one or both the foramina of Monroe.

Table 1. Indication for Torkildsen operation. The total material (*middle column*) is divided into an early group 1953-1961 (*first column*) and late group 1962-1977 (*last column*)

| Indication | 1953-1961 LANG and PIA series N = 81 % | 1953-1977 N = 239 % | 1962-1977 N = 158 % |
|---|---|---|---|
| Aqueduct stenosis (nontumorous) | 3.7 | 7.9 | 10.1 |
| Aqueduct occlusion (nontumorous) | 14.8 | 11.2 | 9.4 |
| Basal ganglia and lateral ventricle tumors | 16.0 | 7.1 | 2.5 |
| IIIrd ventricle | 28.3 | 37.2 | 41.7 |
| Upper pontine tumors | 13.5 | 12.1 | 11.3 |
| IVth ventricle tumors | 22.2 | 18.8 | 17.0 |
| Others | 1.5 | 5.7 | 8.0 |

## Complications

Intraoperative complications occurred in a total of 12.6% of patients, mostly in the form of epidural or subdural hematoma or ventricular (Table 2). The overall percentage of postoperative complications is high, hemorrhage occurring in 43.6% of the patients.

The most frequent postoperative complication was meningitis and ventriculitis with bacteriologically confirmed infection. This was found in 14.9% of cases (Table 3), CSF-fistula was found in 12.1%, and local infection in 7.1%. Foreign body reaction developed in an insignificant number of cases. In two cases brain abscesses developed after prolonged infection. As seen in Table 3, the percentage of infectious complications decreased in the course of last 25 years. Of the total number of patients, 9.1% died because of complications.

Technical particulars of the procedure, such as the tube material used, fixation of the drain, and epidural or subgaleal placing of the catheter were considered in possible complications and proved to be of no great importance. There was also no difference in the complication rate between the uni- and bilateral shunts. However, two technical aspects of the procedure seem to be connected with the incidence of postoperative CSF-fistulas (Table 4):

Table 2. Intra- and postoperative complications of ventriculocisternostomy in percent

| | 1953-1961 N = 81 | 1953-1977 N = 239 | 1962-1977 N = 158 |
|---|---|---|---|
| Intraoperative | 8.7 | 12.6 | 14.6 |
| Postoperative | 55.4 | 43.6 | 38.0 |

Table 3. Postoperative complications of Torkildsen drainage in percent

| | 1955-1961 N = 81 | 1953-1977 N = 239 | 1962-1977 N = 158 |
|---|---|---|---|
| Local infections | 13.5 | 7.1 | 3.7 |
| CSF fistula | 22.1 | 12.1 | 8.1 |
| Meningitis and ventriculitis | 19.6 | 14.9 | 10.0 |
| Foreign body reaction | - | 0.8 | - |
| Death due to complications | 13.5 | 9.1 | 6.9 |

Table 4. CSF fistula and operative technique

| | No. of cases | CSF fistula No. of cases | % |
|---|---|---|---|
| Tube 3 cm in spinal canal | 29 | 1 | 3.4 |
| Tube in the cisterna magna | 108 | 14 | 12.9 |
| Midline incision | 95 | 13 | 13.6 |
| T-shaped incision | 90 | 6 | 6.6 |

1. The introduction of the spinal end of the tube for more than 3 cm into the spinal canal was connected with a markedly lower rate of CSF-fistulas (3.4%) than when the tube was placed into the cisterna magna (12.9%).
2. In 13 of 95 patients with midline incisions, CSF-fistula occurred, whereas this complication took place in only 6 of 90 patients with a T-shaped incision of the skin and muscles.

Early and late insufficiency of the ventriculo-cisternostomy observed clinically, at a second operation, in isotope or X-ray study, was found in 43 cases, that is, 18% (Table 5). The functioning of the shunt was not always studied in detail and therefore this figure may be higher. Twenty-one patients, 8.8% of the total number of cases, had a ventriculo-atrial or ventriculo-peritoneal shunt performed because of insufficient Torkildsen drainage.

The comparison of patients with nontumorous aqueduct stenosis with the group of patients with occlusive hydrocephalus of tumor origin showed that early failure of drainage function (up to 4 weeks) was observed in 26% of patients with aqueduct stenosis and in only 5.5% of the patients of the tumor group.

Table 5. Patency of the shunt in percent, N = 239

| | |
|---|---|
| Shunt insufficient: | |
| within 4 weeks | 6.6 |
| within 1 year | 3.7 |
| within 10 years | 4.1 |
| Time not precised | 3.6 |
| Total | 18.0 |

(Table 6) In spite of functioning drainage 11.2% of the patients continued to deteriorate and 19.6% died within 6 months because their disease progressed.

Postoperative lumbar punctures were performed in 70.3% of all the patients with the aim to remove the blood-stained CSF and to ensure the patency of the shunt. The frequency of lumbar punctures varied from 2 per week to daily. Persistent CSF-fistulas were treated by continous lumbar drainage. The analysis of the frequency of lumbar punctures with the incidence of CSF-fistulas showed however, that frequent lumbar punctures could not diminish the incidence of CSF-leak.

## Discussion

Previous reports (1, 2, 4, 5, 6) discussed mainly the indication and prognosis of patients with Torkildsen operation. Long term survivals and favorable results in patients with nontumorous aqueduct stenosis and in patients with slow growing tumors were reported by GERLACH (1). He considered Torkildsen-drainage a reliable shunt provided that the resorption of the CSF is assured. In the series of GRÖSCHEL and MARGUTH (2) 15% of patients developed infectious complications which were the cause of death in 3%. CSF-fistulas also occurred in 15% of patients. The overall early mortality in the series was 30%. In the largest series published so far, the mortality due to meningitis or CSF-fistula was 10.7%. LANG and PIA (3) and LORENZ (4) stressed the importance of a T-shaped incision of the skin and muscles to prevent the occurrence of postoperative CSF leak through the wound. This suggestion, as well as placing the tube not into the cisterna magna but more distally into the cervical spinal canal, should certainly be followed. CSF-fistula and meningitis were the main complications of the procedure. Our analysis showed that their incidence diminished considerably in the last years. Early insufficiency of the shunt, particularly in patients with nontumor aqueduct stenosis suggests that careful estimation of the absorption capabilities should be performed before a Torkildsen shunt is considered. Obviously, the high percentage of early insufficiency of the shunt in our series was due to this factor. On the other hand, this study seems to show that the late complications are rare and the risk of nonfunctioning of the Torkildsen shunt after 6 months or later is small.

There is still little information about the effect of Torkildsen drainage upon intracranial pressure. We were able to record intracranial pressure in 6 patients submitted to Torkildsen operation before and after the procedure. In two of them (both patients with occlusive hydrocephalus caused by tumor) dangerous rises of intraventricular pressure occurred in the immediate postoperative phase (Fig. 1a, b), whereas preoperatively, only a minimal pressure increase was recorded. In the second one, similar increase of pressure was observed, a moderate elevation persisting for days (Fig. 2a, b). We have no definite explana-

Table 6. Postoperative course in percent

| | 1953-1961 N = 81 | 1953-1977 N = 239 | 1962-1977 N = 158 |
|---|---|---|---|
| Deterioration in spite of functioning shunt | 11.1 | 11.2 | 11.3 |
| Death in spite of functioning shunt | 25.9 | 19.6 | 16.2 |

tion for this phenomena: either blockade of the shunt or diminished absorption may be responsible. Some so far unexplained deterioration followed Torkildsen operation might have been due to this secondary postoperative intracranial pressure increase.

## Conclusions

1. A relatively high rate of early complications is connected with Torkildsen operation (approximately 40%).
2. CSF-fistula and meningitis are the most frequent complications and occurred in 12.1% and 14.9% of cases, respectively. Because of the complications connected with the shunt procedure, 9.1% of patients died. The incidence of the both complications can be diminished by observing the principle of T-shaped skin and muscles incision as suggested by TÖNNIS, and placing the tube more distally into the cervical canal.
3. If a Torkildsen procedure is considered in patients with aqueduct stenosis, the study of CSF absorption should be done if early failure is to be avoided.
4. For patients with adequate absorption capabilities and patients with slow-growing tumors causing hydrocephalus, the Torkildsen shunt permits long survival without the necessity of the patient being subjected to revisions, as the incidence of late complications of the shunt is very small.

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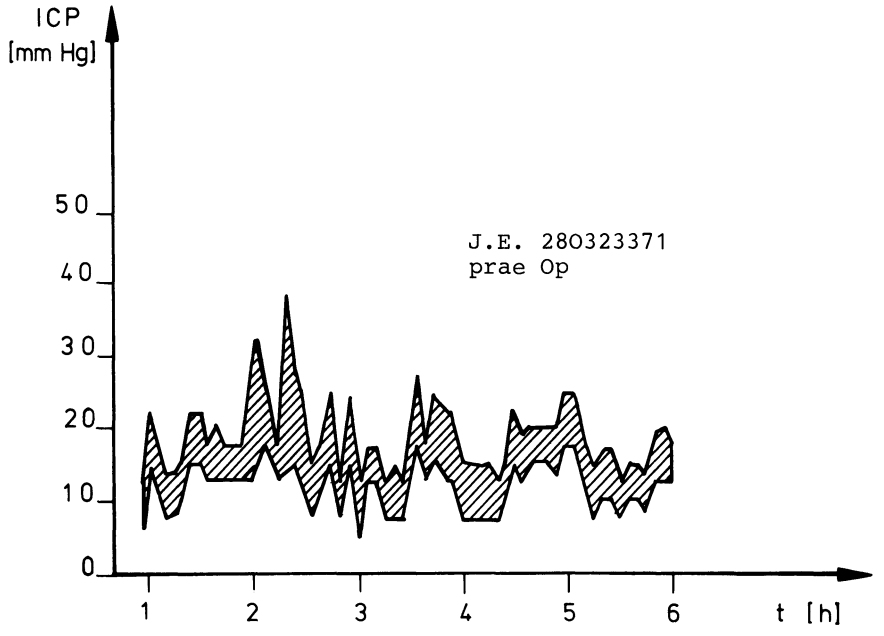


Fig. 1.a IVP recording in a patient with hydrocephalus and thalamic tumor. Pressure curve before the operation

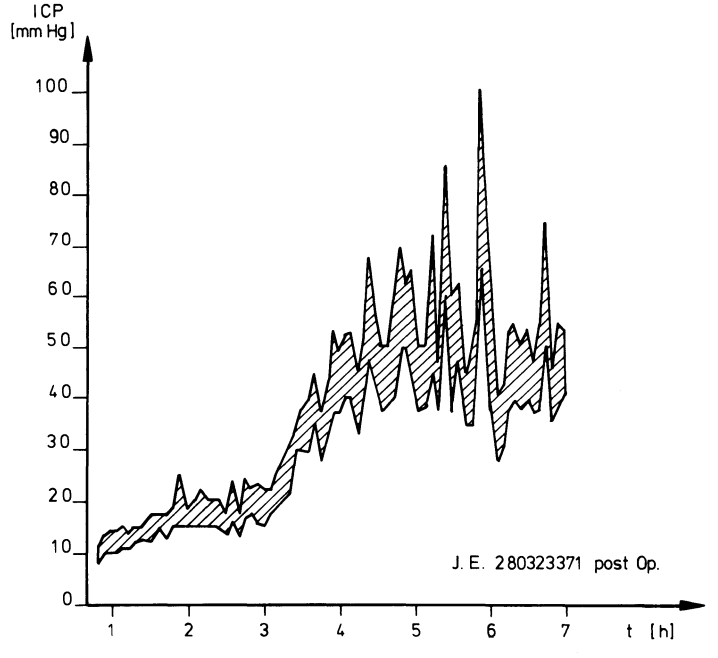


Fig. 1.b IVP recording immediately after Torkildsen operation. High intracranial pressure 4 hours after the operation. No clinical deterioration

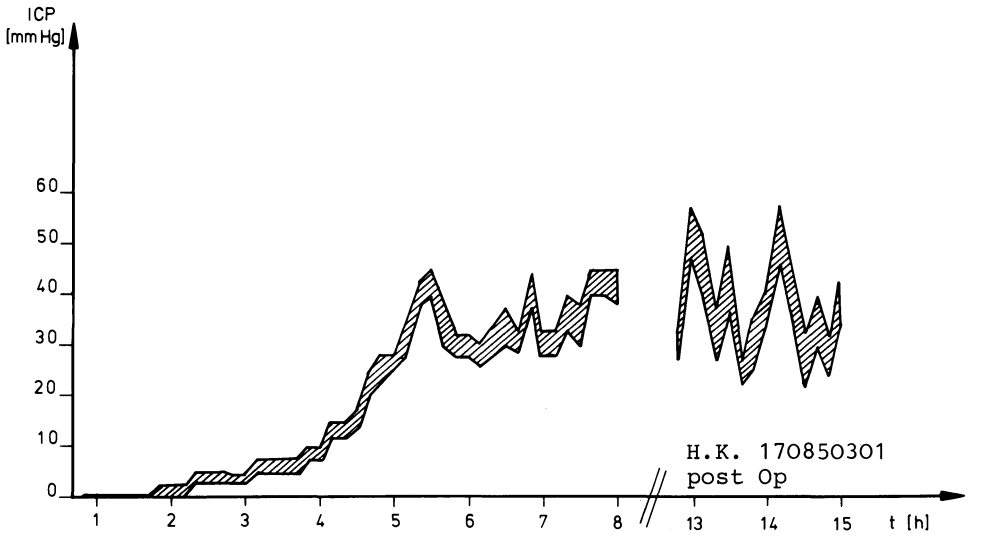


Fig. 2.a IVP recording after Torkildsen operation. Pressure increase 5 hours after the operation

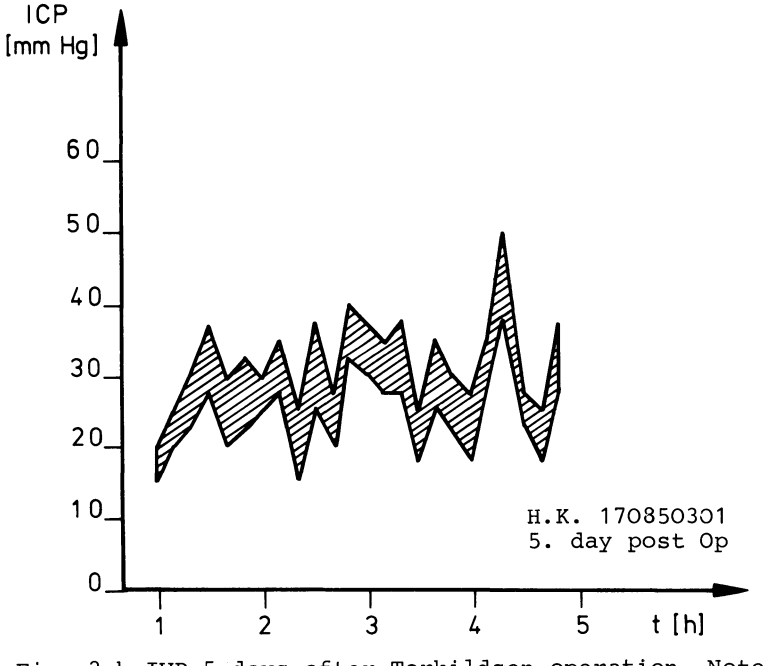


Fig. 2.b IVP 5 days after Torkildsen operation. Note persisting moderate elevation of pressure

# The Complications of Ventriculo-Atrial Shunting in Hydrocephalus

R. V. JEFFREYS and M. CHIR

## Introduction

Since the inception of neurosurgery, the treatment of uncontrolled hydrocephalus has presented considerable problems. Although many different procedures have been tried, it is true to say that at the present time the majority of neurosurgeons favour ventricular drainage to either the right atrium of the heart or the peritoneal cavity, through a variety of one-way valve systems. However, it has been apparent that these systems are far from perfect and a variety of complications have been reported (2, 4). The present author, in an attempt to cut down the incidence of complications, has developed a protocol for such patients and is reporting a retrospective personal series of 56 patients treated during the years 1975-1977.

## Clinical Material

Of the 56 patients, 19 were male and 37 female. The mean age for the group was 39 years in the range 8 months-79 years. The main diagnosis and cause for hydrocephalus for these patients is listed in Table 1. Direct neurosurgery was carried out in all ten patients with benign tumours and eight of the patients with malignant tumours as well as clipping of ruptured intracranial aneurysms in five patients with subarachnoid haemorrhage. In all patients, hydrocephalus was confirmed by either lumbar air encephalography, contrast ventriculography or more latterly, computerized axial tomography. Intracranial pressure monitoring by indwelling ventricular transducer was carried out in the 17 patients with pre-senile dementia and three of the other patients. 51 patients (91%) underwent some form of ventricular cannulation in the form of either ventricular drainage, ventriculography or intracranial pressure (I.C.P.) monitoring, and in only 5 patients (9%) was the ventricular system not explored prior to ventriculo-atrial shunting. Due to obstruction of either one or both foramina of Munro, a double ventricular catheter, attached to a common atrial catheter shunt was inserted in four operations (7%).

Table 1. Primary diagnosis in patients with v.a. shunts

| | Number | Alive | Dead |
|---|---|---|---|
| Presensile dementia | 17 | 16 | 1 |
| Benign tumours | 10 | 7 | 3 |
| Malignant tumours | 15 | 7 | 8 |
| Ruptured aneurysm | 5 | 4 | 1 |
| Aqueduct stenosis | 3 | 3 | 0 |
| Meningitis | 3 | 3 | 0 |
| Head injury | 2 | 1 | 1 |
| Sarcoid | 1 | 0 | 1 |
| Total | 56 (100%) | 41 (73%) | 15 (27%) |

## Operative Technique

In all patients a Pudenz-Heyer ventriculo-atrial shunt was inserted with the exception of the first two patients in whom a Spitz Holter system was inserted. All operations were performed under general anaesthesia. Through a high right hemi-thyroid incision, either the common facial vein or the internal jugular vein were cannulated. An atrial catheter with an opening pressure checked at the time of operation of 5-8 mms. of mercury was then advanced under electro-cardiographic (E.C.G.) control and a final position accepted where the tall bifid P wave had shrunk to just above the iso-electric line. This position was confirmed radiographically as lying within the body of the right atrium just above and to the right of the tricuspid valve. A 'J' shaped scalp incision was then made just above and behind the right ear and the scalp turned forwards. A semi-circular pericranial flap was then cut and also turned towards and the ventricular catheter and reservoir then inserted and connected to the atrial catheter. The pericranial flap was sutured over the top of the reservoir. Throughout the operation, all tubing was kept clamped and spillage of C.S.F. was kept to a minimum of 3-5 ml. In a small number of patients suffering from pseudo-meningocele following posterior fossa exploration and in whom the ventricular system was relatively small, the ventricular catheter and reservoir were then inserted through a right frontal approach into the right frontal horn of the lateral ventricle. Post-operatively, the patients were kept flat with one pillow for three to four days and then gradually mobilised over the next four days, before finally becoming ambulant or allowed to sit up in bed. Since the system inserted was considered to be self-regulating, no external pumping of the reservour was carried out.

## Results

Of the 56 patients, 42 remained alive and without shunt complications for at least the minimum period of 4 months. Fourteen patients died, 13 due to continuing primary intracranial disease and only 1 as a result of a shunt complication. Shunt complications developed in 11 patients (20%). The complications were as follows (Table 2):

1. *Infection.* There was one case of primary infection of a shunt occuring within 3 months of operation. This child had an inoperable brain stem glioma and steadily deteriorated and died within 2 months of surgery. In three patients (5%) there was delayed infection. One case was due to skin erosion over the reservoir and one case due to

Table 2. Complications in 56 patients with v.a. shunt

| | | Numbers |
|---|---|---|
| Infection | (a) early | 1 (2%) |
| | (b) late | 3 (5%) |
| Intracranial haemorrhage | | 1 (2%) |
| Blocked shunt | | 4 (7%) |
| Skin erosion | | 3 (5%) |
| Aerocele | | 1 (2%) |
| Epilepsy | | 1 (2%) |
| Total = 14 complications in 11 patients | | |

an aerocele allowing bacteria into the C.S.F. pathways. The third case developed bacteraemia with colonisation of the atrial catheter, following a severe chest infection 18 months after the insertion of a shunt.

2. *Intracranial Haemorrhage*. The same patient who developed an aerocele and a delayed C.S.F. infection also developed an extradural haematoma during the first 24 h after shunt insertion. There were no other cases of either extra- or subdural haematoma formation.

3. *Shunt Blockage*. Four patients (7%) developed blockage of the shunt system necessitating revision. Three complications were due to an upper end block and one to a lower end block. All were successfully revised.

4. *Skin Erosion*. Three patients (5%) developed skin erosions over their shunts. Two of these patients were the first in the series in whom Spitz Holter valves had been inserted. Both patients were cachectic and it was felt that this was the main contributing factor, as well as the rather bulky nature of the Holter valve. The third patient with a right frontal shunt developed erosion and this was directly attributed to a pericranial flap which in this instance had not been placed over the reservoir. The first two patients were treated by removal of their Spitz Holter shunt and re-insertion of a Pudenz-Heyer system. In the third case, a plastic scalp operation was carried out to effect skin cover.

5. *Aerocele*. One patient with a malignant pineal tumour developed a spontaneous air ventriculogram following a minor head injury some six months after the insertion of a Pudenz-Heyer shunt. It was unfortunately impossible to trace the source of the leak and the patient went on to develop meningitis and became the only fatality directly attributable to a shunt.

6. *Epilepsy*. One patient who before insertion of a shunt had not suffered epilepsy, suffered a series of major seizures for 36 h after the operation, which settled with anti-convulsant treatment. Other patients had suffered from epilepsy before the insertion of a shunt and continued on anti-convulsant therapy after the shunt. Therefore, it was not considered that epilepsy was a complication of shunt treatment in these patients.

## Discussion

There are many pathological paths leading to the development of an obstructive hydrocephalus which in its turn may necessitate an internal ventricular drainage operation. Both these various pathological processes and the age of the patient may influence the development of complications after shunting treatment. Children suffering from meningo-myelocele will be particularly prone to shunt infection. By contrast, elderly patients may be more prone to the development of subdural haematoma formation because their brains may be slightly atrophic and the skull fused, whereas a neonatal child will have a skull that may fold in on the brain, olbiterating any potential subdural space after shunting treatment. It is not surprising, therefore, that the infection rate of shunt treatment in children may be as high as 14% (6) or 27% (5). It should be stressed, therefore, that there are no children with meningo-myelocele in this present series.

Table 3 indicates the main factors that may lead to infection of the shunt. In this series, the insertion of a ventriculo-atrial shunt has

Table 3. Possibilities for infection in v.a. shunt

| | |
|---|---|
| Preoperative | 1. Meningitis<br>2. Chest infection<br>3. Sepsis elsewhere in the body |
| Operative | 1. Repetitive shunt revisions<br>2. Placement of atrial catheter<br>3. Erosion of shunt through scalp |
| Postoperative | 1. Chest infection<br>2. Sepsis elsewhere in the body |

been delayed where possible until the patient was free of sepsis elsewhere in the body. In the case of patients with bacterial meningitis, a shunt was not inserted until the patient had been apyrexial, off all antibiotic treatment, and the C.S.F. normal in regard to cell count, for at least one week following the last dose of antibiotics. Ventricular catheterisation for either drainage of C.S.F. or I.C.P. monitoring did not lead to problems of shunt infection. Frequent shunt revisions due to blockage obviously increase the risk of infection. In the case of upper end blockages, the risks may be minimised by the insertion of the ventricular catheter into the frontal horn. For lage ventricles, the posterior temporal approach is perfectly adequate. However, in those patients with relatively small ventricles and a large pseudo-meningocele in the posterior fossa consequent upon surgery at that site, it has been felt that a right frontal ventricular catheter will maintain its patency much better than a temporal catheter. Since this series is essentially concerned with shunting in adult patients, lower end blockages due to body growth have not been a major factor, and therefore it has simply been essential to position the atrial catheter at the right site during the primary operation. The E.C.G. method of placement of the atrial catheter was felt to be very important, in that not only can the atrial catheter be placed with great accuracy away from heart valves, thereby diminishing the possibility of valvular vegetations, but in addition, there is no disturbance to the drapes, as can happen in a radiographic method. It has also been observed that covering the reservoir with pericranium adds an extra layer which prevents both skin erosion and infection. In this series, skin erosion occured in the one case of a Pudenz-Heyer reservoir not covered by pericranium and in two cases of cachectic patients using the Spitz Holter system. For this reason alone it is felt that a Pudenz-Heyer valve being less bulky than the Spitz Holter valve, is more suitable and provided this is covered with pericranium then skin erosion should not be a problem. In the postoperative phase any infection elsewhere in the body needs early and energetic treatment with antibiotics. It has been pointed out that there are two types of shunt infection (5). The first group of patients develop evidence of shunt infection within two months of surgery and it is probable that the infecting organisms were introduced durint the operation. In this series there has been only one example or primary intra-operative infection. The second group develop infection consequent upon bacteraemia from infections elsewhere in the body. Three cases in this series developed such an infection and it is important to warn the patient and his general practitioner that if the patient develops sepsis elsewhere in the body, early and energetic antibiotic treatment should be instituted.

Table 4 indicates the possibilities for intracranial haematoma formation following shunting. It is important to avoid shunting patients with cerebral atrophy and such patients may be screened out by the various methods outlined (3). Patients with raised I.C.P. as judged by the

Table 4. Possibilities for intracranial haematoma formation in v.a. shunt

| | |
|---|---|
| Preoperative selection | 1. Best results in patients with raised intracranial pressure<br>2. Avoid patients with cerebral atrophy |
| Operative | 1. Excess C.S.F. spillage during operation<br>2. Valve pressure too low<br>3. C.S.F. tracking around ventricular catheter into sub-dural space |
| Postoperative | 1. Sudden postdural changes during early days<br>2. Head injuries |

evidence of papilloedema or ventricular pressure monitoring are most suitable, as are patients who develop a meningocele after posterior fossa surgery and in whom the meningocele does not settle after a series of therapeutic lumbar punctures. During operation it is important to avoid spillage of C.S.F. with the consequent sudden drops of I.C.P. During this series, no more than 5 ml were allowed to escape per patient. In a review of 31 cases of extradural haematoma occuring as a complication of intraventricular pressure release operations, it was found that most of these patients had very severely raised I.C.P. and that this pressure was suddely dropped during the operation (1). It has been suggested that anti-syphon shunts might be of benefit. This type of shunt was not used in this series and normal Pudenz-Heyer ventriculo-atrial shunts were used, although the opening pressure lay between 5-8 mm of mercury. In retrospect, it would seem that this range of pressure is both suitable in treating the hydrocephalus and in preventing gross sub-atmospheric reductions in I.C.P. leading to the development of intracranial haematoma. In addition, collections of C.S.F. in the subdural space have been reported. In this series, therefore, during operation the dura was fused to the leptomeninges at the site of the burr hole with the use of coagulating diathermy in an attempt to prevent C.S.F. running around the ventricular catheter and into the subdural space. A successful ventriculo-atrial shunt wll result in a dramatic drop of I.C.P. within the first 24 hours. It is important to keep patients flat for several days following this major change and allow them to sit up slowly over the next few days in an attempt to minimise the sudden pressure changes. Although no patient is likely to deliberately sustain a head injury, it is important to warn the patient and his relatives to view a blow to the head with some concern and seek immediate medical attention.

## Conclusion

Although the insertion of a ventriculo-atrial shunt can be viewed as a technically easy operation, there are probably very few operations in the neurosurgical armamentarium that have such widespread effect upon the whole of the central nervous system. It is disastrous to regard a ventriculo-atrial shunt as "just a burr hole connected to a cut down" (4). Scrupulous attention to both preoperative selection, intraoperative technique and careful postoperative treatment and follow-up is essential.

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# Complications Following Shunt Operations for Post-Meningitic Hydrocephalus

J. C. DE VILLIERS, P. F. DE V. CLÜVER, and L. HANDLER

## Introduction

This communication deals with the management of children under the age of 12 years, who were admitted under our care with post-meningitic-hydrocephalus, the type of hydrocephalus most frequently encountered in our environment. It offers particular problems of management in the acute, as well as the late phases, and has a high mortality. The problems offered by the causal disease to a large extent determine the complications of the treatment, so that management has to be shaped accordingly.

## Clinical Material

Four hundred and nineteen patients were investigated for hydrocephalus from 1966 to 1977. One hundred and five (25%) suffered from post-meningitic hydrocephalus and another ninety-five (22%) had hydrocephalus due to basal cistern block. Of the 105 children with post-meningitic hydrocephalus, 74 (70%) presented within the first two years of life, while 21 (20%) had hydrocephalus following on an attack of neonatal meningitis.

Neuroradiological investigations in the form of percutaneous ventriculography and air encephalography, revealed the following distribution of the site of block to flow of cerebrospinal fluid (Table 1).

Attention should be drawn to the high incidence of septa in the ventricles. These are usually attributed to previous infection (6), but have occasionally been seen by us in patients withour previous infection.

Table 1. Sites of obstruction in post-meningitic hydrocephalus as demonstrated neuroradiologically

| | |
|---|---|
| Foramen of Monro | 6 |
| Aqueduct | 9 |
| Fourth ventricle outflow | 9 |
| Basal cistern block | 56 |
| Multiple sites | 1 |
| Uncertain sites | 25 |
| Septa in ventricles | 9 |

## Treatment and Its Problems

### *Acute Management*

Many patients are admitted with hydrocephalus associated with active, or inadequately treated meningitis. This dual problem of active meningitis in the presence of progressively expanding hydrocephalus, may demand urgent action in the form of external ventricular drainage to tide the patient over the acute crisis. In the less acute situation, the insertion of a ventricular reservoir may be called for to allow repeated ventricular tapping, or, even continous external drainage and also for the installation of antibiotics locally in the management of ventriculitis (5). Table 2 indicates the extent to which these methods have been employed in the present series.

The high mortality following temporary external ventricular drainage, indicates the serious condition of the patients for whom this procedure was used. Following this acute regime, a number of patients did not come to a shunting procedure for the reasons indicated in Table 3.

### *Postacute Management*

In the treatment of hydrocephalus in general, ventriculo-atrial (VA) shunting was used almost exclusively from 1966 to 1972. In time it became obvious that we were not able to provide the accurate follow-up which is so essential for a child with one of these shunts. Many of the patients live very far away in sparsely populated parts of the country and not within easy reach of medical attention.

Table 2. Methods of treatment of acute infective hydrocephalus and outcome

| | Total | Eventually shunted | Died |
|---|---|---|---|
| Omaya reservoir | 15 (6) | 7 (4) | 4 (2) |
| Temporary ventricular drainage | 15 (3) | 4 (1) | 7 (1) |

(Figures in brackets indicate neonatal meningitis)

Table 3. Reasons for patients not being shunted

| | | |
|---|---|---|
| Referred to paediatricians after initial management | | 19 |
| Temporary ventricular drainage | 10 | |
| Omaya reservoir | 4 | |
| Poor clinical state (blindness, contractures) | | 5 |
| Arrested | | 6 |
| Died of uncontrolled meningitis | | 14 |
| Tuberculous meningitis | 7 | |
| Neonatal | 3 | |
| ? Cause | 4 | |

Figure 1 shows that we did a great number of revisions but nevertheless, there were also serious complications apart and aside from the usual problems of growth shortening, disconnection, and so forth.

From 1972, we gradually changed over to the ventriculo-peritoneal (VP) shunt which did not prove to be a trouble-free procedure as can be seen from Figure 1. The need for accurate follow-up, however, is less critical in the child with this kind of shunt from the point of view of growth shortening and thrombo-embolic phenomena (4). It is not an entirely risk-free shunt as Table 4 indicates.

## Discussion

It is worthy of note that colonisation occurred in four infants, who, in each instance, had suffered from neonatal meningitis as the cause of their hydrocephalus. It would also seem from our experience that ventriculo-peritoneal shunts have fewer complications than VA shunts in post-meningitic hydrocephalus and when they do, they are of a lesser nature.

Three early deaths in ventriculo-peritoneal shunting were due to re-activated meningitis probably due to an error in judgement on our part. In one's anxiety to treat a child with a rapidly expanding head in the presence of active meningitis, one may resort to shunting before the local process has completely settled down and in these 3 instances, the insertion of the shunt stirred up latent infection which could not be controlled. Although seizures tend to be more common in patients with post-meningitic hydrocephalus than in other forms of hydrocephalus, an unexplained feature in our experience is the fact that seizures were relatively more frequent in patients with VA shunts (seven cases), than with VP shunts (one case).

The complications of VP shunting were those common to all kinds of shunt procedure, that is obstruction of the ventricular catheter by choroid plexus or debris and disconnection of tubing with or without migration of the distal catheter into the abdomen and beyond. Erosion of the pump through the scalp in a wasted, undernourished child with a large head and poor head control, is a very real risk. This complication has a multifactorial causation and calls for special nursing care by the mother when the child is discharged from hospital (1).

Table 4. Comparison of revisions and mortality of VA and VP shunts in post-meningitic hydrocephalus

| | VA shunts | VP shunts |
|---|---|---|
| Number done | 31 | 28 |
| Early mortality | 0 | 3<br>(re-activated meningitis) |
| Late mortality | 5<br>(colonisation 4)<br>(1 acute pressure) | 1<br>(renal failure) |
| Lost to follow-up | 11 | 7 |
| Revisions | 21 | 8 |

Pressure areas on the scalp before or after operation may preclude transcranial operation and be an absolute indication for the co-peritoneal shunting in the presence of a communicating hydrocephalus.

The erosion of a rigid abdominal catheter through the skin, is not uncommon and has influenced us in changing from the rather stiff RAIMONDI catheter to a simple soft silicone rubber tube.

Three features of the primary disease are of particular importance in the post-meningitic child and determine the eventual outcome more than the shunting procedure.

1. The presence of ventricular loculation where one ventricle becomes blocked off at the foramen of Monro and may assume space demanding qualities. This requires the establishment of a communication between the two ventricles either by craniotomy or ventriculoscopy and careful assessment of the child afterwards to determine whether shunting is necessary or not.
2. The presence of ventricular septa may make any shunting procedure impossible, particulalry if the ventricles have become converted into multilocular cystic spaces where the spinal fluid has a high protein content and at times a high cellular content as well. Where the ventricular CSF is normal and only isolated septa are present, this may be no bar to a good end-result. Isolated septa may be broken down by craniotomy or by ventriculoscopy and adequate cerebro-spinal fluid flow re-established or a shunt may be performed (3).
3. Inflammatory obstruction of the third ventricle or aqueduct is no bar to the use of ventriculo-cisternostomy provided that surface pathways have been demonstrated to be present. Ventriculo-cistern-ostomy has proved to be the ideal shunt in our environment.

The fact that relatively few shunt revisions had to be done in this group of post-meningitic hydrocephalics as compared to the other patients with obstructive lesions, tends to support the impression that communicating hydrocephalus has a smoother course than other forms of hydrocephalus when treated by a shunting device (2).

Twenty patients with aqueduct stenosis in the same age group and adequately followed-up had 68 revision procedures done whereas in 41 post-meningitic hydrocephalus, the 20 treated by VA shunting had 21 revisions and the 21 treated by VP shunting had 8 revisions.

There is little doubt that after meningitis in some patients, cerebro-spinal fluid pathways may be restored to normality but in these instances a shunting procedure maintains intracranial pressure at an acceptable level until the restorative process has been completed. In post-meningitic hydrocephalus therefore, the dictum of once a shunt always a shunt, need not of necessity hold. Indeed, 9 of our shunted patients in this series have become shunt-independent and have arrested at an acceptable level of ventricular size and intellectual function.

## Conclusion

Post-meningitic hydrocephalus offers some as yet unresolved problems such as the management of active ventriculitis in the presence of expanding ventricles. Earlier diagnosis and earlier, enthusiastic ventriculostomy with intraventricular antibiotics, may salvage more and better quality lives. The simpler the shunting procedure, the better when one practices in an environment where follow-up cannot be perfect. Although the early mortality is high, some children attain excellent results from shunting procedures and may gain shunt independence.

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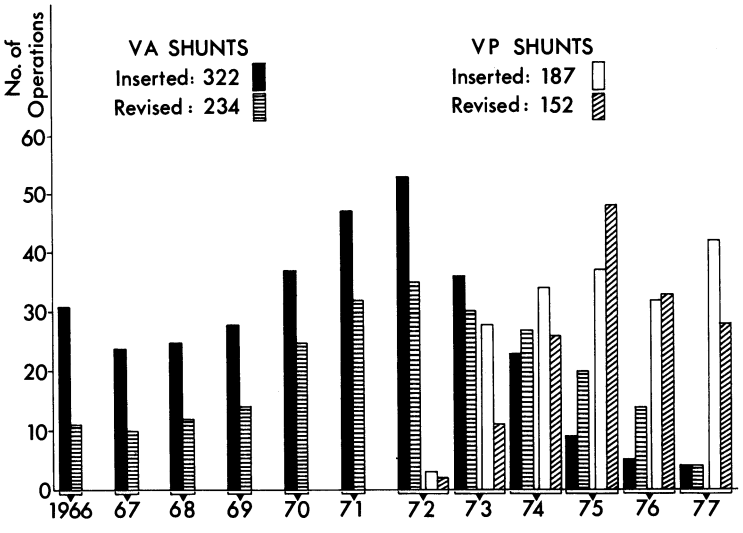


Fig. 1. Comparison between the number of ventriculo-atrial and ventriculo-peritoneal shunts performed and the frequency of revisions for each type of shunting procedure

# Complications in Hydrocephalus Shunting Procedures

G. TH. VAN BEUSEKOM

In a 6-year-period, 1972-1977, 260 shunt procedures were performed at the Sophia Hospital, Zwolle, The Netherlands. From this total, 149 operations were performed by the author on a total of 74 patients. In the first 4-year-period, only the Pudenz system was used. From 1975 on, the Hakim system was preferred. Primary treatment of hydrocephalus has always been ventriculocardial shunting. Only when no venous entrance could be found did I shift to a ventriculoperitoneal shunt. For this VP shunt, the Ames system, as well as the Pudenz-Raimondi-system, were used.

Obstruction of the cardiac canula due to relative shortening through growth is not considered to be a complication in this study, but it allows for the discrepancy between 149 operations and the placement of only 92 shunting systems. When the cardiac canula is no longer located in the atrium, it usually becomes blocked by endothelium. Therefore the reintroduction of the canula into the atrium with a flexible stylet will often be impossible.

The indication for the implantation of a shunting system was *perinatal hydrocephalus* in ten cases. This was usually communicating hydrocephalus. *Obstructive hydrocephalus* was the indication in 19 patients. Some of these suffered from midline cerebral tumours. Sixteen babies with *myelomeningocele* and hydrocephalus received a shunt. In 29 patients the indication was the presence of a *normal pressure hydrocephalus* (NPH). The indication to shunt N.P.H. patients was based on air-studies and RIHSA cisternography. They all suffered from dementia and from ataxia and/or complained of chronic headache. No long term intracranial pressure recording was performed on these patients. However, since 1977 this has been done. The result of the shunting N.P.H. patients is frequently very poor, but sometimes dramatic improvement is obtained.

Of the 74 patients who received treatment, 11 died, nearly 15%. However, with the exception of one patient, the cause of death was not complication of the shunting procedure nor was it related to the surgical procedure. The most frequent complication was the obstruction of the ventricular canula, occurring in 14% of the patients. In these cases, we usually found adhesions to the chorioid plexus. Only in a few cases was the position of the canula in the midline. To prevent adhesion to the chorioid plexus, I now make an occipital burrhole instead of the former parieto temporo-occipital one. At the same time the position of the canula in the frontal horn is ascertained under fluoroscopy. I have found that obstruction of the ventricular canula was more frequent in patients with the Pudenz system or a VPD (20%), than in patients with a Hakim shunt (4%), this notwithstanding the use of Portnoy canulas. The colonisation of a shunting system is a complication of secondary importance in my experience. It occurs in 8.5% of the cases and most frequently with the VPD system. It is least common with the Pudenz system. However, the subdivided numbers are too smal to be significant. Antibiotics were never administered as prophylaxis but only when there was an indication because of earlier infection. The micro-organism causing the infection was usually Staphylococcus epidemidis, or in babies and VPD patients, an enteric micro-organism. For the pre-

vention of infection the utmost operative aseptic precautions must be taken or the procedure must be performed under laminar airflow conditions. VENES of Yale University states that colonisation can be prevented with care and antibiotics. When VPD shunts are used, the occurrence of abdominal adhesions must be considered. These may lead to the formation of peritoneal cysts with malabsorption and subcutaneous reflux of CSF along the abdominal canula, even as high as the skull. This may simulate canula or connection leakage. Never have I encountered obstructive ileus as a complication of the abdominal adhesions. When the adhesions become too extensive, a position of the canula between diaphragm and liver must be considered. This has proven useful in my experience.

Of those cases with ventriculocardial shunting, I had only one patients with thrombosis and multiple pulmonary embolism. This child finally died from this complication. Nevertheless, anticoagulant therapy was never instituted in patients with cardiac shunts.

Technical complications include leakage within the system (three cases) and rupture or kinking of canula (three cases). These can usually be prevented with more operative skill. Skin decubitus over a Pudenz chamber occurred only once. In one case of canula rupture, the canula became dislodged and slipped into the heart ventricle, but with no unfavorable results. With VPD shunting, it appears that secondary displacement of the canula into the lumen of the bowl is possible. I had one such case, and I now have two other cases in our joint practice. They are responsible for a low-grade retrograde pericanular infection. When we look over the total of all complications, this ranks rather high (38%). This is even more impressive if we look at 149 interventions in 74 patients, that is, two operations per patient on the average. The extreme is one myelomeningocele infant that needed nine surgical corrections of the shunt and its complications.

Revisions of the shunt are performed only when there is a clear indication. Therefore, we are sure that many young patients have compensated hydrocephalus.

In conclusion, I consider the shunting procedure for hydrocephalus a mode of treatment which should be discarded when better methods of treatment are developed. In comparison to other procedures of treatment, however, shunt placement is simple and can be easily revised. By comparing our results and complications, we may be able to choose the best policy of treatment with shunting procedures.

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# Surgically Treated Infantile Hydrocephalus and Predictability of Later Intelligence

C. N. TROMP and W. VAN DEN BURG

## Introduction

The relationship of hydrocephalus (HC) and a subsequently impaired intelligence is well documented. In most of the larger follow-up studies (1) a substantially lower mean IQ is found in HC patients. Another frequent finding is the large standard deviation of IQ in this group. To illustrate this: in 117 unselected HC patients we have tested so far, a mean IQ of 76.5 is found, with a standard deviation of 26.6. The distribution of the normal population has a mean IQ of 100 and a standard deviation of 15. This large IQ-variability in HC patients suggests that a number of factors, other than HC alone, have an influence on later intelligence.

Variables reported to be relevant in the literature include
*a) The nature of HC.* Among others, GUTHKELCH and RILEY (3) found that communicating HC carries a better prognosis than non-communicating HC.
*b) The etiology of HC.* Higher rates of mental retardation have been reported in HC following spina bifida (SB) and in HC following meningitis (9), but this is not a general finding (3).
*c) The nature and sensory level of the lesion in SB.* Meningomyeloceles and encephaloceles have been mentioned as the more unfavorable forms of SB (10). HUNT and HOLMES (4) have stressed the importance of sensory level as a predictive factor for intelligence.
*d) The severity of HC.* Different measures to indicate severity have been employed, some are based on ventricular dilatation, others on residual brain mass. The relevance of this factor is disputed. Some authors (6) find no correlation at all with the later IQ, while others suggest "critical borderlines," which range, however, between 5 and 35 mm. For a review, see (8).
*e) Age at the time of shunting.* YOUNG et al. (11) suggest that surgery after the age of 6 months is attended by insufficient cottical reconstitution.
*f) Sex.* HC boys have been found to develop more favorably than HC girls (10).

No study has been reported in which a presumably very important variable was taken into account, namely the IQ of the patient's parents, which can be expected to have a bearing upon the IQ of the patient himself.

The first objective of the present investigation is to establish a control for this variable when evaluating the importance of the factors mentioned above. The second purpose is to correlate later IQ with a large number of plausible indices of ventricular dilatation - or (residual) brain mass - in order to single out the more relevant ones. It must be noted that in this study only those data are considered which the neurosurgeon has at his disposal at the time of shunting.

## Material and Approach

To ensure comparability, the patient group to be studied was restricted to surgically treated HC patients who had their shunt placed within

365 days of birth. Both ventricolographic data at the time of first surgery and IQ's assessed at least 5 years later had to be available. Tumor cerebri cases were exluded. These criteria resulted in a group of 59 patients (32 boys, 27 girls), with a mean age of 10.7 years at the time of testing (range: 5-21 years). Two tests of intelligence were used, both well standardized in the Dutch population: one for patients up to 12 years of age (S.O.N.), and one for older patients (G.I.T.). The IQ of the parents was estimated on the basis of educational and occupational level, according to a method described by LUTEYN (7). Nature and etiology of HC in the patient group under study is shown in Table 1. It will be noted that the proportions of non-communicating HC's appear to be different for SB and non-SB cases. Three relative measures of ventricular dilatation were employed (Fig. 1). Brain mass was expressed as the difference between the volume of the upper part of the skull and the volume of the lateral ventricles, thereby assuming that both forms resemble half elliptoids (Fig. 2). All measures of severity used are shown in Table 3.

The strategy of the analysis was first to evaluate each of the several possible variables independently, taking into account, however, estimated IQ's of the parents. (Statistically speaking, partialing out the influence of estimated IQ's of the parents.) When absolute indices of brain mass or ventricular dilatation were employed, date of shunt placement was also checked. Next, various combinations and interactions of variables were considered in order to trace more optimal prediction possibilities. General multiple regression procedures were employed (2). Plots were made in order to spot possible non-linear relations between the variables (but no such relations were found).

In a further stage of the analysis, the group was restricted to patients who had their shunts placed a) within 110 days of birth (n = 47), and b) 35 days of birth (n = 31).

Table 1. Nature and etiology of hydrocephalus in 59 patients

| | NCHC | CHC | Unknown | Total |
|---|---|---|---|---|
| HC after trauma | 1 | 1 | | 2 |
| HC after meningitis | 1 | 4 | | 5 |
| Congenital HC | 5 | 12 | 1 | 18 |
| HC and encephalocele | 2 | | 1 | 3 |
| HC and meningocele (lumbosacral lesions) | 2 | 2 | | 4 |
| HC abd myelomengocele (cervical lesions) | 1 | 1 | | 2 |
| HC and myelomingocele (thoracal lesions) | 5 | 2 | | 7 |
| HC and myelomeningocele (lumbosacral lesions) | 13 | 4 | 1 | 18 |
| Total | 30 | 26 | 3 | 59 |

NCHC = non-communicating hydrocephalus; CHC = communicating hydrocephalus.

## Results

In the complete group, the only factor significantly related to later intelligence was the nature of the HC: non-communicating HC patients having lower IQ's (Table 2). When estimated IQ's of the parents were investigated statistical significance was even more pronounced ($t = 3.04$, $p$ .005, one-tailed). As Table indicates, IQ's of the parents correlated .355 with IQ's of the children ($p < .01$, one-tailed), and thus is a variable which must certainly be taken into account. Table 3 also shows the lack of any correlation with measurements of brain mass or ventricular dilatation. No "critical cortical thickness" could be assessed. The picture was not altered by a substantial amount of multiple regression analyses which took into account various factors simultaneously, and their interactions. There was a tendency for SB patients with the higher lesions to have a lower IQ (Table 2), and for girls to have lower scores, but these differences were not significant.

When restricting the group to the patients shunted within 110 days of birth, and looking for correlations with IQ's of the parents, the type of HC emerged again as a significant factor ($t = 2.12$, $p < .025$, one tailed): non-communicating HC's having the poorest prognosis. Third ventricle width, relative and absolute also appeared to be significant in this group, but only when both IQ's of the parents and time at shunting were taken into account. Furthermore, the results are comparable to those of the prior analysis.

In the group that required early surgery (within 35 days of birth), no significance whatever could be found, but many analyses had to be based on only very few cases. For instance, only six communicating HC patients were included in this group. The trend of the results, however, was in

Table 2. Possible predictive factors for intelligence

| | N | M.IQ | SD | Sign. |
|---|---|---|---|---|
| *Etiology of HC* | | | | |
| Trauma | 2 | 56.00 | 29.6 | |
| Meningitis | 7 | 88.86 | 34.0 | |
| Congenital | 18 | 83.50 | 30.0 | n.s. |
| Spina bifida | 32 | 73.81 | 20.7 | |
| *Nature of HC* | | | | |
| Non-communicating | 30 | 70.70 | 21.7 | $t = 2.31$ |
| Communicating | 26 | 86.54 | 29.4 | ($p < 0.05$) |
| *Nature of spina bifida* | | | | |
| Encephalocele | 3 | 58.00 | 21.6 | |
| Meningocele | 4 | 68.00 | 34.7 | n.s. |
| Myelomeningocele | 27 | 78.70 | 18.§ | |
| *Level of lesion* | | | | |
| Cervical | 5 | 66.40 | 21.6 | |
| Thoracal | 7 | 72.57 | 21.1 | n.s. |
| Lumbosacral | 22 | 78.68 | 21.8 | |

N = number of patients; M.IQ = mean IQ; SD = standard deviation of IQ; Sign. refers to statistical significance; n.s. = not significant.

Table 3. Correlation of intelligence with measurements of HC severity and estimated IQ of parents

| | |
|---|---|
| IQ-ventricle width: | -.093 n = 59 n.s. |
| IQ-3rd ventricle width: | -.003 n = 52 n.s. |
| IQ-ventriculo-skull distance: | -.143 n = 59 n.s. |
| IQ-rel. cortical thickness: | -.208 n = 49 n.s. |
| IQ-evans index: | .124 n = 59 n.s. |
| IQ-3rd ventricle index: | -.042 n = 42 n.s. |
| IQ-brain mass: | .185 n = 59 n.s. |
| IQ-brain mass ratio: | -.142 n = 59 n.s. |
| IQ-estimated IQ-parents: | .355 n = 47 (p .01, one-tailed) |

The measurements are depicted in Figures 2 and 3. Brain mass ratio-(LHW-1Hw)/LHW (Fig. 3).

the direction described above. An interesting finding was that a "critical cortical thickness" could now be delineated at 23 mm, above which the patients had significantly higher IQ's. (The *correlation* with cortical thickness was not significant.) The significant difference between the groups, however, disappeared when the IQ's pf the parents were taken into account.

## Conclusions

The possibilities of predicting intelligence in HC children from clinical data on the nature, etiology and severity of HC are found to be astonishingly small.

Non-communicating HC's appear to carry the more unfavorable prognosis, but the difference is certainly too small for any practical prediction purposes.

Third ventricle dilatation may be of some additional relevance. The IQ of the parents is a variable that should be considered in the investigation of factors presumed to relate to the later intelligence of HC children. In this study it appeared to be one of the best indicators.

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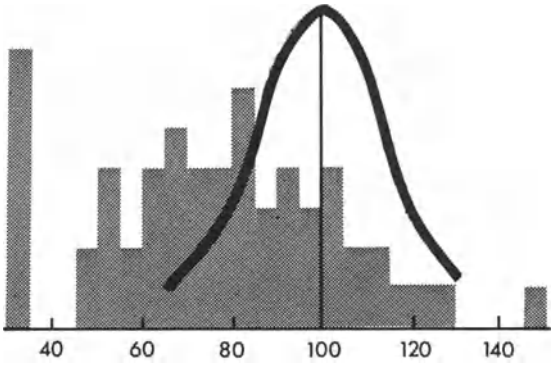


Fig. 1. The distribution of IQ in 59 hydrocephalic children contrasted with the distribution in the normal population (*black line*). Minimum IQ was taken to be 35

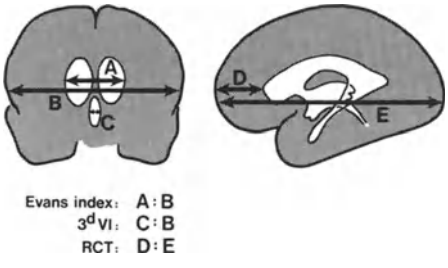


Fig. 2. Indices of ventricular dilatation. A = max. width lateral ventricles; B = max. width inner table of the skull; C = max. width 3rd ventricle; D = ventricle-skull distance; E = max. skull length; 3rdVI = 3rd ventricle index; RCT = relative cortical thickness

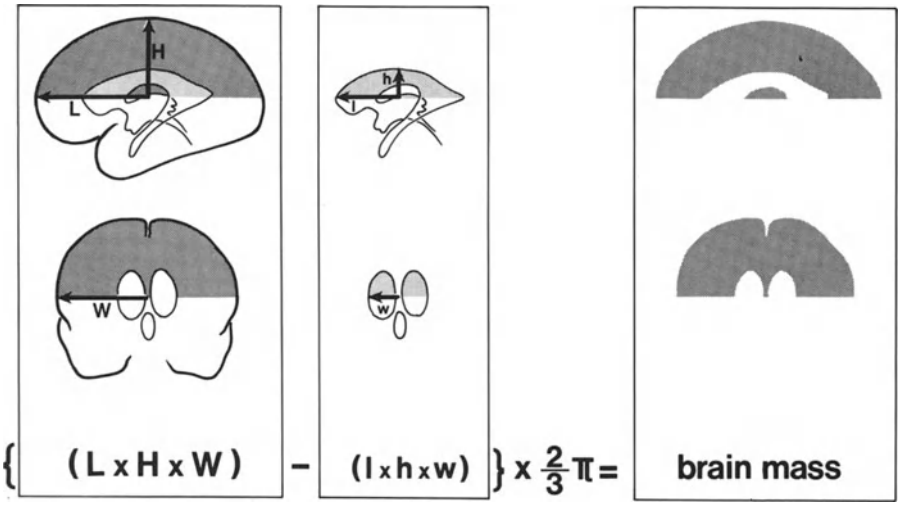


Fig. 3. The estimate of brain mass

# Hydrocephalus and Epilepsy

G. BLAAUW

It is not surprising that hydrocephalus, a disease of the brain, coincides with a greater incidence of epileptic seizures than may be found in the general population. This is confirmed by standard textbooks of pediatrics and neurology (2, 5), but these books do not state the factors which are important for the development of epilepsy. Furthermore, systematic information on the incidence of epilectic seizures in hydrocephalic children is not available. Epilepsy was thought to be more common after the insertion of a CSF-shunt (6).

Importance has been assigned to the etiology of hydrocephalus in the incidence of epilepsy (8).

## Material and Methods

A study was made of 323 children who were treated in the Academic Hospital Rotterdam from 1960 to 1977 and who had either hydrocephalus in combination with spina bifida, hydrocephalus due to bacterial meningitis, or other non-tumoral causes of hydrocephalus, including intra-uterine infection with toxoplasmosis and hydrocephalus after prematurity or perinatal hemorrhage.

## Results

Table 1 shows the composition of the group according to their etiology. For the sake of convenience, they are rearranged into three groups. The patients with an encephalocele were included in the first, the spina bifida, group. The second group consists of children with postmeningitic hydrocephalus. A third miscellaneous groupd included, among others, the children with intra-uterine toxoplasmosis, prematurity and perinatal hemorrhage, since it is difficult to assess in which cases difficult labor and perinatal cranial trauma were caused by existing congenital hydrocephalus. The ages of the children are given in Figure 1. The incidence of seizures in the three groups is shown in Table 2. The overall frequency in the 323 children studied was 34%. The occurrence of fits in the spina bifida group was significantly less (chi square 7.90, $p < 0.01$) and in the postmeningitic group this was significantly higher (chi square 21.39, $p < 0.001$) than the average incidence.

Table 1. 323 children with hydrocephalus arranged according to their etiology. Between brackets are the number of children who had epilepsy

| | | |
|---|---|---|
| Spina bifida | 152 | (35) |
| Congenital hydrocephalus | 98 | (35) |
| Postmeningitic hydrocephalus | 47 | (31) |
| Toxoplasmosis | 13 | (5) |
| Encephalocele | 13 | (4) |
| Total | 323 | (110) |

Table 2. The incidence of epilepsy

| | |
|---|---|
| Overall | 34% |
| Spina bifida group | 24% |
| Postmeningitic group | 66% |
| Miscellaneous group | 36% |

The ages at which seizures occurred are grouped in Table 3. When the seizure took place shortly before or after insertion of the valve system or when seizures were seen during the active phase of neonatal meningitis, this was called early epilepsy. Epileptic manifestations which occurred later than one week after drainage or after the adequate treatment of meningitis were called late epilepsy. It can be seen that 40% of the seizures fall in the early epilepsy group. Only 42 (38%) of the 110 children who had epilepsy were noted to have a second or more seizures later. There was no singificant difference between the occurrence of more than one seizure in the early and late epilepsy groups.

The number of valve revisions in the children who had seizures and those who did not, and in the children who had shunt-related infections and those who did not are shown in Table 4. The average number of revisions in the group of children with seizures was not significantly greater than in those who did not have seizures. But the average number of revisions in the group of children who had had infected shunts was significantly greater than in those who did not have had shunt related infections (more than three times the standard error of the difference).

Epilepsy occurred in 39 of 88 children, who had a history of shunt-related infections and in 28 children the first seizure occurred at the time of the infection. The occurrence of epilepsy in such infected cases was highly significant (chi square 28.7, $p < 0.001$). Thus it appears that late epilepsy was closely connected with shunt-related infections and 59% of the patients with late epilepsy had a history of such infections. The incidence of epilepsy in children who did not have early epilepsy and who had shunt related infections was 44%, and in those who did not experience such infections, it was 14%.

There seems to be no relationship between epilepsy and the protein content of the CSF at the time of the valve insertion, asymmetry of the ventricles, the finding of hemiparesis, diplegia or tetraparesis,

Table 3. The age of inception of the seizure or seizures arranged in early and late groups (see text). Between brackets are the numbers with recurrent epilepsy

| | Spina bifida | Post-menigitic hydrocephalus | Miscellaneous causes of hydrocephalus | |
|---|---|---|---|---|
| *Early epilepsy* | 10 (4) | 24 (11) | 10 (3) | 44 (18) |
| within 1 year after drainage | 8 (4) | 4 (2) | 14 (4) | 26 (10) |
| *Late epilepsy* | | | | |
| later than 1 year after drainage | 21 (8) | 3 (1) | 16 (5) | 40 (14) |
| | 39 (16) | 31 (14) | 40 (12) | 110 (42) |

Table 4. The average number of valve revisions per patient in relation to epilepsy and to infected CSF shunts

| | | | |
|---|---|---|---|
| Overall | 1.98 | SD ± 2.32 | SE ± 0.13 |
| Had seizures | 2.26 | SD ± 2.73 | SE ± 0.26 |
| No seizures | 1.84 | SD ± 2.08 | SE ± 0.14 |
| Had shunt-related infections | 3.05 | SD ± 3.39 | SE ± 0.36 |
| No infections | 1.62 | SD ± 2.21 | SE ± 0.14 |

although in those children with a hemiparesis, the seizures were a reason for major concern because their epilepsy was often difficult to manage. A significant relationship between the occurrence of seizures and valve blockage was not present. The incidence of epilepsy was the same in the children who died (33.75%) as in the children who were alive (34.16%).

## Discussion

The average incidence of seizures in the general child population appears to be about 3% in the group under five years old (1, 3, 10, 12). A comparison of the incidence of seizures from EEG studies in cases of hydrocephalus is given in Table 5. These are from selected patient groups. In a study of 200 hydrocephalic children HOSKING (1974) found that the overall frequency of epilepsy in case of hydrocephalus is about 30%, and the etiology of hydrocephalus appeared to be an important factor in the production of epilepsy. In HOSKING's series, the incidence in the postmeningic group was 54%, and in the spina bifida group it was 26%.

Early epilepsy appeared to be related to progressive hydrocephalus and its operative treatment, and to neonatal meningitis. In late epilepsy there was a close connection to shunt-related infections, and apparently a hydrocephalic child who has no history of neonatal meningitis or early epilepsy and who does not have a shunt-related infection has a low risk of seizures. It is remarkable that the first convulsion occurred during the diagnosis and treatment of a shunt-related infection in such a great number of children. Thus drug prophylaxis of epilepsy during the treatment of shunt-related infections should be

Table 5. Figures from EEG studies in children with hydrocephalus from the literature. The number of children studied is shown, and between brackets are the children who were reported to have seizures. The average incidence from these figures is 18.5%

| | | |
|---|---|---|
| FOIS et al. (1958) (4) | 32 | (2) |
| PAMIGLIONE and LAURENCE (1962) (11) | 50 | (3) |
| GREITZ et al. (1971) (7) | 124 | (23) |
| STERNBERG et al. (1971) (13) | 50 | (15) |
| GRAEBNER and CELESIA (1973) (6) | 39 | (18) |
| VAN HUFFELEN (1974) (9) | 60 | (5) |

considered. The overall incidence of seizures in our series was 34%. However, chronic drug treatment of epilepsy is rarely necessary because only 42 (38%) of the 110 children with epilepsy had a second or more seizures. One of the children died as a result of his first seizure. In this series, valve revisions were not significantly more frequent in children who had seizures. Only in two patients who were admitted with seizures was there clear clinical evidence of shunt dysfunction. This is in contradiction to earlier publications (6, 8), in which frequent valve revisions appeared to increase the tendency towards seizures. It may be that convulsions in those cases were wrongly attributed to valve blockage causing unnecessary shunt revisions, which was not apparent later.


## Summary

A retrospective study of 323 hydrocephalic children suggests that the incidence of seizures is related to the etiology of hydrocephalus. Epilepsy which occurred during the treatment of meningitis or which was observed shortly before and until one week after the insertion of a CSF shunt was called early epilepsy. Seizures observed later than one week after drainage were called late epilepsy. Forty percent of the seizures fall in the early epilepsy group. Late epilepsy appeared to be closely connected to shunt-related infections, and 59% of the patients with late epilepsy had a history of such infections. The incidence of epilepsy in children who did not have early epilepsy and who had shunt related infections was 44%, and in those who did not experience such infections it was 14%.


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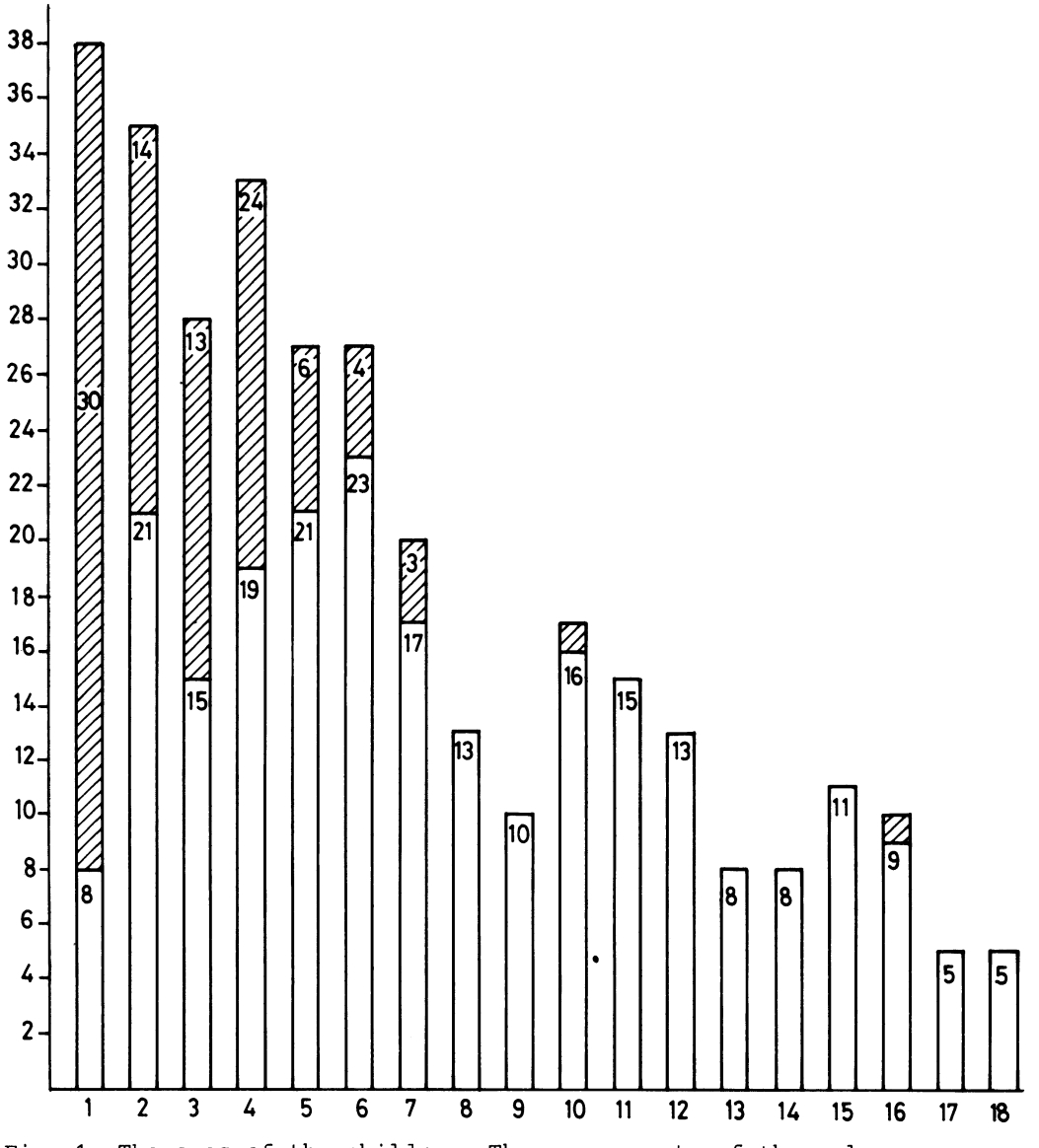


Fig. 1. The ages of the children. The upper parts of the columns are the children who died

# The Use of the Anti-Siphon-Value in Prevention of Functional Complications of Shunting System

S. SALAH, M. SUNDER-PLASSMANN, F. ZAUNBAUER, and W. KOOS

Among the potential complications following shunting procedures in hydrocephalic patients, those due to dynamic and hydraulic propterites of the system used are of major importance. Clinically, an excessive reduction in intraventricular pressure undoubtedly constitutes a significant problem. Table 1 shows those side effects and complications which we found to be most common.

In the erect position, intracranial pressure is known to be physiologically subatmospheric (1, 3, 7). At the level of the Foramen of Monro, the intraventricular pressure was shown to be approximately -70 mm $H_2O$. This is roughly equivalent to the vertical distance to the foramen magnum and the cisterna magna.

All currently available shunting systems are equipped with differential pressure valves. This means that the valve will open (or close) as soon as the hydrostic opening (or closing) pressure is reached. Whether the requisite pressure level is reached by positive valve inlet pressure or negative valve outlet pressure is irrelevant. This explains why the available systems are all associated with a siphoning effect of the tubing. According to FOX et al. (3) the perfusion pressure within the shunting system is

$$P = IVP + HP - (AP + CP),$$

P = perfusion pressure in the system, IVP = intraventricular pressure, HP = hydrostatic pressure, AP = intra-auricular (peritoneal) pressure, CP = valve closing pressure.

In the erect position, HP constitutes a significant value, particularly in peritoneal shunts which have become increasingly common, since this value is proportional to the length of the distal catheter. This formula implies that IVP will be *negative* as soon as HP exceeds the sum of AP

Table 1. Most common effects and complications caused by uncontrolled lowering of IVP

| Infants | Adults |
|---|---|
| Irritability | Headache |
| Sunken fontanel | "Low-pressure syndrome" |
| Overriding bones | |
| Secondary craniosynostosis | |
| Ventricular collapse | |
| Subdural: Hematoma / Effusion | |
| Secondary aqueduct: Stenosis / Occlusion | |

and CP, because a valve will remain open until the perfusion pressure becomes zero (8).

The anti-siphon valve developed by PORTNOY and SCHULTE (8) closes as soon as the pressure in the system drops to subatmospheric levels, so that a siphon effect is precluded. To prevent a very high positive pressure build-up in the ventricular system, the membrane surface at the valve inlet is eight times that at the valve outlet (Fig. 2).

However, in clinical application a correct choice of the differential pressure valve is, naturally, a prior condition for obtaining adequate results. We have been using Pudenz and Hakim valves, which are placed proximally to the anti-siphon valve. Considering our experience to date, the combination with an anti-siphon valve appears to be extremely useful, particularly in the treatment of severe hydrocephalus in infancy and normal pressure hydrocephalus in adults (9). Cases of normal pressure hydrocephalus constitute a singularly profitable indication, because the balance of intracranial pressure in such patients is persistently unstable and thus uncontrolled variations in intracranial pressure often produce unpleasant side effects and complications: Headache, vegetative disturbances, vomiting, fever, disturbances in consciousness and above all subdural hematomas occurring in as much as 23% of cases (2, 4, 5, 6, 10). In fact, these have often enough prompted us to discontinue shunting and treat what amounted to a "low-pressure syndrome" by various measures.

Shunting with valve combinations, including an anti-siphon valve, has so far been done in 38 cases. Except in three of them, the postoperative course in regard to the intracranial pressure situation proved to be very satisfactory. One patient developed increased intraventricular pressure on the third postoperative day. On revision of the shunt, incorrect implantation of the system was found to be the underlying factor. Once this was corrected, the further postoperative course was uneventful. In spite of the implantation of an anti-siphon valve, another adult female patient with long-standing benign aqueduct occlusion and extreme internal hydrocephalus complained of headache as long as four days after surgery. Her symptoms disappeared immediately with horizontal positioning and infusion therapy. Another patient suffering from posttraumatic normal pressure hydrocephalus was found to have minimal asymptomatic subdural effusion at cranial computer tomography about one month postoperatively. A repeat CCT 8 weeks later showed that the effusion had not increased in size.

No definite assessment can as yet be made in terms of anti-siphon valve combinations and their effect on late complications, e.g., conversion of a communicating into an occlusive hydrocephalus or development of secondary craniosynostosis, since both the number of patients available and the postoperative follow-up period are insufficient for significant conclusions.

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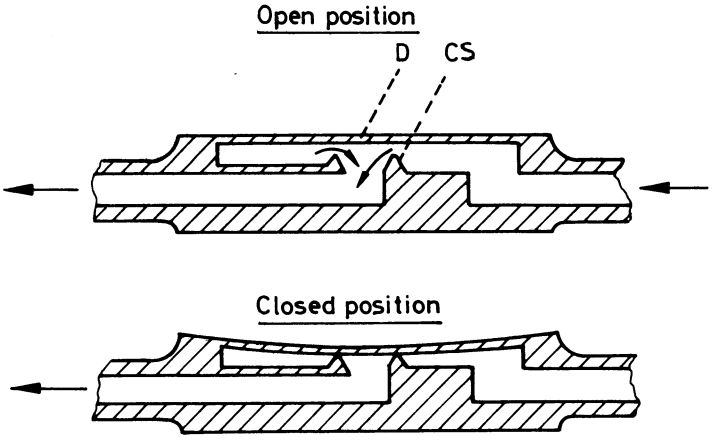


Fig. 1. Diagram of anti-siphon valve (8)

# Neurosurgical and Neurological Applications of the Ommaya Reservoir System

Z. D. GOEDHART, R. E. M. HEKSTER, and B. MATRICALI

The clinical application of the OMMAYA system (OS) was investigated in the Department of Neurosurgery of the Leiden University Medical Center (1). OMMAYA tested many kinds of material, and gave preference to a permanent ventriculostomy made of Silastic. His first description of this device was published in *The Lancet* of November, 1963 (2). A year later, RICKHAM described a comparable but much smaller device (3). At present, three types of OS are available:

1. One with a side inlet
2. One with the entrance of the ventricular drain situated centrally in the base of the reservoir
3. One with a convertible side outlet tube as well

In general, we prefer the reservoir of the second type with an I.D. of 1.5 cm (Fig. 1).

## Indications

Over the past five years we have used 110 OS in 98 patients on the basis of the following indications:

1. Neuroradiological diagnostic procedures (80 times)
2. External CSF drainage (49)
3. ICP registration (12)
4. Therapeutic purposes (12)
5. CSF investigation (10)

Thus, it is evident that in many cases more than one indication was present at the same time. In 10%-20% of the cases, the original indication proved incorrect, but this error was more than compensated for by the results obtained when the OS was used for an indication provided by the unexpected initital findings. Retrospectively, ICP registration and CSF investigation provided essential information which could not have been obtained without the OS in about half of the cases. In four cases, the OS proved inadequate, which was ascribed to insufficient experience with the surgical technique used for the introduction of the device. For this procedure we had a complication rate of 10%, the complications being due to infection and drain dysfunction.

## Surgical Procedure

We prefer a right parasagittal precoronal site. If the enlargement factor of the radiogram of the skull is known, this site can be determined easily. We use the crescent-shaped incision with the base facing medially to reduce tension on the edges of the wound and to obtain a hyp- or anasthetic puncture field. The skin flap is folded back in the median direction, and the dissected periosteum in the temporal direction (Fig. 2). The ventricular drain is cut to the correct lenght, connected to the reservoir, and with the aid of the stylet, is introduced as a unit. Blind positioning of the drain tip in the third ventricle is difficult, due to the lack of landmarks (Fig. 3), and maintenance of the correct direction of the drain tip is hampered because the stylet slides along the radiopaque ball and thus deflects the drain tip.

## Discussion

External drainage and reservoir puncture are technically much easier and safer with the OS than with the RICKHAM system. The incorporation of a puncturable reservoir into a drain system or separate implantation is, in our opinion, advantageous in a drainage patient. I shall restrict myself here to two special causes of hydrocephalus in which technical difficulties can be encountered in the use of an OS.

In patients with an intraventricular tumor, especially those with obstruction of the foramen of Monro, the OS is introduced into the usually dilated ventricle via a burr-hole which has been made, slightly more posterior than routinely, outside the field of the subsequent frontal craniotomy, thus avoiding the effects of any leakage, infection, and so on. To insure effective functioning of the drain, the position of the tip should be checked by injecting contrast medium under image-intensified fluoroscopy and simple radiography. It was wise to start the drainage after, rather than before this visualization. Our series included nine cases of obstructing ventricular tumors.

In cases of craniopharyngeoma, an OS is introduced preoperatively on the right coronal side. An attempt is then made to remove the tumor radically via subfrontal approach. At the end of the operation, a second OS is placed via a left procoronal burr-hule, leaving the drain tip in the surgical field. If a cyst forms, it can be drained. Sometimes the tissue lining the cyst can be inactivated by repeated injection of a 10% saline solution, but only in the absence of communication with the subarachnoidal space.

## Conclusions

The use of the OMMAYA system makes it possible to objectivize and quantify the parameters of the various clinical pictures. For us it has become an indispensible method in the treatment of drainage patients and of patients with a suprasellar or posterior fossa lesion. Our retrospective study has shown a much greater need for a safe ventricular access rout than could have been anticipated. When the OS is used on the basis of strict indications, a complication percentage of about 10% is considered acceptable.

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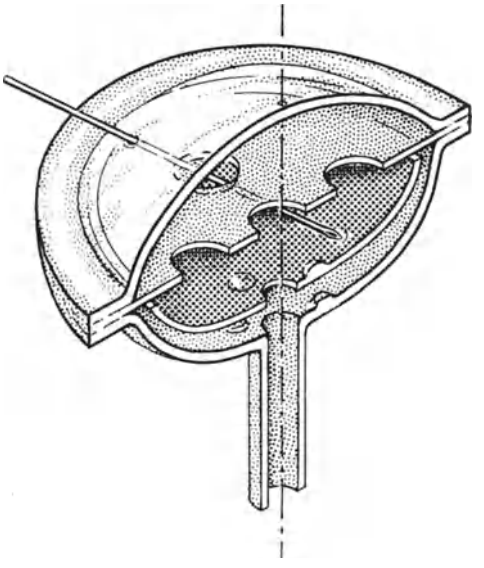

Fig. 1

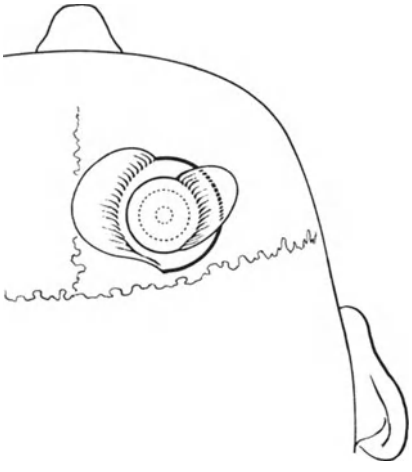

Fig. 2

Fig. 1. Cross-section of a burr-hole reservoir with an inner diameter of 1.5 cm. The reservoir is self-sealing at 50-100 punctures with a 23 or 25G needle. The darkest area is a floating hard shell which prevents perforation of the dome on the dural side

Fig. 2. Precoronal parasagittal burr-hole situated on a line between the two ends of the incision in the skin. The incision in the periosteum is curved in the opposite direction

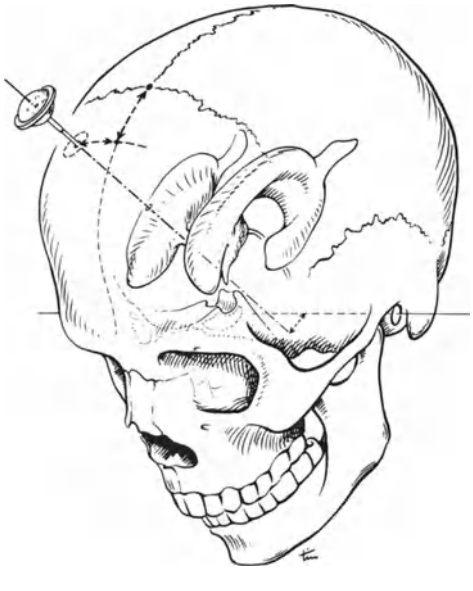

Fig. 3. This three-dimensional drawing shows the parameters applied for the positioning of the drain tip of the OS in the 3rd ventricle

# Computerized Axial Tomography and Shunt Dependency

A. D. HOCKLEY and A. E. HOLMES

## Introduction

As a non-invasive method of demonstrating the ventricular system, computerised axial tomography (CAT) is well established in the investigation of hydrocephalus (1-4). When this is symptomatic, the patient is treated by a shunting procedure. Reappearance of symptoms may be related to shunt malfunction and quite ofte this can easily be determined by palpation of the flushing device, but this is not always infallible. In other instances the hydrocephalus may well be capable of resolution in which case the patients will not remain shunt dependent for the rest of their lives. We have tried to evaluate CAT as a means of determining shunt dependency and thus establish whether or not shunt revision is essential and at the same time we have found that CAT is very helpful when shunt revision is contemplated.

## Material and Approach

This paper describes our experience in Cambridge from 1976 with CAT scanning in cases where hydrocephalus was not associated with an intracranial tumour (Table 1). The diagnosis was established by CAT alone in 39 out of 44 cases, the other patients also having conventional investigations. Thirty-three patients were subsequently treated by Pudenz-Heyer ventriculo-atrial shunts. The hydrocephalus was mild in 11 patients in whom it was felt shunting should be deferred and none of these have subsequently required a shunt. Follow-up scanning was carried out in 76 patients who were asymptomatic following the insertion of ventriculo-atrial shunts for the conditions described in Table 2. The youngest was 9 days old and the eldest 78 years. Thirty-six of these scans were performed within 3 months of the shunt insertion and several many years later. Twenty-three partients with symptoms suggestive of shunt malfunction were investigated prior to shunt revision, this group comprising 21 cases of congenital hydrocephalus.

## Results

We have correlated ventricular size with the clinical assessment of shunt function by palpation of the Pudenz reservoir. In the asymptomatic group the ventricles were of normal or subnormal size in 48 and symmetrically dilated in 20 patients (Fig. 1). Disproportionate enlargement

Table 1. C.A.T. scan in hydrocephalus

| | |
|---|---|
| Diagnosis | 44 |
| Response of ventricular system to shunting | 76 |
| Investigation of shunt malfunction | 23 |
| Total | 143 |

Table 2. Causes of hydrocephalus in follow-up study

| | |
|---|---|
| Infantile | 53 |
| Post-traumatic | 10 |
| Normal pressure hydrocephalus | 4 |
| Fourth ventricular outflow obstruction | 4 |
| Post-haemorrhagic | 3 |
| Vein of Galen aneurysm | 1 |
| Toxoplasmosis | 1 |
| Total | 76 patients |

of the occipital horns was seen in five instances (Fig. 2), and the left lateral ventricle opposite to the side of shunt insertion remained more dilated in three cases. It can be seen from Table 3 that 48 out of 76 patients with functioning shunts had normal or subnormal sized ventricles. Five patients from this group had their shunts temporarily removed because of problems of infection, and in four cases the previously normal-sized ventricles became dilated within a few days. Symptoms developed requiring external drainage until the infection was controlled and a new shunt inserted. There are ten patients who may be said to be shunt independent, in whom the ventricles either have returned to normal size (in three) or were the same size or smaller (in seven) as compared with the initial study.

In 23 patients examined prior to revision, only 2 patients did not show evidence of partial or symmetrical ventricular dilatation. In one patient the cause of symptoms was a large subdural haematoma contralateral to the shunt, and the patient improved following its removal. In the other case the ventricles were normal in size, although the reservoir was slow to refill. Because of persisting symptoms, the shunt was revised and following replacement of the ventricular catheter the child became symptomfree. In three cases the shunt reservoir felt normal to palpation but when revised the valve was found to be imcompetent.

In addition to assessing ventricular size, CAT also provides useful information regarding the position of the catheter in relation to the ventricular contour. This is illustrated in Fig. 3 where the catheter tip is buried in the wall of a dilated ventricle in a patient with acquired craniostenosis.

Table 3. Ventricular size after shunting

| Ventricular size | Asymptomatic | | Symptomatic | |
|---|---|---|---|---|
| Normal | 48 | (45) | 2 | (0) |
| Large | 20 | (16) | 13 | (3) |
| Partial dilatation | 8 | (5) | 8 | (0) |
| Total | 76 | (66) | 23 | (3) |

(Numbers in brackets refer to those patients whose shunts were thought to be functioning clinically).

## Discussion

Our evidence confirms that
a) patients with normal or subnormal sized ventricles after shunting are likely to be shunt dependent.
b) Ventricular size unaffected by shunt insertion, malfunction or removal suggests compensated hydrocephalus or shunt independence.

In patients with symptoms, who required shunt revision, CAT demonstrated there was nearly always partial or symmetrical ventricular dilatation. This facilitates accurate placement of the ventricular catheter and hopefully this may reduce the likelihood of future revisions. In no cases have we had to seriously contemplate a subtemporal decompression to encourage focal dilatation of the ventricular system.

It is now our policy to combine CAT with proposed shunt revision. In many instances the scan can be done without general anaesthetic, but where necessary the two procedures can be easily combined. The single instance of a patient with persistent symptoms, although with normal sized ventricles, indicates that the most important single factor remains the clinical picture, and therefore the CAT scan should not prevent or delay surgical intervention if required.

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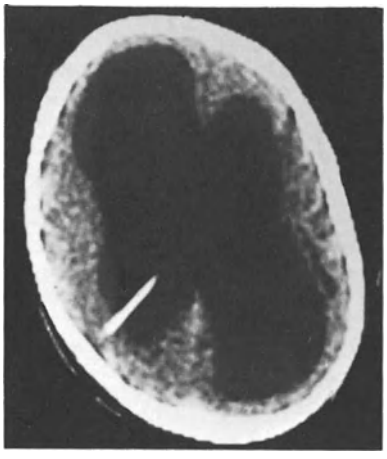

Fig. 1

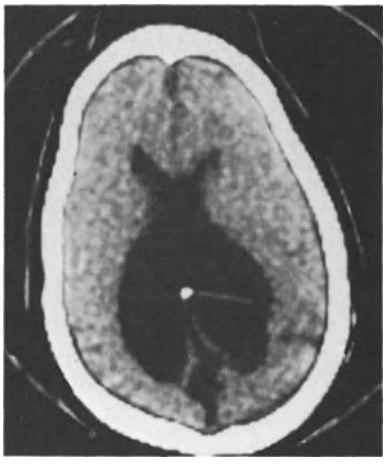

Fig. 2

Fig. 1. CAT scan in an asymptomatic 15-year-old patient with grossly dilated ventricles. Normal shunt function clinically

Fig. 2. Disproportionate enlargement of occipital horn in 10-year-old child with functioning shunt

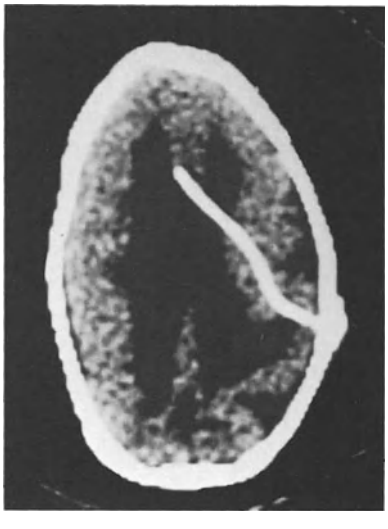

Fig. 3. CAT scan in 2-year-old child with acquired craniostenosis and symptoms of shunt malfunction. The catheter tip is extra-ventricular, and the ventricles dilated

# Perioperative Chemoprophylaxis in Shunting-Infections

H. FRIEDRICH and G. HAENSEL-FRIEDRICH

Infections are a common and serious complication of cerebrospinal fluid shunts. The existence of a foreign implant perpetuates the infection. Moreover, the shunt valve, particularly with its somewhat stagnant medium, is an ideal culture medium for infectious organisms. Thus meningitis and ventriculitis are the most frequent causes for mortality and morbidity in shunted patients (4, 6, 7). Systemic antibiotic therapy after the demonstration of the pathogenic organism and resistance test often fails. Although prophylaxis plays an important part in medicine, antibiotic prophylaxis in surgery remains subject to debate. The routine use of antimicrobial prophylaxis does not reduce the risk of septicemia and may provoke superinfections with resistant pathogens (5). In most operations therefore, we have avoided chemoprophylaxis during the last 4 years. According to our experience, prophylactic perioperative chemoprophylaxis is adviseable only in the following operative procedures:

1. Operations with insertion of a foreign body, i.e. VA shunts and VS shunts
2. Operations in infected regions, i.e., CSF-fistulas, myeloceles etc.

Antibiotics, to be administered in neurosurgery, especially for a perioperative chemoprophylaxis, should be sufficiently CSF-patent in normal meninges. The few known measurements of antibiotics in patients with normal blood-CSF-barrier are incompletly documented (8, 9). A therapeutically efficient concentration of chemotherapeutics in the CSF with normal meninges could be demonstrated up to now only with Chloramphenicol and Co-trimoxazol (2). We have examined the CSF-levels of Co-trim (Bactrim, Eusaprim etc.) in six patients with normal meninges. The results of our measurements are shown in Figure 1.

The maximum CSF-concentration of non-protein-bound SMZ was approximately 46.5% (36%-69%) of the active serum levels (Fig. 2).

The TMP-CSF-concentration was 68.9% (42%-90%) of the serum concentration. These graphs show a continous Cotrim-level in the CSF during a 12 h period, while the serum concentrations decrease in a linear way within 3-4 h. The elimination out of the CSF-compartment seem to be delayed. Because of the well-known side-effects of Chloramphenicol during the last two years we usedl exclusively the combination Co-trim for a short time prophylaxis, as shown in Table 1.

Table 1. Short time prophylaxis

| | Co-trimoxazol |
|---|---|
| Day before shunting | 2 x 24 h |
| Operative day | 2 x 24 h |
| First postoperative day | 2 x 24 h |
| Only exceptionally longer | |

During this period we operated 403 VA and VP shunts in our clinic. This total includes 192 initial placements and 211 revisions. In 101 patients the shunt system was inserted because of an obstruction in the CSF pathways due to tumors in or near the 3rd or 4th ventricle. After the patients' recovery from increased intracranial pressure and hydrocephalus the tumor was removed with microsurgical technique.

We had 12 infections altogether representing an infection rate of 3%. There were three infected VP shunts out of 46, and nine infections out of 357 VA shunts. The risk of infection for the initial operation was 7:192 and for subsequent revision procedures 5:211, which was approximately in the same range (Table 2). The last report of our clinic in 1967 stated 18 cases of septicemia among 183 children operated upon - an indicence of 10% (3).

Apparently certain infections cannot be avoided, either by surgery or by antibiotic prophylaxis, as can be seen in Table 3. Traumatic lesions were caused by a laceration of the scalp over the shunt system with subsequent exogenous infection. Two patients had already suffered from infections with septicemia prior to shunt operation. In one child the peritoneal catheter perforated into the colon 8 months after surgery. In one case, shunt infection occurred after the operation of a suppurating cholesteatoma, because the atrial catheter was cut intraoperatively. Further reasons were wound infections after the local operation and technical factors during the operation.

In 8 patients the infection appeared within one month, while they were still in hospital. Four patients had to be readmitted because of delayed infection (Table 4).

Table 2. Number of shunt operations (ventriculo-atrial, ventriculo-peritoneal) and infections

| | No. of patients | Infected |
|---|---|---|
| Original insertion | 91 | 4 |
| Revision | 211 | 5 |
| Shunting procedure before tumor operation | 101 | 3 |
| Total | 403 | 12 |

Table 3. Etiology of infections

| | No. of patients |
|---|---|
| Traumatic lesion | 2 |
| Preoperative infections | 2 |
| Bowl perforation by perit. cath. | 1 |
| Shunt catheter cut during ENT surgery | 1 |
| Multiple punctures of the shunt system | 1 |
| Wound infection | 2 |
| Surgical technique | 3 |

Table 4. Interval between operation and infection

| | No. of patients | |
|---|---|---|
| Post-operative ( 4 weeks) | 8 | |
| 1 month to 1 year | 3 | |
| Over one year | 1 | |
| | v-atrial | v-perit. |
| Type of infected shunt | 9 | 3 |

Eleven patients were treated by removing the infected shunt system, external ventricular drainage, and both intravenous and intraventricular antibiotics according to the resistance test. Four patients died during therapy, two of them, suffering from 3rd ventricle tumor and meningeoma of the tentorium were in a poor condition after the local operation. They caught bronchopneumonia and urinary tract infection which led to septicemia and secondary shunt infection. Thus both patients were victims of typical hospitalism. The other two patients were admitted in a moribund state with bowl perforation and shunt sepsis following ENT-surgery. In the 7 patients whose infection could be controlled, the shunt system was reinserted. In one case the infection was controlled by intravenous and intrashunt antibiotic therapy without removal of the shunt system (Table 5).

Though the number of our patients was limited, we believe - due to the results obtained - the the infection rate in shunting procedures can be considerably reduced by a short-term perioperative chemoprophylaxis. It is not likely that the number of our shunt infections during the last years dropped from 10%-3% only because of a better surgical technique and asepsis. The well-considered use of prophylactic chemotherapy during the perioperative period limits the progression from transient bactericemia to postoperative sepsis. Since few complications

Table 5

| Result | Infecting organism | Shunt removal | Antibiotic treatment and external ventricular drainage | Reinsertion of shunt system |
|---|---|---|---|---|
| *Infection controlled* | Staph. aureus (4 pat.) | + | + | + |
| | Staph. epidermidis | + | + | + |
| | -Streptococcus | + | + | + |
| | Micrococcus species | - | + | - |
| | Unknown | + | + | + |
| *Death during therapy* | Pseudomonas, Klebsiella | + | + | - |
| | E. coli | + | + | - |
| | Pseudomonas | + | + | - |
| | Enterococcus | + | + | - |

result from the short time prophylaxis and superinfections as well as changes of infection are unknown, there seems to be no disadvantage to this method.

The final proof whether this method is feasible or not could only be obtained by a double blind study, which seems to be justified in the prophylactic use of antibiotics, not, however, in the case of infection.

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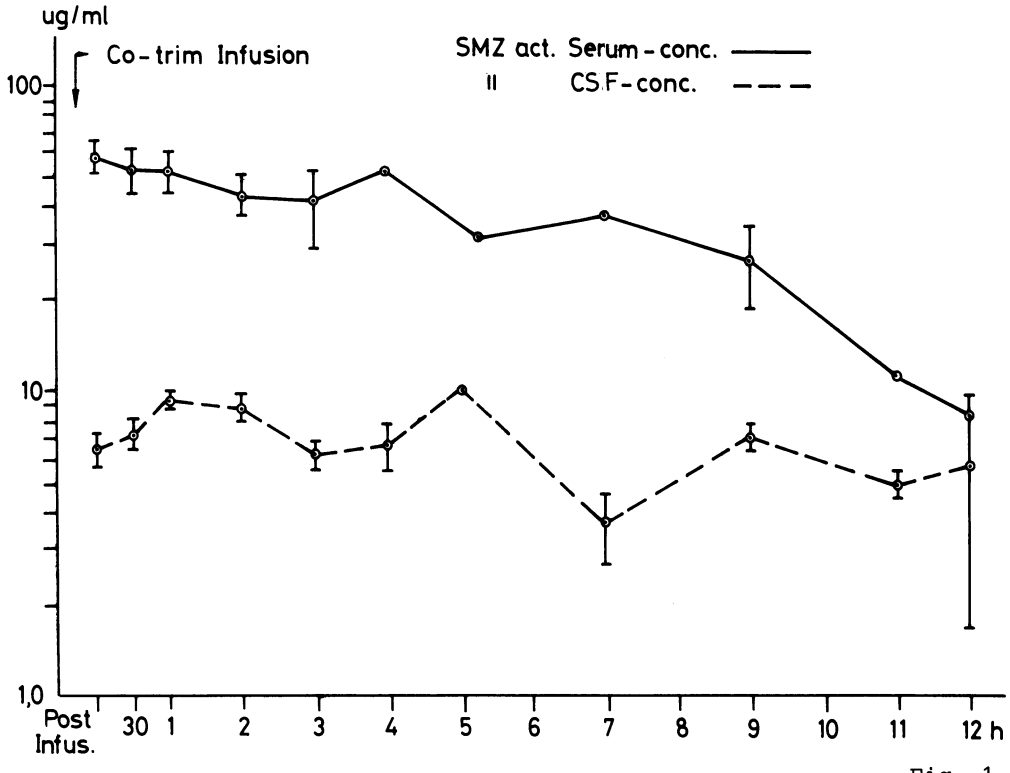
ug/ml
Co-trim Infusion
SMZ act. Serum-conc.
CSF-conc.
100
10
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9
10
11
12 h

Fig. 1

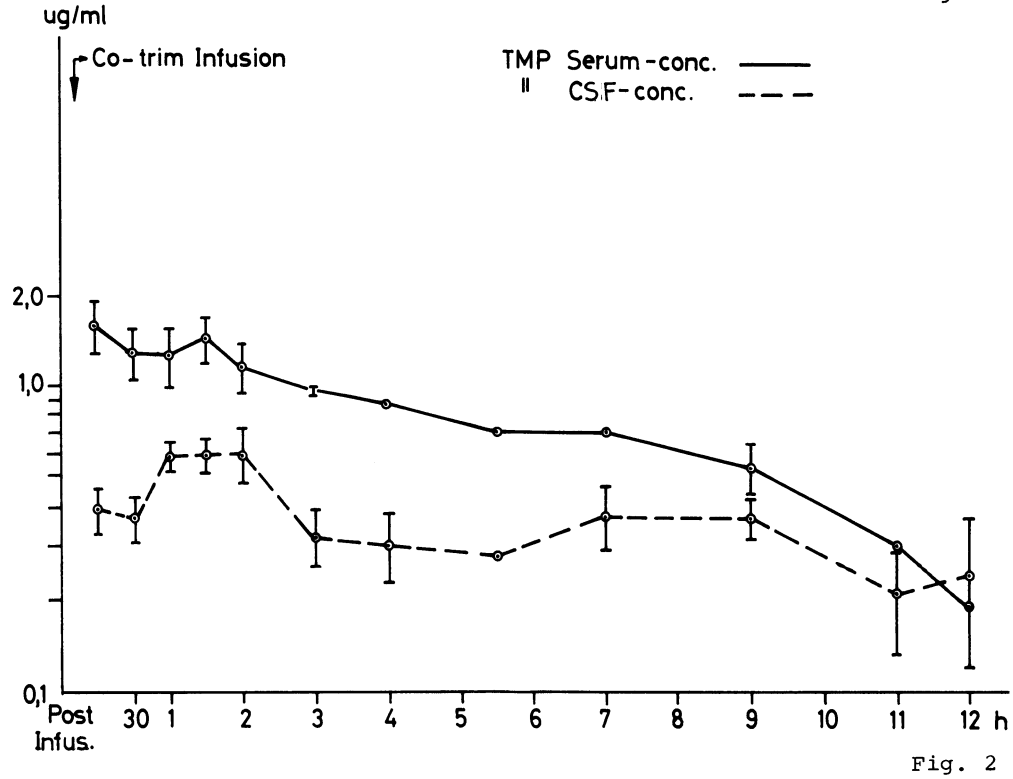
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Fig. 2

# Computer Tomography

# Impressionist Mechanical Mind of Man

J. SMALL

Mr. Chairman, you have devoted this day to problems in computerised reconstructive tomography, the most important technical contribution to diagnostic radiology, not excluding image amplification. This centimetre thick slice is analysed as to density. The positive and negative information of this reconstruction is of amazing value. During the day you will hear a number of papers on the technical defects and errors of the technique, some of which will be of mechanical origin and others man made. Man sees what he thinks he sees and frequently what he has been conditioned to see - fragments of vision, like highlifts, are dangerous to the picture.

It is now just over 100 years since Claude Monet exhibeted a picture entitled "Impression Sunrise" and the word "Impressionist" was employed as a derogatory term for those artists escaping from the contrived compositions of nearly 400 years. Monet insisted that painting should be the rapid recording of the "motif" at the site, in fact, the total picture. Ruskin wrote at about the same time, "A great composition always has a leading emotional purpose, technically called its motive, to which all its lines and forms have some relation." Our motif is the patient, and we must never permit this to be forgotten. This is where our impression, wrongly called intuition, in diagnosis is grown and where intrusive conditioning is excluded. No machine must blur this vital point of application. A young patient of mine was in North Africa when he had his first subarachnoid leakage of blood and a firm diagnosis of sandfly fever was made on the basis that there was a lot of it about. Three weeks later he had another attack in Central Africa where another firm diagnosis of malaria, cerebral in nature, was made again, because there was a lot of it about. While he was being flown to England he had a serious subarachnoid haemorrhage and the country was alerted to the fact that he might have lassar fever because there had been a good deal written about it. A scan was done in England and declared normal, but his retired doctor father thought it would be a good idea if he was sent to me, as he thought he had a subarachnoid haemorrhage. He had indeed, from a middle cerebral aneurysm shown on angiograpy but not on a scan. This is an important area of negative information on scanning. On the morning that I was to operate him, he became deeply unconscious and hemiplegic and a scan showed the reason why. The clot and aneurysm were treated, and you will be interested to know he is back at work in Africa. The value of the scan in distinguishing those people requiring urgent surgical treatment for clot is accentuated in this case.

Throughout the history of intelligence, particularly in war, the importance of positive information has always been correctly valued, but large areas of negative information have so often been the true cause of catastrophe. There is a mid-Atlantic vocabulary song from years ago, "Accentuate the Positive, Eliminate the Negative." The taking of relevant history and clinical examination of the patient has become less fashionable with the approach of that hoped-for machine which will tell the truth, the whole thruth, and nothing but the thruth. It will be Pandora's Box, and it is this I fear from the indiscriminate routine

use of scanning. Taken in context, scanning is a wonderful innovation. It sometimes tells the truth when you don't want to believe it.

The importance of the plane of scanning for correct localisation makes reconstructive sagittal scans desirable, but this will require a large number of slices and considerable increase in irradiation. Here lies a fear for the future - excessive radiation dosage may bring eye cataract formation. Scanning is not designed to determine histology, but we may yet find facts of disturbed physiology by scanning. Protoplasmic astrocytoma, the disturbed physiology of fluid retention within the cell, is clearly observable.

Mr. Chairman, the significance of scanning for the patient's future health and the financial implications in health care is something of which I must speak. There can be no doubt that in trauma and varieties of stroke, scanning enables not only the diagnosis to be established, but earlier appropriate therapy instigated. It has reduced the need for varying introsive contrast procedures and therefore removed the morbidity and mortality of those procedures. Scanning also helps in determining what other investigations should be undertaken. This having been said, I doubt if scanning, under the best circumstance, has failed to declare a treatable condition, but I am also doubtful if, in the medium and long term care of the sick, it has improved the patient's outlook. The value of the investigation is like all other investigations, it is at its best when it is used to look for what is suspected by clinical impression. It is unobtrusive but intrinsic to the gestalt of the patient's picture and future hopes. It was hoped that the enormous expense of scanning installation and procedure would be balanced by a reduction in the number of beds and surgical facilities, and that in the x-ray departments there would be a massive reduction in other procedures. This has not proved to be the case. There is no doubt that scanning has reduced the waiting list for neurosurgical beds and this is a good thing for the patient, but it means that those who enter frequently require the investigations and therapy that scanning was predicted to reduce. There has been a humane and social advance but, as always, at great expense.

What is the force and fire that takes the scientific mind beyond the facts? The secret does not lie in these things alone. The Sphinx is reputed to give the essence of wisdom in a sentence and I leave you with this verse:

The Sphinx when asked about the Scan
Said, "Do not expect too much, my man."

# Problems in Diagnosis With Computerized Tomography (CT)

V. L. McAllister, J. Hankinson, and R. P. Sengupta

## Introduction

There are few reports stressing the limitations of CT scanning (4, 2). In this paper we will discuss some problems in CT diagnosis we have encountered in our everyday practice. They will be discussed in the following categories shown in Table 1.

## Small Tumours near the Skull Base

Small pituitary adenomas, acoustic neuromas or brain stem tumours may escape detection with conventional CT techniques. Greater diagnostic accuracy may be obtained by enhancing the basal subarachnoid cisterns with the water soluble non-ionic contrast medium Metrizamide (12). Small doses (6-10 cc of a 170 mg concentration of Metrizamide) are injected by lumbar puncture and directed immediately intracranially followed by CT scanning. The technique of Computerized Metrizamide Cisternography (CMC) is promising but we would like to emphasize some limitations.

## Acoustic Neuromas

Tumours less than 2 cm in size may be missed at CT (7). A small acoustic neuroma measuring 17 mm in diameter not demonstrated on plain or contrast enhanced CT scans but subsequently shown by CMC is illustrated in Fig. 1. This tumour was predominantly intracanalicular but fortunately there was an associated arachnoid cysts projecting into the cerebellopontine angle cistern producing the filling defect demonstrated at CMC. A small, predominantly intracanalicular acoustic neuroma measuring 13 mm in diameter which was not demonstrated on conventional CT or CMC was subsequently shown by Myodil cisternography. We are at present comparing the accuracy of CMC and Myodil cisternography for detecting small acoustic neuromas. It is our practice at the time of lumbar puncture for CMC to inject a few ccs of Myodil after the Metrizamide has been run intracranially so that Myodil costernography can be carried out subsequently. This combined Myodil and Metrizamide cisternographic

Table 1

| |
|---|
| Small tumours near the skull base |
| Problems in differential diagnosis at CT |
| Isodense lesions |
| Difficulties in tumour localization |
| Vascular lesions |
| Incomplete information for surgical management |
| False negative CT scans due to the timing of the CT examination |

technique is carried out at present in all patients who have a strong clinical suspicion of an acoustic neuroma when the plain and contrast enhanced CT scans are negative. Further experience with CMC is needed to assess its accuracy. We believe that Myodil cisternography is still the procedure of choice for detecting small predominantly intracanalicular tumours. If one fills the internal auditory meatus with Myodil one can positively exclude an acoustic neuroma.

## Sellar and Suprasellar Lesions

The value of CMC in the diagnosis of sellar and suprasellar lesions is now well established (5). Difficulties may arise in the distinction of a suprasellar cistern filling defect due to a tumour from that due to an enlarged third ventricle as illustrated in Figure 2.

We routinely take a lateral skull film of the sellar region after the Metrizamide is run intracranially. Injection of 10 cc of a 170 mg concentration of Metrizamide provides a very clear demonstration of the anatomy of the suprasellar cistern and position of the optic chiasm (Fig. 3A). In addition, small suprasellar extensions may be demonstrated (Fig. 3B).

The diagnosis of an empty sella syndrome at CT is difficult (1). Findings at CMC suggest a diagnosis of an empty sella, but are not conclusive (Fig. 4A). A lateral film of the sellar region while the Metrizamide was run intracranially provided a simple technique for clinching the diagnosis (Fig. 4B).

Spontaneous CSF rhinorrhoea is a difficult diagnostic problem for it is often very difficult to define the site of the leak, and this may lead to more extensive exploratory surgery.

In the case illustrated in Figure 5, an empty sella associated with a spontaneous CSF leak into the sphenoid sinus was clearly demonstrated on a simple lateral skull film of the sellar region (Fig. 5A). CT showed layering of Metrizamide in the right side of the sphenoid sinus (Fig. 5B) but did not demonstrate the empty sella.

## Problems in Differential Diagnosis at CT

A correct histological diagnosis based solely on the findings at CT is in many cases not possible. A ring shaped CT enhancement pattern is a non-specific finding in our experience. It has been described in gliomas and metastases (7, 13), abscesses (10), infarction (14), meningiomas (3), haematomas (15), and in postoperative cases (6). A specific diagnosis of a ring-enhanced lesion can be made only after consideration of a clinical history or in many cases the use of serial CT scans. Non-neoplastic lesions may be diagnosed as a tumour as illustrated in the following cases:

*Case 1*: A 34-year-old female one month prior to admission had an episode of severe headache, nausea and vomiting. There was a recurrence of the symptoms 2 days later with confusion and drowsiness for 3 days. A CT scan showed a low density area in the left frontal region with a little compression of the left frontal horn. After Conray, an irregular ring-shaped pattern of enhancement was demonstrated in the left frontal region extending into the corpus callosum (Fig. 6A). On the CT findings a diagnosis of glioma was considered but the history was atypical for

a tumour. A left carotid angiogram was therefore carried out and this showed an arteriovenous malformation on the medial surface of the frontal lobe extending into the corpus callosum (Fig. 6B).

*Case 2*: A 56-year-old male with an eight year history of temporal lobe epilepsy and had a grand mal attack one week prior to admission. A CT scan showed a shift of the frontal horn to the left side and a large right temporal calcified enhanceing mass with some surrounding finger-like oedema (Fig. 7). The patient died before carotid angiography could be carried out. At post mortem it was found that the patient had had a subarachnoid haemorrhage associated with a massive right middle cerebral artery aneurysm acting as a temporal tumour.

*Case 3*: A 57-year-old female with a six year history of atypical facial pain affecting the left side of the face and around the left eye. In the three months prior to admission, the patient suffered from frontal headaches and complained of sensation of pins and needles in both legs and a subjective sensation of weakness affecting both legs. There were no abnormal neurological signs. A CT scan showed a high density mass behind the dorsum sellae and upper part of the clivus extending on the left side into the middle cranial fossa and upwards towards the tentorium (Fig. 8A). Differential diagnosis included a tentorial meningioma, 5th nerve neuroma or chordoma. Vertebral angiography showed changes consistent with a space-occupying lesion in the region of the tentorium (Fig. 8B). A left carotid angiogram showed a loculated posterior communicating artery aneurysm with a little spasm of the carotid siphon (Fig. 8C). At surgery a large chronic encapsulated haematoma was found and removed and the aneurysm clipped.

## Isodense Lesions

Intracranial space-occupying lesions with the same density as brain tissue will be described in the paper by GRUMME and his colleagues. Isodense lesions may result in equivocal or false negative scans. In our experience lesions which may be isodense on the plain CT scan include acoustic neuromas, metastases, brain stem tumours, arteriovenous malformations and subdural haematomas. In a recent survey of our posterior fossa tumours, 22% were isodense on the plain scan emphasising the importance of contrast enhancement.

## Difficulties in Tumour Localisation

The accurate anatomical localisation of cerebral tumours may be difficult at CT. This especially applies to the high convexity and fronto-parietal regions. The main difficulties arise from the lack of reliable bony and intracranial landmarks to act as a reference point for each scan section. In addition variable head angulation during scanning may profoundly alter the apparent site of the tumour (Fig. 9). We are in agreement with LOGUE (9) that isotope scanning still has a useful place to show the accurate relationship of a tumour to the skull topography.

In the posterior fossa, difficulties may occasionally arise in the distinction of an intra-axial from an extra-axial tumour especially in the region of the brain stem and cerebellopontine angle. In the case illustrated in Figure 10, CT showed a lesion of increased density in the region of the pons with backward displacement and flattening of the 4th ventricle. There is patchy enhancement after Conray. On the CT appearances alone an intrinsic pontine tumour could not be excluded. Vertebral angiography confirmed this mass to be a largely thrombosed giant sac of a basilar aneurysm.

## Vascular Lesions

Angiography will of course be the investigation of choice in cases with suspected vascular pathology (arteriovenous malformation, arteriosclerosis arteritis, aneurysms and vessel spasm). We have encountered normal CT scans in spite of significant intracranial vascular occlusive disease, ulcerating plaques, stenosis or occlusion of the internal carotid artery, Moya disease and saggital sinus thrombosis.

## Incomplete Information for Surgical Management

Angiography is of course necessary in many types of tumours to show tumour vascularity, feeding vessels, venous sinus occlusion or evidence of vessle encasement by a tumour.

In haemangioblastomas, vertebral angiography is necessary to show multiple mural nodules which may occur in up to 20% of cases and may not be detectable at CT (6).

## False Negative CT Scans due to the Timing of the CT Examination

A normal CT scan does not exclude a tumour. If the clinical suspicion of a tumour is high, other neuroradiological techniques or serial CT scanning should be carried out. We have seen several rapidly growing gliomas negative on the initial scan but clearly demonstrated after a short interval. In cerebral infarction there is often a latent interval of 12-24 h before the CT scan becomes abnormal.

## Conclusions

CT is extremely accurate in detecting a cerebral lesion but it is not always possible to arrive at a specific histological diagnosis. Optimal diagnostic information from CT necessitates close attention to clinical details and often the use of other complementary neuroradiological procedures.

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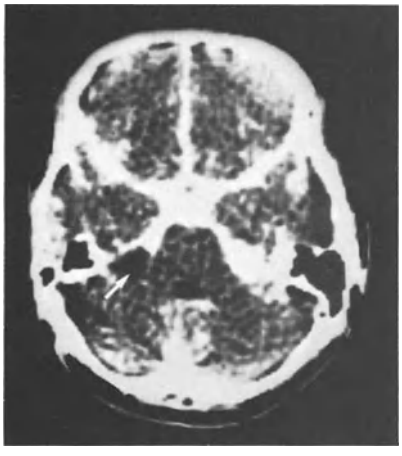


Fig. 1. Computerized Matrizamide Cisternogram showing a small filling defect (*arrow*) in the left cerebollepontine angle cistern. At surgery a left acoustic neuroma was found with an arachnoid cyst projecting into the cerebellopontine angle cistern

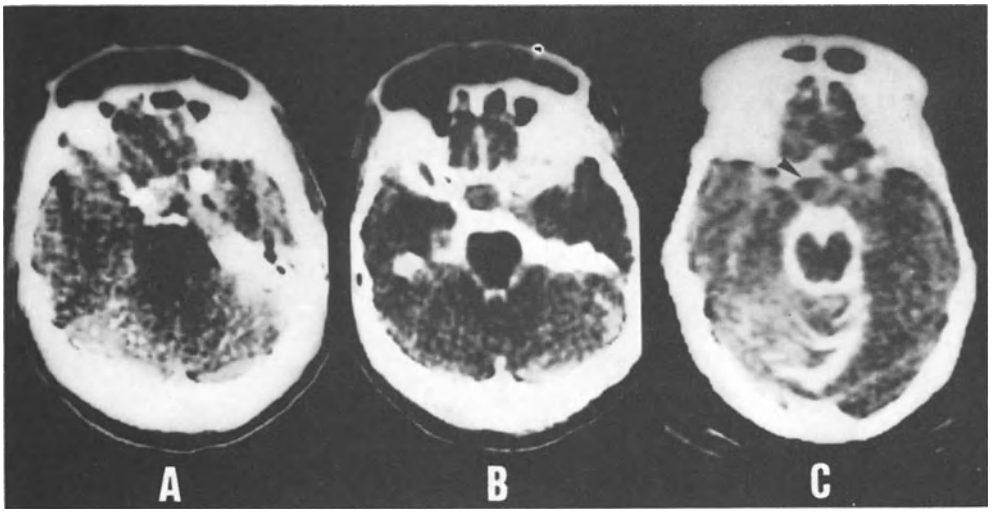



Fig. 2. A Contrast-enhanced CT-scan in a patient with a chromophobe adenoma showing a suspicious area of enhancement in the sellar region. B Computerized Metrizamide Cisternogram same case as clearly showing a suprasellar mass. C Computerized Metrizamide Cisternogram, in a patient with a dilated 3rd ventricle showing a filling defect (*arrow*) in the suprasellar cistern indistinguishable from a tumour

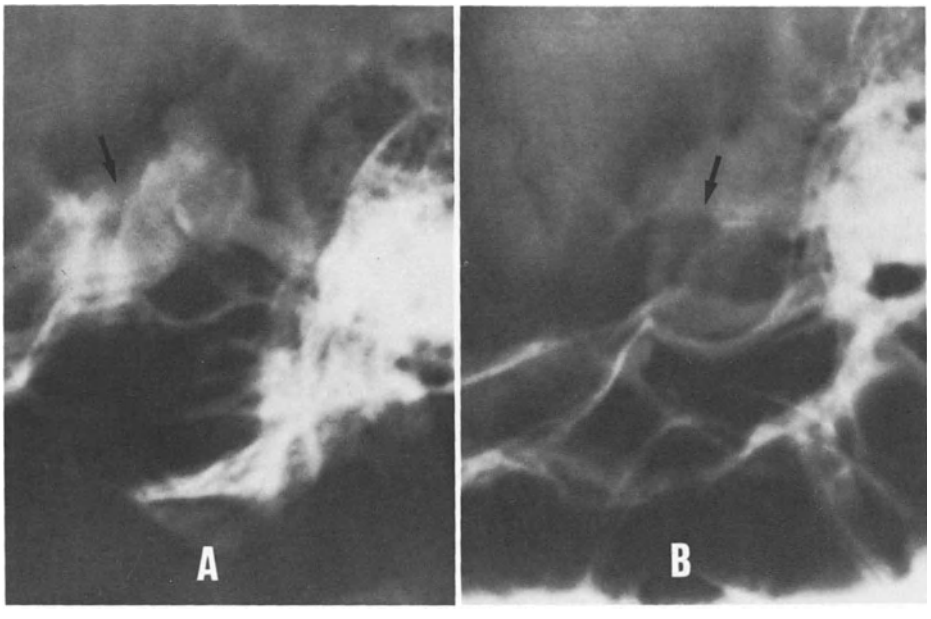



Fig. 3. Lateral skull films of the sellar region taken during intracranial flow of Metrizamide. A The anatomy of the suprasellar cistern, optic chiasm (*arrow*) and optic nerve are clearly shown. B The suprasellar extension of a chromophobe adenoma is clearly outlined (*arrow*)

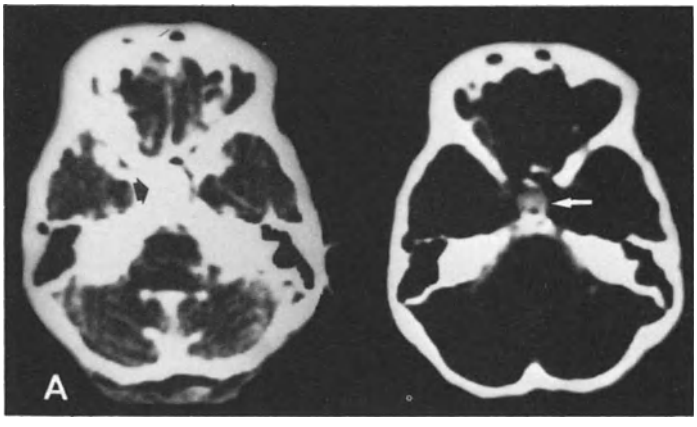



Fig. 4. A At Computerized Metrizamide Cisternography contrast was shown in the region of the pituitary fossa, this was suggestive but not conclusive of an empty sella

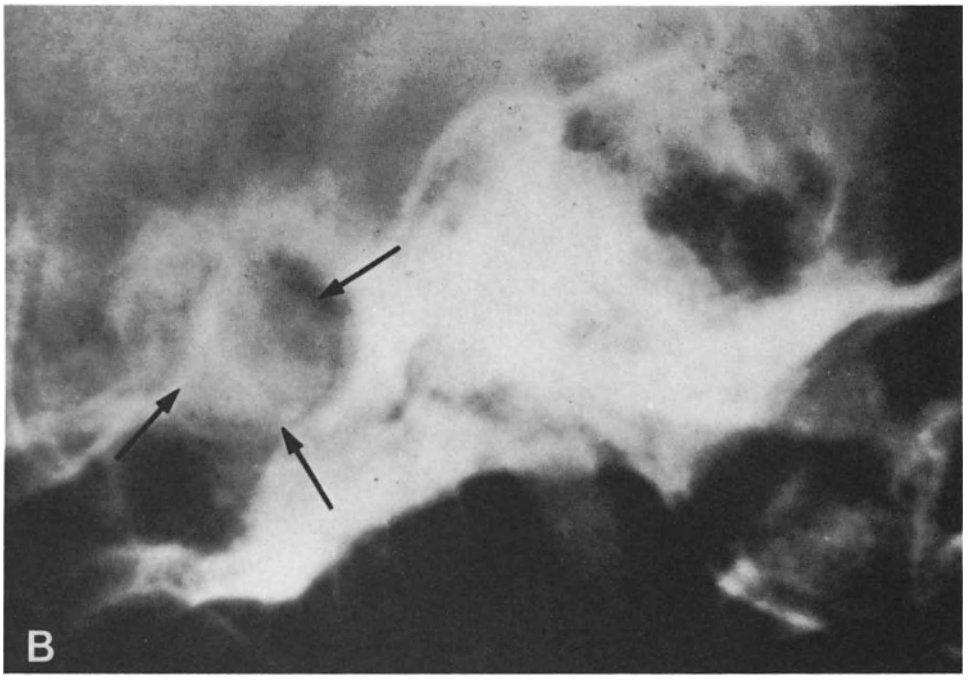



Fig. 4. B Lateral film of the sellar region showing Metrizamide completely filling the pituitary fossa (*arrows*) thus clinching the diagnosis of an empty sella

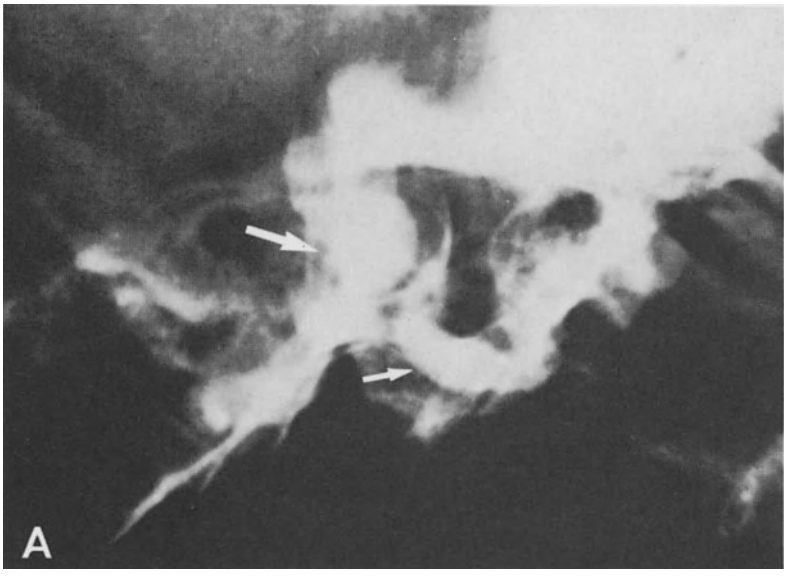



Fig. 5. A Lateral film of the sellar region taken during the intracranial flow of Metrizamide. This shows an empty sella (*large arrow*). Associated with a spontaneous CSF leak into sphenoid sinus (*small arrow*)

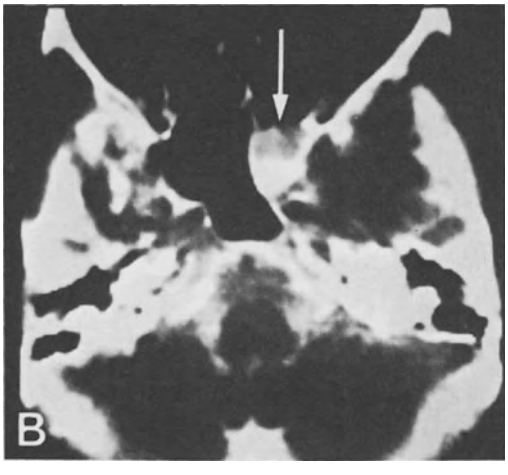



Fig. 5. B Computerized Metrizamide Cisternography (CMC) shows layering of contrast in the right side of the sphenoid sinus (*arrow*)

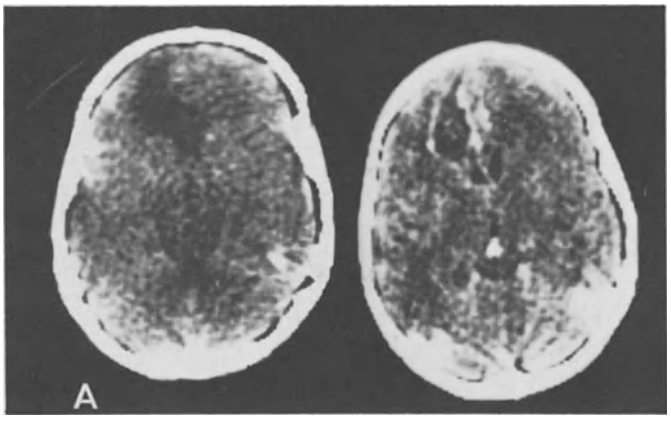



Fig. 6. A CT scan showing a low density area in the left frontal region. After Conray there is irregular ring enhancement extending into the corpus callosum suggestive of a glioma

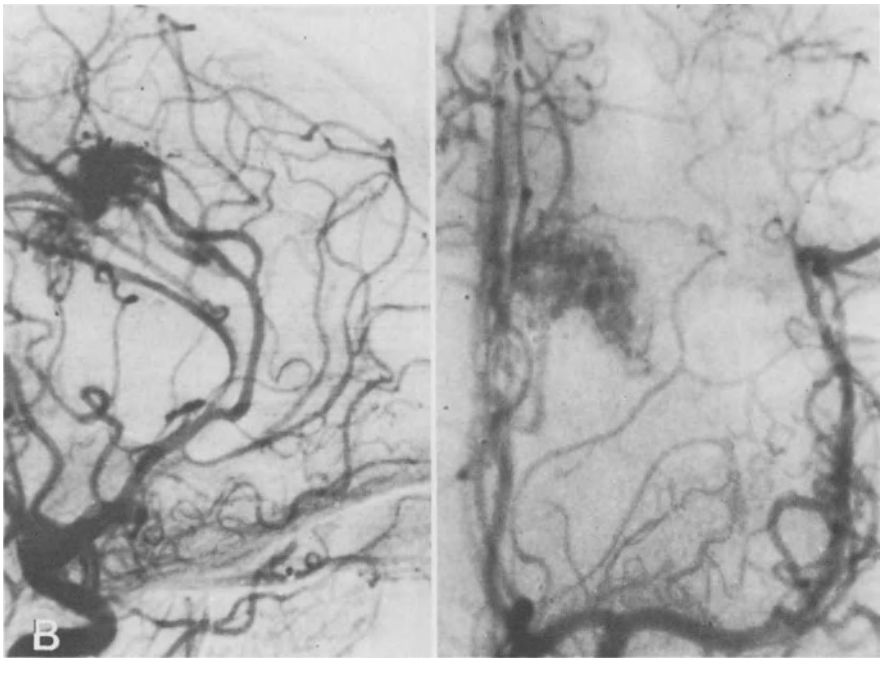



Fig. 6. B Carotid angiogram lateral (*left*) and AP (*right*) views show an arteriovenous malformation on the medial surface of the frontal lobe

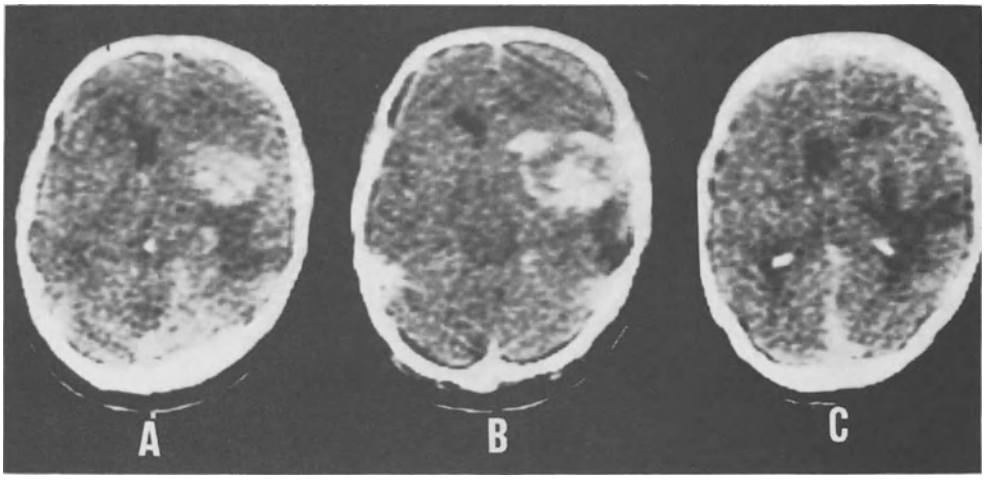



Fig. 7. A, B. CT scan shows a calcified ring enhancing mass in the right temporal lobe with some surrounding finger-like oedema (C). A tumour was considered the most likely diagnosis

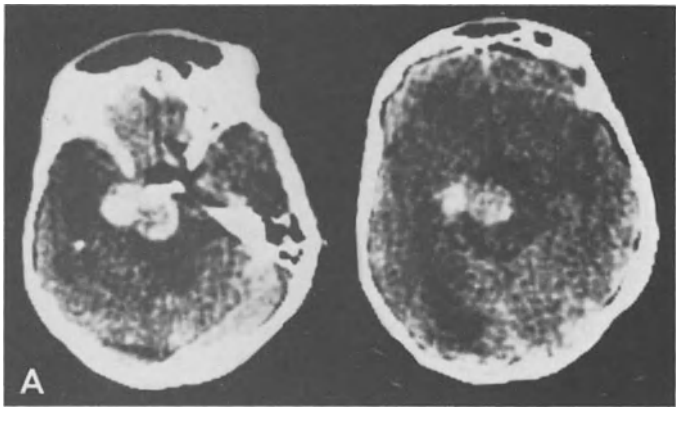
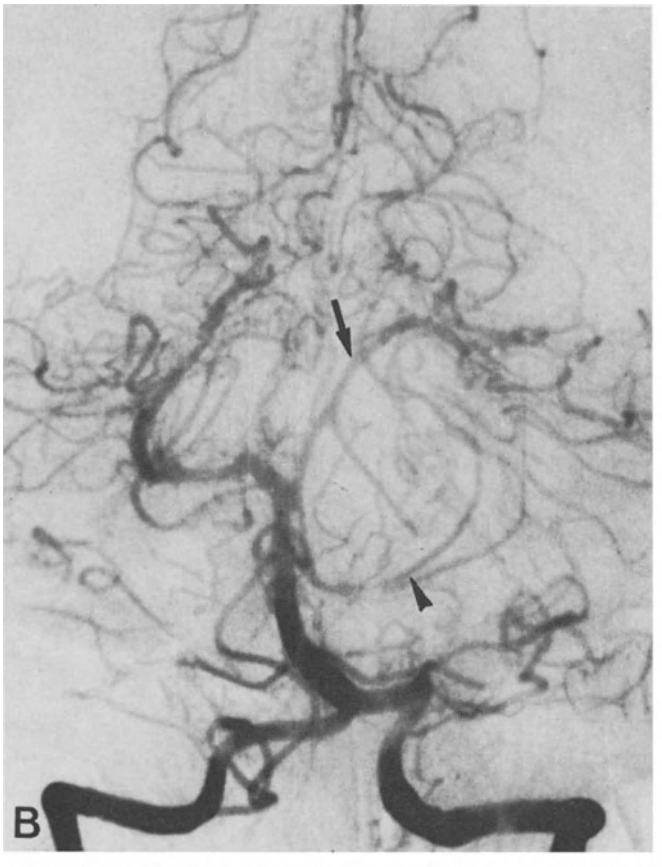



Fig. 8. A CT scan shows a high density mass behind the dorsum sellae and upper clivus extending into the middle cranial fossa on the left side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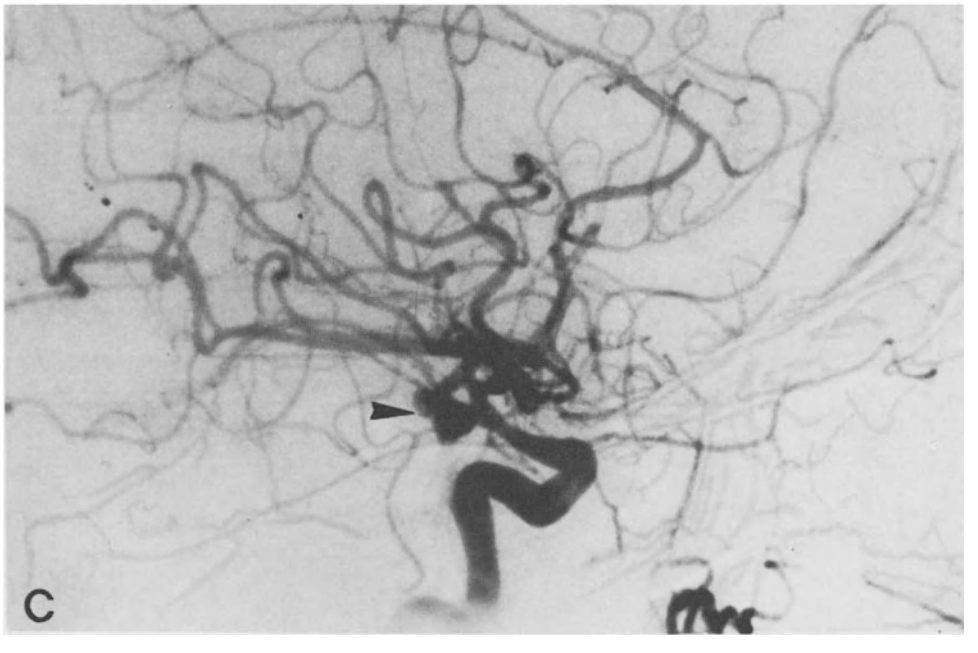
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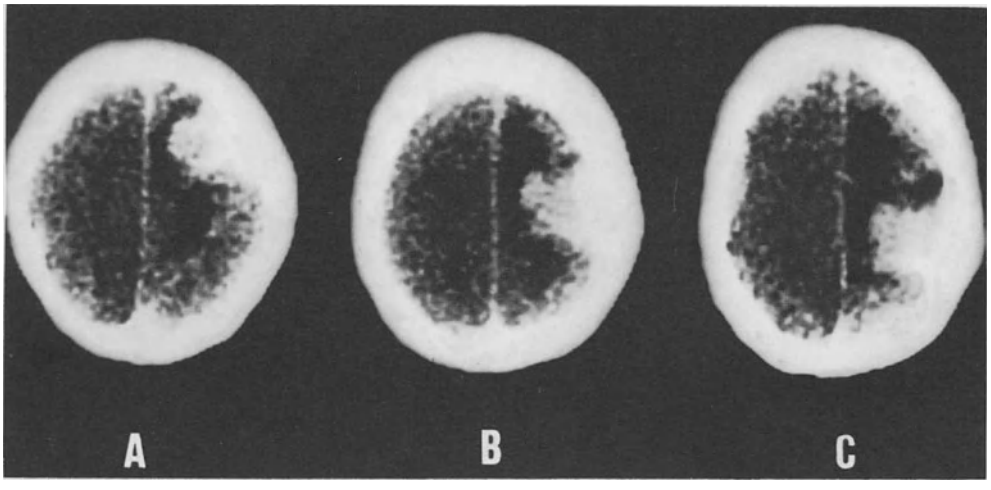


Fig. 9. Contrast-enhanced CT scan of a meningioma showing how changes in the position of the head (*A* flexion, *B* parallel to the orbital meatal line and *C* extension) can profoundly alter the apparent site of a tumour

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Fig. 8. B Vertebral angiogram showing elevation of the posterior cerebral artery (*large arrow*) and depression of the superior cerebellar arteries (*small arrow*). There is some displacement of the basilar artery to the right side

Fig. 8. C Left carotid angiogram shows a bilocular posterior communicating artery aneurysm. There is some spasm of the supraclinoid portion of the internal carotid artery

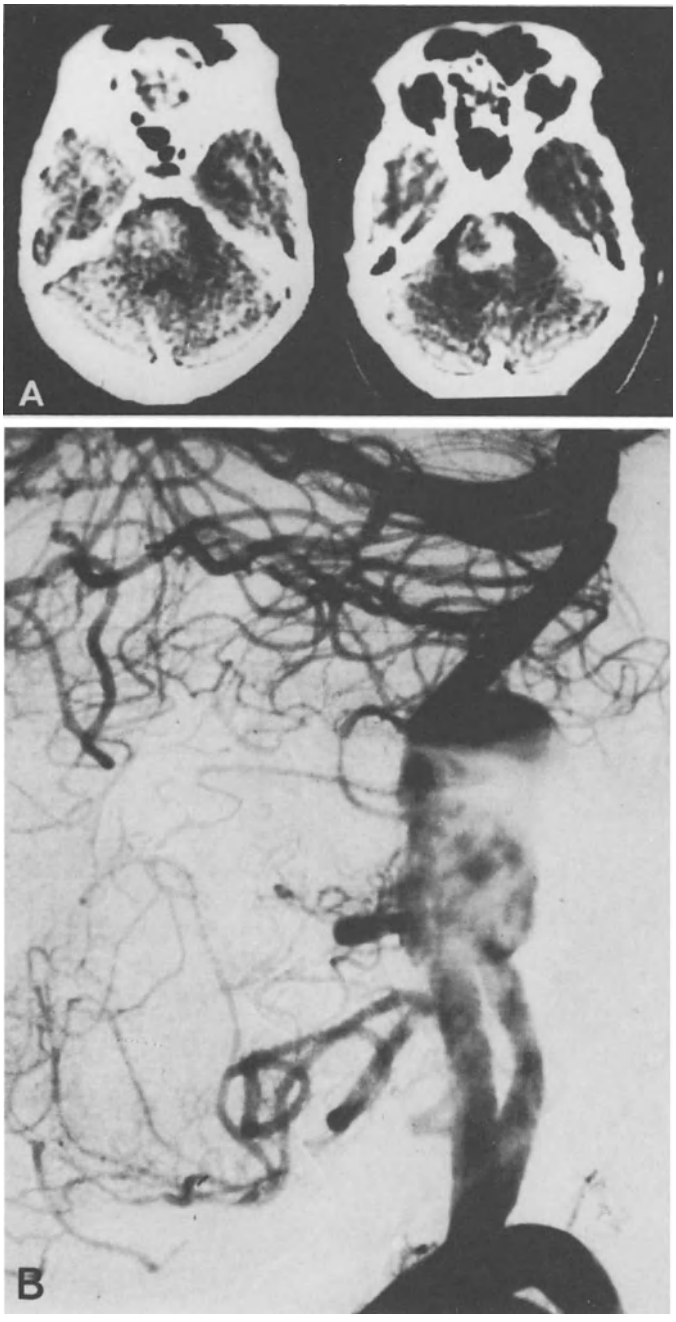


Fig. 10. A CT scan shows a mass of increased density in the region of the pons which shows patchy contrast enhancement. The 4th ventricle is flattened and displaced posteriorly. B Vertebral angiogram shows a largely thrombosed giant sac of a basilar aneurysm

# Intracerebral Space-Occupying Lesions With Brain Density (Isodense Lesions)

TH. GRUMME, K. KRETZSCHMAR, G. EBHARDT, W. LANKSCH, and S. LANGE

Intracranial space-occupying lesions can be demonstrated directly in the CT scan as a result of differences in density between these lesions and adjacent brain tissue, or they may be demonstrated indirectly through the presence of perifocal cerebral edema or shifts in such intracranial structures as the internal and external C.S.F. spaces and physiological calcifications. Diagnosis of an intracranial mass lesion can be extremely difficult if these criteria are not fulfilled in the CT scan.

Before we discuss isodense space-occupying lesions as the latter appeared in the material compiled by the CT study groups in Berlin, Mainz and Munich, a few preliminary remarks are in order. Tumors and hemorrhages smaller than 1.5 cm in diameter may escape detection in the CT scan, because they may not be depicted in a single slice and because density resolution may not be adequate. We cannot classify such lesions as isodense, since their real density is not measured. Nor is it permissible to classify tumors as isodense when the CT scans themselves are inadequate and the tumors escape direct demonstration.

Brain tumors, chronic subdural hematomas, acute complications of head injuries and intracerebral hemorrhage will be discussed in the following report.

## Brain Tumors

Table 1 shows that, of 2581 brain tumors, 403 (15.6%) appeared isodense in the plain CT-scan. However, in more than half of these cases, perifocal edema and/or evidence of a shift in normal brain structures suggested the presence of a space-occupying lesion. It was impossible to demonstrate the lesion in the plain CT scan in only 166 cases (6.4%). One ml of a 60%-66% contrast medium per kg of body weight was injected, and this procedure raised the percentage of tumors demonstrated directly, so that finally only 42 tumors (1.7%) escaped detection in the CT scan. The tumors which proved to be too small or which were inadequately scanned are included in this figure. The pilocytic astrocytomas are most frequently isodense in the plain CT scan (19.7%), followed by medulloblastomas, anaplastic (grade II) astrocytomas and glioblastomas each with 13%-16%. Low grade astrocytomas, oligodendrogliomas and ependymomas can be recognized with the highest degree of certainty. Brain stem gliomas are isodense in 15% of cases and can be suspected because of the distended pons, even in the rare cases in which they fail to take up contrast medium.

Acoustic neuromas appear isodense in 52.5% of cases in the plain scan and cannot be recognized at all, not even through indirect signs, in 40.5% of cases. All tumors with a diameter of 1.5-2.0 cm and more were demonstrated after contrast enhancement. All of the undetected acoustic neuromas were smaller than 15 mm in diameter. Meningiomas appear isodense in 13.6% of cases, and this behavior is especially true of meningiomas of the posterior fossa. Eleven meningiomas escaped detection. The correct slice was missed in five cases, and six tumors at the base

Table 1. Isodense absorption of tumor tissue in 2581 patients with brain tumors

| | No. of patients | Plain CT-scan | | | | Contrast-CT-scan | |
|---|---|---|---|---|---|---|---|
| | | Isodense absorption | | Tumors not detected | | Enhancement negative; Tumor not detected | |
| | | n | % | n | % | n | % |
| Astrocytoma Grade I | 121 | 1 | 0.8 | - | - | - | - |
| Astrocytoma Grade II | 109 | 18 | 16.5 | 5 | 4.6 | - | - |
| Glioblastoma | 523 | 65 | 12.4 | 14 | 2.6 | 2 | 0.4 |
| Oligodendroglioma | 117 | 5 | 4.2 | - | - | - | - |
| Pilocytic astrocytoma | 76 | 15 | 19.7 | 4 | 5.2 | - | - |
| Ependymoma | 37 | 3 | 8.1 | - | - | - | - |
| Medulloblastoma | 51 | 7 | 13.7 | 3 | 5.8 | 1 | 1.9 |
| Neuroma | 138 | 72 | 52.2 | 56 | 40.5 | 17 | 12.3 |
| Hemanioblastoma | 30 | 2 | 6.6 | 1 | 3.3 | - | - |
| Meningioma | 410 | 56 | 13.6 | 31 | 7.5 | 11 | 2.7 |
| Pituitary adenoma | 243 | 47 | 19.3 | 13 | 5.3 | 1 | 0.4 |
| Craniopharyngioma | 67 | 5 | 7.4 | 4 | 5.2 | 1 | 1.5 |
| Metastases | 363 | 60 | 16.5 | 15 | 4.1 | 5 | 1.3 |
| Other brain tumors | 185 | 20 | 10.8 | 7 | 3.7 | 3 | 1.6 |
| Unknown histology | 111 | 18 | 16.2 | 13 | 11.7 | 4 | 3.6 |
| Total | 2581 | 394 | 15.2 | 166 | 6.4 | 45 | 1.7 |

of the skull and at the cerebellopontine angle were too small for detection, as they measured less than 15 mm in diameter. Isodense in the plain scan were 19.3% of pituitary adenomas, but pathological changes in the region of the so-called pentagon suggested the diagnosis of a tumor in all but 5.3% of cases. One tumor appeared isodense, even after contrast enhancement and escaped detection. Craniopharyngiomas were isodense in plain scan in 7.4% of cases, only one tumor remained undetected after contrast enhancement; a cystic craniopharyngioma with isodense contents was found at operation.

Metastases appear to have the same density as normal brain tissue in the plain scan in 16.5% of cases. Five tumors went undetected. The correct slice was missed in two cases, two metastases were too small, and the fifth case involved diffuse cerebellar carcinomatosis. Multiple metastases are often so small that they escape detection. It is likely that the percentage of undetected brain metastases is greater than our material would suggest. A summary of all tumors that escaped detection in the CT scan is presented in Table 2.

The question of whether a tumor in an early phase of development will continue to appear isodense after contrast enhancement is still unresolved after more than 50 patients with follow-up studies. Certainly a number of tumors classified initially as normal findings, remain undetected because of lack of follow-up studies. Recurrence of a surgically treated glioma can fill a surgical defect with isodense tumor material. The importance of contrast enhancement cannot be estimated.

## Chronic Subdural Hematoma

Table 3 provides a summary of 142 chronic subdural hematomas, of which 31.7% were isodense. One may assume a unilateral isodense chronic subdural hematoma when the CT-scan fails to show contrast enhancement in the presence of a space-occupying lesion which does not differ in density from brain tissue. Membranes are demonstrated only in rare cases. Angiography is recommended if the CT findings are questionable. In three cases, supposed chronic subdural hematomas proved to be a carotid artery occlusion, a brain tumor in early stage and diffuse posttraumatic cerebral edema of one hemisphere respectively. Bilateral isodense chronic subdural hematomas pose special problems, for they may remain undetected if a midline shift is not present. We observed two cases of this type. Often bilaterally compressed frontal horns can be observed in such cases and can lead to correct diagnosis. GRAU speaks of the rabbit ear phenomenon.

Table 2. Undetected tumors

| | |
|---|---|
| Glioblastoma | 2 |
| Medulloblastoma | 1 |
| Neuroma | 17 |
| Meningioma | 11 |
| Pituitary adenoma | 1 |
| Craniopharyngioma | 1 |
| Other tumors | 7 |
| Metastases | 5 |
| | 45 |

Table 3. Isodense absorption in 142 patients with chronic subdural hematoma

| n | Isodense absorption n | % | Bilateral; isodense absorption; not detected |
|---|---|---|---|
| 142 | 45 | 31.7 | 2 |

## Acute Head Injuries

Acute extracerebral hematomas pose a diagnostic problem only in exeptional cases. In one patient with a serious coagulation disorder, almost the entire fronto-temporal extradural hematoma appeared isodense, since the blood was not coagulated.

Ninety-eight patients with head injuries showed a generalized increase in brain volume in the CT scan as a result of diffuse traumatic cerebral edema, recognizable by compression or obliteration of the ventricles and cisterns. The brain tissue appeared homogeneously grey, with normal absorption values.

## Intracerebral Hemorrhage

CT diagnosis of a freshly coagulated intracerebral hemorrhage presents no special problems. However, the situation changes considerably in the course of a week. Resorption in and around the hematoma may cause inhomogeneous changes in density which can render the differential diagnosis between hemorrhage, tumor and infarction considerably more difficult, especially if the first CT studies are made 10 days or more after the event. In a large percentage of cases, an intracerebral hemorrhage will pass a phase in which the hemorrhage appears to have the same density as adjacent brain tissue.

It may be impossible to recognize an older intracerebral hemorrhage if there is no accompanying edema or if there is no perifocal resorption zone. Both conditions seem to be rare. This is documented by a case of recurrent cerebellar hemorrhage. The CT scan was performed 14 days after the first and second days before the second, fatal, hemorrhage. It was not possible to demonstrate the old hemorrhage, since it had the same density as the adjacent brain tissue. Similarly, intracerebral hemorrhages which develop as postoperative complications may escape detection in the CT scan. We have observed 460 intracerebral hematomas and we have missed only 12 isodense hemorrhages as far as we know. The actual number could be greater, however, since follow-up CT studies were not made routinely, and operation or autopsy was not performed in every case.

## Concluding Remarks

Direct demonstration of lesions which alter tissue density has undoubtedly added a new dimension to the diagnosis of neurosurgical diseases. However, it must also be understood that cerebral lesions with the same density as normal brain tissue pose new diagnostic problems.

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# Ring-Shaped Lesions in the CT Scan – Differential Diagnostic Considerations

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## Ring Blush in Brain Tumors and Abscesses

Ring-like appearances in the contrast-enhanced CT scan are a common finding with various intracranial space-occupying lesions and must be regarded as a nonspecific finding. This CT pattern is found most frequently in *brain abscesses* (86%), followed by *gliobastomas* (54%). Anaplastic astrocytomas, pilocytic astrocytomas, and metastases relatively often present as ring-shaped lesions in the CT (see Table 1).

Apart from low-grade gliomas *nearly all types of intracranial tumors* may occasionally show a ring blush, thereby making harder the correct interpretation. Thus, the very first meningioma of our series appeared as a ring caused by central liquefaction of tumor tissue (1). Plain skull films helped to identify the nature of the lesion since an enostosis and an enlarged channel of the middle meningeal artery were present. Among 410 patients with proven *meningiomas*, a ring-like CT pattern could be observed in 2% of the cases. Such menin iomas may easily be confused with a glioblastoma.

Another tumor type in which one would expect homogeneous contrast medium uptake, the *acoustic neuroma*, not so rarely presents a central lucid area. According to our observations, this was usually caused by cystic degeneration or, as in one case, by an old liquefied hematoma within the tumor. From the viewpoint of differential diagnosis, metastasis must also be considered in such cases, especially in older people.

With *ring-like lesions in the cerebellum*, the age of the patient will contribute to the correct prediction of the underlying process. In

Table 1. Incidence of ring-shaped CT pattern in brain tumors and brain abscesses

| Diagnosis | No. of patients studied by CT | Ring blush | |
|---|---|---|---|
| | | n | % |
| Pilocyt. astrocytoma | 76 | 25 | 33 |
| Anaplast. astrocytoma | 109 | 25 | 22.9 |
| Oligodendroglioma | 117 | 12 | 10.3 |
| Glioblastoma | 523 | 282 | 53.9 |
| Ependymoma | 37 | 5 | 14 |
| Neurilemmoma | 138 | 13 | 9.4 |
| Haemangioblastoma | 30 | 4 | 13 |
| Meningioma | 410 | 8 | 2.0 |
| Pituitary adenoma | 243 | 12 | 4.9 |
| Craniopharyngioma | 67 | 4 | 6 |
| Other tumors | 468 | 24 | 5.1 |
| Metastases | 363 | 102 | 28.1 |
| Total | 2581 | 516 | 20.0 |
| Brain abscess | 44 | 38 | 86 |

children and adolescents pilocytic astrocytoma is the most probable diagnosis, since medulloblastomas present a similar feature only in exceptional cases. Among 51 patients with medulloblastomas this occurred only twice. In middle age, a haemangiomblastomas may appear as a ring (3). Four out of 30 cases exhibited such a finding (see Fig. 1). The attenuation values of the central hypodense, cystic part of the tumor are similar to those of necrotic tumor tissue or abscess contents. In older people again metastasis should be considered first. In one case of our series, a cerebellar infarct had caused a similar appearance after contrast enhancement.

In the *suprasellar region, pituitary adenomas as well as craniopharyngiomas* may show ring-like enhancement if central cysts or old liquefied hematomas are present. The differentiation, however, is possible by looking at the endocrinological status and the configuration of the sella. In hypothalamic astrocytomas, only rarely a symmetric ring-like pattern can be observed which may be confused with one of the above-mentioned tumor types.

## Differentiation Brain Tumor - Abscess

To our experience, in adults the *differentiation between glioblastoma and brain abscess* poses a great problem in CT diagnosis (2, 6). Without knowledge of the history, a certain CT picture showing a ring-like lesion cannot be interpreted as an abscess with absolute certainty in many cases. The qualitative assessment of the CT findings on the postcontrast scans reveals a wide variety of CT patterns with glioblastomas as well as with anaplastic astrocytomas, metastases and brain abscesses. However, a scalloped configuration of solid tissue, representing a subgroup of ring-shaped lesions, seems to be most typical of glioblastomas (6). Neither a thin enhanced circular or non-circular rim nor a thick enhanced rim is typespecific for any of the lesions under discussion. In agreement with NEW and co-workers (5), we observed that irregularity of the thickness of the enhanced rim is a CT finding which is uncommon with abscesses with the exception of multichambered abscesses. On the other hand, a certain constant thickness of the rim does not speak only in favor of an abscess. According to our experience, solid parts of the rim projecting into the central translucent part of the lesion point to a tumor.

Multiplicity of ring-type lesions does not always facilitate the differentiation since glioblastomas may also occur multifocally, not to mention multiple metastases.

The *quantitative evaluation* of contrast enhancement of solid tumor tissue in glioblastomas or metastases as well as of the capsule of an abscess did not reveal significant differences in our experience. For all these tissues, the average increase in attenuation ranged from 4 to 7 EMI units, 8 to 14 Hounsfield units respectively. The attentuation values of the central, more lucent parts of the lesions, on the one hand necrotic tumor tissue, on the other hand pus, also did not differ markedly from each other (7). The EMI units of these central structures ranged from +9 to +12 in the average. For the new Hounsfield scale these figures have to be doubled. Thus the final diagnosis is often based more upon clinical data than on the CT appearance. Carotid angiography will remain irreplaceable in most cases.

A very small rim can be observed in some anaplastic astrocytomas, occasionally also in children with supratentorial ependymomas or sarcomas.

## Infarction and Intracerebral Hematoma

*Infarcted areas* only seldom show ring-like contrast medium uptake. In most cases, a somewhat irregular garland is visible, representing the brain cortex which takes up contrast because of the breakdown of the blood brain barrier (8). However, in the individual case it may be difficult to distinguish such a lesion from a tumor.

According to the literature intracerebral hematomas in the resorptive phase may present a ring blush around the isodense or hypodense hematoma (9). This would not be unexpected because of the blood-brain barrier disturbance in a certain stage of this disease. In our own series we have more than one patient in whom such an observation has been made, probably since we have not studied many intracerebral hematomas with contrast enhancement at follow-up studies.

## Postoperative Ring Blush

In *early postoperative CT controls* we have been confronted with many cases with ring blushes, especially between the second and the sixth week after the neurosurgical intervention. It turned out that the origin of such rings is not uniform. At first we thought that this CT finding represents an abscess within the former tumor cavity. Accidentally, the first two observations indeed showed an abscess at reoperation.

During the last years we learned that a *ring structure in the postoperative CT is usually a sign of a completely normal course* (4). In the patient whose CT scans are shown in Fig. 2, we had removed an anaplastic astrocytoma from the left precentral region 6 weeks before. The CT picture showed a ring blush and perifocal edema (Fig. 2a). By the clinical course we have not been urged to proceed more actively in this case. A second CT follow-up study two months later exhibited only a small area of decreased attenuation as a result of tumor removal (Fig. 2b). No contrast medium uptake could be observed. The explanation of this typical finding lies in a temporary breakdown of the blood-brain barrier at the margin of the operative field and in productive and resorptive mechanisms.

In one case of postoperative ring blush we had the opportunity to study the lesion directly. This was a patient, who had been operated on a fibrillary astrocytoma in the left fronto-precentral area, and developed aphasia and motor paresis of the right arm four weeks after the removal of the tumor. Assuming this to be an abscess from the CT picture, we decided to reopen the bone flap and to inspect the situation. To our surprise we could only find encapsulated xanthochromic fluid - obviously CSF - under high pressure within the former tumor bed. The wall of the cavity was covered by a brownish green spongy hypervascularised granulation tissue, thereby explaining the CT finding.

## Conclusions

Computerised tomography has really opened new dimensions for the diagnosis of various intracranial space-occupying lesions. However, during the last years we had to learn how difficult it can be to evaluate a certain CT finding correctly. Especially the group of ring-type lesions still pose some unsolved problems even if clinical information available in the individual case is considered. The ring blush is a nonspecific finding which occurs in primary and metastatic neoplasms, abscess, infarction, certain stages of intracerebral

hematomas and even after neurosurgical operations. The ring blush is caused partly by breakdown of the blood brain barrier, partly by hypervascular pathologic tissue or by both factors.

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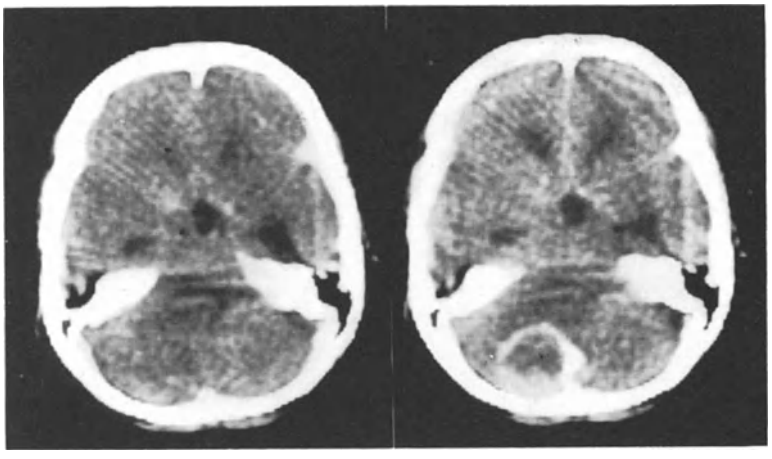

Fig. 1. Ring blush in case of haemangioblastoma. The left CT scan is before contrast administration (Pat. Helga K., 38 y., CT-Nr. M 6996/77)

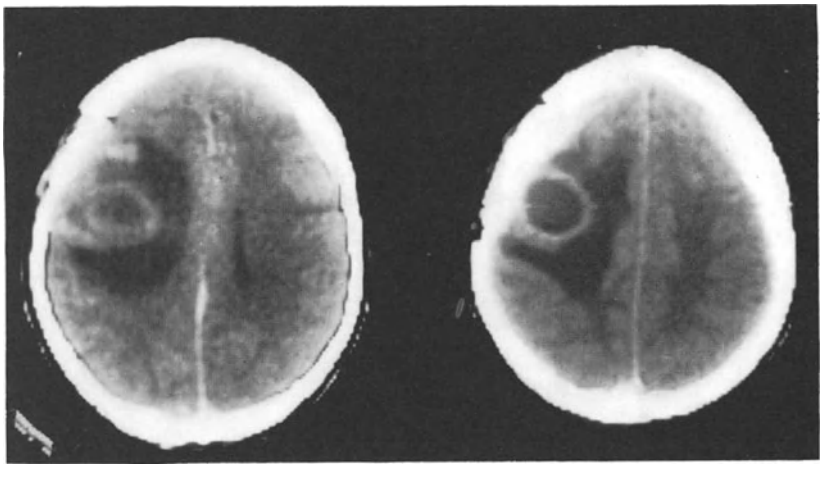

Fig. 2a, b. Postoperative ring blush after removal of an anaplastic astrocytoma in a 37-year-old female. a 6 weeks after operation a ring blush is present due to temporary breakdown of the blood brain barrier and hypervasluclar granulation tissue

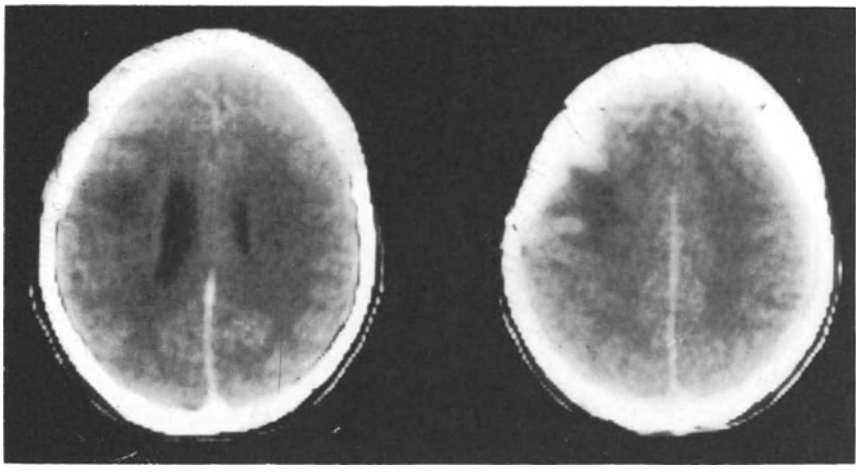

Fig. 2b. Two months later nearly complete normalisation, no contrast enhancement (by courtesy of Dr. Backmund, Max-Planck-Institute of Psychiatry, Munich)

# Diagnostic Errors in the Interpretation of Cerebral Infarction

CH. B. OSTERTAG and F. MUNDINGER

## Introduction

Cerebral infarcts resulting from long past vascular occlusions present no difficutlies in diagnosis (5) by computer-tomography. The diagnosis of recent vascular occlusions, on the other hand, poses greater problems in computed tomography (CT) because of the large numer of possible alterations and the danger of misinterpretation.

## Material and Method

Among more than 8,300 CT examinations, 226 cases were diagnosed as "cerebral infarction." The majority of these 226 infarcts (n = 140) were clearly older than 28 days, according to the symptoms and the computer-tomographic picture. Eighty six cases had to be regarded as recent infarcts. On the basis of different CT alterations, the recent infarcts were subdivided into two groups: the first group consisted of those which were examined up to 7 days after the infarct occurred; in the second group, the infarct dated back 8-28 days before the examination (the validity of this classification was established on the basis of further clinical development).

The examinations were carried out with an EMI scanner Mark I, matrix 160 x 160. Although no contrast medium was used as a rule, when the infarct was assumed to be older, a native scan as well as a scan with a contrast medium were made (Conray 60% 100 ml infused intravenously) in the case of a recent infarct.

## Results

### *1. Typical Findings in the Case of Cerebral Infarction*

(Table 1): An older infarct shows a CSF-filled defect caused by tissue necrosis. As a rule, this is sharply demarcated and its borders coincide with an arterial vascular supply area. Neighboring ventricles and sulci are frequently drawn out (Fig. 1, 2).

Table 1. Time dependence of typical CT findings in cerebral infarcts

| | 1 week (n = 36) | 2-4 weeks (n = 50) | 4 weeks (n = 140) |
|---|---|---|---|
| Mass effect | 52% | 22% | - |
| Low density | 36% | 76% | - |
| Enhancement | 16% | 40% | - |
| Vascular-distribution | 20% | 40% | 95% |
| Cavitation | - | - | 90% |

A recent acute cerebral infarct (1-7 days) as well as a subacute cerebral infarct (8-28 days) show, on the other hand, signs of a space-occupying lesion and usually only an unevenly demarcated zone of slightly reduced density or even isodense values. The high lipid content of subacute infarcts appears to be the most important histological factor for attenuation reduction (4). Isodense values of absorption, the cause of nonvisualization, are possibly a symptom of a reactive blood flow. The transition into a necrosis is marked by a renewed hypodensity caused by lipid, protein and water storage (Fig. 3) (1, 2, 4).

The borders of a necrosis show increased density values in the form of a ring-like structure indicating a blood brain barrier disturbance. This does not occur until after contrast medium has been administered. However, contrast media can also make an infarcted area that was originally hypodense appear to be isodense. Recent infarcts, therefore, must always be examined first by means of a native scan.

Only in approximately 10% of the patients who were examined within the first week did we find hemorrhagic infarcts. These are not difficult to detect because of the increased density values due to blood coagulation (Fig. 5a).

### *2. False Negative CT-Results*

20% of the patients who displayed clinical symptoms of an infarct showed no recognizable density alterations within the first week in comparison with normal cerebral tissue. Approximately 10% also showed no density alterations in the weeks that followed. The infarcted tissue area remained isodense compared with healthy cerebral tissue, and thus was not identifiable. Moreover, symptoms of the space-occupying lesion do not necessarily have to appear in the first 4 weeks. The percentage of recent infarcts that are not detected at the beginning is 12%.

### *3. Misinterpretations of Cerebral Infacts* (Table 2)

Whereas isodense infarcts are frequently overlooked, hypodense as well as as hyperdense CT alterations occasion typical misinterpretations with a frequency of 8%. Hypodense lesions without sharp demarcation showing symptoms of a space-occupying lesion are also characteristic for focal, infectious and inflammatory lesions (encephalitis, Fig. 4c), gliomatous tumors (astrocytomas, Fig. 4b) and leukemic-lymphatic infiltrations of the brain. Occasionally a hypodense zone can be found underneath an isodense subdural hematoma.

Hyperdense focal lesions can indicate an intracerebral and subarachnoid hemorrhage, bleeding into a malignant glioma, a metastasis, an a.v. malformation, or an hemorrhagic infarct (Fig. 5). Ring-shaped hyperdense zones with centrally reduced density are also found in the case of cerebral abscesses, small glioblastomas and metastases (Fig. 5c, d, Fig. 6).

## Discussion

The knowledge of the extent and site of the infarcted area is indispensable for the diagnosis as well as for the prognosis. CT is currently the appropriate, i.e., the non-invasive examination. But CT alterations observed in recent infarcts show a changing pattern and inconstant

Table 2. Common pitfalls in the diagnosis of cerebral infarcts

| |
|---|
| *1. Isodense lesions* |
| false negative CT results |
| *2. Low density lesions* |
| - Gliomas (Astrocytoma) |
| - Encephalitis |
| - Leucaemic-lymphatic infiltrations |
| *2. High density lesions* |
| - intracerebral hematoma |
| - a.v. malformation |
| - focal inflammatory lesion |
| - brain abscess |
| - glioblastoma (necrotic) |
| - metastases |

characteristics up to the fourth week of onset: mass effect and low density areas are common, a definable vascular distribution is not always present; after enhancement there are round, triangular or ring-like areas of hyperdensity; hemorrhages may be present. There characteristics are similarly found in many other tumorous and nontumorous cerebral lesions (Table 2). The differential diagnosis must distinguish between infarct and common cerebral lesions such as gliomas, metastases, intracerebral hematomas, brain abscesses and those lesions infrequently found: demyelinizing disease, focal atrophy due to head injury, viral encephalitis, subarachnoid hemorrhage, arterio-venous malformations, subarachnoid cysts and inflammatory lesions. The patterns of the negative scan as well as of the enhanced scan are equally variable. Fortunatly a typical clinical history will frequently obviate the need to establish the definitive diagnosis based on the CT scan alone. The CT diagnosis can only be corroborated by taking the anamnestic data and the clinical development into account. CT follow-up examinations in 2-3 week intervals, isotope diagnosis, and possibly angiography assist in establishing a probable diagnosis. Although a time dependence of typical CT findings is evident (Table 1), the numer of misinterpretations (8%) and of false negative results (12%) is nevertheless high and corresponds with those found by other investigators (3, 6). A cerebral infarct may not be ruled on the basis of a single CT examination. Tissue necrosis can only be excluded after consecutive examinations with native scan and enhancement. Technical factors (incomplete scan series, incorrect window width, artefacts due to motion, exclusively transverse scans that do not completely cover a supply area) contribute to an increased number of false diagnoses of recent infarcts.

Summary

Acute and subacute infarcts show a changing pattern and inconstant characteristics up to the fourth week of onset. There is a wide spectrum of other lesions presenting identical alterations in the CT scan. These include tumorous lesions, mostly gliomas (glioblastomas, astrocytomas), demarcating inflammatory lesions (abscesses, viral encephalitis), metastatic lesions, resolving intracerebral hematomas, subdural hematomas, subarachnoid hemorrhages and a.v. malformations. False negative results are frequently found in cases of recent infarcts

that present isodense lesions without mass effect. Only consecutive CT scans - native as well as with enhancement - and other diagnostic procedures can help to establish a definite diagnosis.

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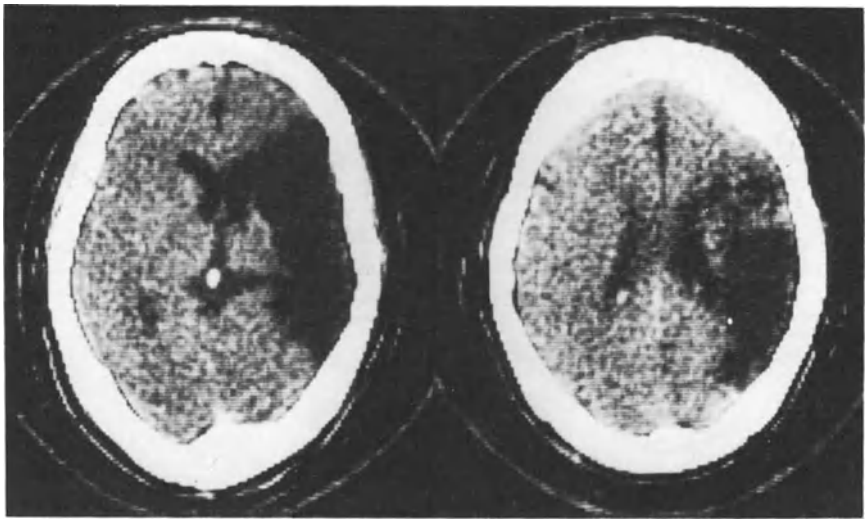


Fig. 1. Demarcated infarct of right middle cerebral artery

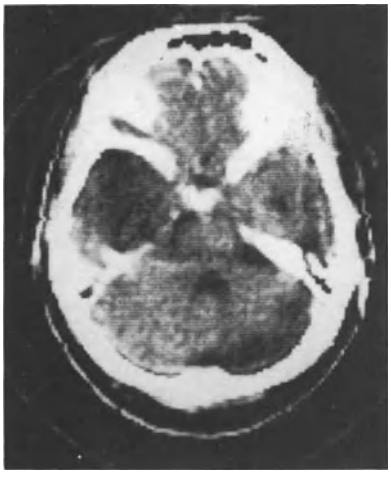


Fig. 2. Infarct of right posterior inferior cerebellar artery

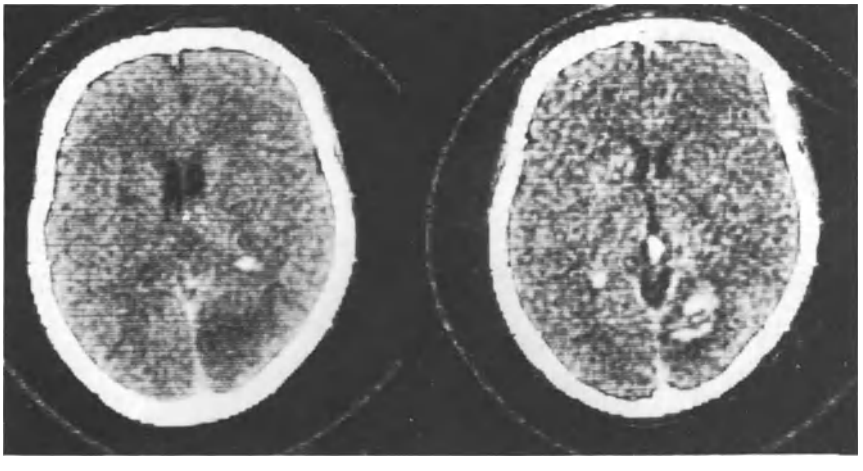


Fig. 3. Acute infarct of cortical branches of right posterior cerebral artery with sudden homonymous hemianopia. *Left*: CT scan 48 hours after acute onset of symptoms; *right*: CT control scan 4 days later

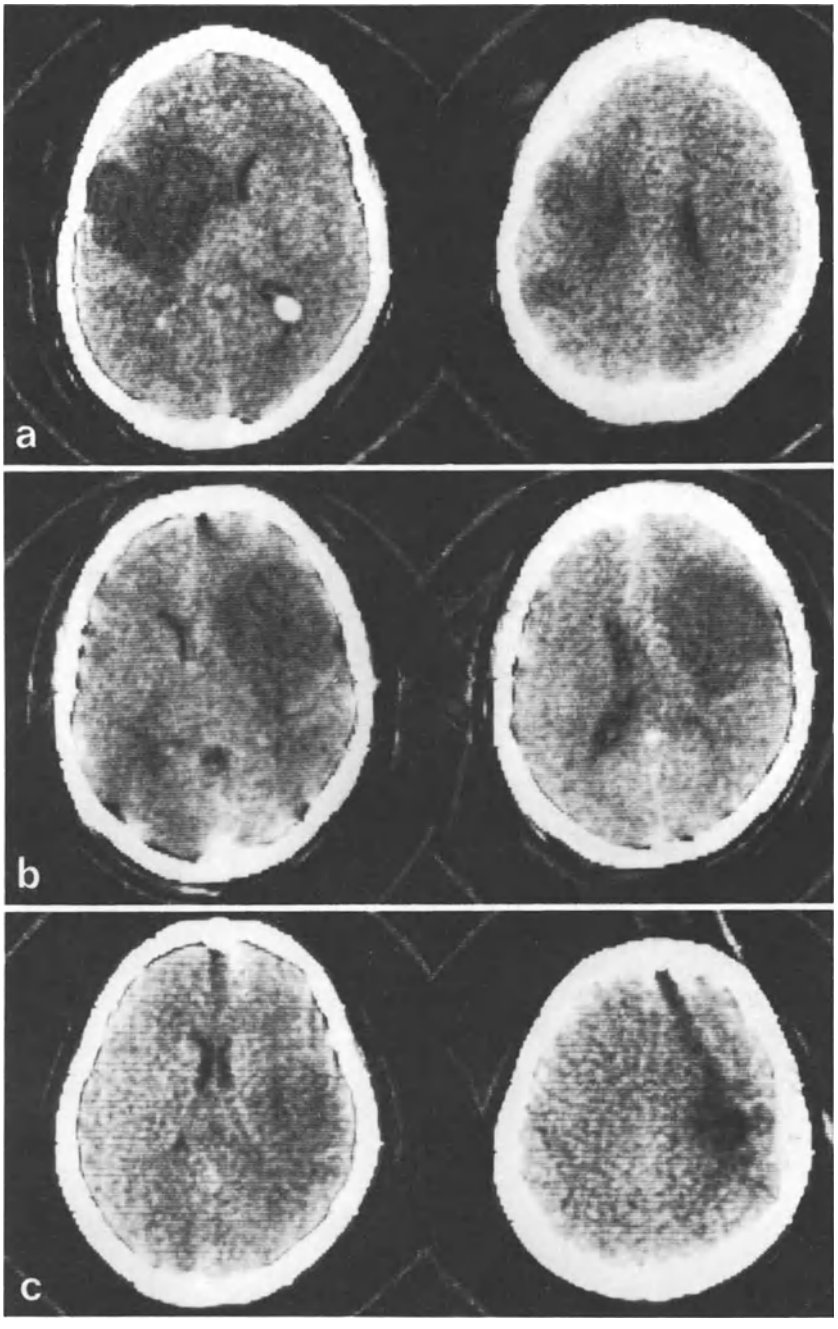



Fig. 4. Hypodense lesions with mass effect (proven by autopsy). a Acute infarct of the left hemisphere; b Fibrillary astrocytoma of right fronto-temporal region; c Lymphatic leukemia, cerebral infiltration

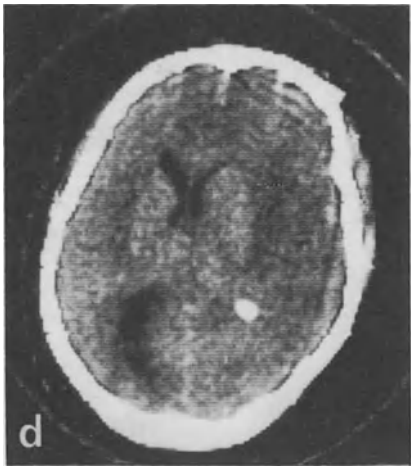
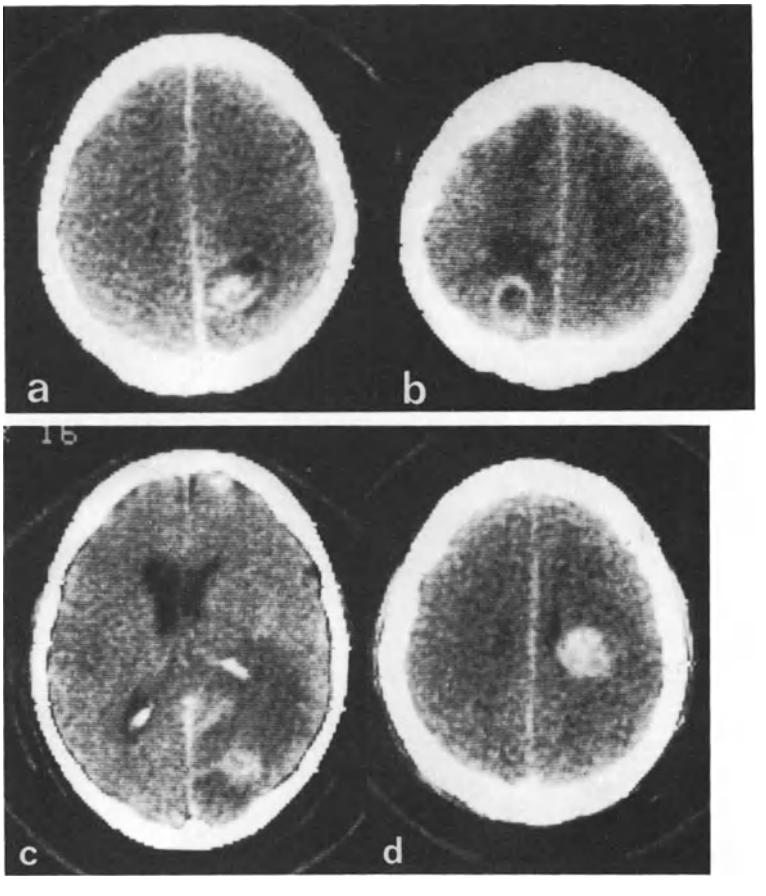



Fig. 4d. Viral encephalitis of right temporal region



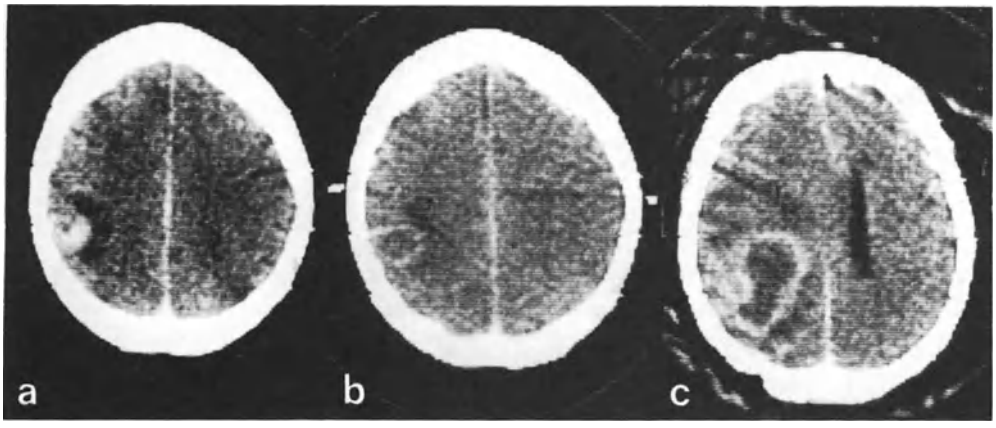



Fig. 6. Follow-up of a glioblastoma, primarily misdiagnosed as an infarct: a first CT scan 10 days after acute onset of seizures; b Ring formation 25 days later, supporting the diagnosis of an infarct; c Three months later, findings of a glioblastoma were evident and proven by biopsy

◁

Fig. 5. Hyperdense lesions: a Ring formation of a hemorrhagic infarct of parieto-occipital artery; b Small solitary brain abscess; c Small solitary metastasis with marked mass effect; d Intercerebral hemorrhage from an arteriovenous malformation

# A Clinical Study of the E.M.I. Scanner With Implications for the Future Distribution of Neurological Services

J. R. BARTLETT and G. NEIL-DWYER

## Introduction

The installation of an EMI Scanner at the Brook General Hospital in February 1976 provided a unique opportunity to study the scanner as the primary tool for the investigation of intracranial disease. The retirement and resignation of all the consultant neuroradiologists ensured that only those angiograms, air encephalograms, and ventriculograms that were absolutely necessary for patient management could be carried out. As in other units there was a reduction in the number of contrast investigations performed but the extent to which this was possible, without reducing the standard of patient care, surpassed expectations. We found that the accurate demonstration of distorted anatomy and the alteration of densities when interpreted in the light of a careful history and physical examination was often sufficient evidence on which to plan treatment. This paper analyses the change in demand for contrast investigation and the resultant implications for the provision and organisation of neuroradiology.

## Clinical Material

The records of 570 patients, admitted to the Neurosurgical Unit in the year immediately following the installation of the scanner, were examined.

## Method of Analysis

The patients were divided into the following groups according to their clinical presentation: 1) Intracranial tumour; 2) vascular disease; 3) head injuries; 4) intracranial infection; and 5) a group which included degenerative disease, congenital abnormalities, orbital tumours, disseminated sclerosis and so forth.

The number of neuroradiological contrast procedures that would have been carried out on each patient without access to the scanner was estimated. These results were compared with the actual number of investigations performed during this period.

It is possible that the estimated number of contrast investigations was high; therefore, the estimate was compared with the figures for 1974 and 1975 (Table 1). There is no significant difference.

A few patients were excluded on the grounds that the scan had been performed for the purposes of the evaluation of the equipment or self-instruction. Some were excluded where there were insufficient grounds for carrying out a contrast procedure. The remained, the large majority, were divided into two groups: those with positive and those with negative scan findings. Two tables were constructed in which the estimated number of investigations required without a scanner are compared with what was actually done.

Table 1. Comparison of estimate for the scanner year with figures for 1974 and 1975

| The number of intracranial contrast investigations per patient | |
|---|---|
| 1974 | 1.677 |
| 1975 | 1.504 |
| Estimate (scan year) | 1.567 |

## Results

Table 2 shows the percentage reduction in contrast radiology correlating the provisional diagnostic category and scan finding.

Table 3 shows the percentage reduction of special test correlated with the scan findings.

Table 4 shows the effect the scanner has had on the work of the Regional Neurosurgical Unit at the Brook Hospital.

## Discussion

The division of findings into positive and negative groups is of particular importance where the scan findings are positive. If the scan

Table 2. Percentage reductions of special tests correlated with scan findings

| | Positive scan | Negative scan | Total |
|---|---|---|---|
| Angiography | 69 | 37 | 61 |
| Air studies | 85 | 96 | 90 |
| Isotope studies | 98 | 95 | 97 |
| Exploratory burr-holes (head injuries only) | Definitive procedures only | 100 | 100 |

Table 3. Percentage reductions in contrast radiology correlating provisional diagnostic category and scan finding

| Provisional diagnostic category | Positive scans (%) | Negative scans (%) |
|---|---|---|
| Tumours | 86 | 87 |
| Vascular | 43 | 9 |
| Head injuries | 95 | 98 |
| Infection | 87 | 100 |
| Degenerative disease | 92 | 100 |
| Congenital abnormality | 64 | 33 |
| Miscellaneous | 68 | 83 |

Table 4. Effect of scanner and estimated space capacity

Population served: Neurosurgically 2 million
Neurologically 1.25 million

| | | Introduce Scanner | | | |
|---|---|---|---|---|---|
| | 1974 observed | Reduce by (%) | Number of tests required | Maximum no. of investigations reasonable in each room | Percentage utilisation (%) |
| Angiograms | 875 | 50 | 437 | 1.500 | 29 |
| Air studies and ventriculograms | 260 | 90 | 26 | 750 | 3.5 |

examination determined only which patients required contrast investigation, there would be no reduction in the number of radiological tests performed in this group. A significant reduction indicates that patients were treated on the basis of the scan findings.

In the group with negative scan findings, patients presenting symptoms of vascular disease show the reduction in the requirement for other tests is small, that is, only 9%. However, it is worth emphasising that in cerebrovascular disease, angiography is the definitive investigation required to demonstrate treatable lesions. Patients with positive scans had fewer radiological contrast procedures than those in whom the scan was negative because the scanner often revealed changes which, taken with the clinical picture, indicated that no neurosurgical operation was either reasonable or feasible. In these patients it was possible to dispense with contrast investigations whereas a normal finding was generally followed by another procedure.

Table 3 shows that a) isotope studies have been virtually abandoned and b) no burr-hole was made for the diagnosis of an intracranial haematoma following head injury during the year.

By providing knowledge of the nature of the intracranial event in the head injured patient, the scanner has made it possible to dispense with exploratory burr-holes for intracranial haematoma; this is a change of management which is not possible without a scanner.

Table 4 shows the impact on demand for conventional contrast neuroradiology at the Regional Neurosurgical Unit. This unit serves a population of two million people. In 1974, before the installation of the EMI Scanner, 875 angiograms and 260 air studies were performed. From the evidence of our study it is possible to reduce the number of angiograms by more than 50% and reduce the number of air studies and ventriculograms by at least 90%. The number of tests required in this population is reduced to 437 and 26 respectively. The maximum number of tests possible in a room is based on the assumption that three air studies and six angiograms are reasonable daily workloads. Before the installation of the scanner at the Brook Hospital these numbers were often exceeded. The percentage utilisation of specialist rooms with the arrival of the scanner is now 29% for angiography and 3.5% for air studies and ventriculograms. Therefore, a single neuroradiological department, provided with adequate computerised axial tomography scanning capacity, could serve a population of at least four million people.

These observations have important implications for the organisation of neuroradiology.

## Conclusion

The introduction of the CAT scanning service leads to a reduction in the requirement for angiography and air studies. The magnitude of the change implies that fewer departments are required. If the number of departments were reduced there would be cash savings. It is suggested that the cash savings are spent by introducing CAT scanners to major general hospitals. This could have two important effects: 1) a proportion of the patients can be investigated in the general hospitals; 2) more efficient use of expensive specialist services becomes possible.

# The Correlation Between E.M.I. Scan Appearances and the Pathologic Findings in a Small Group of Patients Submitted to Anterior Temporal Lobectomy for Intractable Epilepsy

C. E. POLKEY[1]

## Introduction

The criteria used to select patients with intractable temporal lobe epilepsy for the operation of temporal lobectomy are well known. The clinical history, neurological findings, and the results of neurophysiological, psychometric and neuroradiological investigations should all be consistent with a single or principal temporal lobe focus which is responsible for the patient's seizures. It has also been shown that when the resection is carried out in such a way that the mesial temporal structures are preserved for pathological examination, then those patient's in whom a definite pathology can be demonstrated do better than those in whom only nonspecific changes are found (1). Thus in FALCONER's series (1), when the lesion of mesial temporal sclerosis (MTS) was found, 60% of the patients became seizure-free, with other specific lesions 62%, but when only nonspecific changes were seen this figure reduced to 32%. Since the majority of these patients neurological examination is normal and very experienced clinical neurophysiologist find difficulty in being absolutely sure of structural changes on their records, any firm evidence of specific pathology prior to operation in the majority of cases must come from the neuroradiological findings. Methods used prior to the introduction of the EMI scanner were abnormal in only 60% of cases (2). It was hoped that computerised axial tomography (CAT) would help to resolve this problem and this small series of patients in whom the pathology is definitely known is presented to define a little the role of this technique in that report.

## Methods and Materials

A group of nine patients with intractable temporal lobe epilepsy, who, after investigation had 5-6 cm "en bloc" anterior temporal lobectomies carried out is described[1]. Their clinical details and the results of their investigations are summarised. All were selected for surgery on the basis described in the Introduction, but had, in addition, CAT scans carried out, three on CT 1000 machines, and the other six on CT 1010 machines. Enhancement was carried out on most occasions. The high accuracy and high definition options on the latter machine were used additionally on a few occasions, but were found to yield no further information. The resected temporal lobes were examined by Dr. I. JANOTA of the Department of Neuropathology of the Institute of Psychiatry.

Of the nine patients, six were adults over the age of 15, three men and three women, and three were children. All had suffered with temporal lobe or other minor seizures for at least five years, except for one child aged 3 years and 6 months, who had attacks for 3 years. Four of the nine had minor neurological signs on examination. In seven of the nine the electrical abnormalities were predominently in one temporal

1 My thanks are due to my colleagues in clinical neurophysiology, Drs. M.V. DRIVER and R. HARRIS, and in neuroradiology, Drs. R.D. HOARE and J. DAWSON, for their invaluable assistance in the investigation of these patients.

lobe with sphenoidal spikes. In two cases, both tumours, the electrical disturbance was more widespread but still indicated a structural lesion in one temporal lobe. In four of the nine the psychometric abnormalities were consistent with the laterality as suggested by other investigations. These details are summarised in Table 1.

## Results

The results of CAT scanning are best dealt with in relation to the pathology found and the other neuroradiological investigations. All nine resected specimens, five left-sided and four right-sided, contained a specific pathology. There were three patients with established MTS, three with tumours, and one with an angioma, one with cortical dysplasia, as described by TAYLOR et al. (3) and one in whom the hippocampal structures were placed too far anteriorly in the temporal lobe. The results of the investigations are summarised in Table 2. It can be seen that all nine had normal skull radiographs and that the results of lumbar air encephalography (AEG) and CAT scanning parallel each other with certain limitations which will be analysed in the discussion section.

## Discussion

There is now considerable experience of the assessment of ventricular size by CAT scanning, but this is chiefly related to the lateral ventricles. The temporal horns, by virtue of their size will be difficult to demonstrate. SHAH and NEWCOMBE (2) suggest 4 mm as the upper limit of normal for the width of the lateral cleft of the temporal horn. In addition, their proximity to the floor of the middle fossa and the fact that they lie in the plane of the slices also adds to this difficulty. RUGGERIO (4), in 100 negative CAT scans was only able to visualise the temporal horn in 18%. Furthermore, in 22 of the 73 patients described by SHAH and NEWCOMBE, the dilatation of the temporal horn on AEG was either bilateral or on the contralateral side to that operated upon. The three patients with MTS and the one with the anatomical anomaly all had dilated temporal horns on AEG. Where the

Table 1. Summary of clinical details

| Pathology | No. and Sex | Age at Opn. | Duration of seizures | Neurol. Exam. | EEG | Psychometry |
|---|---|---|---|---|---|---|
| Mesial | 3M | 12 | 10 | Positive | S.F.[a] | Consistent |
| Temporal | | 17 | 7 | Negative | S.F. | Inconsistent |
| Sclerosis | | 26 | 12 | Negative | S.F. | Not available |
| Tumour | 1F | 3.5 | 3 | Positive | M.F.[b] | Retarded (consistent) |
| | 2M | 14 | 9 | Positive | M.F. | Consistent |
| | | 16 | 5 | Negative | S.F. | Inconsistent |
| Angioma | 1F | 41 | 18 | Negative | S.F. | Consistent |
| "Cortical | 2F | 25 | 9 | Positive | S.F. | Normal |
| Dysplasia" | | 41 | 21 | Negative | S.F. | Consistent |

[a] S.F. = Single focus; [b] M.F. = Multiple foci.

Table 2. Correlation between pathology and neuroradiological findings

| Pathology | MTS N = (3) | Tumour N = (3) | Other N = (3) |
|---|---|---|---|
| *Skull XR* | | | |
| Normal | 3 | 3 | 3 |
| Calcification | 0 | 1 | 1 |
| *AEG* | | | |
| Dilated temporal horn | | | |
| Ipsilateral | 2 | 0 | 1 |
| Contralateral | 1 | 0 | 0 |
| Mass effects | 0 | 3 | 1 |
| Calcification | 0 | 1 | 1 |
| *CT scan* | | | |
| Visible temporal horn | | | |
| Ipsilateral | 2[a] | 0 | 1[a] |
| Contralateral | 1[a] | 0 | 0 |
| Mass effects | 0 | 3 | 2 |
| Calcification | 0 | 2 | 1 |

[a] This is based on any visualisation of the temporal horn, indicating its enlargement and all of these could be passed as normal.

temporal horn could be visualised on the CAT scan, it was always the same temporal horn which was dilated on the AEG. However, as can be seen in the accompanying figures, it was always difficult to visualise and these scans could all have been passed as normal and any action based on them alone would have been speculative. Even on occasions when the hippocampus was clearly shrunken from MTS as in Figure 1, this could not be demonstrated with the CAT scan.

In contrast, the pathology in the three patients with the tumours and the one with the angioma were easily seen on the CAT scans, mainly because of the increased density of three of the four lesions as seen in Figures 2 and 3, and the obvious mass effect in the fourth. In general, increased radiolucency is easily seen on CAT scans, and indeed in one case no calcification could be seen on any of the radiographs, and the other two lesions with calcification were less easily demonstrated on AEG, including tomography of the temporal horns. The patient with the cortical dysplasia, shown in Figure 4, presented the single false diagnosis in that all the preoperative radiography, including CT scan, AEG and arteriography, suggesting an indolent tumour. Studies of epileptics with CAT scanning is beginning to show that the proportion with identifiable pathology using this neuroradiological method is higher than with previous methods. GASTAUT and GASTAUT (5) report that the number of patients with positive findings rises from 30% to 55% and that the number of tumours discovered from 5% to 10%. The implications in selecting patients for surgery are important. First, there is a group of patients, represented by the child with the tumour, in whom the detection of a structural abnormality may lead one to view other investigations, and especially the EEG recordings, in a different light, having to distinguish between the primary and secondary electrical effects of such a lesion. Secondly, it can be seen that in patients with scarring of the hippocampi, as represented by MTS the CAT

scan may fail to reveal a lesion which is potentially curable. The CAT scan cannot, therefore, be used alone as a screening device for a population of epileptics to judge their suitability for surgery.

## Conclusion

In a group of nine patients undergoing anterior temporal lobectomy for intractable epilepsy, EMI scans are available for direct comparison with other neuroradiological investigations and the pathology found in the resected temporal lobes. The atrophic lesion of mesial temporal sclerosis is difficult to demonstrated by CAT scanning. However, lesions such as tumours and angiomas containing areas of increased radiodensity are better indentified by this method. Because of this difference, CAT scanning is not a suitable single investigation for screening epileptic populations for surgical candidates.

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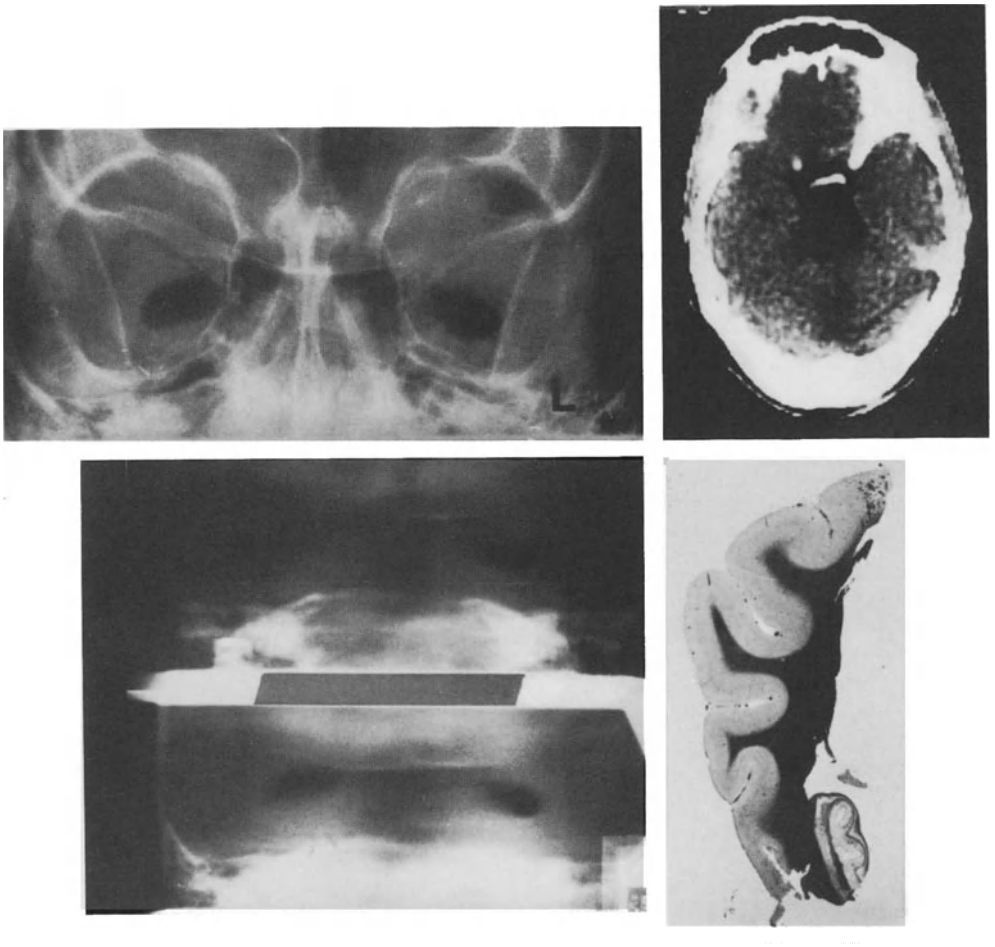

Fig. 1. *Mesial temporal sclerosis*: On the 1B cut of the CAT scan, (*upper right*), the dilated left temporal horn is just visualised, whereas it is clearly dilated in both the straight view (*upper left*), and the tomograms (*lower left*) of the AEG. The shrunken hippocampus, typical of MTS, is seen in the specimen (*lower right*)

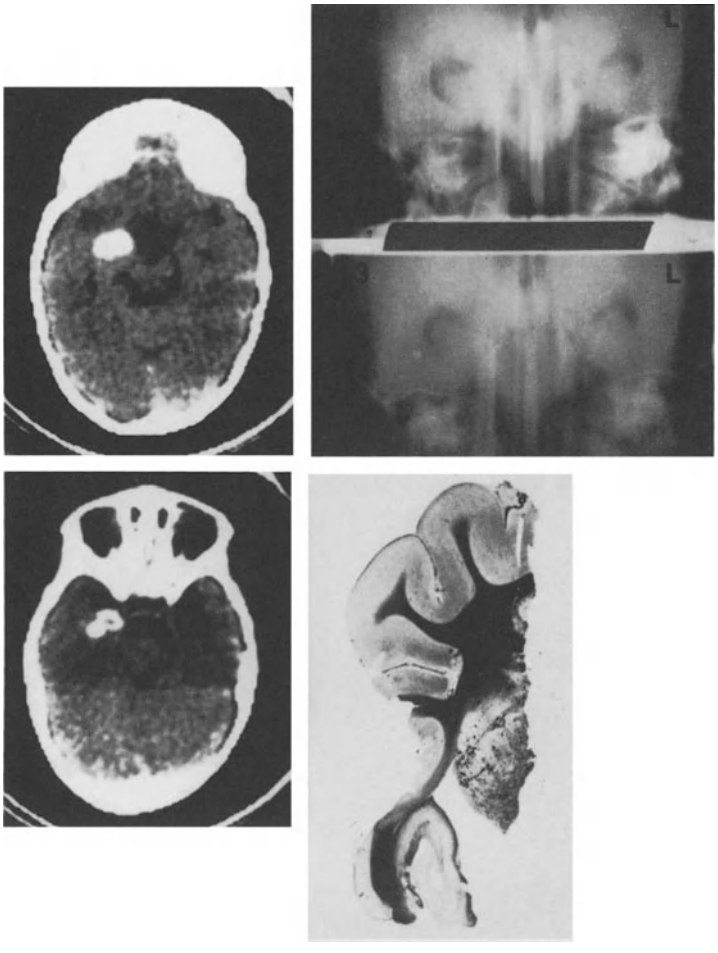


Fig. 2. *Hamartoma*: The CAT scan shows a tumour, visible without enhancement, in the 2B cut (*upper left*) and the 3A cut (*lower left*). It is in the antero-medial part of the left temporal lobe adjacent to the brain stem. By contrast it is only seen as minimal displacement of the left temporal horn in the AEG (*upper right*). The lesion is clearly seen in the section of the lobectomy (*lower right*)

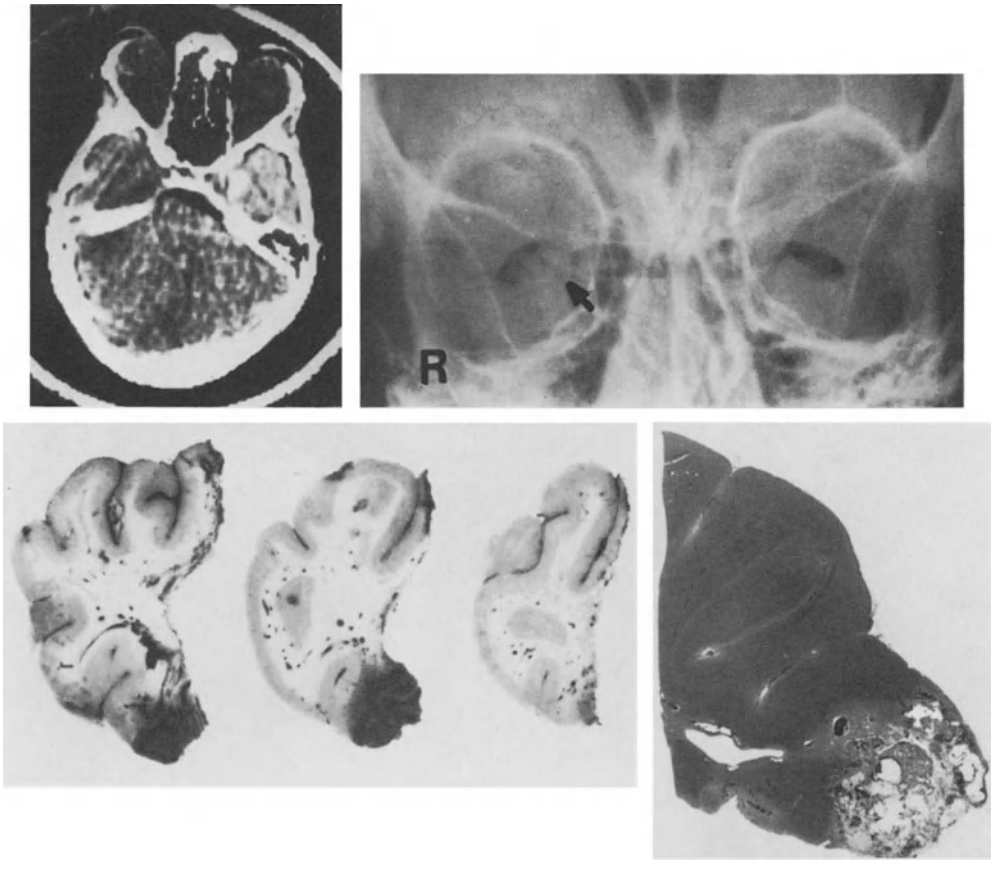


Fig. 3. *Angioma*: On the un-enhanced CAT scan, (1A cut) this lesion is visible in the anteromedial part of the right temporal lobe (*upper left*). It is less easy to demonstrate on the AEG (*upper right*) although the faint calcification and distortion of the temporal horn can be seen (*arrow*). It is well seen in the serial sections of the lobectomy specimen (*lower left*) and in a single section (*lower right*)

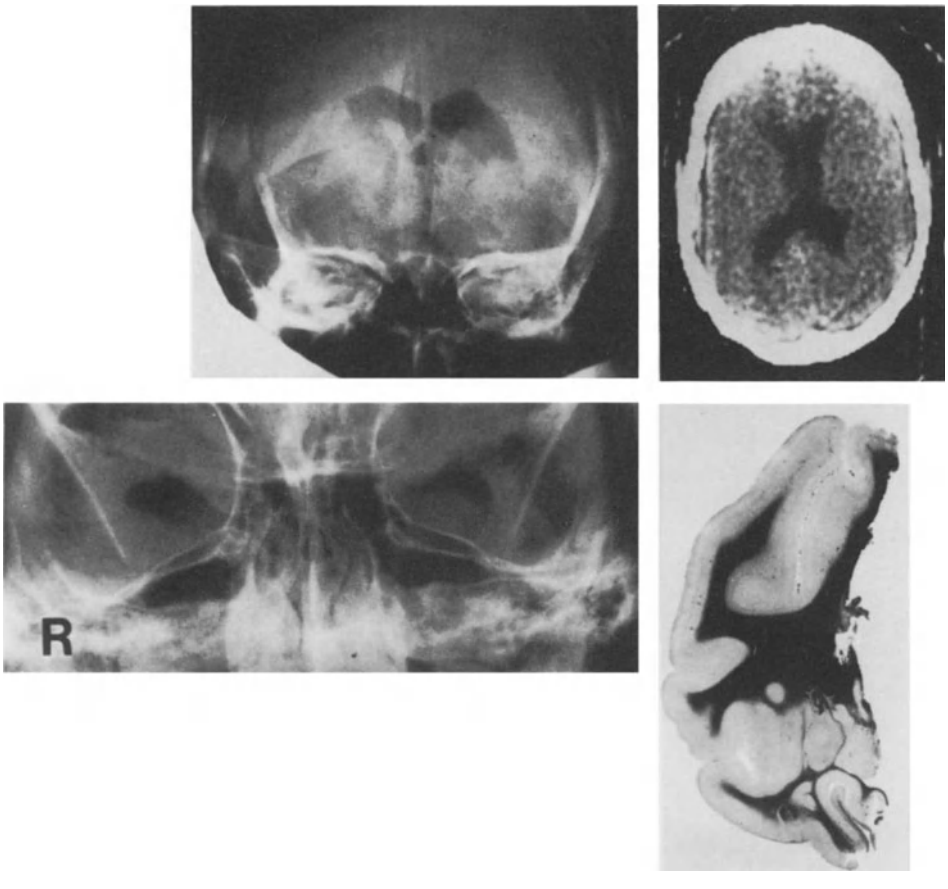


Fig. 4. *Cortical dysplasia*: In the CAT scan (2B cut) only distortion of the body right lateral ventricle can be seen (*upper right*), likewise the AEG shows the same distortion of the ventricle (*upper left*), and a dilated right temporal horn (*lower left*). These appearances, suggestive of tumour, are confounded by the operation specimen which demonstrates cortical dysplasia (*lower right*)

# Computer-Tomography as Applied to Early Posttraumatic Epilepsy

W. I. STEUDEL, J. KRÜGER, TH. GÖLLER, and H. GRAU

## Introduction

It is widely accepted that seizures may appear following head injuries. The origin of early seizures resulting from brain injuries remains unclear. The circumstances under which epileptic attacks occur and their frequency are particularly interesting (3, 7, 9, 10, 14, 16). Since the frequency of posttraumatic epilepsy is highly variable - from 4% to about 30% the question of the actual cause of the seizures has a special meaning (2, 18).

For many years localized brain damage such as edema, ischemic disturbances, and hemorrhages have been considered to be a pathological correlate of early posttraumatic epilepsy (6, 17). Computer-Tomography (CT) has gained an important position in the diagnosis and management of brain injuries since it permits direct visualization of pathological entities such as contusions and hemorrhages (1, 5, 8, 10, 12).

Using a prechosen group of 30 adults with traumatic intracranial hematomas - mostly operative cases - CT and EEG findings were compared as to their localizing value in cases of early posttraumatic seizures in the first week.

## Methods

*Patients*: We studied 30 patients (4 female and 26 male) with severe head injuries between the ages of 18 and 73 (mean 41.5) years. All were in coma for at least 3 days following the injury. The injuries which led to unconsciousness are summarized in Table 1. Twenty-eight patients were operated upon for intracranial hematomas: 15 epidural, 8 acute subdural and 12 intracerebral; 3 of these were operated bilaterally and in 4 others a number of hemorrhages were removed unilaterally. The basic management was the same for all patients. Nasal intubation was maintained for the entire period of coma. The seizures were treated with Hydantoins and Barbiturates.

Table 1. CT findings in 30 patients with severe brain injuries

| CT (n = 30) | Left | Right | |
|---|---|---|---|
| Depressed skull fractures | 2 | 1 | 3 |
| Hemorrhages | | | |
| epidural | 3 | 12 | 15 |
| subdural | 4 | 4 | 8 |
| intracerebral | 10 | 7 | 17 |
| intraventricular | 2 | 2 | 4 |
| Contusions | 2 | 2 | 4 |
| Total | 23 | 28 | 51 |

*EEG*: On the 1st, 2nd, 3rd, 5th and 7th day following injury or operation a standard 8-channel EEG-recording was made with frontal (Fp1-F3, Fp2-F4), temporal (T3-T5, T4-T6) and parieto-occipital (P3-O1, P4-O2) needle electrodes (30 hz filter, 0.3 sec time constant) (Fig. 1). The standard recording time was half an hour, although some recordings lasted several hours.

*CT*: The patients were routinely examined on the day of admission with a SIRETOM-Scanner using a matrix of 128 x 128 (5). CT was repeated in cases of persisting unconsciousness (11).

## Results

*CT*: We differentiated between depressed skull fractures, intracranial hematomas and contusions. Superficial scalp injuries and fractures of other types were no considered. Intracranial bleedings were subdivided, according to their localization, into epidural, acute subdural, intracerebral or intraventricular. A total of 51 findings were made from 30 patients (Table 1).

*EEG and seizure patterns*: Among the 30 cases, 13 had epileptic discharges in the EEG. In nine focal seizures appeared (Figs. 1 and 3). In four no obvious clinical epileptic manifestations could be found (Figs. 2 and 4). For technical reasons two additional patients could not be studied at the time of their seizures (during the night). In all 13 cases in which we were able to record epileptic discharges, CT showed intracranial bleeding on the corresponding side: four epidural, two subdural and seven intracerebral. The first seizure or epileptic discharge was verified twice on the 1st day, four times on the 2nd, 3rd and 5th day and once on the 10th day following operation or injury. The latter is a patient who rebled on the 3rd day, although the first attack appeared on the 7th day after the second operation (Fig. 5). The clinical manifestations of the seizures in the 11 patients were similar: focal attacks beginning in the facial region (five times on the left and six times on the right side) and spreading to one arm and leg. In three cases focal seizures had a rapid generalization.

All patients were treated with hydantoins and barbiturates until cessation of clinical attacks. We were unable to observe spontaneous disappearance of seizures during EEG recordings which lasted from one half to 3 h. The four patients who had obvious clinical manifestations and who merely had epileptic discharges were not submitted to antiepileptic therapy. Seven of the eleven patients with seizures survived the injury. Three among the four patients who only showed discharges in the EEG died.

## Discussion

In all 13 cases, in whom we were able to record epileptic discharges in the EEG, localization of the EEG-foci corresponded to the CT findings. This result supports the assumption that early posttraumatic epilepsy may be the result of local changes caused by edema, bleeding or ischemia around the damaged brain areas (18). This high correlation did not exist for late epilepsy since CT findings may disappear with time (4, 10, 12). On the other hand, a focus may be expected in the CT when seizures appear. In this context, early posttraumatic epilepsy may be considered indicative of an intracranial hematoma (12, 18) and a CT-study should be performed in such cases. Eleven of the 30 patients had clinically observable attacks and four additional patients showed focal epileptic discharges in the EEG. The frequency of early posttraumatic seizures

varies between 4% in closed head injuries and 32% in intracranial bleedings (7, 15). Literature contains variable values as to EEG changes in patients with seizures. They lie around 4% (15, 18). A crucial factor is, no doubt, the frequency of EEG recordings. The fact that EEG was regularly recorded in our patients (for hours and the same day) explains the high correlation to EEG foci in our cases.

We were able to obtain 51 CT scans at the time of admission. It is possible that further bleedings and, particularly, contusion foci may be detected (11, 12) in repeat studies. Additional findings observed during control were not considered in this report.

## Conclusions

In cases of early posttraumatic epilepsy CT is the most important diagnostic tool. EEG recordings should be done in the first week after the injury or operation. Our findings show that an early posttraumatic seizure is a good indication of an intracranial bleeding.

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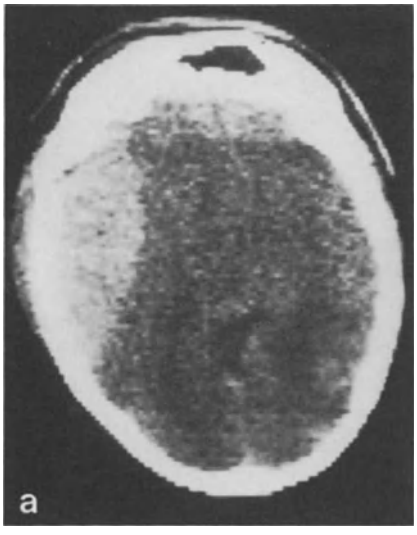



Fig. 1a. CT: Left temporal epidural hematoma

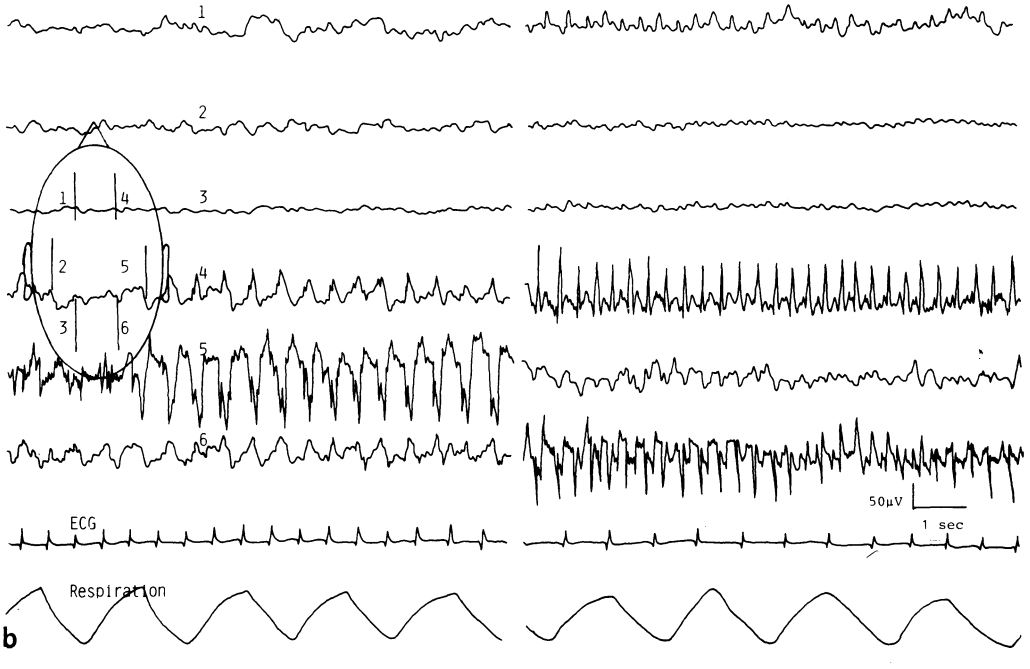



Fig. 1b. EEG: 2 segments from the recordings of successive discharges. Incipient focal attacks could be observed on the right side of the face

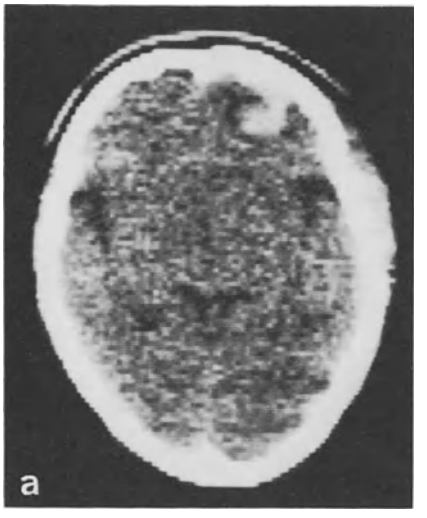



Fig. 2a. CT: Small right frontal intracerebral hematoma (H)

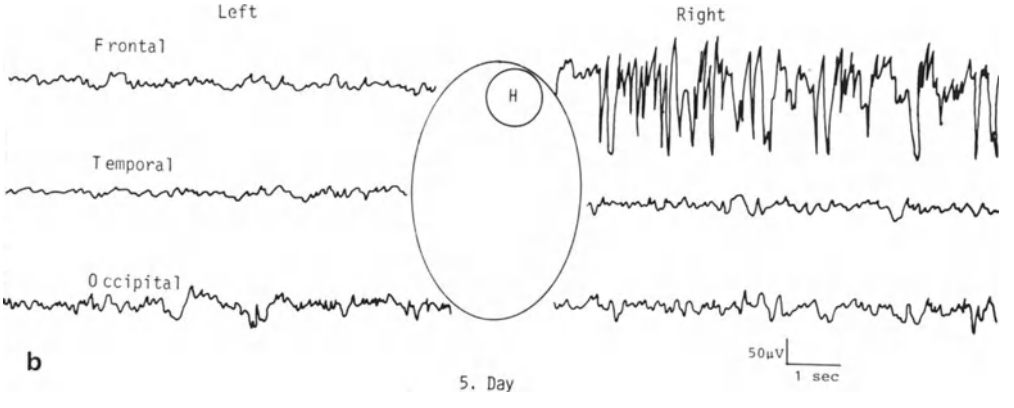



Fig. 2b. EEG: Right frontal discharges on the 5th day after the injury. No clinical attacks were visible. The 80-year-old woman died on the following day

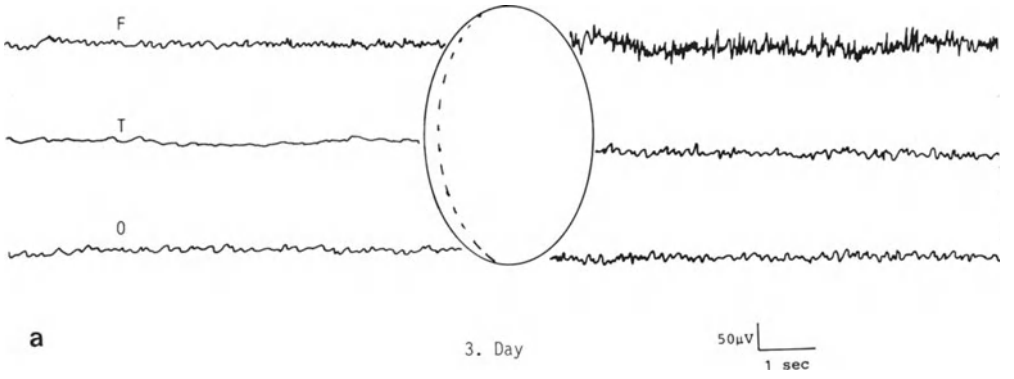



Fig. 3a. EEG from the 3rd postoperative day recording of a discharge (*right frontal*) in a patient with an acute left sided subdural hematoma. On admission no CT was made since an angiography had already been performed

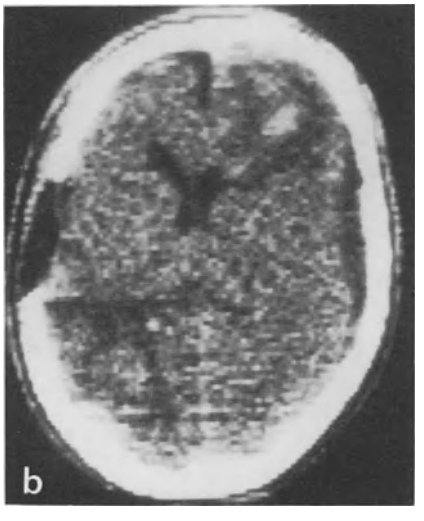
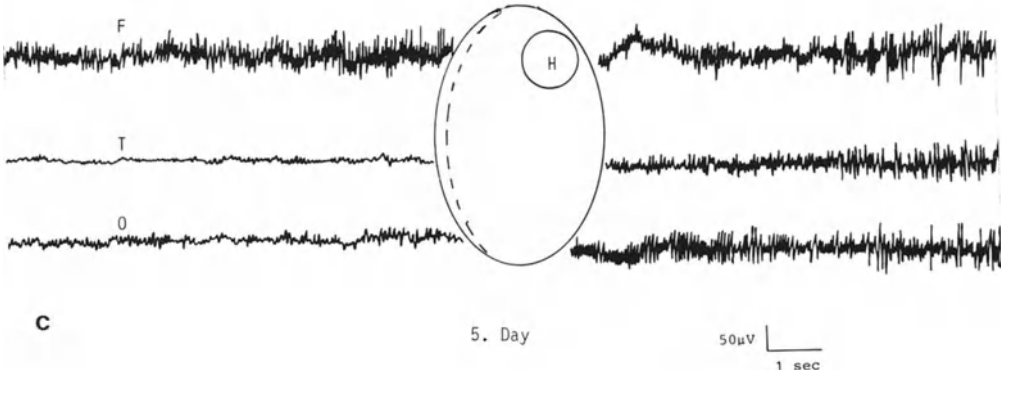



Fig. 3b. CT: As a result of the above EEG findings CT was made and showed a right frontal contusion and an intracerebral hematoma (H). The site of operation is seen in the left temporal region

Fig. 3c. EEG from the 5th postoperative day recording during a seizure. Focal attacks were observed on the left side



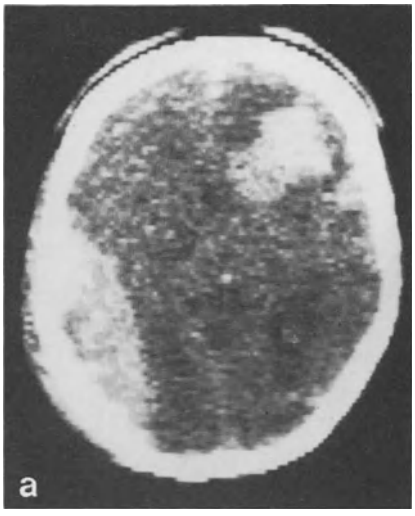



Fig. 4a. CT: Bilateral findings: right frontal intracerebral hematoma (H) and left temporal epidural hematoma (H)

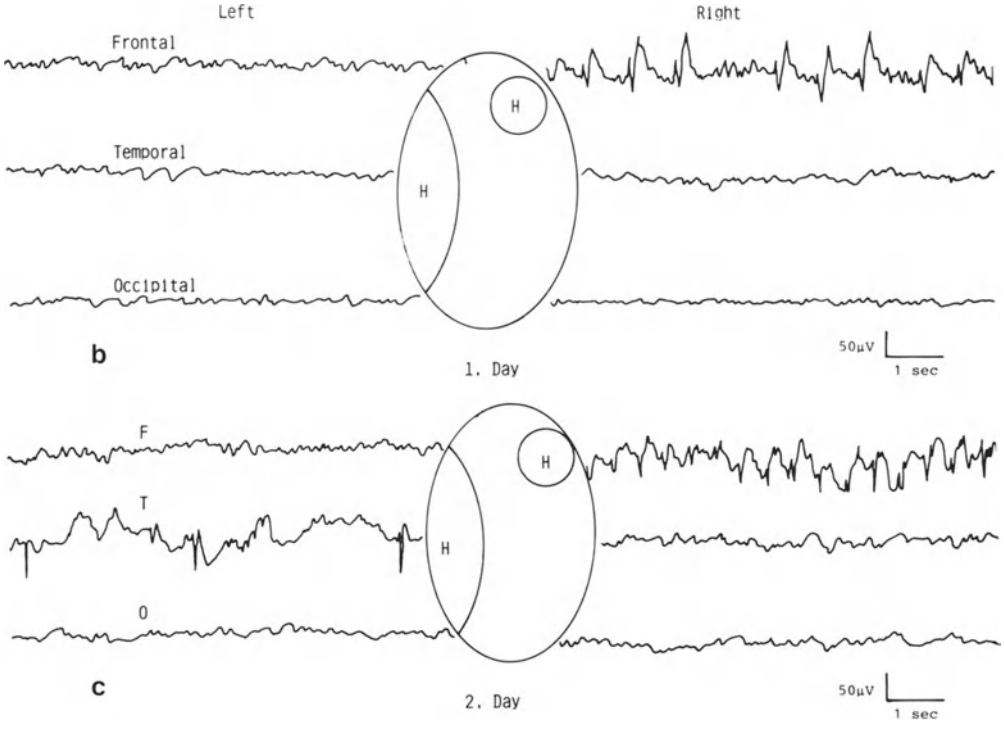



Fig. 4b. EEG from the 1st postoperative day right frontal spikes and waves

Fig. 4c. EEG from the 2nd postoperative day right frontal discharge and some left temporal spikes. No clinical manifestation. The patient died on the 3rd day (note the severe bilateral brain damage)

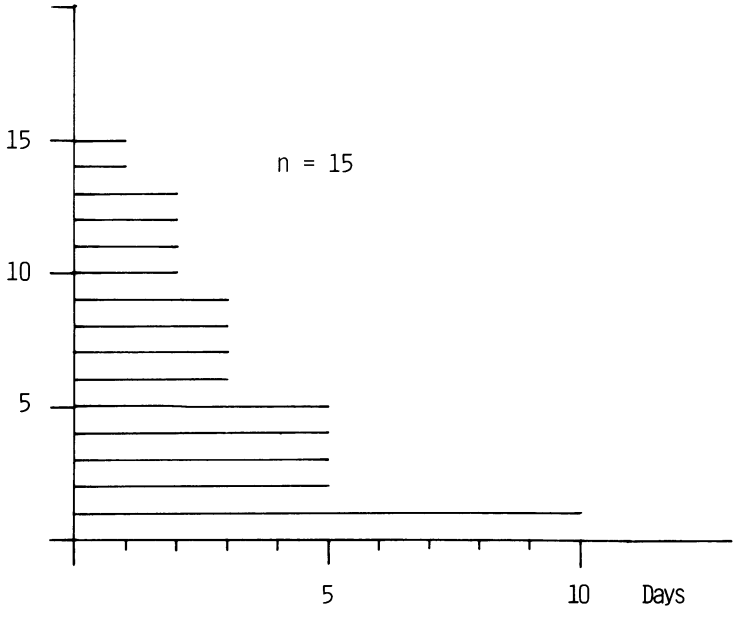



Fig. 5. Time histogram of the first clinical seizure or the first epileptic discharge in the EEG

# Diagnostic Errors in Computerized Tomography in the Differential Diagnosis of Cerebrovascular Lesions

R. MÜKE and D. KÜHNE

There are two alternatives to demonstrate diagnostic errors in computerized tomography: statistics and the evaluation of single cases. Statistics are necessary but they cannot disclose the reason and the consequences of misinterpretations for the individual case. Nine out of 4200 patients subjected to computerized tomography were selected to discuss some problems of diagnosis of cerebrovascular lesions by computerized tomography. This report is divided into three parts:

## Part 1

*Case 1*: A 13-year-old girl with increased intracranial pressure of half a year's duration had a hypodense formation in the left temporal lobe in the CT (Fig. 1). No contrast enhancement. CT diagnosis was intracerebral tumoral cyst. Since one would hesitate to perform surgery on the dominant hemisphere, cyst puncture was considered. Angiography, however, revealed a large vascular malformation (Fig. 2). At surgery we had the impression of a cystic angioblastoma but histologic diagnosis was: hemangioperycytoma or angioblastoma.

*Case 2*: A 34-year-old female complaining headache, dysphasia and apathy had the CT diagnosis of fronto-temporal subarachnoid cyst (Fig. 3). The angiogram, however, showed a small vascular lesion at the medial wall of the cyst (Fig. 4). A cystic angioblastoma was discussed. At surgery only a subarachnoid cyst was found not, however, the vascular lesion, which must be intraparenchymatous. We proposed treatment with the Gamma Unit in Stockholm.

*Case 3*: In a 35-year-old female with increased intracranial pressure and posterior fossa symptoms CT revealed a not well defined hypodensity zone (28 Hounsfield units) in the middle of the posterior fossa (Fig. 5). Without contrast enhancement, thought to be a spongioblastoma or an epidermoid. The vertebral angiogram, however, showed an angioblastoma (Fig. 6), confirmed at surgery.

Consideration of the above three cases leads to two conclusions:

1. Even larger vascular lesions may not be detected by CT.
2. One should not hesitate to perform a preoperative angiogram if the diagnosis is not clear (3).

## Part 2

The diagnosis of an acute intracranial bleeding is generally easy. Sometimes, however, it is difficult to recognize a co-existing tumor or metastasis in cases of tumor bleeding (Fig. 7). In a subacute or chronic hemorrhage differential diagnosis as to a tumor or abscess is even more difficult.

*Case 5*: Figure 8 shows a finding rather resembling an abscess (1) than a hematoma. There were no signs and no history of infection. The ring pattern shown by CT disappeared without therapy in a short time. We be-

lieve it could have been the capsule of an old hematoma. Similar rings were found upon contrast enhancements in the follow-up of several cases with a known bleeding, one case having been confirmed at surgery.

*Case 6*: A 37-year-old male was admitted in comatous state. History was unknown. An accident was suspected. CT diagnosis was: older subdural hematoma (Fig. 9). At surgery, however, a subdural empyema was evacuated. Following operation the patient did not recover completely. The CT was repeated, showing no abnormality in the frontal region but an occipital lense shaped lesion, the periphery of which enhanced by contrast medium. We suppose contrast injection at the first CT investigation could have led to the correct diagnosis earlier (2). Thus we propose that contrast medium injection be used if there is an uncertain extracerebral lesion.

Part 3

*Case 7*: A 50-year-old female complaining of headache, facial pain and hypertonus had undergone surgery for cancer of the breast one year before admission. CT (Fig. 10) disclosed a zone of higher density with nodular enhancement in the right parasellar region as well as a hypodense in the right occipital lobe with no clear enhancement. There was also a symmetric hydrocephalus. The first impression was of a cerebral metastasis. To our surprise the angiogram revealed a large carotid aneurysm on the right side (Fig. 11) and, in addition, two small aneurysms of the middle cerebral artery and of the basilar artery. The occipital lesion was now explained as an infarct of the posterior cerebral artery (5, 6, 7). This case underlines the importance of the angiogram (8) to complete diagnosis even if CT finding and history are in agreement.

*Case 8*: The CT of a 60-year-old male referred to us because of headache, hemianopia and left sided weakness (Fig. 12) showed a lesion thought to comprise a diffuse edema. The marked shifting of brain structures should rather indicate a tumor than an infarct. However, the patient recovered and the occipital lesion had disappeared 6 weeks later. At this time we recognized two frontoparietal lesions of lower density without contrast uptake. Considering the history, we believed these to be also vascular lesions. The condition of the patient worsened again and the next CT, 3 months later, revealed a large right frontotemporal tumor (Fig. 12e, f). It was a glioblastoma. One notices that sometimes differential diagnosis between tumor and infarct can be extremely difficult (5, 6, 7) and only follow-up studies may permit diagnosis.

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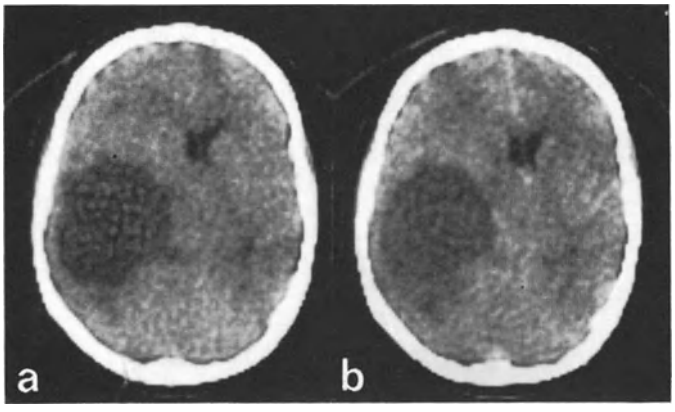


Fig. 1a, b. Large sharply circumscribed cystic lesion in the left hemisphere. No contrast enhancement

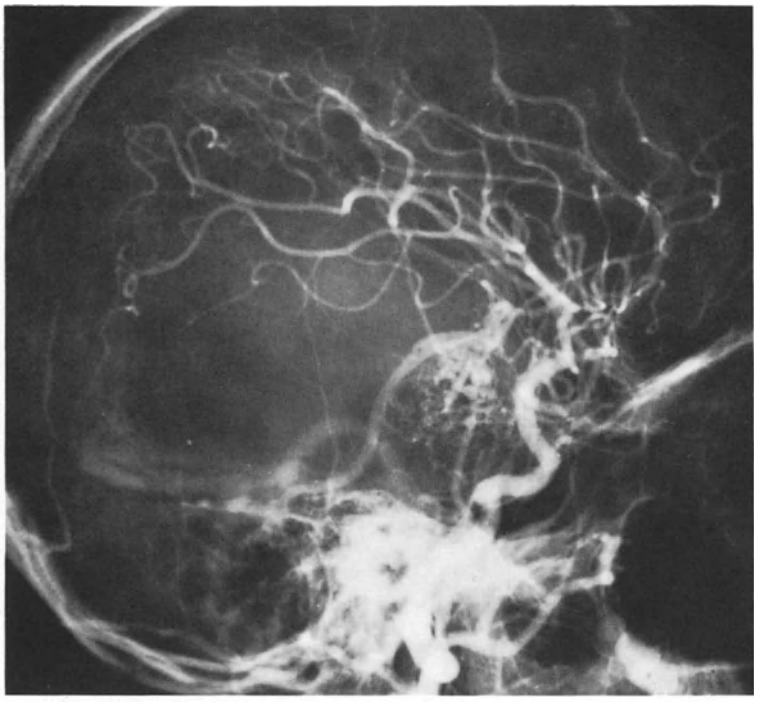

Fig. 2. Left carotid angiogram, lateral projection. Midarterial phase. Arterio-venous malformation fed by the middle cerebral artery and drainage into an enlarged superficial cerebral vein

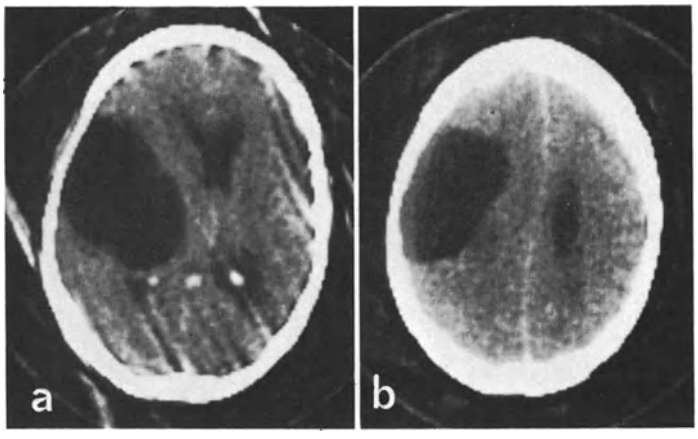


Fig. 3a, b. Subarachnoid cyst of the left hemisphere

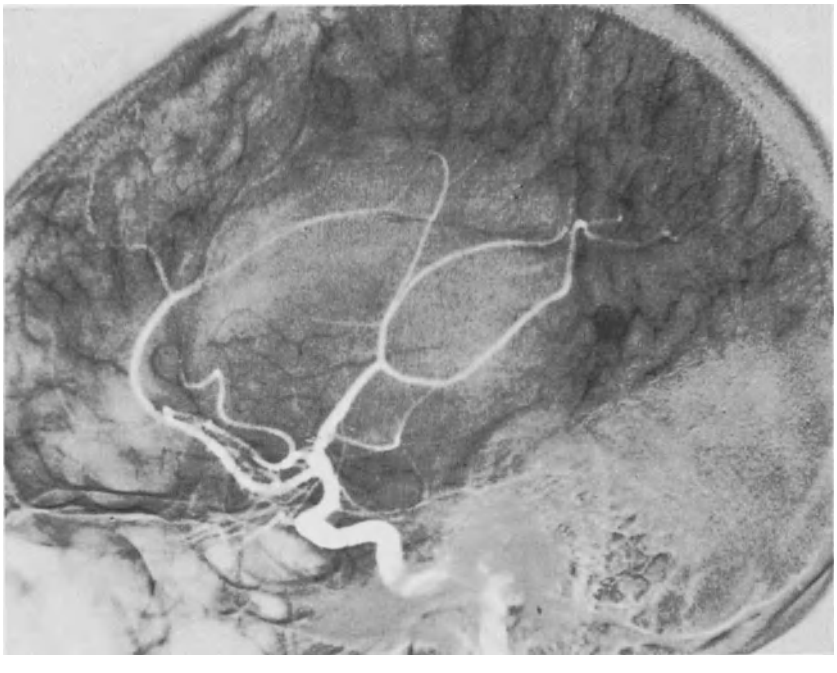

Fig. 4. Left internal carotid angiogram, lateral projection, late arterial phase. Small arteriovenous malformation in the deep temporal region

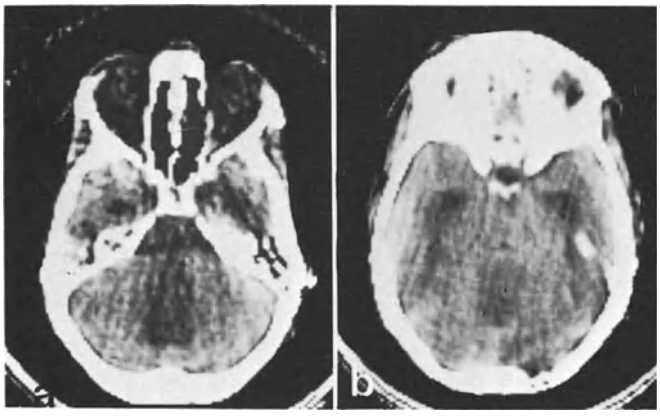

Fig. 5a, b. Heterogeneously hypodense zone in the middle of the posterior fossa. No contrast enhancement

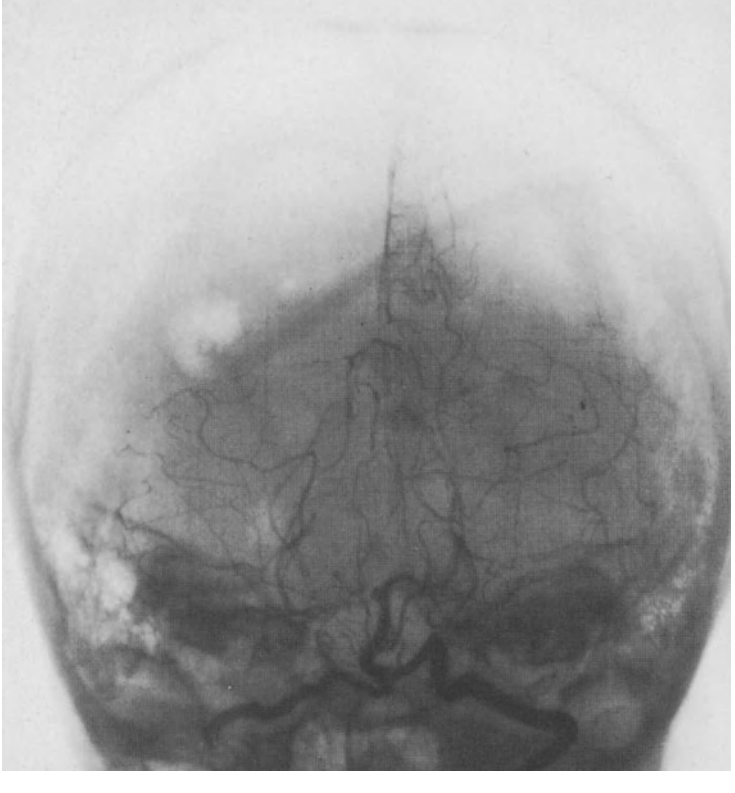

Fig. 6. Selective vertebral angiogram. Small, sharply delimitated vascular mass in the right cerebellum (hemangioblastoma)

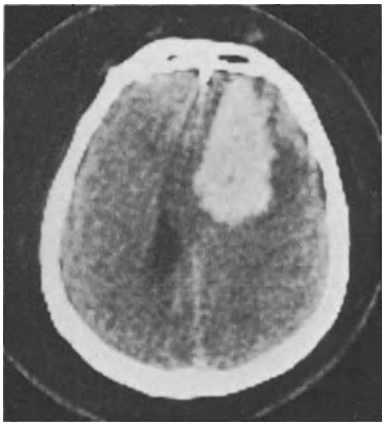

Fig. 7. Tumor bleeding in the right frontal area

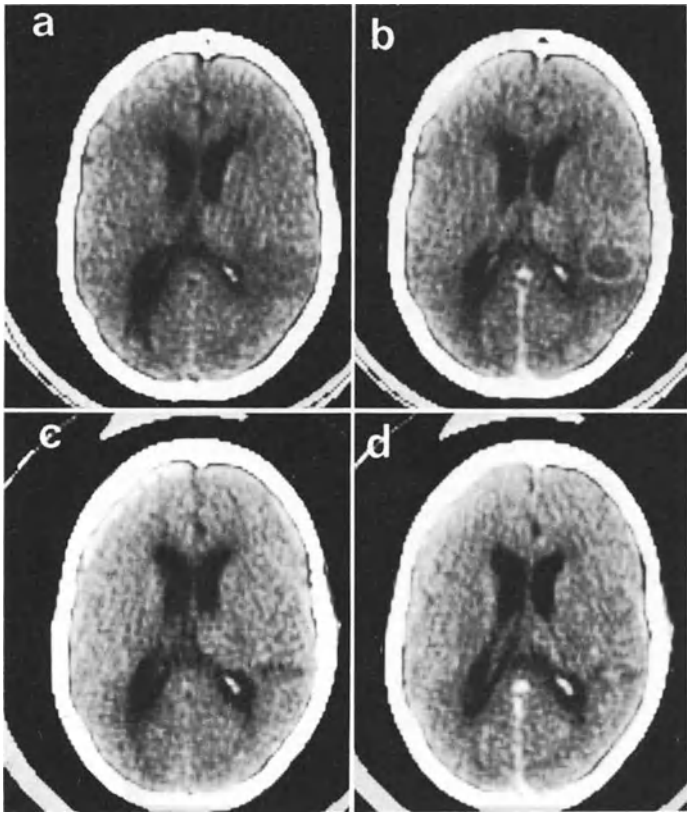


Fig. 8a, b. Right temporal hypodense lesion. Smooth ring of contrast enhancement. c, d Two months later. CT findings appear normal

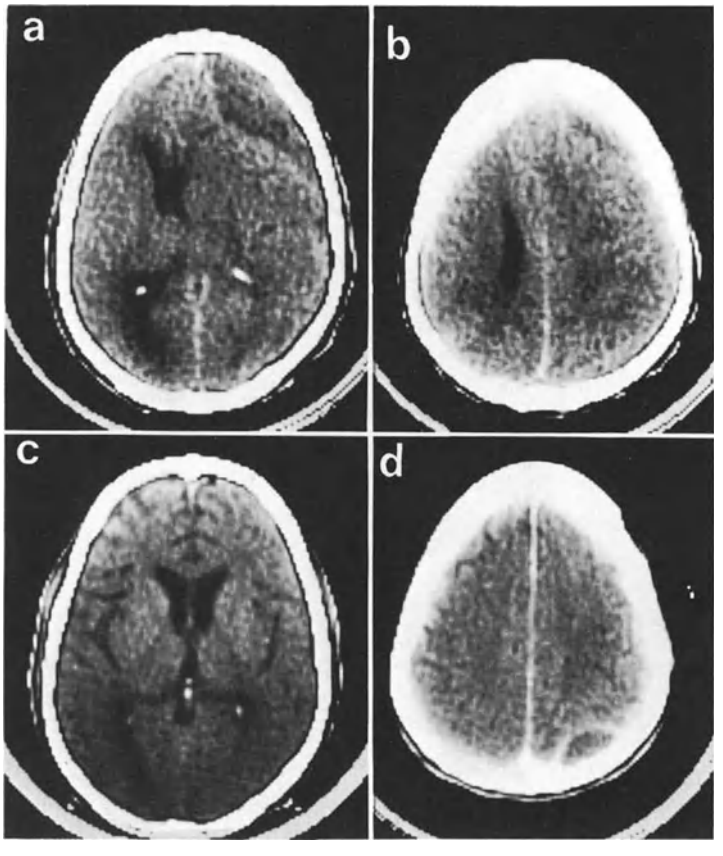


Fig. 9a, b. Right frontal subdural collection. Septum pellucidum markedly displaced towards the left. c, d Three weeks later: regression of the frontal lesion. Lens-shaped lesion with peripheral enhancement in the right occipital area.

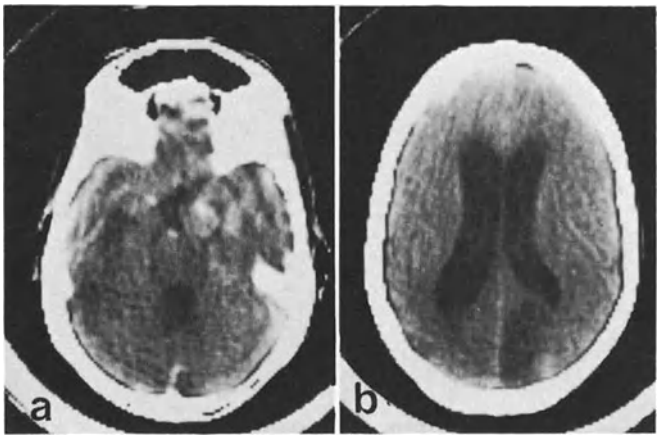


Fig. 10a, b. Hyperdense zone in the right parasellar region with nodular contrast enhancement. Hypodense right occipital area with focal contrast enhancement. Symmetric hydrocephalus

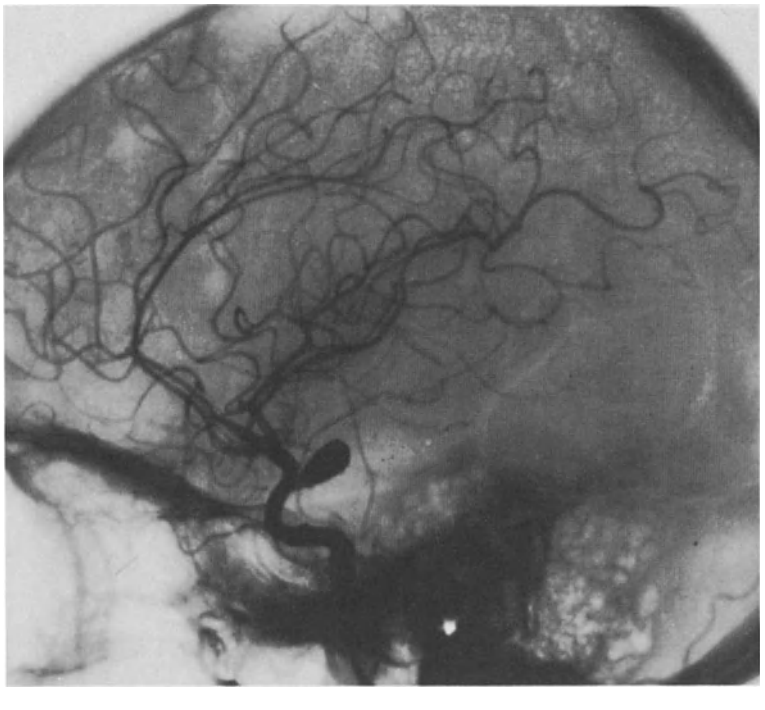

Fig. 11. Right carotid angiogram, lateral projection, arterial phase. Large aneurysm of the carotid artery

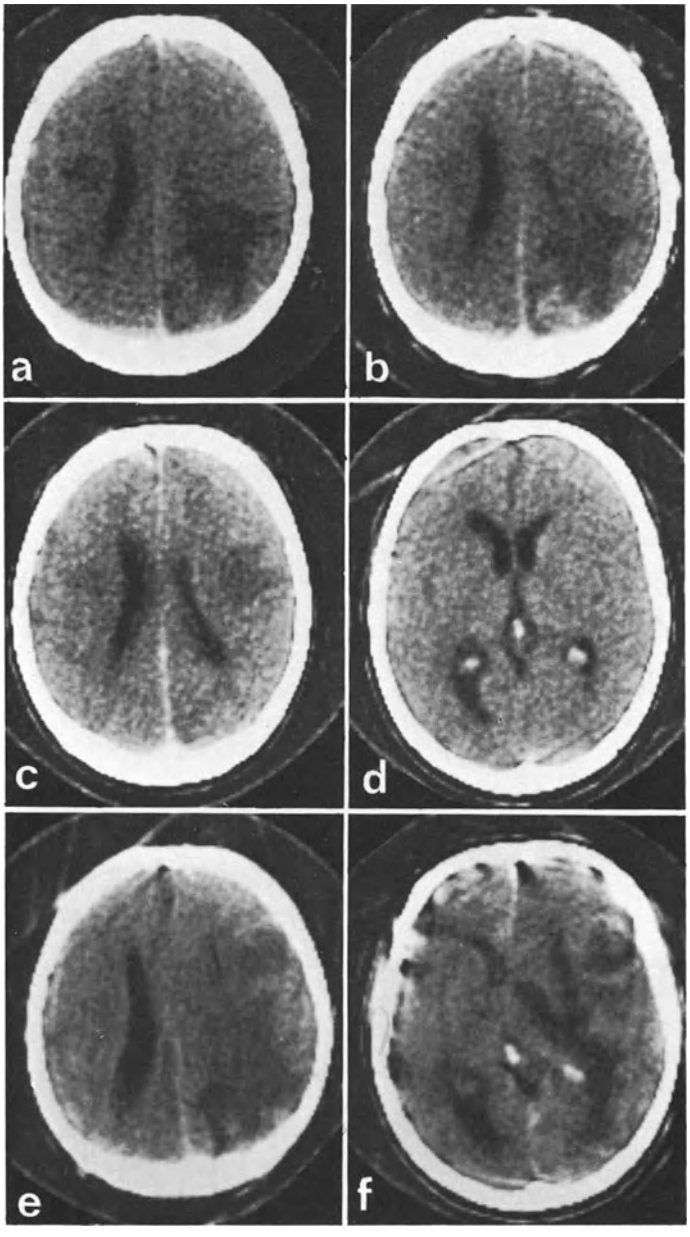


Fig. 12a, b. Hypodense lesion in the right parieto-occipital region, compressing of ventricles. Focal contrast enhancement. Small hypodense lesion in the left frontal region without contrast enhancement. c, d Two months later: Complete regression of the occipital lesion. Development of a low density lesion without enhancement in the territory of the right middle crebral artery. e, f Three months later: Development of a glioblastoma

# Correlation of Hyperdense and Hypodense Areas in the Computerized Tomogram of Subdural Hematomas

H. E. CLAR, W. J. BOCK, and H. C. WIECHERT

## Introduction

The computerized tomogram is of great value in the diagnosis of intracranial hemorrhages. This is true for acute hematomas. Contrary to this, subacute and chronic subdural hematomas can cause diagnostic problems, first because the clinical symptoms are often uncharacteristic and second because the interpretation of the CT findings may present difficulties (1, 5). The scope of this paper is to correlate the CT findings with the time course of subdural hematomas.

## Patients and Method

Fifty-one patients with surgically proven subdural hematomas have been treated during a period of 22 months. Of these hematomas, 10 were acute (0-24 h), 22 were subactue (2-40 days), 19 were chronic (more than 40 days). In the group of acute hematomas, onset of hemorrhage could be determined exactly in all cases. In the subacute group, the time elapsed between trauma and CT investigation was clear in 12 cases and uncertain in 10 cases. A known trauma occurred in only four cases with chronic subdural hematoma. CT investigations were performed using a SIEMENS SIRETOM 1 with a 128/128 matrix. Scans were taken in the usual plane parallel to orbito-meatal line.

## Results

In the analysis of our CT findings we distinguish direct and indirect signs (Table 1):

1. *Direct signs* of attenuation value are hyperdensity, isodensity, and hypodensity, compared to normal brain tissue. These areas were unileteral or bilateral and had the shape of a sickle on a lens.
2. Bilateral and unilateral hematomas show different form of *indirect signs*: In cases of unilateral hematomas, a mass dislocation is the dominant finding, with compression of the ipsilateral ventricle and occlusion of the subarachnoid space. Furthermore, a midline-shift is visible. In cases of brain atrophy, cortical sulci are not visible on the side of the hematoma. In cases of bilateral hematoma the midline-shift is missing, but there are signs of an expansive lesion too, with symmetrical compression of the ventricles and obliteration of subarachnoid spaces. Indirect signs allow no conclusion as to the age of a subdural hematoma. The main feature is the attenuation value of hematoma.

*Hyperdense areas* are found in all cases of acute hematoma. However, subacute hematomas may also show hyperdense areas, which often contain clots of higher density within an isodense region. Hyperdense areas are scarcely seen in chronic hematomas. *Isodense areas* are observed in subacute or chronic, but never in acute hematomas. *Hypodense areas* are more often seen in chronic hematomas, less evident in subacute hematomas, but seldom in acute hematomas (Table 2). Intravenous

Table 1. Different signs of CT findings in subdural hematoma

| CT findings | Subdural hematoma Acute | Subacute | Chronic |
|---|---|---|---|
| Direct signs | Hyperdense Sickle shaped | Isodense and clots Sickle- or lens shaped | Hypodense Lens shaped |
| Contrast medium | | Brain tissue: Increased density Hematoma: Unchanged | |
| Indirect signs | | | |
| Unilateral | Midline shift<br>Compression of ispilateral ventricle<br>Obliteration of subarachnoid spaces<br>In brain atropy: invisible cortical sulci (ipsilateral) | | |
| Bilateral | Symmetrical compression of ventricles<br>Obliteration of subarachnoid spaces | | |

Table 2. CT findings in correlation to the different forms of subdural hematomas

| CT findings | Subdural hematoma Acute | Subacute | Chronic |
|---|---|---|---|
| Hyperdense | + | (+) | |
| Isodense | | (+) | + |
| Hypodense | | + | + |

application of a contrast medium leads to an increase in density of the brain tissue, while the capsulae of the hematoma are also enhanced (Table 2). The hematoma itself is not enhanced by application of a contrast medium.

## Discussion

The CT signs in subdural hematomas can be grouped as direct and indirect signs. The acute hematoma shows hyperdense sickle shaped areas as a direct sign of high diagnostic value. Indirect signs (ventricular compression, mass expansion and midline-shift) are seen in all cases (Fig. 1a, b). Therefore, the CT diagnosis of acute hematomas is very reliable. Contrary to this, subacute and chronic hematomas present major diagnostic problems. Hypodense areas and indirect signs can be detected by CT in most cases (Fig. 2). Also, hyperdense clots are visible within hypodense or isodense areas. These findings can be signs of subacute or chronic hematomas as reported by GRUMME et al. for chronic hematomas. Most difficulties and misdiagnosis may occur in cases with isodense areas. These hematomas can only be recognized from indirect signs (Fig. 3a, b). After injection of contrast medium, the brain tissue or the capsule of the hematoma may be enhanced (Fig. 4a-c). This fact is also observed by DAVIS et al., while FORBES et al. state that contrast medium is not helpful in subdural hematomas.

The results of our investigation confirm other findings of FORBES et al. who stated that the attenuation values decrease with increasing age of the hematoma. NORMAN et al. demonstrated in vitro a direct cor-

relation between the CT attenuation value and hemoglobin contents of blood samples. The known progressive resorption of hemoglobin explains the decreasing attentuation value with increasing age of a hematoma. The various density values in the subacute and chronic stage depend either on different Hb-values at the time of trauma or its variable resorption. Moreover, repeated bleeding and trivial trauma may cause different attenuation values in the same hematoma. Hyperdense clots may represent coagulated and non-resorbed parts of the hematoma.

## Conclusion

These results prove that subdural hematomas may present diagnostic difficulties. We therefore propose the following diagnostic conduct:

1. In cases which show both direct and indirect CT signs no further diagnostic exploration is necessary.
2. In cases without direct signs, application of contrast medium may lead to diagnosis.
3. Cases without direct or indirect signs having clinical symptoms require further investigation by angiography, or CT control (Fig. 5).


## Summary

CT findings of 51 patients with acute, subacute and chronic subdural hematomas were studied. The results showed that direct signs (hyperdensity, isodensity, and hypodensity) and indirect signs (midline-shift, expansive lesion, compression of ventricles, and occlusion of the subarachnoid space) can be distinguished. The following diagnostic procedure is proposed in cases of subdural hematoma:

1. In cases with both, direct and indirect CT signs, no further diagnostic exploration is necessary.
2. In cases without direct signs, application of contrast medium may lead to diagnosis.
3. Cases without direct or indirect signs showing clinical symptoms require further investigation by angiography, or CT control.


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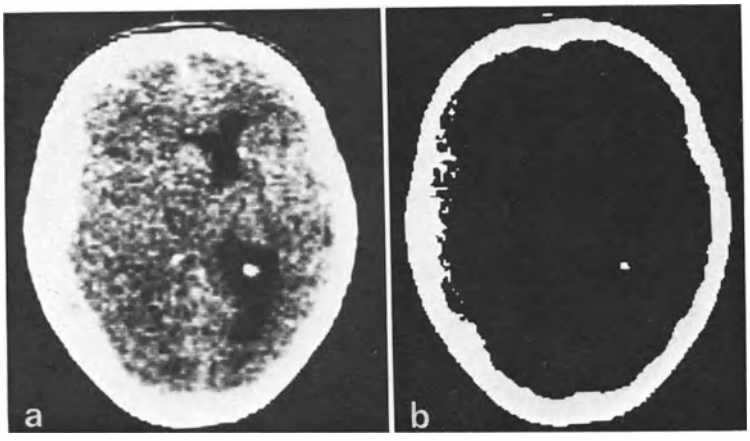


Fig. 1a, b. CT of 40-year-old woman with acute subdural hematoma (14 hours after trauma). a Direct sign: sickle shaped hyperdense area on the left side; indirect sign: midline-shift, compression of left lateral ventricle. b The attenuation value in the hematoma is 50 Hounsfield units

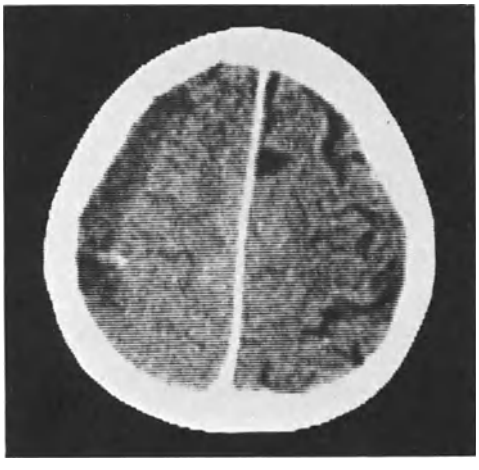

Fig. 2. CT of a 79-year-old woman with a chronic subdural hematoma (7 weeks after trauma). Direct sign: sickle shaped hypodense area on the left side; indirect sign: midline-shift, compression of the left lateral ventricle, missing cortical sulci

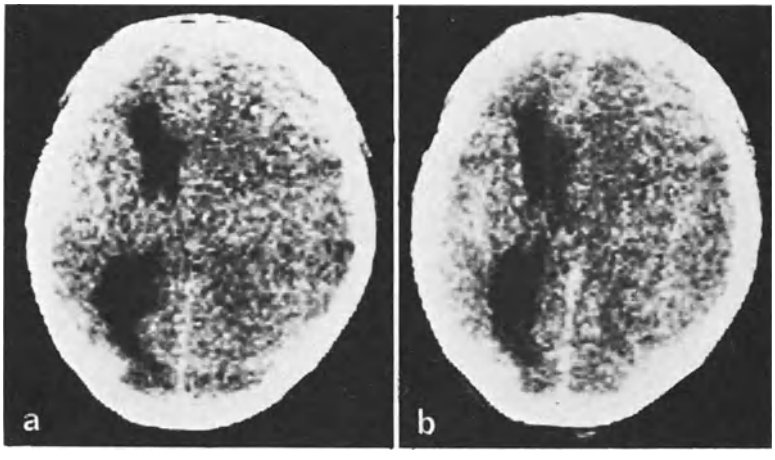


Fig. 3 a, b. CT of a 37-year-old woman with a chronic subdural hematoma (1 year after trauma). a Direct sign: not visible; indirect sign: midline-shift, compression of the right lateral ventricle. b CT after injection of contrast medium: enhancement of the capsule of the hematoma

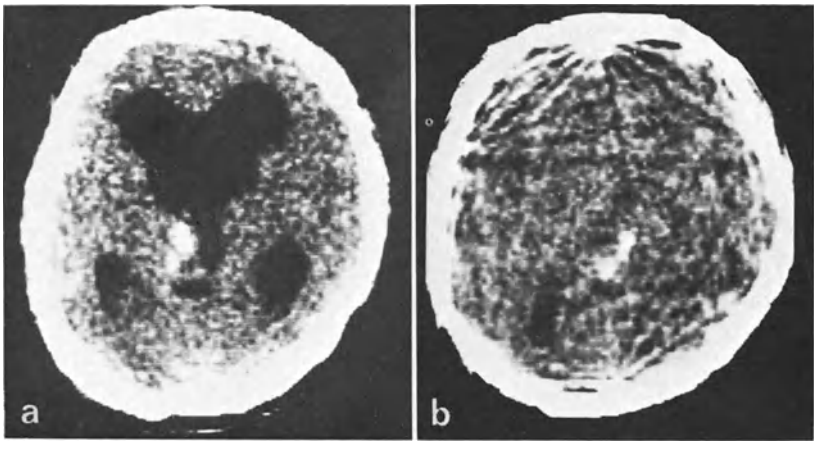


Fig. 4a-b. CT of a 28-year-old man. a Tumor of the pineal region with hydrocephalus occlusus. b Subacute subdural hematoma (less than 6 weeks after ventricular drainage). Direct sign: not visible; indirect sign: shift of the calcified pineal region, compression of both lateral ventricles

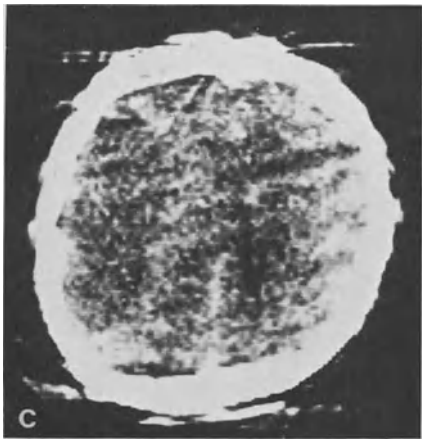


Fig. 4c. CT after injection of contrast medium three days later. Direct sign: isodense lens shaped area with enhanced capsula; indirect sign: compression of the lateral ventricles

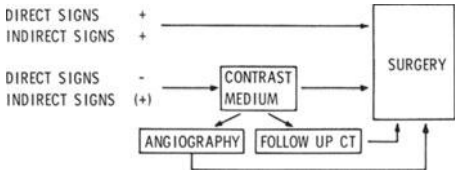


Fig. 5. CT diagnosis of subdural hematoma

# Difficulties in the Interpretation of Computerized Tomography

I. SCHÖTER and J. WAPPENSCHMIDT

Computerized tomography may avoid complications due to more invasive techniques (1, 3, 4, 12, 15). Nevertheless, this investigation is limited by the physical conditions of the tomograph as well as by the size and nature of the pathological process (2, 4, 8, 9, 13, 17).

Prior to discussing some cases of difficult interpretation, we wish to demonstrate the excellent correlation between CT and post-mortem findings: A 52-year-old male died 3 weeks after subarachnoid hemorrhage without regaining consciousness. A small aneurysm of the anterior communicating artery was demonstrated by angiography. Autopsy revealed complete necrosis of the territory supplied by both anterior cerebral arteries, without occlusion. Due to subarachnoid hemorrhage and consecutive arteriospasm, a luxury perfusion is visualized by CT as a sharply defined cortex of increased density with surrounding edematous lucency (Fig. 1).

Several authors have pointed out that differential diagnosis by CT may be very difficult (4, 5, 9, 16). CT scans of rare diseases may lead to misinterpretation. Three examples have been selected: the CT pictures of a 9-year-old Libyan boy scanned after intravenous Telebrix administration, demonstrated an extensive area of increased density, ranging from the left temporal region across the midline, as well as a huge hydrocephalus. The case history comprised only psychomotor retardation and progressive spastic hemiparesis of seven years duration. At surgery a 350 g hematoma was removed (Fig. 2).

The second patient, a 31-year-old male, experienced subarachnoid hemorrhage with consecutive spastic paresis of the right arm. CT revealed a small rounded area of increased density in the posterior part of the left ventricle, probably an enhancement due to a large aneurysm. This lesion proved to be a small meningeoma of the ventricle (Fig. 3).

In the third partient, with a 3 month history of headaches, CT demonstrated an encapsulated epidural hematoma. At surgery a non-specific granuloma was removed.

CT failure is inevitable whenever two pathologic processes - one of them being isodense - coexist: A 60-year-old female with a 2 year history of hypertension and gradual loss of vision underwent CT after intravenous administration of Telebrix. A large subdural hematoma was visualized. The concomitant pituitary adenoma, suspected from ophthalmological data had to be verified by pneumencephalography.

It has been emphasized that CT may offer information concerning vascular lesions of the brain (6, 10, 14, 15). The exact diagnosis of aneurysms and arteriovenous malformations, however, is reserved to angiography (4, 7, 11, 14). As a screening method, CT may lead to misinterpretation of vascular lesions and delay adequate therapy. Here are three examples:

The CT of a 46-year-old male demonstrate a space-occupying ring-shaped lesion which might be a malignant tumor or an abscess. Only knowledge of the clinical data, with sudden onset of headache, neck stiffness and

somnolence, give a hint to the diagnosis of hemorrhage. Arteriography revealed an aneurysm of the right middle cerebral artery.

The CT of a 58-year-old male with a 30 year history of nocturnal epilepsy and bitemporal hemianopsy delineated a ring-shaped process in the sellar region, obviously a pituitary adenoma. Angiography disclosed a small aneurysm, concomitant to a space occupying process, At surgery, thrombotic mural plaques and calcification were seen within a huge aneurysm (Fig. 4a, b).

A 21-year-old female experienced grand mal seizures for 14 years. CT demonstrated a non-expansive lesion of increased density in the fronto-temporal region. Although angiography did not reveal the arteriovenous malformation, a globular angioma, 3 cm in diameter, was removed.

Finally, CT misinterpretation is possible as a consequence of technical failure: CT picture of a 38-year-old male, suffering from headaches and seizures since childhood and presenting multiple neurological signs demonstrated a rounded area of decreased density in the region of the longitudinal fissure. This proved to be an artefact caused by the film-material, which might have led to surgical intervention, had the original tape not been reviewed (Fig. 5).

It has been recommended that CT interpretation include case history, neurologic findings and the results of other investigations (9, 11, 16, 18). As a consequence of our experiences, we tend to perform additional angiography in cases of doubtful scan findings.

## Summary

Value of CT is demonstrated by excellent correlation of CT with autopsy findings in a case of subarachnoid hemorrhage and consecutive arterio-spasm. As a screening investigation CT has its limitations, due ot the physical conditions of the tomograph and to the size and nature of the pathological process. Some examples have been selected to demonstrate difficult interpretation:

1. Rare diseases
2. Coexistence of two pathological processes
3. Vascular lesions
4. Technical failure

Since misinterpretation of CT may delay adequate therapy, the authors tend to perform additional angiography in cases of doubtful findings.

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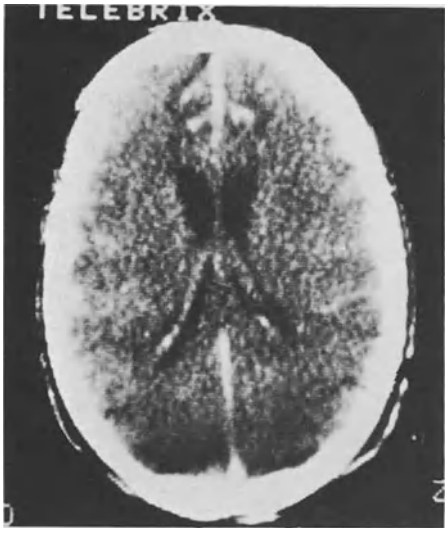

Fig. 1. Luxury perfusion of the cortex as consequence of arteriospasm

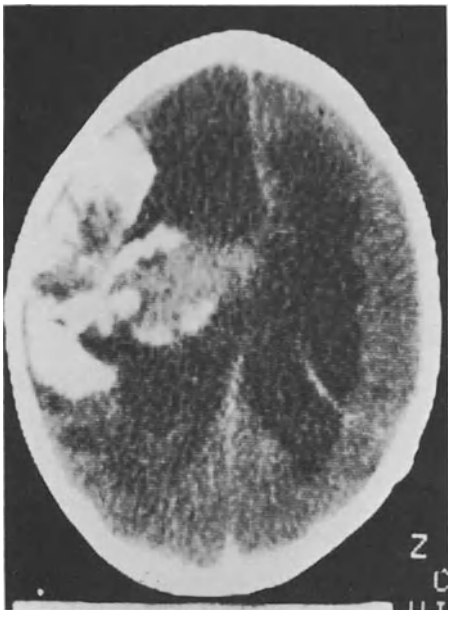

Fig. 2. Hamartoma of the left hemisphere causing marked hydrocephalus

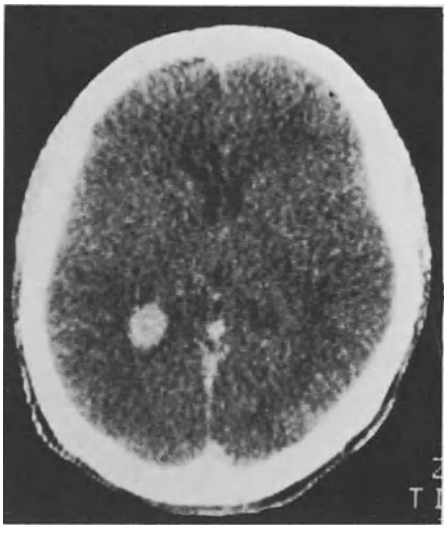

Fig. 3. Meningioma of the left ventricle

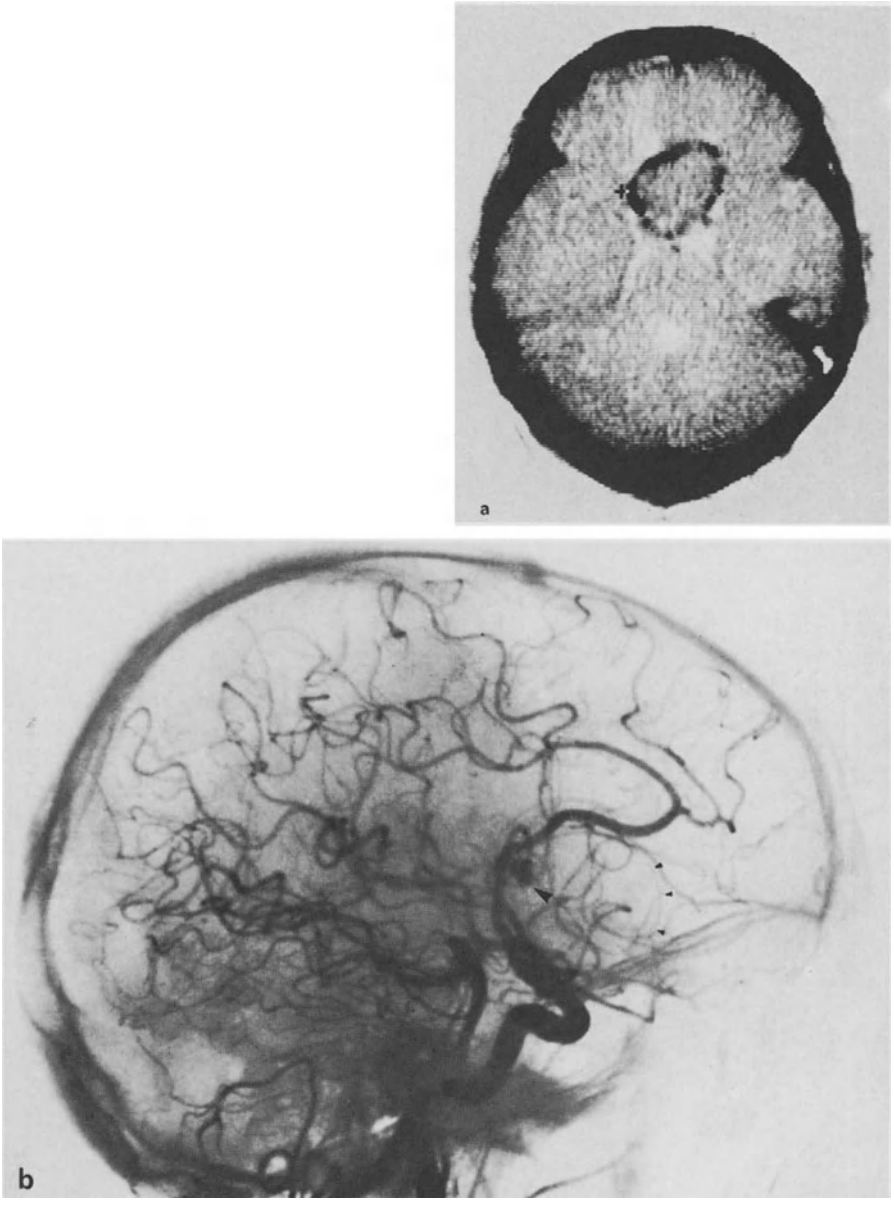


Fig. 4a, b. Huge aneurysm with thrombotic mural plaques and calcification

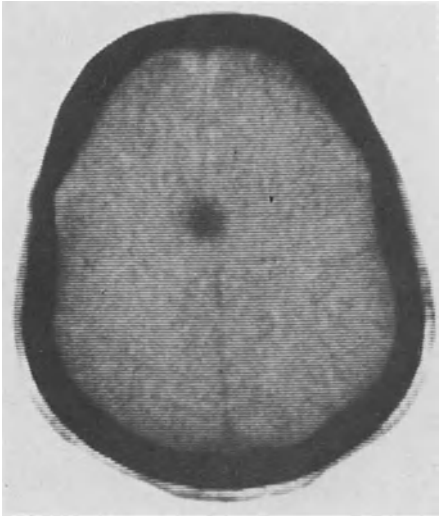

Fig. 5. Artefact of the film-material

# Free Topics

# Clinical Deterioration and Intracranial Pressure Patterns Following Aneurysm Surgery[1]

M. R. FEARNSIDE and C. B. T. ADAMS

## Introduction

The problem of postoperative deterioration following a successful direct operation for intracranial aneurysms remains a problem, despite technical advances in the surgery. The postoperative course in 55 patients was studied to determine patterns of deterioration.

## Material and Approach

All patients were treated in The Radcliffe Infirmary, Oxford. Of 56 procedures performed in the 55 patients, 53 followed rupture of the aneurysm with a subarachnoid haemorrhage. Two procedures were for a second unruptured aneurysm and one was for temporal lobe epilepsy. The patients who presented with a subarachnoid haemorrhage were graded using the classification of HUNT and HESS (3) and surgery was offered only to those in good condition. At surgery, 33 patients were grade 1, 13 were grade 2, and 7 were grade 3. Microneurosurgical techniques and controlled hypotension with sodium nitroprusside were used during the surgery.

Postoperatively, the intracranial pressure (ICP) was measured in all patients using an intraventricular or subdural catheter, an externally mounted transducer, and a heated stylus pen recorder.

Routine clinical observations were supplemented by a neurological score chart, scaled 20-0 and indicating a progressively deteriorating level of response (Table 1). Patients with a score of 16 or less for most of the postoperative course were classified as having "deteriorated." This technique, which allowed a quantitative measurement of the clinical response, was used statisfactorily by both medical and nursing staff.

In addition, in 60% of the patients it was possible to pass a cannula retrograde via the superficial temporal artery to the common carotid bifurcation. Postoperative angiograms were performed using the cannula. The arterial $CO_2$ ($PaCO_2$) and arterial bood pressure (ABO) were monitored prior to each angiogram. Serial changes in the arterial calibre were studied by taking measurements of the internal carotid artery near its bifurcation and of the proximal anterior and middle cerebral arteries. The percentage change from the preoperative values were then calculated. These techniques have been described elsewhere (1).

## Results

The ICP pattern were divided into three groups based on the mean intracranial pressure (MICP) for most of the postoperative course (Table 2). The MICP was calculated using the definition of BROCK and HARTUNG (2).

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1 Section of a thesis submitted by Mr. FEARNSIDE for the degree of Master of Surgery in the University of Sydney.

Table 1. Neurologic scale of level of response

| | |
|---|---|
| 20 | Fully alert. Orientated. Not confused. Headache. |
| 18 | Alert. Grossly orientated but slighly confused. Meningism. |
| 16 | Drowsy. |
| 14 | Partially disorientated. Restless. |
| 12 | Difficult to rouse. Grunts. Disorientated. |
| 10 | Very drowsy. Rousable to pain. |
| 8 | Purposive response to pain |
| 6 | Flex limbs non-purposively to pain. |
| 4 | Extend limbs non-purposively to pain. |
| 2 | Reflex response to pain. |
| 0 | No response to pain. |
| | (Add 'A' for focal signs) |

Table 2. Postoperative intracranial pressure groups

| | |
|---|---|
| Group I | MICP less than 15 mmHg for most of the postoperative course. |
| Group II | MICP between 15 mmHg and 25 mmHg for most of the post-operative course. |
| Group III | MICP greater than 25 mmHg for most of the postoperative course. |
| | IIIA - MICP reached 25 mmHg in less than six hours |
| | IIIB - MICP reached 25 mmHg in greater than six hours |

In Group I, the MICP was less than 15 mmHg for most of the postoperative course. In Group II, it lay between 15 mmHg and 25 mmHg and in Group III, the MICP was greater than 25 mmHg. When the rate of rise in the first six hours after surgery in Group III patients was studied, two sub-groups were identified.

*Group I*

The mean of the MICP levels among 14 patients in this group showed that the pressure was low for several hours after surgery, tending to rise slowly to a plateau after eight to ten hours. Of the 14 patients, 13, including the three with unruptured aneurysms, had uneventful post-operative courses and a good result. One patient in this group who developed spasm died.

*Group II*

Of the 21 patients in Group II, 12 had uneventful courses but nine deteriorated and one of these died. Comparison between these revealed that in those who had deteriorated, the MICP was significantly higher immediately postoperatively ($p < .05$), the early rate of rise was more rapid ($p < .05$) and the plateau level was reached earlier ($p < .05$). All the patients who deteriorated in this group had spasm.

*Group III*

All the 21 patients in Group III had an eventual MICP greater than 25 mmHg and all deteriorated postoperatively. There was no difference between the MICP levels in Group IIIA and Group IIIB immediately after operation. However, in the 14 patients in group IIIA, the MICP rose more rapidely, reaching a level of 25 mmHg within six hours, a mean rate of rise of 7.9 ± 4.6 mmHg/h (1SD). There was a slower rate of rise in the seven patients in Group IIIB and the mean rate of rise was 3.1 ± 1.89 mmHg/h (1 SD).

*Arterial Spasm*

The time course of angiographic spasm was studied by measuring changes in the arterial calibre in the serial angiograms. We found that the arterial calibre in uncomplicated patients varied by ± 25% and considered that spasm was present when the mean of the percentage decreases in the three basal arteries exceeded 25%.

When comparison was made between those patients with and without spasm, the first angiogram, which was performed within two hours after surgery, revealed significant differences in the arterial calibre ($p < .02$). When spasm developed, there was a further rapid decrease in calibre, maximal after eight to ten hours and thereafter changing little. In all patients with spasm, a simultaneous rise in the ICP accompanied the angiographic changes, although the actual levels varied.

*Postoperative Pathology*

Fourteen patients had an uneventful postoperative course (Table 3). Of the complications, the most common was early spasm and 18 of the 20 patients with this finding deteriorated postoperatively, although the intracranial pressure levels varied. Five patients died. Two patients developed spasm 50-60 hours postoperatively, but this was less clinically significant.

Hydrocephalus, usually characteristed by a fluctuating neurological condition, was a major factor in eight patients. All required CSF diversion.

Table 3. Major postoperative factors

| | |
|---|---|
| Uncomplicated | 14 |
| Spasm | |
| Early | 20 |
| Late | 2 |
| Hydrocephalus | 8 |
| Raised ICP, small ventricles, no spasm | 7 |
| Other | |
| Intraop. pulm. embolus | 1 |
| Extradural clot | 1 |
| Operative factors | 3 |

A group of seven patients who had no angiographic spasm nevertheless showed a Group III ICP pattern, usually with a slower rate of rise in the ICP postoperatively and reaching an MICP of 25 mmHg more than six hours after surgery. All had surgery within a week of the most recent haemorrhage. Five of the seven had hypertension which was inadequately treated or untreated on admission and six had an aneurysm of the anterior cerebral or anterior communicating artery. There were no deaths in this group.

A complicated postoperative course could be predicted if the ICP one or two hours after surgery was greater than 5 mmHg, if the rate of rise was greater than 3 mmHg/h, or if the ICP rose above 25 mmHg. Clinical deterioration, however, was also noted in some patients whose ICP was less than 25 mmHg and who had spasm.

## Discussion

This study confirmed the findings of others (4, 6) that raised intracranial pressure following aneurysm surgery is associated with a poor clinical state. However, some patients in ICP Groups I and II deteriorated postoperatively, and all of these had spasm. A possible explanation of the observation of clinical deterioration with a relatively normal intracranial pressure may be the involvement by spasm of the small perforating vessels which supply the diencephalon and brain stem reticular formation.

Angiographic deterioration was noted within hours of surgery in those patients who developed spasm. Most of these had an early, rapid rise of intracranial pressure within hours of surgery, although the actual level if ICP finally reached, varied. Clinical deterioration almost always followed. These data suggest that an intraoperative factor or factors contributed to the development of spasm. A combination of factors, e.g., "primed" arteries (5), operative manipulation and "low flow" states resulting from brain retraction and induced hypotension may contribute. The early onset of angiographic spasm has abvious therapeutic implications.

Raised intracranial pressure was also noted to occur on occasions in the absence of angiographic spasm or ventricular enlargement. Most of these patients had untreated or inadequately treated hypertension and all underwent early surgery. Spasm of angiographically invisible vessels may be responsible.

## Conclusions

A number of correlates with postoperative deterioration following aneurysm surgery were found in a study of 55 patients. Cerebral arterial spasm was the most common complication and was usually associated with neurological deterioration, although the actual level of the ICP varied. The spasm was angiographically visible within 2 hours of surgery and reached a maximum in 6-8 hours, thereafter changing little. Clinical deterioration followed the angiographic changes by six to ten hours. The early onset of angiographic spasm has abvious therapeutic implications. Other causes of deterioration were hydrocephalus and raised intracranial pressure without angiographic spasm. This latter complication was commonly seen emong hypertensive patients who underwent early surgery.

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# Aneurysms in the Elderly

R. A. C. JONES

While the highest incidence of subarachnoid haemorrhage occurs in the 5th and 6th decades of life, approximately 20% of such haemorrhages present in the 7th and subsequent decades (3), when consideration of age assumes increasing importance in treatment, since the increasing incidence of other diseases, particularly ischaemic heart disease and hypertension, may modify attitudes to the risk of re-bleeding.

Any group of older patients is necessarily selected, because physicians do not refer patients of poor risk, and the referral unit tends to be selective in admission. Thus, such groups favour the better quality patient, those without major deficit, or those better preserved for their age. "Elderly" patients are not readily defined as a group, but those in axcess of 60 years form a significant entity, and review of such group, having in common a ruptured aneurysm, should enquire whether operative intervention contributes to the subsequent quality of life of those surviving the haemorrhage causing referral.

## Materials and Methods

An otherwise unselected group of 56 patients, over age 60 years at first presentation, and shown to have suffered from a ruptured intracranial aneurysm, were found to constitute 14% of all adult patients with identified ruptured aneurysm, admitted under one surgeon over a decade. There was a strong female bias of 46:10; all but one between the ages of 60 and 69; 50% presenting with a coma-producing subarachnoid haemorrhage. Thirty (53%) of these patients were admitted within 7 days of first leakage, 44 (78%) within 2 weeks. They were assessed by neurological grade (1), and policy was dictated largely by the neurological grade recorded during subsequent observation. Favourable grades I, II, and III were submitted to diagnostic angiography, latterly preceded by tomographic scan (CT scan). During investigation the neurological grade was reviewed for change - there was stability of grade in 45 (of which 34 underwent operation), improvement in 5 (3 operations), while 6 showed deterioration (no operation, one survivor). This continuing assessment of grade during investigation allowed 37 patients (68%) to be submitted to operation (36 intracranial clipping, 1 carotid ligation). Of the 37 undergoing operation, 8 (22%) did so within 7 days of leakage, 22 (68%) within 2 weeks. The 34 survivors at 6 months from haemorrhage (27 postoperative) were submitted to a functional grade which related to their level of activity. At this age, patients are less likely to resume employement, many accept early retirement; thus, full activity may be equated with good quality domestic life. The level of independence or dependence may depend upon the social situation of the patient or the presence of a relative able to provide an environment compatible with their functional level.

Patients were allotted to four functional groups, namely Grade I: Full activity (good quality retirement); Grade II: Light work or domestic duty, independent; Grade III: Significant deficit, neurological or intellectual, producing dependence, but capable of home care; Grade IV: Gross deficit, institutional care.

Patients were followed for up to 10 years, assessment of functional capacity commencing at 6 months after treatment.

## Results

The group of 56 patients was submitted to a retrospective comparison of the admission grade against status at 6 months (Table 1). Of those entering at Grade I there were two deaths, those entering at Grade II showed three deaths (with six survivors in dependent grades). There was a high mortality in Grade III patients, with 16 deaths, 6 surviving in a dependent state, while 7 returned to an independent life. One of two Grade IV patients gained admission died. Thus, viewed after 6 months, there were 22 independent patients, 40% of the whole group (64% of survivors), there were 12 survivors (20%) in dependent Grades III and IV, and 22 deaths (40%).

The group was then reviewed for outcome related to aneurysm site (Table 2). The anterior communicating group included peripheral (pericallosal) lesions, while multiple aneurysms were not subdivided, though the majority of these showed an internal carotid aneurysm as one component when it was most often the site of leakage (7 cases). While the anterior communicating aneurysm was the site of leakage in the majority of cases of single aneurysm (21) the commonest single site of aneurysm rupture was the internal carotid artery since there were 18 single lesions with 7 from multiple aneurysm complexes (totalling 25 ruptures). Of 21 patients with anterior communicating and pericallosal lesions, 11 underwent operation (with 4 deaths), 10 were treated convervatively (5 deaths). Of single internal carotid aneurysms (18) there were 15 operations with 3 deaths, and 3 conservative regimes (3 deaths). Of 11 patients harbouring multiple aneurysms, 7 underwent operation for the responsible lesion, with 2 deaths.

The 37 operated patients were assessed for comparison of preoperative grade with final outcome at 6 months (Table 3). There was an increase in the number of Grade I patients before operation, with one death in this group. There were 20 independent survivors after operation (54% of the operated group), 7 dependent, and 10 deaths, 6 of them from a pre-operative Grade III. The major cause of death was cerebral infarction, but there was one operative myocardial infarction, and one late death shown at autopsy to be due to cerebellar metastatic neoplasm. In the longer term review of survivors there were two late deaths from malignant disease, both over 3 years from first treatment, both after good quality survival.

Of the 19 conservatively treated patients, a non-operative policy was dictated by their unfavourable neurological status, there were 2 early

Table 1. Admission grade compared with final grade: 56 patients - 37 operations; 19 conservative regimes

| Admission grade | Final grade I | II | III | IV | Dead |
|---|---|---|---|---|---|
| I | 12 | 1 | - | - | 2 |
| II | 1 | - | 5 | 1 | 3 |
| III | 6 | 1 | 6 | - | 16 |
| IV | 1 | - | - | - | 1 |

Table 2. Outcome assessed by aneurysm type: 56 patients

| Aneurysm type | All cases by site | Operated | | Conservative | |
|---|---|---|---|---|---|
| | | Total | Deaths | Total | Deaths |
| ACA | 21 | 11 | 4 | 10 | 5 |
| ICA | 18 | 15 | 3 | 3 | 3 |
| MCA | 6 | 4 | 1 | 2 | - |
| Multiple aneurysms | 11 | 7 | 2 | 4 | 1 |

deaths from re-bleeding, 13 patients died without leaving hospital, and there was 1 further late death from re-bleeding. There were three survivors who led good quality lives.

## Discussion

While the elderly patient, having survived the initial haemorrhage, remains at risk from recurrence, it is necessary for the surgeon to take account of the special considerations of the age group in formulating a policy for treatment. In the considerable literature concerning selection and treatment of ruptured aneurysm, the elderly have not been separately reviewed, so their treatment continues to present a dilemma. This review was undertaken in an attempt to resolve the dilemma by providing evidence supporting operative intervention in defined groups of patients.

It is accepted that prognosis after ruptured aneurysm is dictated largely by the neurological state after haemorrhage, and that this group shows some deviation from the anticipated pattern, with both unexpected deaths in favourable patients and qualities of surival better than the initial grading would have suggested. The further selection process, based on grade, by which the operated group was formed, shows greater conformity to expectation, with exceptions. The influence of aneurysm site was most marked in the anterior communicating (ACA) territory, where there was a mortality of 40%, involving both operated and unoperated patients, whereas the internal carotid territory carried a relatively more favourable prongosis, and contributed to the 54% of independent postoperative survivors.

The implications seem clear, therefore, that selection of operative treatment in this group should depend on both neurological status and anatomical location. While cerebral angiography remains the crucial

Table 3. Operated patients - 37; preoperative grade compared with final grade

| Preoperative grade | Final grade | | | | |
|---|---|---|---|---|---|
| | I | II | III | IV | Dead |
| I | 14 | 1 | 1 | - | 1 |
| II | - | - | 3 | - | 3 |
| III | 4 | 1 | 2 | 1 | 6 |
| Total | 20 Independent | | 7 Dependent | | 10 |

investigation before a diagnosis or ruptured aneurysm can be made, it would be valuable to obtain information about the site of haemorrhage to allow modification of the extent of angiography, and there is evidence (2) that the CT scan provides this. The present group contains too few patients (7) in the era of the CT scan to give clear guidance, but were the expectations of the CT scan to be realised, older patients shown to have ruptured an ACA might be considered best treated conservatively, while lateralised abnormalities should lead to unilateral carotid angiography (since in this age group, multiplicity of aneurysms is less significant, and requires treatment only of the ruptured lesion).

## Conclusion

Though the elderly patient is less resilient to the effects of aneurysm rupture and operative intervention than younger ones (showing a 40% overall mortality), of those selected for operation over 50% can become fully independent when assessed after recovery. Those shown to have ACA do not fare well compared with other sites, and it is suggested that the CT scan should aid selection for definitive study.

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# The Backward Technique in the Total Excision of Cerebral Arteriovenous Malformations: Experience With 86 Cases

B. PERTUISET and J. P. SICHEZ

## Introduction

Regarding the radical excision of the cerebral arterovenous malformation (AVM) all textbooks advise isolating and clipping or ligating first the arterial trunks supplying the malformation before dissecting it, and to terminate the operation by coagulating or clipping the venous drainage. This strategy was recommended by DANDY (1) who reported a disastrous issue in one case of a primary ligation of the venous drainage. One must remember that at that time angiograms were not available, and the patient was under local anesthesia. Nevertheless, since then everyone has followed this apparently rational recommendation.

## Material and Approach

A careful analysis of the angiograms led us to a different opinion:

1. The arteries supplying the AVM are tortuous and serpentine. It is therefore rather impossible to cut them at the site where they enter the AVM. Consequently, a softening of the brain around the AVM can occur when a feeding artery is cut too far from the AVM border since, in this case, some collaterals are deprived of their blood supply. This can provoke a postoperative swelling and, moreover, neurological deficit when the patient survives.
2. With the exception of very few cases (4 in a series of 145), there is always more than one vein for drainage. Therefore it is feasible to use one of the veins to begin the dissection without impairing the total drainage of the AVM.

At the same time we experienced that such malformations were consistent enough to be pulled out with a forceps thus making straight the tortuous arteries which can be severed at the site of their penetration into the AVM. We have called this particular approach the "backward technique" and the senior author has reported his first experience using this technique in 1968 on the basis of a series of 15 cases. Since then, all patients were operated on using the backward technique when the angiogram demonstrated the presence of more than one vein for drainage. In a series of 150 AVM operated on from 1958 to 1977, 86 AVM have been removed in this way in conscious patients:

| | |
|---|---|
| Superficial hemispheric | 77 |
| Grey nuclei | 5 |
| Intraventricular septum | 1 |
| Cerebellar | 3 |

This technique has not been used in the following cases:

| | |
|---|---|
| Superficial hemispheric (before 1965) | 38 |
| AVM with hematomas and coma | 17 |
| AVM with one drainage vein | 4 |
| AVM of the external carotid territory | 5 |

In order to perform a more careful dissection, mainly in the motor and speech areas, we have been using profound hypotension during the last two years. The patient is under neuroleptanalgesia, and hypotension is

obtained by a permanent intravenous injection of sodium nitroprusside with the aid of an electric injector. It is sufficient to lower mean arterial pressure, monitored in the radial artery, to 50 mmHg. During hypotension the brain is protected by autoregulation as demonstrated by the recording of the cerebral blood volume (Fig. 1). It shows clearly that blood volume increases in the brain when arterial pressure is lowered, while it decreases in the AVM. This phenomenon has been demonstrated also by comparing angiograms obtained without hypotension and under hypotension (Fig. 2). Seventeen patients were operated on under hypotension using the operating microscope. This permits coagulating the malformation from the veins towards the arteries and, thus, achieves a faster excision. Following total excision it is necessary to wait until mean arterial blood pressure is again at the 80 mmHg level prior to beginning the closure. It is usually at this level that small arteries begin to bleed. In three cases we have been forced to reinduce hypotension in order to achieve hemostasis. Control angiograms showed that by this technique the feeding arteries are severed at the border of the AVM (Figs. 3, 4). Hypotension is of great help during the dissection of the AVM but it does not facilitate much the hemostasis within large cavities (when the AVM is of great volume). This particular problem is under research at present in our clinic.

## Results

### *Mortality*

In the first series, published in 1968, there was no mortality in 15 cases. In the series of 86 cases published today there were 3 fatalities (3.4%). The causes of these fatalities were:

- one postoperative softening due to thrombosis of the vein of Galen;
- two blood perfusion problems, one of which could have been avoided easily.

### *Morbidity*

None of the patients developed seizures which did not exist before operation. Sixty percent of the patients who presented seizures before surgery were cured after 2 years of antiepileptic treatment. Forty percent of these patients required a permanent antiepileptic treatment. None of them have been aggravated. None of the patients operated on in the speech area of the dominant hemisphere developed permanent aphasia. All patients operated on in the motor area developed hemiplegia, the prognosis of which is related to the age of the patient despite intensive rehabilitation. In fact, the two last patients operated on in this territory, presented only a monoplegia.

## Discussion

The backward technique is certainly not the final answer regarding the radical treatment of AVMs. This technique, however, facilitates the dissection of the malformation and, moreover, avoids sectioning of the feeding arteries far from their penetration into the AVM. In this respect the backward technique certainly represents an improvement. The use of profound hypotension and of the operative microscope added more safety, as may be particularly appreciated by the young neurosurgeons.

## Conclusion

In a series of 150 AVM operated on between 1958 and 1977, 86 cases have been radically removed using the backward technique which consists of beginning the dissection from one of the draining veins and proceeding towards the feeding arteries, thus avoiding the difficult and dangerous localization of these arteries when one begins the operation with them. There was a general mortality of 3.4% and an excellent result regarding seizures. Morbidity was higher for AVMs in the motor area than for those in the speech area.

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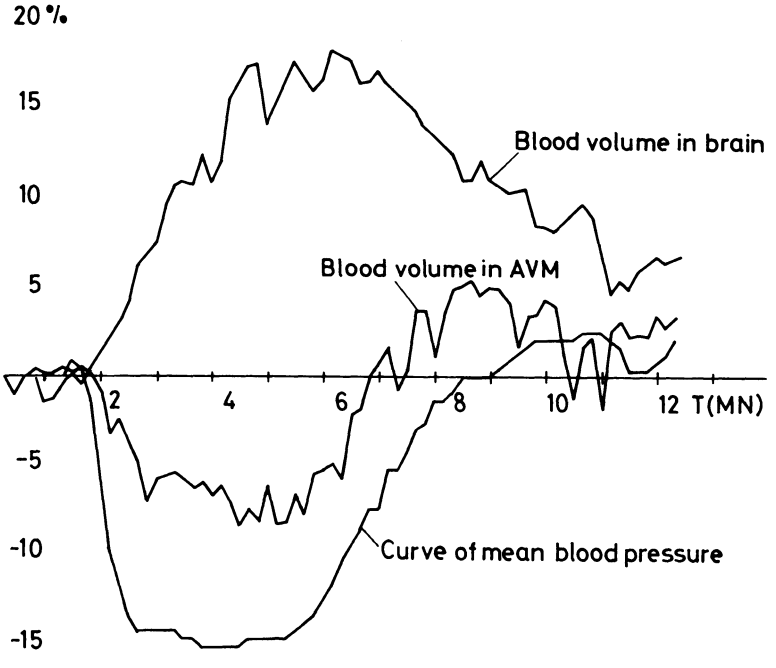


Fig. 1. Cerebral blood volume variations in an AVM and in the opposite hemisphere during hypotension induced by sodium nitroprusside

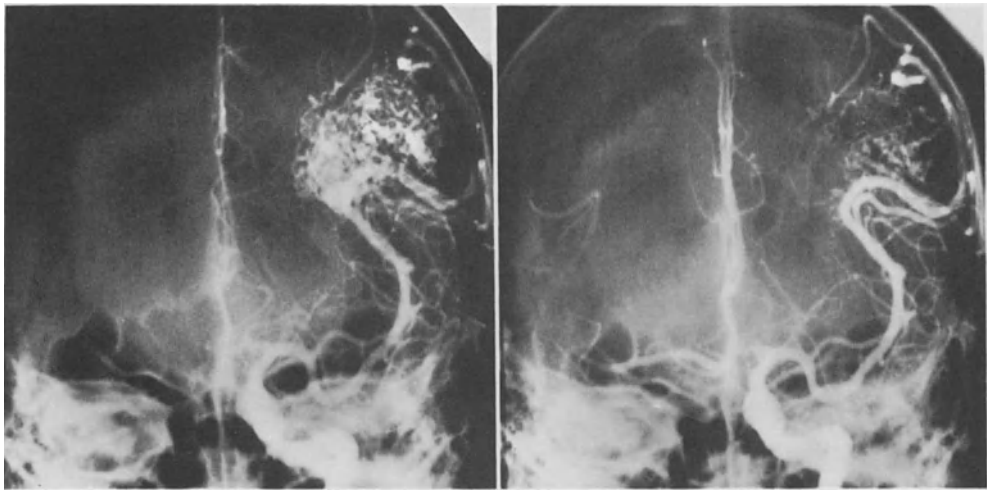

Fig. 2. Right parietal AVM (1977). Selective angiography (Telebrix 38-10 ml), injection rate: 6 ml/s. *Left*: without hypotension; *right*: with hypotension (40 mmHg mp) induced by sodium nitroprusside

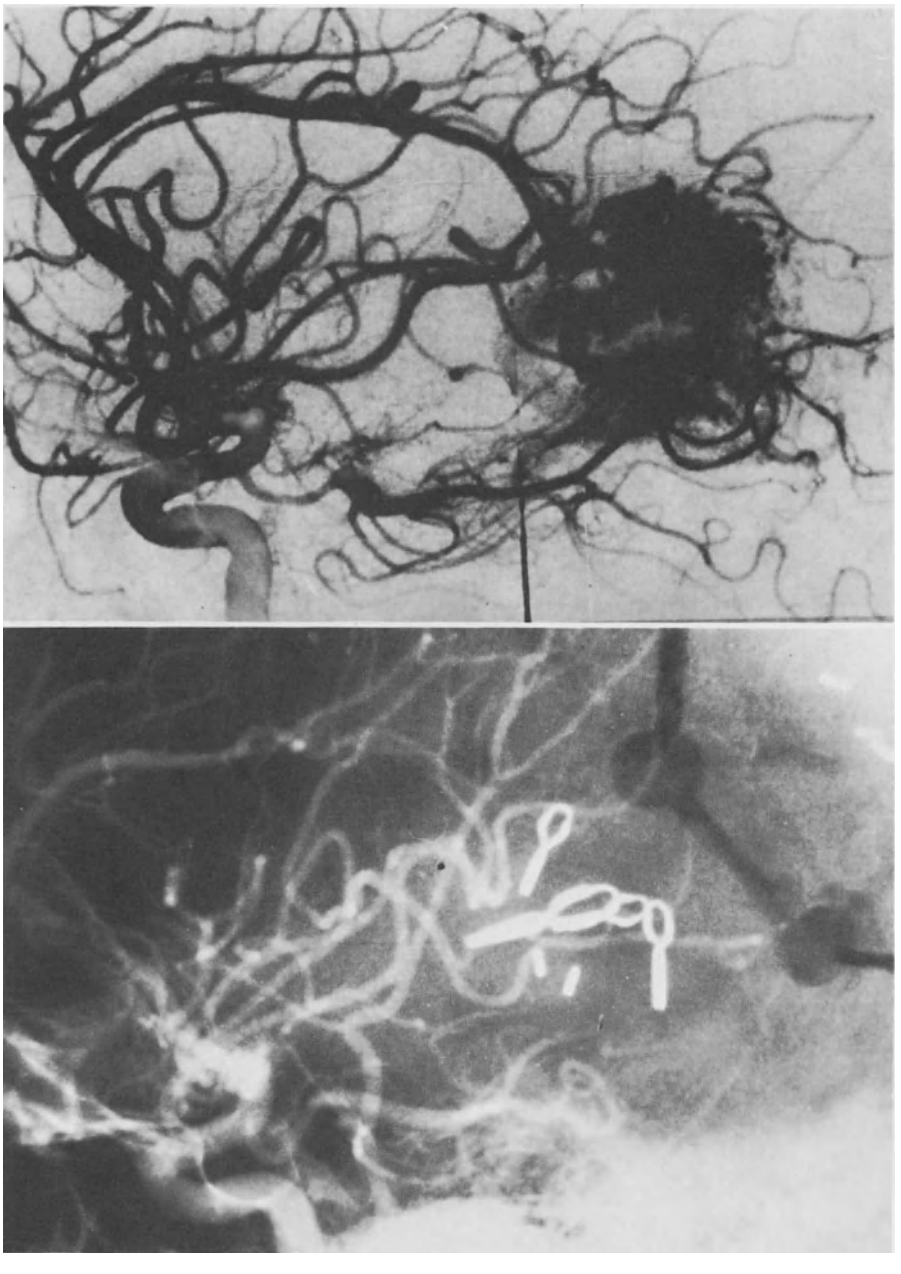

Fig. 3. Left AVM medial to the lateral ventricle's junction. *Above*: 16.11.1976; *below*: 14.12.1976

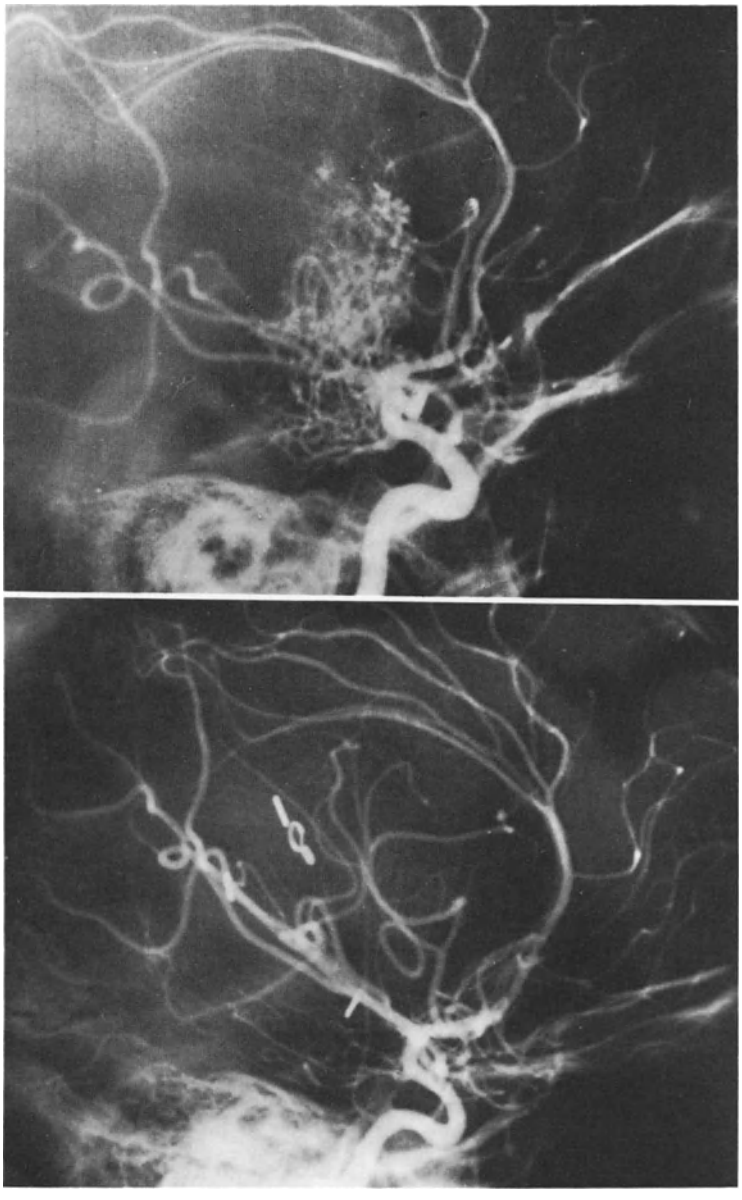

Fig. 4. Pallido-caudate AVM (1976). Lateral view. *Above*: preoperative angiogram; *below*: postoperative angiogram

# Fibromuscular Dysplasia

T. A. LIE

Fibromuscular dysplasia (FMD) is a nonatheromatous stenosing disease of medium-sized arteries with unknown etiology. The first description of this condition was on the renal artery by LEADBETTER and BURKLAND in 1938. In 1965 CONNET and LANSCHE reproted the first case (angiographically and histologically demonstrated case) of FMD of the internal carotid artery. Widespread use of cerebral angiography has allowed the discovery of this disease in an increasing number of cases. The condition is not so rare. HOUSER et al. observed 42 cases in a review of 7000 cerebral angiograms. However, in the last two years they encountered just over 20 cases, so that they expect that FMD of the carotid arteries might be observed in 1% of all cerebral angiograms.

Histologically, any of the layers of the arterial wall may be involved, but the media is affected in most cases. Areas of hyperplastic rings of fibrous tissue and muscle producing narrowing of the vessel lumen alternate with segments of medial thinning, giving rise to dilatations. These alternating zones of widening and narrowing of the arterial lumen produce the characteristic appearance of a string of beads or loose stocking irregularities. The abnormal arterial pattern is usually confined to the extracranial internal carotid at the level of C2-C3. Involvement of the intracranial portion of the internal carotid is rare. FMD of the middle cerebral artery has been recorded by HUBER and FUCHS (5) and by OSBORN and ANDERSEN (9). The external carotid, vertebral, and occipital arteries may be also affected.

Clinically, the angiopathy may be an incidental finding, but, as the disease produces stenosis of the internal carotid artery, about two-thirds of the reported cases had definite cerebrovascular symptomatology. Symptoms include headache, spells of dizziness, black-outs, focal seizures, etc. Transient ischemic attacks or infarction may occur. It is striking that in about 25% of the series of HOUSER et al., and of OSBORN and ANDERSEN (9) the condition was associated with a saccular aneurysm. In 12 of the 42 cases of HOUSER et al., the disease was discovered in combination with an intracranial neoplasm.

Diagnosis is made on the basis of angiography. There are three characteristic angiographic appearances of FMD:

1. The string of beads deformity, covering 89% of all the published cases and associated with FMD of the medial type;
2. The unifocal of multifocal tubular stenosis type, occuring in 7% of the recordings and found in any type of FMD;
3. Only one wall of the segment is involved, known as "atypical FMD."

Surgical treatment is limited only to patients with a significant angiographic narrowing of the carotid lumen. MORRIS et al. (8) introduced a technique of graduated internal dilatation of the carotid artery. Their good results, with clinical and angiographic follow-up ranging from two months to four years, led them to consider this procedure as the preferred mode of treatment.

## Case Reports

1. A 62-year-old woman was sent to the neurologist by the ophthalmologist because of slight bilateral papilledema and visual disturbances. On examination no neurological symptoms were found other than a suspect Babinski's sign on the left. Bilateral percutaneous carotid angiography revealed a temporal process on the right side. Since there was no upsweeping of all branches of the middle cerebral artery, an extracerebral process, most probably an outer ridge meningeoma, was diagnosed. There were also the typical strings of beads, pathognomonic for FMD of both internal carotid arteries at the subpetrosal level (Fig. 1). She was transferred to our department on March 3, 1978, operated upon, and the meningeoma totally removed. The postoperative course was uneventful.

2. A 23-year-old female was admitted to a hospital on August 2, 1977. She was found on the street, where, after a severe headache, she had suddenly fallen and become unconsious. On admission she was thought to be in a terminal state, being in deep coma with wide unreactive pupils and total areflexia. Respiration worsened. Lumbar puncture revealed bloody CSF. She was transferred to the intensive care unit and treated with assisted respiration and high doses of dexamathasone. Two days after admission she regained consciousness, and no neurological symptoms were found. A four vessel angiography using Seldinger's technique was performed. Both internal carotids showed the typical appearances of FMD, with mural aneurysms at the level of C2-C3, except for the left, where the abnormality extended into the intracranial portion of the siphon (Figs. 2, 3). It was thought that a true supraclinoidal berry aneurysm was present on the left side (Figs. 2, 3). The left vertebral artery was small, but the right showed definite wall irregularities. She was admitted to our department. An operation was performed on August 15, 1977, using the microscope. Signs of an old hemorrhage were found. No saccular aneurysm was seen, but the intracranial portion of the internal carotid consisted of two mural aneurysms with a very thin wall through which the blood stream could be observed. During dissection of the arachnoid one of the mural aneurysms ruptured and the internal carotid artery had to be trapped with two clips. The patient developed a right-sided postoperative hemiparesis and aphasia, which gradually improved after several months.

## Discussion

Both patients presented the characteristic angiographic appearance of FMD. In the first, the string of beads was found incidentally, in association with a temporal meningeoma. In the second, the left internal carotid artery was affected up to the intracranial carotid siphon, which is very exeptional. Unlike what has been reported in most publications, the dilatations were so wide that a wrong diagnosis of an associated saccular aneurysm had been made. The wall of the mural aneurysm was so thin that it caused severe subarachnoid hemorrhage. Although diagnosis is based on the typical appearance of the angiograms in most instances, STANLEY et al. (10) stressed that arteriography alone is not sufficient to diagnose FMD. One must also differentiate from stationary arterial waves (1), from collagen disease, from giant cell arteritis, and from hereditary disorders such as Marfan's syndrome and homocystinuria (3). In homocystinuria, MOREELS et al. (7) showed a rippled angiographic appearance of an iliac vessel, almost similar to that seen in FMD. We have also been able to study a patient with homocystinuria. Unilateral internal carotid angiography revealed the string of beads appearance.

We have seen one case of tubular stenosis type of FMD in which the left internal and external carotid artery, and both vertebral arteries showed multifocal tubular stenosis. However, follow-up angiograms after several months showed a normal image of the affected arteries again.


## Summary

Two cases of bilateral fibromuscular dysplasia of the internal carotid arteries are described. In one patient the string of beads on the left side proceeded intracranially as far as the carotid siphon, causing severe subarachnoid hemorrhage. In the second, the condition was found incidentally in association with a right-sided temporal meningeoma. It is stressed that angiography alone is not sufficient for the diagnosis of FMD in some cases.



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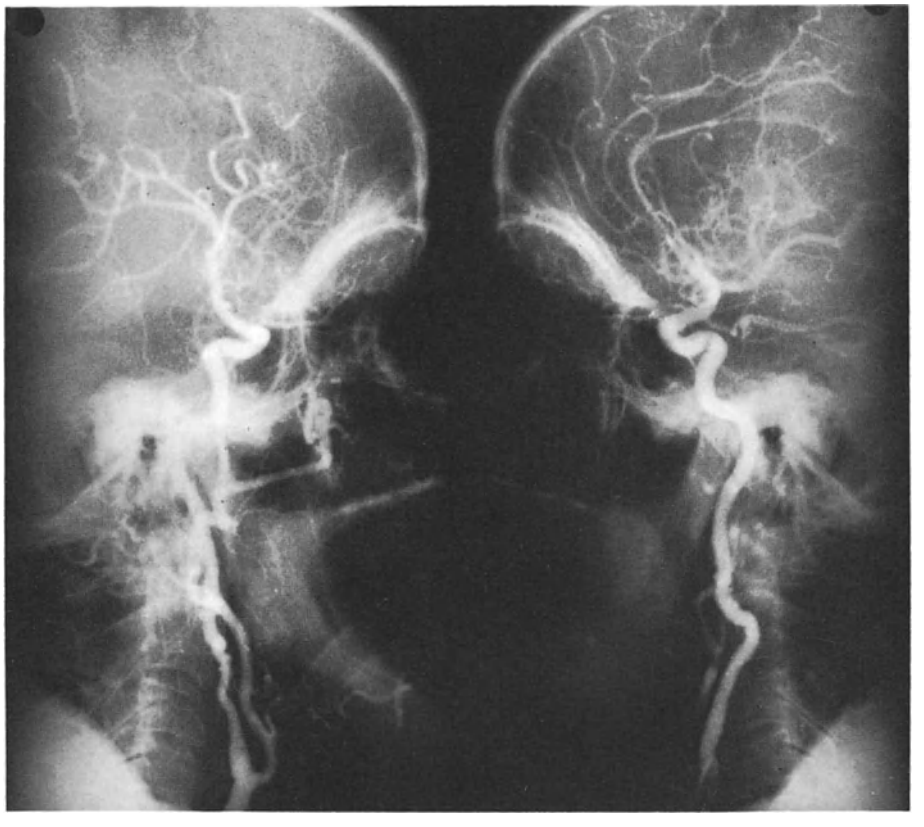

Fig. 1. The right middle cerebral artery is elevated. At the level of C2 the internal carotid artery on both sides showed the typical "string of beads"

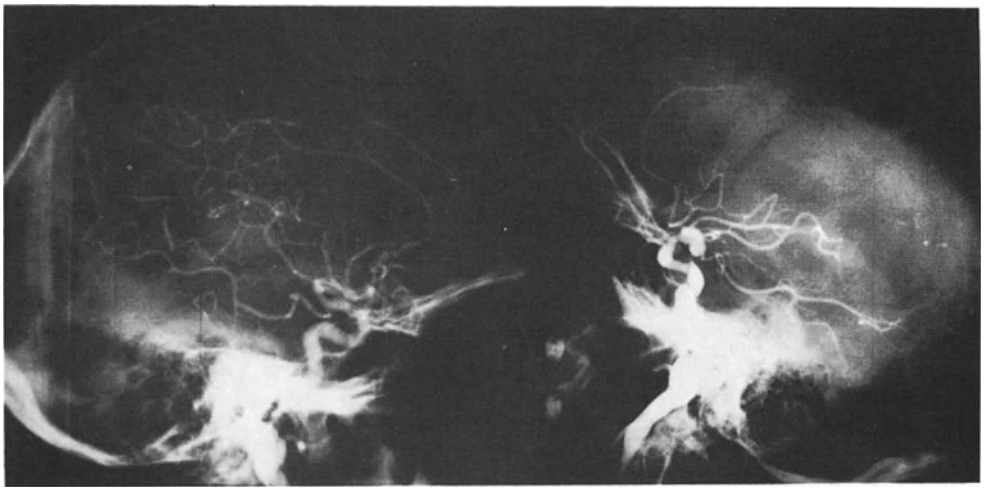

Fig. 2. The lateral view demonstrates multiple dilations and constrictions. There is also left-sided intracranial involvement of the internal carotid. A supraclinoidal aneurysm is diagnosed

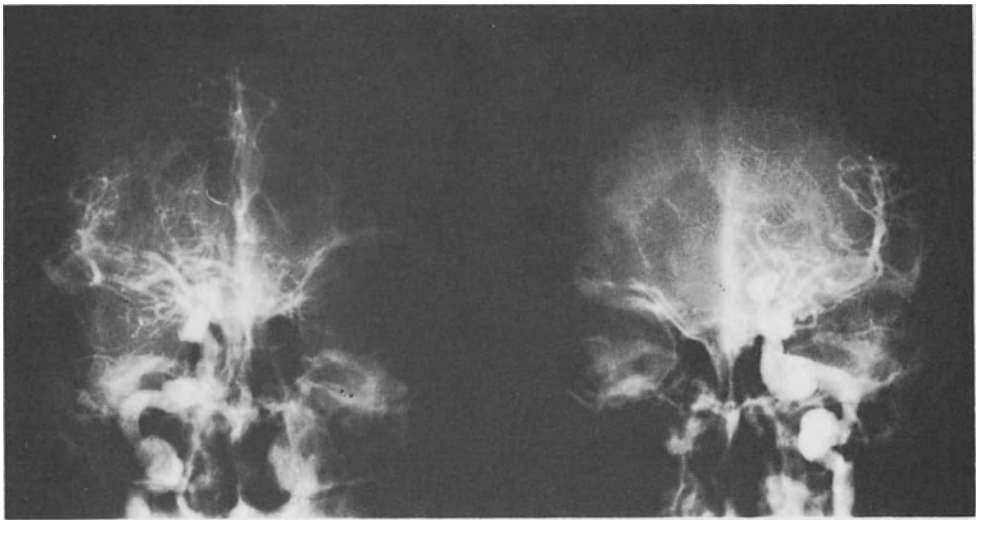

Fig. 3. Antero-posterior view of the same patient

# Disseminated Intravascular Coagulation in Head-Injured Patients

W. PONDAAG

Disseminated intravascular coagulation (DIC) is classified as an intermediary mechanism of disease (16), and defined as an acute, transient coagulation occurring in blood flow throughout the vascular tree, and potentially obstructing microcirculation. It may or may not result in an accumulation of fibrin, but does involve the transformation of fibrinogen into fibrin. It includes the agglutination of platelets and red cells, and the sticking of leucocytes (11). The DIC syndrome has been described in detail by McKAY (16) and HARDAWAY (11). There are recent reviews by DEYKIN (4), BLAISDELL and LEWIS (1), CASH (2), and others. The association between DIC and trauma is known, as is the relation to prognosis (22). However, so far DIC in cases of head trauma has received scant attention. Only a few case reports and short communications, and one or two more detailed studies are found in the literature (3, 5, 6, 8, 10, 13, 14, 15, 17, 21, 23, 24). Discussion with colleagues of the department of general surgery about the joint treatment of polytraumatized patients with headinjury aroused interest in this condition. The present study was undertaken to determine if DIC regularly occurs in brain-injured patients, and if so, if a relationship can be established with outcome and therapeutic consequences drawn.

## Patients and Methods

In retrospect, data of all patients admitted to a 600-bed community hospital with the diagnosis of head injury with or without associated lesions from January 1974 through March 1977 were reviewed. The diagnostic classification was: 1) commotio cerebri; 2) contusio cerebri; 3) skull fracture; 4) epidural hematoma; 5) subdural hematoma, and 6) intracerebral hematoma, according to the coding list of the Stichting Medische Registratie (this institute processes the medical data of the majority of Dutch hospitals).

Blood coagulation studies, performed immediately upon admission and serially afterwards, included a platelet count, determination of thrombin time (TT), levels of factor I (fibrinogen), factor V (proaccelerin or labile factor), and fibrin degradation products (FDP), and the ethanol gelation test (EGT). The following values were regarded as normal: TT 20-25 sec, thrombocytes 100-350 $10^9$/L, factor I 2-4 g/L, factor V 80%-120%, FDP negative, EGT negative. DIC was called positive (+), when the platelet count was below normal or the TT lengthened, factor I or V decreased, and FDP or EGT positive. DIC was called strongly positive (++), when both factors I and V were decreased, and FDP and EGT both positive or the FDP-level strongly raised (> 80µg/L). All other abnormal findings were considered to be inadicative of dubious DIC (±). Blood gases were determined according to the Astrup-method. Outcome was graded: I - complete recovery, II - recovery with deficit, or necessity of prolonged rehabilitation treatment, III - survival in persistent vegetative state, IV - no recovery.

## Results

During the period mentioned above, 924 patients with head injury were admitted. The findings are summarized in Tables 1-4. It should be stressed that not a single case of missile injury was admitted.

Coagulation studies were performed in 11.1% of all patients. In 33% they yielded abnormal results. It may be deduced that the frequency of DIC + and ++ in this whole population will be between 2.5% and 13.5%. Although the commotio/contusio concept is rather ill-defined, and not too useful, in the context of this study it can be safely assumed that, in this way, at least a distinction is made between a group of patients with slight brain injury and a group of severely brain-injured patients, with perhaps some overlap in the group with moderate injuries.

There seems to be a trend for DIC to occur more frequently in the more severely injured patients, but this is not yet statistically significant. However, there is a significant ($p < 0.01$) difference in mortality between the groups with positive and negative findings. In patients with DIC ±, + and ++, there also exist significant relationships between grades of DIC and grades of injuries and outcome.

In reviewing the blood-gas values obtained immediately at admission, it was found that in patients presenting a pH < 7.30, the mortality was 100% ($p < 0.001$). There was no relation between the first $paO_2$ value obtained, or for that matter between the lowest $paO_2$ value in serial studies, and mortality. All patients with DIC ± had an initial pH between 7.35 and 7.50. All patients with an initial pH < 7.35 and > 7.50 showed DIC + or ++.

Table 1. DIC in 924 head injured patients

| | DIC | No DIC | Unknown DIC | Total |
|---|---|---|---|---|
| Commotio | 8 | 29 | 574 | 611 |
| Skull fracture | 1 | 0 | 42 | 43 |
| Contusio | 23 | 37 | 154 | 214 |
| Epidural hematoma | 1 | 0 | 11 | 12 |
| Subdural hematoma | 1 | 3 | 36 | 40 |
| Intracerebral hematoma | 0 | 0 | 4 | 4 |
| Total | 34 | 69 | 821 | 924 |

Table 2. Degrees of DIC in different kinds of injuries

| DIC | Commotio | Fracture | Contusio | Hematoma | Total |
|---|---|---|---|---|---|
| ± | 6 | 1 | 3 | 1 | 11 |
| + | 2 | 0 | 7 | 0 | 9 |
| ++ | 0 | 0 | 13 | 1 | 14 |
| Total | 8 | 1 | 23 | 2 | 34 |

Table 3. Mortality in relation to DIC and type of injury

| | DIC | No DIC |
|---|---|---|
| Commotio | 3+/29 | 0+/ 8 |
| Contusio | 6+/37 | 12+/23 |
| Hematoma | 2+/ 3 | 11+/69 |
| Fracture | 0 | 0+/ 1 |
| Total | 11+/69 (15.9%) | 14+/34 (41.2%) |
| Total mortality | 25/103 = 24.3% | |

Table 4. Relation between degree of DIC and outcome

| DIC | I | II | III | IV | Total |
|---|---|---|---|---|---|
| ± | 8 | 1 | 0 | 2 | 11 |
| + | 2 | 3 | 0 | 4 | 9 |
| ++ | 3 | 3 | 0 | 8 | 14 |
| Total | 13 | 7 | 0 | 14 | 34 |

## Discussion

DIC is in essence a state of hypercoagulability, followed by a secondary fibrinolysis, with intravascular coagulation being triggered by various stimuli such as capillary stasis, acidosis, release of catecholamines, or procoagulants. A close association does exist between DIC and "clinical shock," the latter being defined as an inadequate tissue-perfusion at the microcirulatory level. The DIC syndrome may have a fulminating clinical course, with a tendency toward abnormal bleeding as the most prominent feature, but it may also express itself as a successive organ failure (lung, brain, kidney, liver), sometimes remaining unrecognized as such, unless specific laboratory studies are performed.

Our findings imply that DIC plays a part in the pathophysiology of brain injury, especially in the more severe cases. DIC may be responsible for secondary brain damage, along with the damage caused by direct impact, as also suggested by others (3). The pathogenesis may be twofold: in brain injury (laceration) thromboplastins might be released into the systemic circulation, possibly acting as a procoagulant (5, 10). It has also been experimentally documented that total brain ischemia causes DIC (12). On the other hand, there are many reports on brain damage caused by DIC associated with extracranial trauma or disease. The ominous combination of (metabolic) acidosis and DIC observed by us suggests an intimate relationship between traumatic shock, DIC, and intractable traumatic brain edema at least in a number of patients.

An important therapeutic consequence is the vigourous treatment of shock, which is the best way to treat and prevent DIC (19). Ample fluid should be given intravenously (dehydrating therapy is contra-indicated in the initial phase at any rate and ventilation should be adequate. Pharmacological doses of dexamethasone (100-200 mg) should also be used in other conditions of shock and DIC, e.g., in septic

shock (18). The recently reported beneficial effect of high-dose steroid therapy in severe head injury (7, 9) might be related to its systemic effect. One should avoid administration of procoagulants, and use only fresh blood (20), infused through a micropore filter. For evident reasons, heparin therapy is seldom, if ever, indicated.


## Summary

The retrospective review of the records of 924 patients admitted for head injury, revealed a 2.5%-15.3% incidence of disseminated intravascular coagulation in such cases. There tends to be a relation between DIC and the grade of brain injury. DIC is associated with a raised mortality, especially when accompanied by acidosis. Pathogenesis and therapeutic implications are discussed.


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# Operative Angiography

W. J. OVERBECK

In neurology and neurosurgery, preoperative angiography of carotid and vertebral arteries is generally used to demonstrate vascular anomalies and saccular aneurysms. Likewise, postoperative angiography, although less generally used, is a means of evaluating the result of an operation. To every neurological surgeon, cases are known where postoperative angiography proved the operation had not been completely successful. It would have been much better if the surgeon had known this before putting an end to the operation. Some surgeons have tried to achieve that goal. One of the first papers on this subject was by MOORE et al. (6) in 1948. They injected fluorescent dye intravenously 1-2 h before the operation and examined both the removed tumor tissue and the residual cavity under ultraviolet light to see whether the tumor had been totally removed.

LOOP and FOLTZ (5), in 1966, were the first to perform operative angiography. Thereafter, VLAHOVITCH et al. (11, 12), published a report on a series of 50 gliomas in 1967 and a series of 19 vascular malformations in 1969. TURNER and GROSSART (3, 9, 10) reported on the same subject in 1970, 1971 and 1974, ultimately completing a series of 50 patients with aneurysms or A.V. malformations. Less extensive experience as well as single cases were described by BARTAL (1), LAZAR (4), CUMMINS (2) and PEETERS (7) and their co-workers. All agreed on the following value of the method: localization of the lesion is facilitated; approach to the lesion is made easier; the various stages of the procedure can be checked. Also, unwanted complications such as the clipping of a major vessel can be recognized easily. There are no technical difficulties in performing operative angiography. The operating time is not necessarily prolonged. In the neurosurgical clinic of the University of Groningen, operative angiography has been a routine procedure in most cases of saccular aneurysms and A.V. malformations since 1973. So far the method has been used approximately 80 times.

## General Procedure

Operative angiography is performed by means of catheterisation as introduced by SELDINGER in 1953 (8).

## Premedication

A sedative cocktail, composed of 0.5 mg atropine, 25-25 mg pethidine, 25 mg phenergan, and 25 mg largactil is routinely administered.

## Local Anesthesia

The site of puncture is locally anesthetized by infiltration with approximately 10 ml of a solution of lidocaine 15 g/v and adrenalin 0.01 mg/ml.

## Needles, Catheters, Instruments

The puncture is performed with a steel needle with a hollow inner needle and solid mandrin (type PE 160, KIFA, ELEMA, STOCKHOLM). The catheters are of radiopaque polyethylene, torque controlled through a steel braiding inside the wall. We generally use a headhunter no. 1 or 3, or a sidewinder no. 4, French 7, length 100 cm, as manufactured by COOK, INDANIA or COOK, DENMARK. The catheter is introduced over a teflon coated guide wire size 0.038, length 145 cm, having either a fixed core and a flexible tip or a movable core and a J-tip of 3 mm diameter. After introduction, the catheter is connected to a saline drip to which 0.5 g/l heparin is added. The catheter is flushed with 3-6 drops per min. This can be done in various ways, be it under pressure or by hanging the saline on a level higher than needed to overcome arterial blood pressure (5-8 feet).

## Injector

The contrast material is injected by a Medrad Mark II injector.

## X-Ray Unit

The x-ray unit is a Philips Diagnost OP-C9 with a superrotalix x-ray tube with foci of 0.15 mm and 1.5 mm, an image intensifier, and a 70 mm film camera.

## Contrast-Material

We have been using Vasobrix which is methylglucamine - ioxithalamate and mono-ethanolamide-ioxithalamate 1:2, containing 32% iodine, yet has a relatively low viscosity, making it suitable for demonstrating small and often spastic vessels.

## Catheterisation

Introduction of the catheter(s) is done in the x-ray room, under local anesthesia, as mentioned before. The introduction site is either one femoral artery or both arteries if two catheters are required. Under fluorscopy the catheter is then moved up into the desired artery. The patient is then transported to the operating theatre and placed on the operating table or, if the operation is to be performed under hypothermia, in the bath tub. The patient's head is placed outside the steel bath tub on a support that is pervious to x-rays. Once the patient is placed in the right position for the operation the first series of spot films is made under an angle of incidence that shows the lesion well. During and after surgery as many series of spot films as needed are made to assist the surgeon in eliminating the lesion as completely as possible.

## X-Ray Technique

Prior to the beginning of the surgical procedure one series of 70 mm spot films is made, the head of the patient being in the position most suitable for the operation. The number of images of one series depends on whether one is interested in the arterial phase only (six to eight films at two per sec), or whether the late venous phase is important

as well (12 ti 16 films may be required). The amount of contrast material is 6-10 ml per series. The number of series may vary from 2 to 13 (our largest number). The catheter will dwell until the operation is completed and the patient is back in the recovery room. Our longest permanence time for a catheter is 8.5 h so far. A good average is 4 h. We have not seen any complications at yet. The personnel will receive no radiation, as they leave the operating theatre while the series is being made. A series will be ready for viewing within 2-5 min, the x-ray department being next to the operating theatre. Viewing may be done without magnification. The following case histories demonstrate the advantages of operative angiography.

The first patient is a 25-year-old man whose right-sided carotid angiogram demonstrated an A.V. malformation in the fronto-temporal region, apparently fed by two dilated branches of the Sylvian artery, and with early venous drainage to the deep venous system and to the superior sagittal sinus. Figure 1 shows the pre-operative situation in a.p. and lateral projections. The malformation was approached by a fronto-parietal craniotomy. Figure 2 shows four successive operative angiograms demonstrating the various stages of this approach. The first picture, upper left, is the situation after removal of the bone flap. The second picture, upper right, shows a clip on an artery presumed by the surgeon to be one of the supplying vessels. It turns out not to be the right one. The third picture, lower left, again marks a wrong artery. Finally, the fourth picture is made: it is not the correct one after all! Figure 3 (upper left). The artery just behind the one being held by the forceps is the right one (Fig. 3, upper right). A clip is placed (Fig. 3, lower left); no more filling of the feeding artery. The same procedure is repeated for the second supplying artery, as is seen in Figure 4 (upper left, upper right, lower left). Finally, an a.p. projection demonstrates the edema caused by the operation (Fig. 4, lower right). Recovery of the patient was undisturbed.

The second patient, a 35-year-old man, had an A.V. malformation of a frontal branch of the sylvian artery. Figure 5 shows a.p. and lateral projections of the preoperative angiogram. The enlarged supplying artery and early venous return are well visible. As in the previous case, the sylvian artery was approached by frontoparietal craniotomy. In Figure 6, the top picture shows compression of the wrong artery, the middle picture demonstrates compression of the main feeder, the bottom picture the same artery clipped. Only minimal filling of the malformation is seen. The patient showed good recovery, the pre-operative epileptic seizures, reason for his hospitalisation, did not return.

The third patient, a 29-year-old woman was found to have a saccular aneurysm of the bifurcation of the left internal carotid, as is seen in Figure 7. The aneurysm having been approached by way of frontal craniotomy, a clip was placed over the base of the aneurysm. In Figure 8 the top picture shows the aneurysm before, the middle one after placing of the clip. Apparently there is no filling of the aneurysm, but neither is there filling of the anterior cerebral artery. However, as there were no signs of disturbance of circulation in the area, a spasm of the artery was likely to have occurred. The bottom picture, the third operative angiogram, made a little while later, showed good filling of the anterior cerebral artery again. There is still a short spastic stretch visible.

The last patient was a 49-year-old man with a saccular aneurysm of the anterior communicating artery (Fig. 9, left side). Following its exposure an operative angiogram was made (Fig. 9, upper right). A clip was placed on the base of the aneurysm with good result (Fig. 9, lower

right). There is still good filling of the anterior cerebral artery, and no more filling of the aneurysm.

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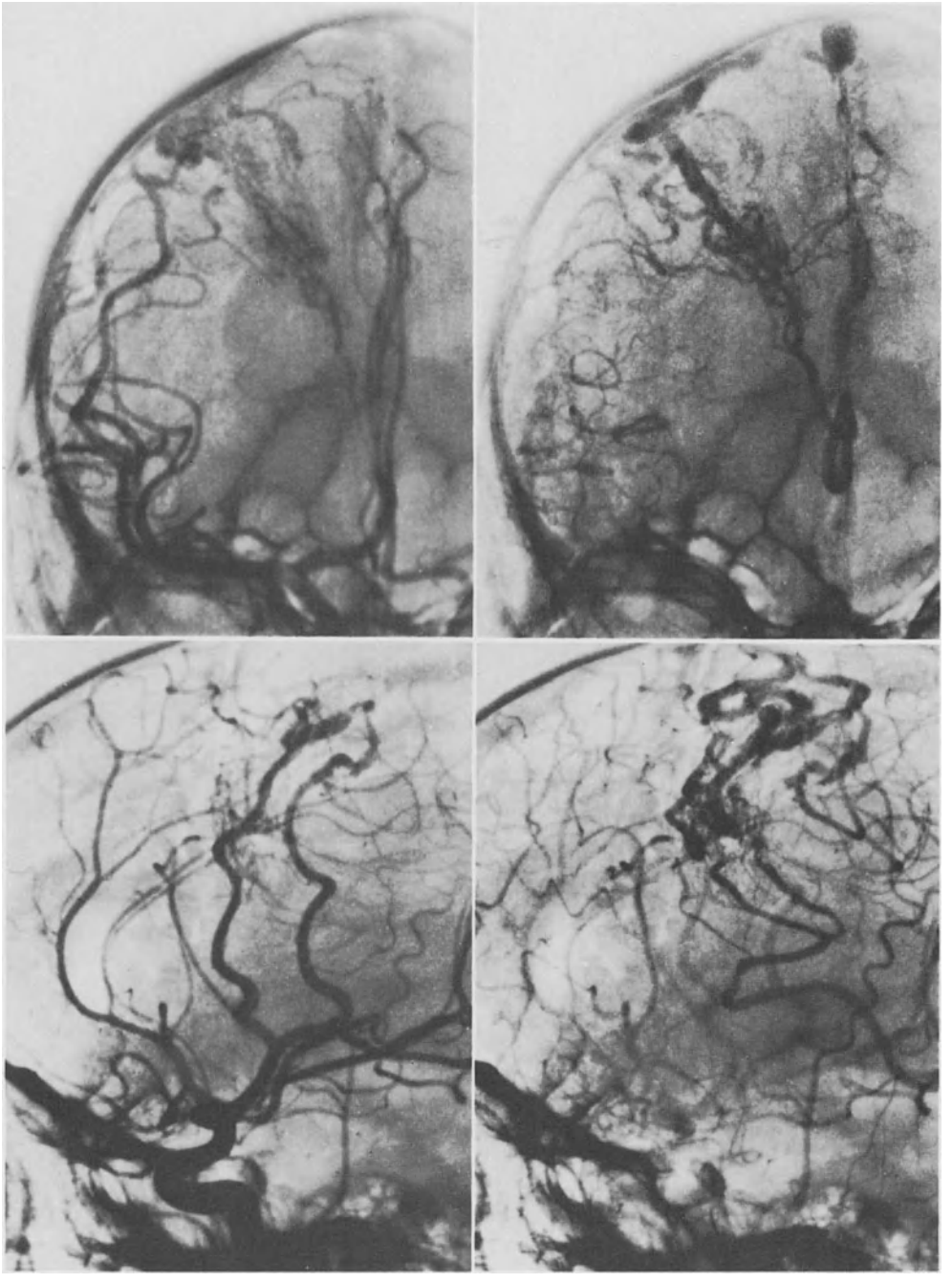

Fig. 1. Right frontoparietal arteriovenous malformation. Preoperative a.p. and lateral views

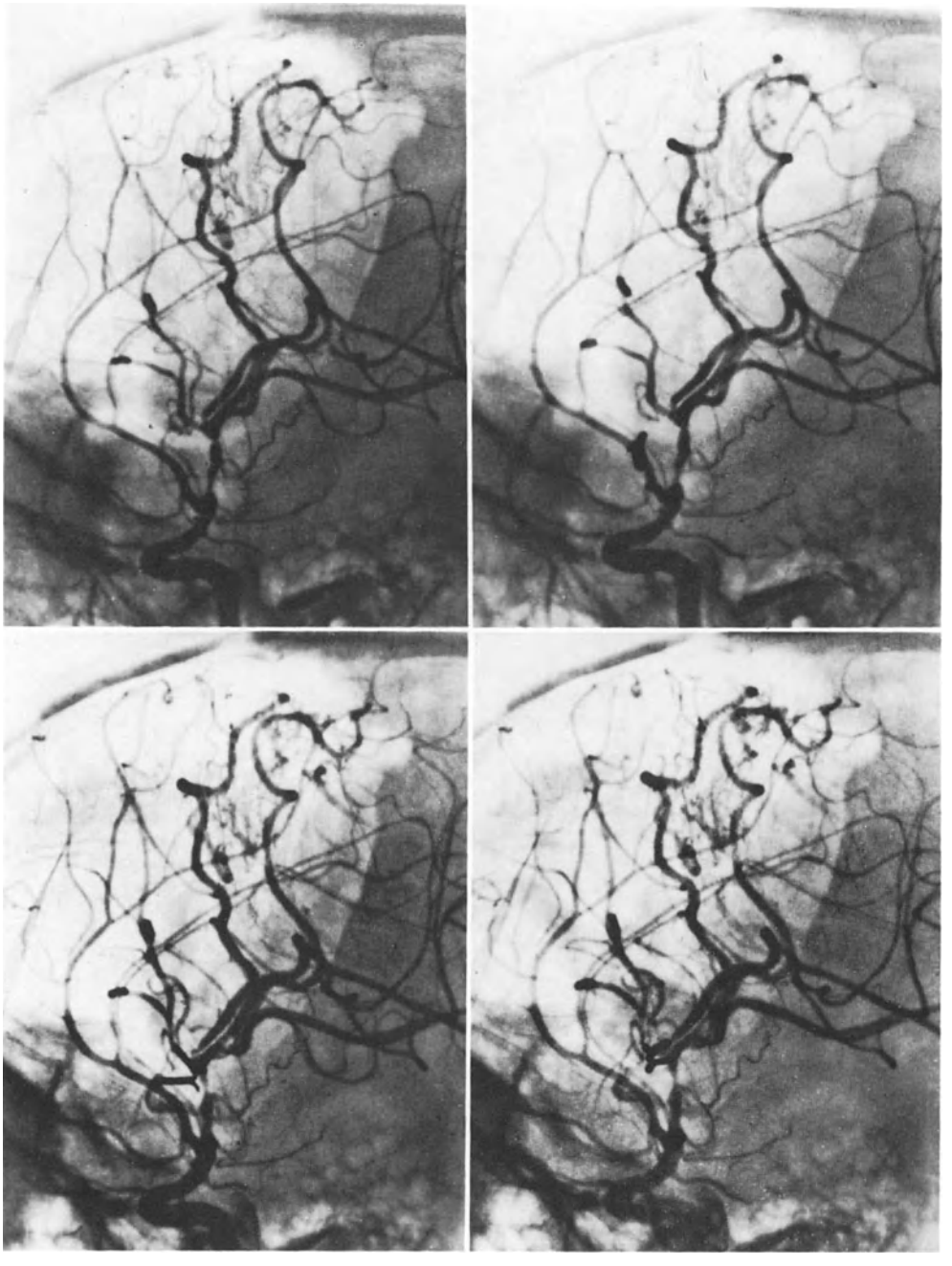

Fig. 2. Operative angiograms. *Upper left*: after removal of bone flap; *upper right* and *lower left*: clips on wrong artery; *lower right*: clip near presumable feeding artery

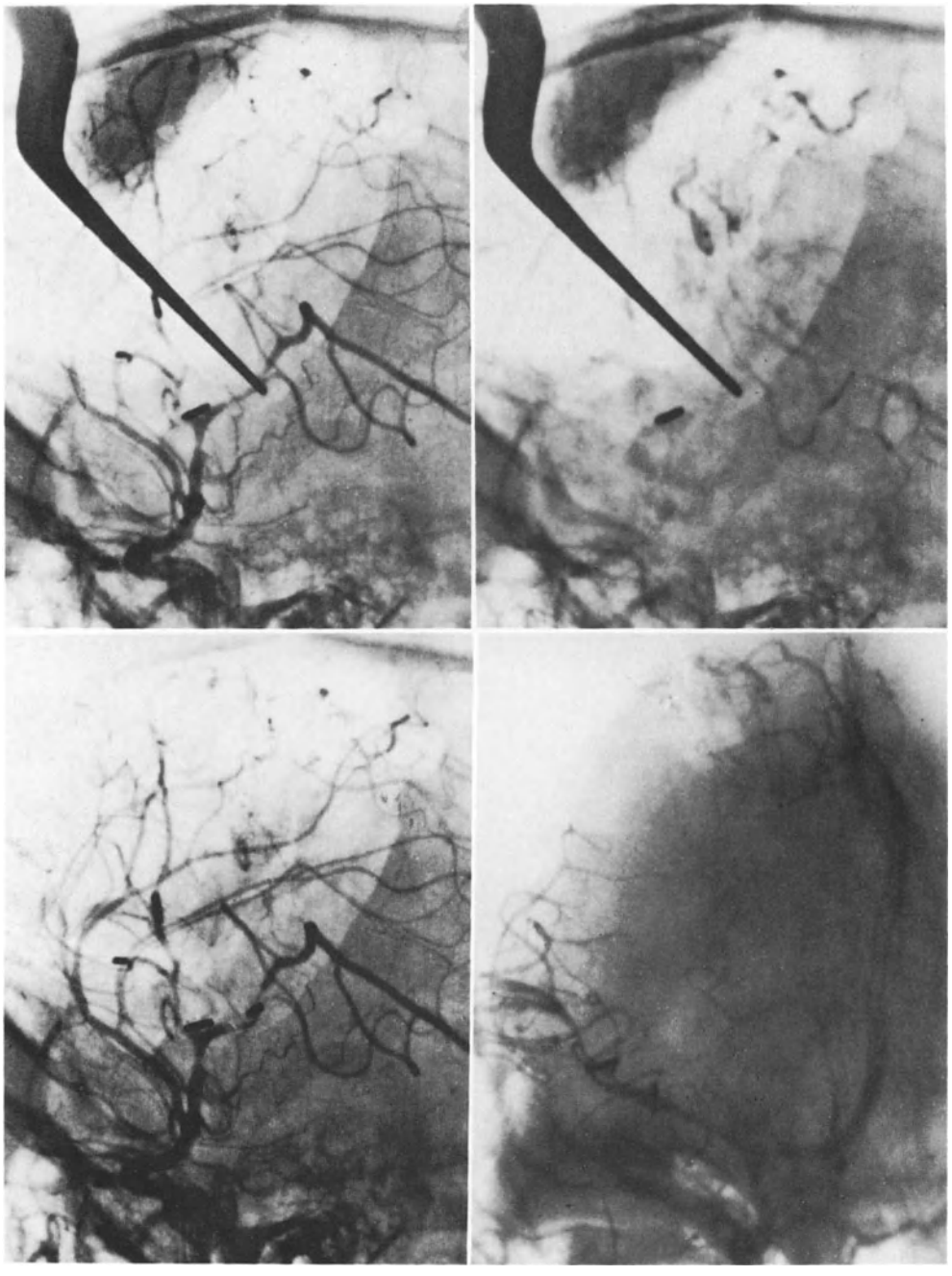

Fig. 3. Operative angiograms. *Upper left*: forceps on artery of Figure 2; *upper right*: forceps on feeding artery; *lower left*: clip on the same artery, no more filling; *lower right*: wrong artery

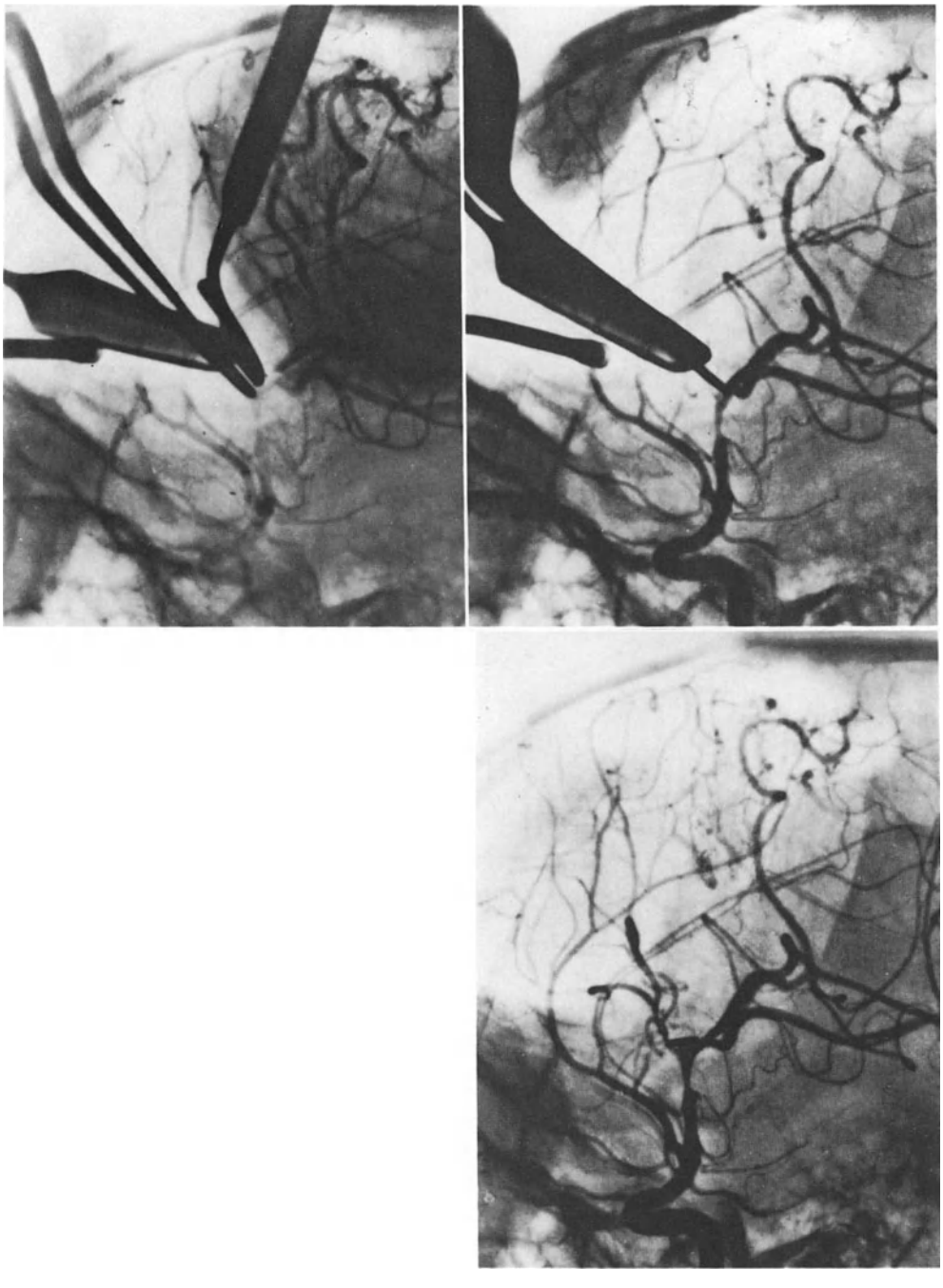

Fig. 4. Operative angiograms. *Upper left*: forceps on wrong artery; *upper right*: forceps on feeding artery; *lower right*: lateral displacement of anterior cerebral artery, caused by edema

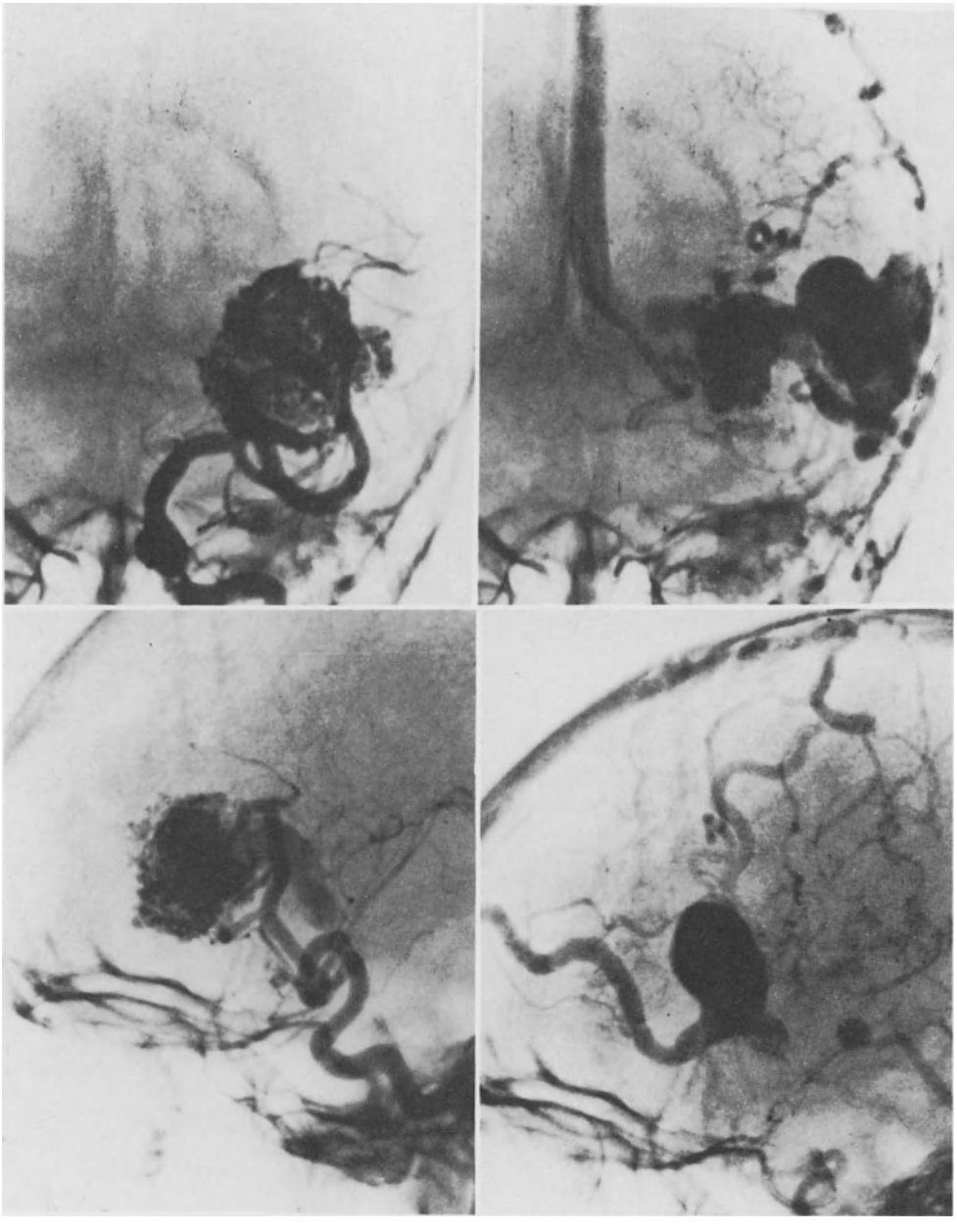

Fig. 5. A.v. malformation in the sylvian area. Preoperative a.p. and lateral views

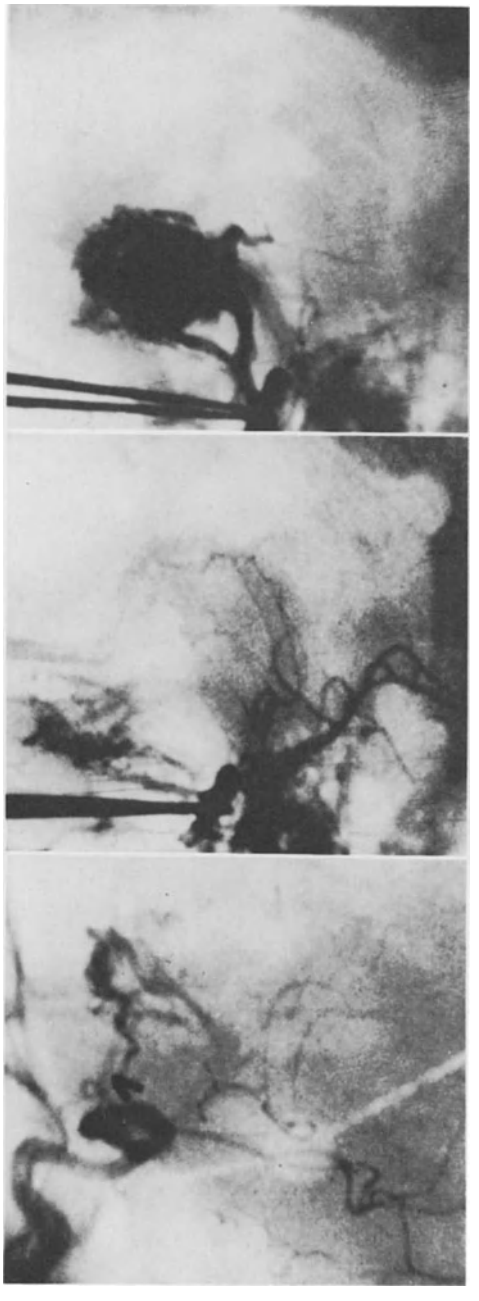

Fig. 6. Operative angiograms. *Top*: compression of wrong artery; *middle*: compression of main feeder; *bottom*: clip on same artery

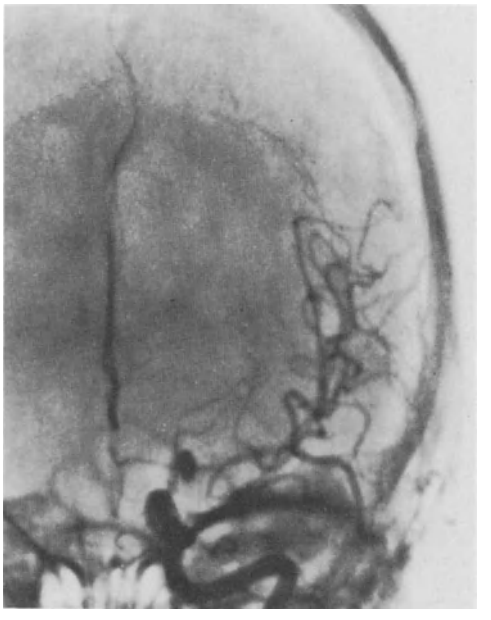

Fig. 7. Saccular aneurysm at bifurcation of left internal carotid; preoperative a.p. views

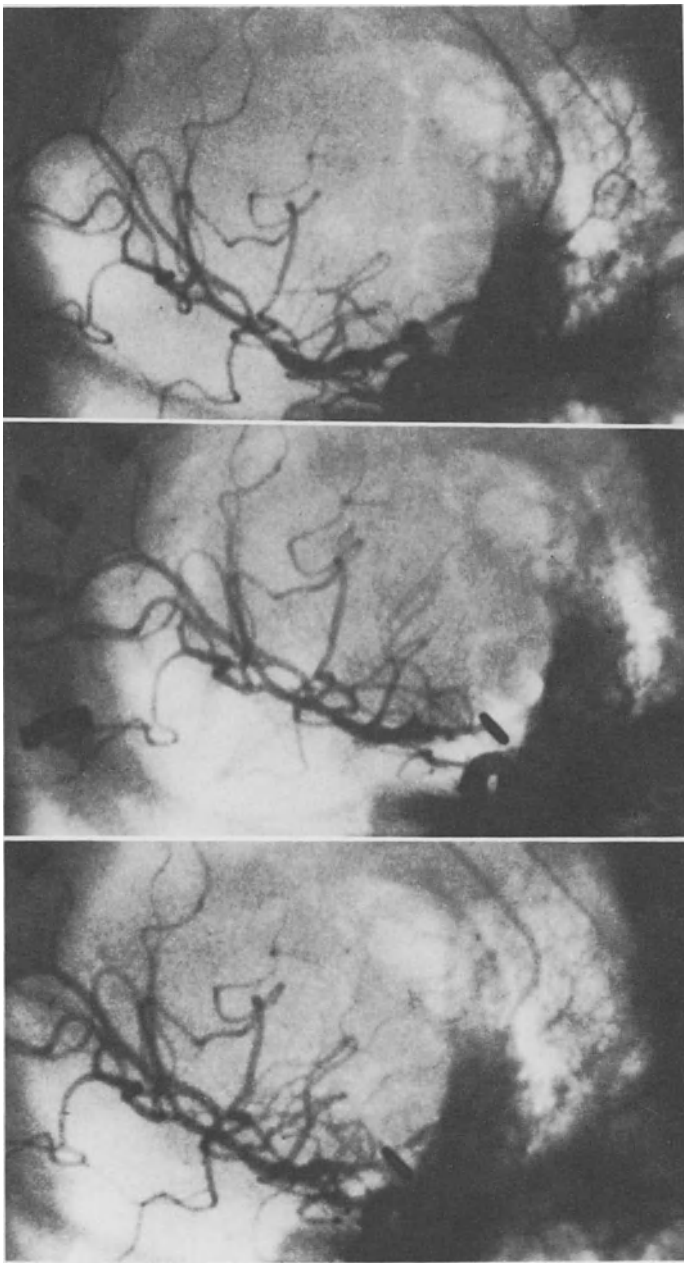

Fig. 8. Operative angiograms. *Top*: after removal of bone flap; *middle*: clip of aneurysm, no more filling of aneurysm, and no more filling of anterior cerebral artery; *bottom*: a little while later: good filling again, no more spasm in anterior cerebral artery

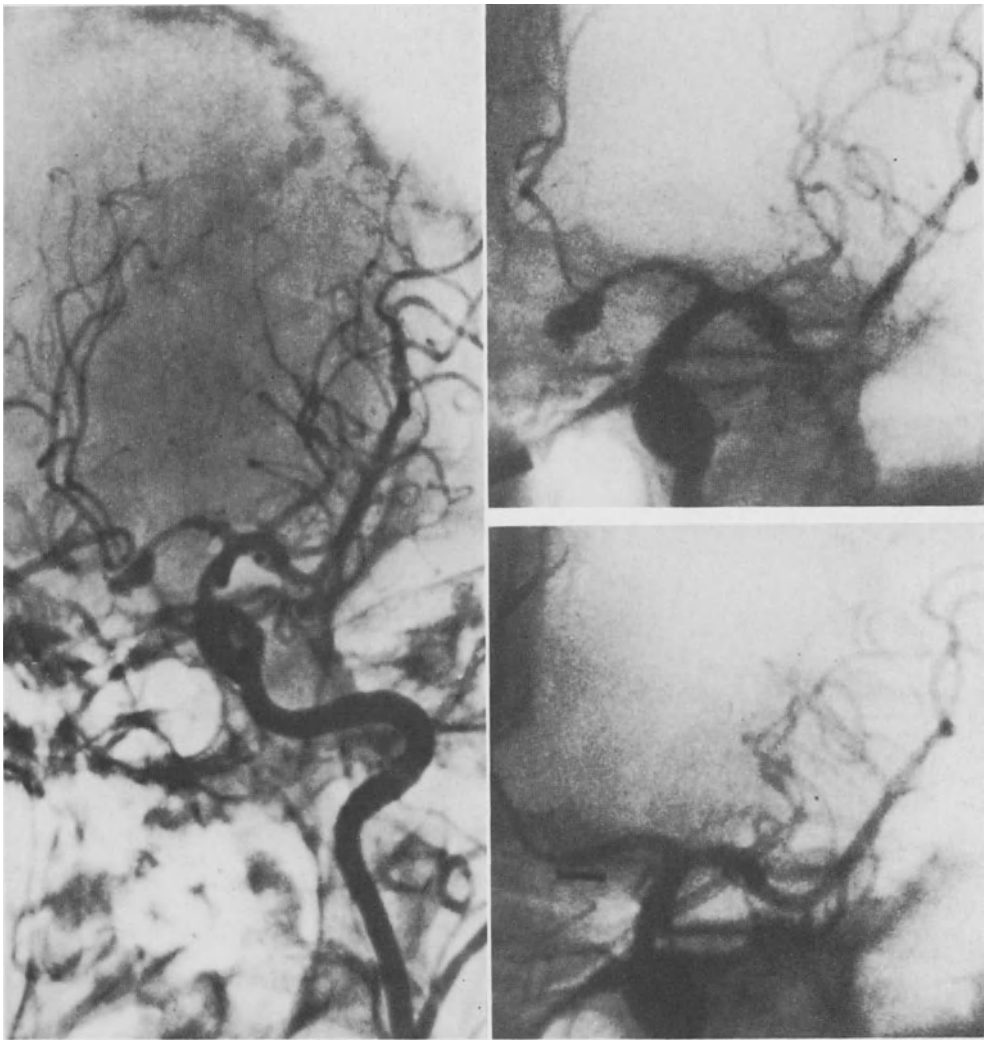

Fig. 9. Saccular aneurysm of anterior communicating artery. *Left*: pre-operative view; *upper right*: operative angiogram; aneurysm well visible; *lower right*: operative angiogram; clip on base of aneurysm, no more filling of aneurysm; anterior cerebral artery well visible

# Basilar Impression in Chondrodystrophy

W. LUYENDYK and R. THOMEER

The typical feature of an achondroplastic or chondrodystrophic dwarf are well-known. They were first described by PARROT in 1876, and extensively studied by my compatriot Murk JANSEN in 1912. Most of the particular features, due to inadequate enchondral bone formation, are demonstrated by skeletons kept at the Anatomical Laboratory in Leiden and originally studied by Murk JANSEN. They illustrate the relatively large neurocranium, the saddle nose and the short extremities, the arms usually reaching not farther than the tronchanters. In most instances the fingers are equally long, showing a trident hand.

However, another important feature is not demonstrated by these skeletons, namely the high intelligence of such individuals. Due to this and to their comic appearance, they were popular buffoons in former times.

In chondrodystrophic dwarves neurological disturbance can develop, mostly as a result of the congenital narrowness of the vertebral canal associated with a thoraco-lumbar kyphosis and osteophyte formation or one or more prolapsed discs. This may lead to radicular or even medullar ompression. Additionally, the anomalies of the cranio-cervical region may also be the cause of, or contribute to neurological disorders. However seldom, quadriparesis is described. This may be due to a craniocervical anomaly with a basilar impression of the so-called anterior type, which is rather frequently seen in chondrodystrophy. X-rays show an oblique upward position of the petrous bones, a strikingly short clivus of Blumenbach and a high position of the dens, its tip reaching above the line of Chamberlain. Moreover, the foramen magnum is usually narrowed. The following case demonstrates the improtance of logical disturbances in these cranio-cervial anomalies.

A 15-year-old girl with the characteristic appearance of chondrodystrophic dwarfism developed a steadily progressive paraparesis 9 months before admittance, which incapacitated her to the point that she needed a wheelchair 6 months later. From that time on, muscle power of her arms and fingers also decreased, obliging her to use an electric typewriter instead of the mechanical model she had used before. Disturbance of sensitivity and micturition also developed. She was admitted because of progressive tetraparesis. X-rays of the skull showed a typical anterior basilar impression. The vertebral canal appeared to be relatively narrow especially in the lumbar region. Electromyography of the legs showed bilateral signs of denervation, especially in the distal muscles. No EMG abnormalities were found in the paretic arms. However, these findings were not in keeping with the tetraparetic syndrome, and for this reason it was thought necessary to examine the occipito-cervical region neuroradiologically. Oxygen ventriculography, performed via an Ommaya system, revealed ventricular dilatation. No filling of the great cistern was achieved and there appeared to be only minimal space available dorsal to the spinal cord at the levels of C1 and C2. It seemed reasonable to consider this occipito-cervical obstruction to be the cause of the patient's quadriparesis, and surgical decompression was performed. The arches of C1 and C2 were removed and the narrowed foramen magnum was enlarged

dorsally. At this level, the dura appeared to constrict the craniospinal canal, having formed a transverse fold. This was carefully cut open, and the distal part of the posterior fossa was decompressed. The dural defect was covered with Lyodura. The postoperative course was uneventful. Ten months later the patient could walk considerably better, not needing a support. She could climb stairs and get into and out of a car without help. The muscle power of the arms and hands was completely restored. The legs were still slightly paretic, while EMG showed no divergence from the pre-operative findings. Sensitive disturbances had fully vanished and micturition had been restored. Although the signs and symptoms of this patient seem to be due to a combination of causes, it clearly appears that surgical enlargement of the foramen magnum and of the upper part of the cervical vertebral canal led to an interruption in the course of progressive quadriparesis and to a satisfactory recovery.

In order to evaluate the frequency and degree of basilar impression in chondrodystrophy we studied 22 typical cases of achondroplstic dwarfism so far. They were volunteers and did not suffer from severe neurological disorders. Four of them were younger than 10 years; these cases were excluded from comparison with normal adults. Although this study has not been concluded yet, some interesting data can be reported on the 18 older dwarves. First of all, the clivus of Blumenbach appears to be too short in all cases: its size is at least 8 mm below the normal adult average. The size of the dens, however, appears to be completely normal as compared to normal adults. The position of the tip of the dens is very important and was carefully determined in relation to the line of Chamberlain (extending from the posterior end of the hard palate to the posterior rim of the foramen magnum).
Normally, the tip of the dens is situated 1 mm below this line, variation being ± 2.3 mm. When these values are exceeded the diagnosis of basilar impression has to be considered. In our study it appeared that this was the case in all 18 chondrodystrophic dwarves. We compared the degree of basilar impression in our material to the data of SAUNDERS, who made a random study of the occipito-cervical configuration in a population of 200 white individuals. He found upward displacement (tip lying 5 mm or more above the Chamberlain line) in 5%. In the chondrodystrophic dwarves we studied, the incidence was tenfold. i.e. in 50%. An upward displacement of 2 mm or more existed in 20% of the cases of SAUNDERS, while this incidence was much higher in the group of chondrodystrophic dwarves we studied, amounting to 83%.

The purpose of this study was to emphasize the high incidence of basilar impression in chondrodystrophic draves. Every neurologist and neurosurgeon should consider basilar impression as a possible cause of or contributing to neurological disorders in chondrodystrophic patients.

# Incidence and Mortality of Abscesses of the Central Nervous System in England and Wales – Results of a Survey

P. GORTVAI, J. DE LOUVOIS, and R. HURLEY

## Introduction

Suppuration of the central nervous system (CNS) remains a relatively frequent condition in neurosurgical practice. In spite of surgical treatment combined with the administration of powerful antimicrobial agents, the mortality remains regrettably high. Table 1 records the mortality rates in some of the larger published series.

In this communication the frequency of the condition is described and mortality established for England and Wales rather than for one or a few neurosurgical centres.

## Material and Approach

A list of neurosurgical centres was obtained from the Society of British Neurological Surgeons. The 38 centres in England and Wales were asked to state the number of patients with intracranial abscess and with intraspinal abscess treated in 1973 with the mortality. Thirty-six replies were received. Figures from Scottland or Ireland were not used because their population data are recorded separately.

For yearly mortality the Registrar General's review (9) was consulted. The population figures for England and Wales are found in the Registrar General's tables (10). We examined copies of the death certificates of all persons for whom intracranial or intraspinal abscess was mentioned as an additional cause of death in 1973. Copies of the death certificates were provided by the Registrar General. We examined the internal records of two London neurosurgical centres for the number of admissions of patients with suppuration of the CNS between the years 1951-1973 inclusively.

Table 1. Mortality with abscesses of the central nervous system

| | No. of cases | % Mortality Overall | % Mortality Treated |
|---|---|---|---|
| PENNYBACKER (6) 1938-1949 | 110 | - | 36 |
| BELLER et al. (1) 1941-1971) | 89 | 45 | 40 |
| MORGAN et al. (5) 1946-1971 | 88 | 36 | 29 |
| TUTTON (8) 1948-1962 | 68 | 30 | 13 |
| GARFIELD (2) 1961-1967 | 200 | 40 | - |
| SAMSON et al. (7) 1961-1971 | 42 | 45 | 18 |
| KAO (3) 1964-1972 | 26 | - | 23 |
| DE LOUVOIS et al. (4) 1973-1976 | 46 | 24 | 20 |
| This survey 1973 England and Wales total | 200 | 36 | - |
| Neurosurgical centres | 157 | 18.5 | - |

## Results

The number of deaths primarily ascribed to intracranial and intraspinal abscesses (1CD No. 322) and to late effects of intracranial abscess or pyogenic infection (1DC No. 324) are shown in Table 2.

From examination of the death certificates, we found 20 additional cases where the main cause of death was intracranial suppuration. Adding this figure to the 52 reported by the Registrar General, we obtained 72 as the total number of deaths for that year.

The number of admissions necessitated by CNS suppuration to two London neurosurgical centres, and the sex distribution, is given in Table 3.

In 1973, according to our survey, 157 patients were treated for CNS suppuration in the neurosurgical centres for England and Wales - 148 with intracranial abscesses and 9 with spinal abscesses. There were in the same year 43 deaths caused by CNS abscesses outside the neurosurgical centres. Thus the total number of proven cases in 1973 was 200. Since the number of deaths was 72, the overall mortality became 36%. In the neurosurgical centres there were 29 deaths in 1973, a mortality rate of 18.5%.

Using a map of England and Wales which shows the Area Health Authorites as constituted in 1973, we divided the area and the population into two: the North of England and Midlands, and the South of England and Wales.

Table 2. Deaths caused by suppuration affecting the central nervous system in England and Wales in the Registrar General's Review (9) (1CD 322: Intracranial and intraspinal abscess; 1CD 324: Late effects of intracranial abscess or pyogenic infection)

| | 1CD 322 | 1CD 324 | Total |
|---|---|---|---|
| 1964 | 73 | 18 | 91 |
| 1965 | 83 | 18 | 101 |
| 1966 | 76 | 14 | 90 |
| 1967 | 67 | 18 | 85 |
| 1968 | 67 | 30 | 97 |
| 1969 | 65 | 15 | 80 |
| 1970 | 44 | 12 | 56 |
| 1971 | 66 | 14 | 80 |
| 1972 | 56 | 25 | 81 |
| 1973 | 52 | 24 | 76 |

Table 3. Incidence of intracranial and intraspinal abscess at two London neurosurgical centres, 1951-1973

| | Centre 1 | | | Centre 2 | | |
|---|---|---|---|---|---|---|
| | Male | Female | Total | Male | Female | Total |
| 1951-1955 | 20 | 8 | 28 | 39 | 16 | 55 |
| 1956-1960 | 17 | 2 | 19 | 19 | 11 | 30 |
| 1961-1965 | 20 | 8 | 28 | 15 | 9 | 24 |
| 1966-1970 | 26 | 7 | 33 | 11 | 6 | 17 |
| 1971-1973 | 10 | 2 | 12 | 9 | 2 | 11 |
| Total | 93 | 27 | 120 | 93 | 44 | 137 |

The serpiginous dividing line lies roughly at the latitude of Bedford. The position of Wales is somewhat ambiguous since the two Welsh Centres (Cardiff and Swansea) care largely for Southern Wales, and patients from the North of Wales are treated in the neighbouring Liverpool, which lies to the north.

The number of patients admitted to neurosurgical centres in the North of England and Midlands, and the South of England and Wales, the admission rate for each million of population, and the mortality are given in Table 3.

## Discussion

The mortality caused by CNS suppuration during the 10 years shown in Table 1 shows a tendency to fall. This tendency is less marked when the mortality caused by late effects if intracranial abscess or pyogenic infection is added. Indeed, in this respect, it might be said that mortality remains disappointingly high.

The number of admissions in two London neurosurgical centres over 23 years (Table 4) show that the problem has not been solved. We estimate that each of these centres serves a population of some 1.2 million. The differences between the centres indicate shifts in population and in emphasis of work rather than in incidence or referral rate of the condition.

The truly disappointing figure is the 36% mortality, calculated for England and Wales as a whole, for all cases of proven CNS suppuration in 1973. It is noteworthy that two-thirds of the deaths occurred outside neurosurgical centres presumably without reference to them.

Amongst the 157 patients treated in neurosurgical centres in 1973 the mortality was 18.5% - a more encouraging figure and lower than that seen in many published series. It is of the same order as the 20% treated mortality we reported in the study of cases in 1973-1976 from six co-operating English neurosurgical centres (4). The number of admission for each million of population is higher for the North of England and Midlands compared to the South of England and Wales (Table 4). Reasons for this are speculative - e.g., in the North personal incomes and living standards are somewhat lower than in the South. We would discount the difference in percentage mortality as it is based on only 16 deaths in the North and 13 deaths in the South.

Prevention of CNS suppuration, a goal which is already partially attainable, should be the ultimate aim, but cases still occur. Prompt

Table 4. Admissions to neurosurgical centres with abscesses of the central nervous system

| | North of England and Midland | South of England and Wales |
|---|---|---|
| Admissions/million of population | 4.3 | 3.5 |
| Number of admissions | 79 | 78 |
| Number of deaths | 16 | 13 |
| Mortality | 20.25% | 16.6% |

diagnosis and speedy referral of patients to neurosurgical centres should reduce the overall mortality.

The 18.5% mortality in neurosurgical centres is still high. We hope that the introduction of computerised axial tomography will help in diagnosis and management of intracranial suppuration. Further work is being undertaken to determine factors which contribute to the mortality.

## Conclusions

Deaths from intracranial and intraspinal abscesses in England and Wales for the decade 1964-1973 average 83.7 a year.

In 1973, the year chosen for the survey, there were 200 proven cases in England and Wales; 72 died, a mortality of 36%. Two-thirds of the deaths occurred outside neurosurgical centres. The mortality amongst patients admitted to neurosurgical centres was 18.5%. The admission rate of patients with abscesses of the CNS was 4.3/million population in the North of England and Midlands and 3.5/million population in the South of England and Wales.

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# Immunologic Aspects of Malignant Gliomas

R. E. H. VAN ACKER

## Introduction

Malignant gliomas are universally fatal, and severe physical and psychological damage are the most striking aspects of this destructive illness. Disappointment and frustration accompany the present modalities of treatment, and once the diagnosis of malignant glioma is made, surgical intervention, irradiation and cytotoxic drug therapy provide at best a mean survival of 51 weeks (7). However, some patients survive much longer than others (16). What factors may influence the outcome?

The postulated link between cancer and immunology is a line of approach which may shed a new light on this problem. Humoral and cellular immune factors can regulate growth and development of neoplasms. Spontaneous regression, exceptionally long intervals prior to the development of metastases, appearance of metastases following immune suppression, and regression of metastases after vaccination, are evidences of immunological resistance against tumor cells. The assumption is that the immune system in each of us is adequate to eliminate all incipient neoplastic lesions unless we develop "blocking factors." Thymus dependent and independent lymphocytes play a dominant role in cell-mediated immunity and the humoral immune response.

## Gliomas

Evidence is accumulating that immune factors may be important in the development and growth of tumors of the nervous system. Regression of neuroblastomas and melanomas have been recorded in man, but has never been documented in a case of human glioma. Unfortunately, certain immunological pecularities of the brain have tended to discourage investigations in this field. The absence of lymphatics in the brain gives it an "immunological privileged site" (9), and the blood-brain barrier makes it less likely that immunology will be able to contribute to the glioma problem as it does in the field of cancer in general. On the other hand, there are indications that the central nervous system has an autonomous immune system (13), independent of lymph glands and the thymus. Furthermore, in gliomas clinically detectable, vascular permeability in and around the tumor is increased, leading to a local break-down of these barriers.

Several investigations have been carried out in man to prove that malignant gliomas give rise to a host immune response. Up till now these experimental and clinical investigations provided a rather pessimistic picture. The first attempt to identify glioma-specific tumor antigens was made by SIRIS in 1936 (14), using extracts of glioblastoma tissue. However, 42 years later, no glioma-specific antigen has been found. Autogenous sera have been shown to inhibit and stimulate the growth of explanted gliomas. Immune complexes in serum, cerebrospinal fluid or cyst fluid in glioma patients have never been reported. These immune complexes are a common finding in other types of malignancies. Lymphocytotoxicity tests reveal no apparent specificity of lymphocytes of patients with glioma for the inhibition of tumor growth in vitro.

Lymphocytic infiltration in malignant gliomas does not follow the pattern which occurs in other types of cancer (1), and there is no correlation with a favorable prognosis as is the case with breast cancer (5). Patients with malignant gliomas have subnormal general immune competence, but these findings are very unspecific (7).

## Clinical Data

The largest series of postoperative follow-up of malignant gliomas (17) gives a 10% surival at 2 years. Our results were worse. One patient, however, survived much longer. Mrs. P., born on February 11, 1923, was admitted to our department in 1962. A right temporal cystic tumor was subtotally removed, followed by local radiotherapy of 6000 RAD. Histology then, and on review in 1978, confirmed an astrocystoma III-IV. A CT scan (January 1978) showed no evidence of recurrent tumor.

Several sensitive methods for direct detection of immunoglobulins and of antigen-antibody complexes have been devised in recent years. No systematic research has been done on the immunology of cystic gliomas. It has been suggested (11) that immunoglobulins can enter the cysts of malignant gliomas where they may play a role in tumor inhibition. However, the question is, when the appearance of immunoglobulins in cyst fluid is the result of an immune response or only a transsudation effect. We think the last factor is the most important. Analysis of cyst fluid in nine patients with astrocytoma revealed a direct correlation between these immunoglobulins, serum total protein and albumin, and cyst fluid (Table 1).

Another question is whether cyst fluid contains antigens that can activate lymphocytes. To answer this question the cyst fluid from two patients, one with an astrocytoma grade II and another with an astrocytoma grade IV, was assayed against the lymphocytes of these patients, and also against the lymphocytes of the first patient with a long survival time. It was impossible to show any activation effect in any of these lymphocytes. Considering these findings, it seems improbable that specific immunity plays a major role in the pathology of gliomas.

## Discussion

Much more research both in patients and in experimental animal models is necessary before meaningful therapeutic studies can be planned. Three groups of authors, TROUILLAS et al. (16), TAKAKURA et al. (15), and BLOOM et al. (4) have attempted to treat glioma patients by applying autogenous white blood cells into the tumor cavity following autosensitization. They failed to produce any convincing beneficial effect. The natural cytotoxicity of human lymphocytes in the brain is probably of minor importance, and still poorly understood. Evidence has been presented that intrathecally infused lymphocytes may escape into the systemic circulation (12). It appears that macrophages play a more important role in the immune system of the brain (13). These macrophages have traditionally been assumed to be derived from microglia cells. More recent investigations show they have a monocytic and pericapillar cell origin (2). There is much divergence concerning the possible local or hematogenous origin of microglia cells, but if there is an autodefence against glioma cells, it would be an attractive idea to stimulate immunological response locally. Experimental studies suggest that simultaneous presentation of an unspecific immunostimulant may be the most efficient way to stimulate an immune response against the tumor (3). Studies on the local administration of B.C.G. as an immuno-

Table 1. Albumin, total protein, Ig G and Ig A concentrations in the fluid of tumor cyst and in the plasma of nine patients with cystic astrocytomas

| | mgr.% Albumin | | gr/l Total protein | | mgr.% Ig G | | mgr.% Ig A | |
|---|---|---|---|---|---|---|---|---|
| | Plasma | Cyst | Plasma | Cyst | Plasma | Cyst | Plasma | Cyst |
| Astrocytoma IV | 3660 | 2884 | 68 | 52 | 1336 | 900 | 400 | 300 |
| Astrocytoma III-IV | 3593 | 3488 | 53 | 51 | 878 | 776 | 282 | 163 |
| Astrocytoma III | 3526 | 4069 | 64 | 50 | 917 | 1069 | 187 | 241 |
| Astrocytoma III | 4078 | 2442 | 61 | 41 | 692 | 326 | 200 | 59 |
| Astrocytoma II | 3607 | 3069 | 56 | 40 | 659 | 491 | 110 | 66 |
| Astrocytoma II | 4204 | 1973 | 54 | 41 | 992 | 306 | 205 | 24 |
| Cerebellar Astrocytoma II | 4144 | 3120 | 51 | 49 | 1161 | 708 | 107 | 53 |
| Cerebellar Astrocytoma II | 3806 | 2852 | 64 | 42 | 758 | 491 | 77 | 32 |
| Cerebellar Astrocytoma II | 4300 | 2950 | 68 | 48 | 1450 | 860 | 54 | 30 |

stimulant confirmed the safety of this method. There have been reports of a reduction in the incidence of recurrences and of a prolongation of the disease-free survival in patients with lung cancer (8) and with bladder cancer (10).

## Conclusion

Far from having reached the end of all therapeutic possibilities, immunotherapy is slowly advancing. There is evidence that local immunotherapy improves in cases of extraneural cancer. We believe that in cases of gliomas nonspecific stimulation of macrophages is a more favorable method of treatment because of the priviledged site of the brain from the immunological point of view. Thus, there might be hope for a better prognosis of gliomas in the future.

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# Corticosteroids Stimulate Cerebral Energy Metabolism; Support for a Metabolic Action of Steroids on Brain Edema[1]

A. BAETHMANN, W. ÖTTINGER, B. FLEISCHER, K. MORITAKE, and F. JESCH

## Introduction

Most methods and drugs generally employed today in the treatment of brain edema, such as dehydration by hypertonic solutions, or saluretics, hyperventilation, or surgical methods, do not specifically affect the edema proper, but rather one of its major consequences, the rise in intracranial pressure. An ideal method, acting directly and specifically on brain edema, should be expected a) to normalize the function of the disrupted blood-brain-barrier so as to prevent further influx of edema fluid into the parenchyma, b) to attenuate or prevent potential toxic effects of the edema fluid, which creates an abnormal environment for the cerebral cells, c) to prevent or abolish cell swelling, and, finally, d) to enhance the clearance of edema fluid from the cerebral tissue. To our knowledge, a method fulfilling all these requirements is not available at the moment. It remains to be seen, however, whether corticosteroids or their powerful synthetic derivatives, e.g., dexamethasone, meet such expectations. Experimental data suggest that the central nervous system must be considered a target organ for biological steroid actions. Some observations which might be relevant in steroid mechanisms on brain edema are contained in Table 1. For example, the blood-brain barrier seems to be protected by dexamethasone in traumatic brain tissue damage causing edema (8, 13, 15); formation of cerebrospinal fluid is inhibited not only by dexamethasone, but even more effectively by spirolactone (6, 14, 16). Metabolic studies of several laboratories including ours, revealed that corticosteroids not only induce several enzymes in brain tissue, but also stimulate cerebral energy metabolism (2, 3a, 4, 7, 10, 17, 18).

This point could be very important with respect to the cytotoxic, i.e., intracellular component of brain edema, likely to result from disturbances of active, energy dependent transport mechanisms across cell membranes in one way or the other. In order to advance the understanding of this particular aspect of steroid action on brain edema, cerebral blood flow (CBF) and metabolism were studied in adrenalectomized dogs withdrawn from steroid substitution prior to and after acute administration of either aldosterone, or dexamethasone, respectively.

## Materal and Methods

Adult male mongrel dogs (8-12 kg body weight) were used for bilateral adrenalectomy, performed 2-3 weeks prior to the final experiment. Approximately three days after steroid substitution had been discontinued, CBF was measured under pentobarbital anesthesia in control conditions and in hypercapnia, in order to study $CO_2$-reactivity. In adrenalectomized animals the barbiturate dose necessary to induce anesthesia was found to be approximately only 50% of that necessary in intact animals (i.e., 12 mg/kg, 5). CBF was determined by extracranial

1 Supported by DFG-Sonderforschungsbereich 51: Medizinische Molekularbiologie und Biochemie, München.

Table 1. Some effects of steroids or of steroid deprivation by adrenalectomy on nervous tissue (From BAETHMANN, 1978)

| | | | |
|---|---|---|---|
| ▲ Blood-brain-barrier | ROVIT, 1968<br>PAPPIUS, 1969<br>EISENBERG, 1970 | ▲ Electrophysiology | DAVENPORT, 1949<br>FELDMAN, 1970 |
| ▲ CSF-formation | DAVSON, 1972<br>SATO, 1973<br>POLLAY, 1975 | ▲ Sensitivity to barbiturates | KOBAYASHI, 1976<br>BAETHMANN, 1978 |
| ▲ Specific binding (Receptors?) | DE VELLIS, 1965<br>MC EWEN, 1969<br>ANDERSON, 1976 | ▲ Tissue metabolism | HOAGLAND, 1953<br>WOODBURY, 1972<br>BAETHMANN, 1977 |
| ▲ Induction of enzymes | DE VELLIS, 1965<br>BAETHMANN, 1968<br>STASTNY, 1972 | | |

recording of $^{133}$Xenon-clearance for 10 min (temporal muscle removed at the site of measurement). $CBF_{10}$ (height over area) and the initial slope index were calculated in parallel, yielding almost identical figures. The completeness of adrenal removal was ascertained by the characteristic changes in plasma electrolytes. Two additional CBF-measurements in normocapnia followed 30 min and 3 h after injection of 0.2 mg/kg of either aldosterone (Aldocorten, Ciba-Geigy AG, Wehr, Baden) or dexamethasone (Decadron-Phosphat, Sharp und Dohme GmbH, München) into the common carotid artery. A schematic experimental protocol is given in Figure 1. The cerebral uptake of oxygen ($CMRO_2$, Lex $O_2$Con) and glucose ($CMR_{gl}$, enzymatically) were calculated from the respective concentrations in arterial and cerebral venous blood (sinus sagittalis) and from cerebral blood flow.

## Results

Adrenalectomized dogs withdrawn from steroid substitution had a marked reduction in CBF and glucose consumption (3), while the cerebral oxygen uptake initially remained almost at the control level (Table 2). The response of CBF to changes in $CO_2$ was still present in adrenalectomized dogs, but markedly attenuated as concluded from regression analyses. Autoregulation of CBF, determined by infusion of angiotensin, was found to be maintained. Hence, the reduction in blood flow seems to be independent of the fall in arterial blood pressure (to approximately 70-90 mmHg) regularly encountered in adrenalectomized animals. Aldosterone, or dexamethasone, respectively, were slowly injected into the common carotid artery via the same catheter used for administration of $^{133}$Xenon. CBF determination in normocapnia 30 min after injection did not disclose significant changes as compared to control values. However, 3 h after injection of either compound both CBF and cerebral oxygen uptake were markedly higher than in adrenalectomized animals without steroids after an equivalent period of time (Table 3). On the other hand, as opposed to the initial control run, no differences in cerebral glucose consumption were detected between the adrenalectomized controls and the animals receiving corticosteroids.

## Summary and Conclusions

Deprivation of corticosteroids induces an intriguing pattern of metabolic abnormalities in the brain. CBF is reduced for more than 20% from control levels, parallel to a reduction in cerebral glucose uptake. Cerebral oxygen consumption, which initially is almost normal in adrenalectomized animals without steroids, falls in the course of the

Table 2. Cerebral blood flow, and cerebral oxygen ($CMRO_2$) and glucose ($CMR_{gl}$) consumption in intact and adrenalectomized normocapnic dogs (First measurement, see Fig. 1)

| | CBF (ml/100 g min) | $CMRO_2$ (ml/100 g min) | $CMR_{gl}$ (mg/100 g min) |
|---|---|---|---|
| Controls | 38.2 ± 1.5 (23) | 3.40 ± 0.25 (21) | 6.54 ± 0.77 (22) |
| p | < 0.005 | n.s. | < 0.05 |
| Adx | 29.9 ± 1.9 (17) | 3.26 ± 0.29 (15) | 4.62 ± 0.43 (14) |

Table 3. Cerebral blood flow, and cerebral oxygen and glucose- consumption determined in normocapnia 3 h after injection of steroids. In adrenalectomized animals without steroid injection (Adx only) an equivalent period elapsed before measurement

| | CBF (ml/100 g min) | $CMRO_2$ (ml/100 g min) | $CMR_{gl}$ (ml/100 g min) |
|---|---|---|---|
| Adx + Aldosterone (0.2 mg/kg) | 34.0 ± 3.7 (8) | 3.11 ± 0.17 (7) | 5.16 ± 1.37 (6) |
| p | < 0.05 | < 0.005 | n.s. |
| Adx only | 25.6 ± 1.3 (12) | 2.19 ± 0.20 (9) | 4.30 ± 0.41 (9) |
| p | < 0.005 | < 0.01 | n.s. |
| Adx + Dexamethasone (0.2 mg/kg) | 34.4 ± 2.0 (7) | 3.33 ± 0.32 (6) | 4.17 ± 0.56 (6) |

experiment. This may result form additional disturbances of as yet unknown nature imposed by the experiment (e.g. duration of anethesia). Intact control animals maintain CBF and oxygen consumption throughout the experimental period. Aldosterone and dexamethasone were found to stabilize, or even improve, CBF and oxygen consumption as compared to untreated adrenalectomized controls. Since both compounds have very different mineralo- or glucotropic properties, but still influence cerebral energy metabolism quite similarly, it must be concluded that specific gluco- or mineralotropic actions of both steroids were of no importance in this particular condition. The data obtained in this study support the contention that one action of steroids on brain edema is the stimulation of metabolic processes in brain edema. This could influence beneficially not only the cytotoxic, i.e., intracellular component of brain edema, but also support active mechanisms of clearance of the extracellular compartment from the vasogenic extracellular edema fluid.

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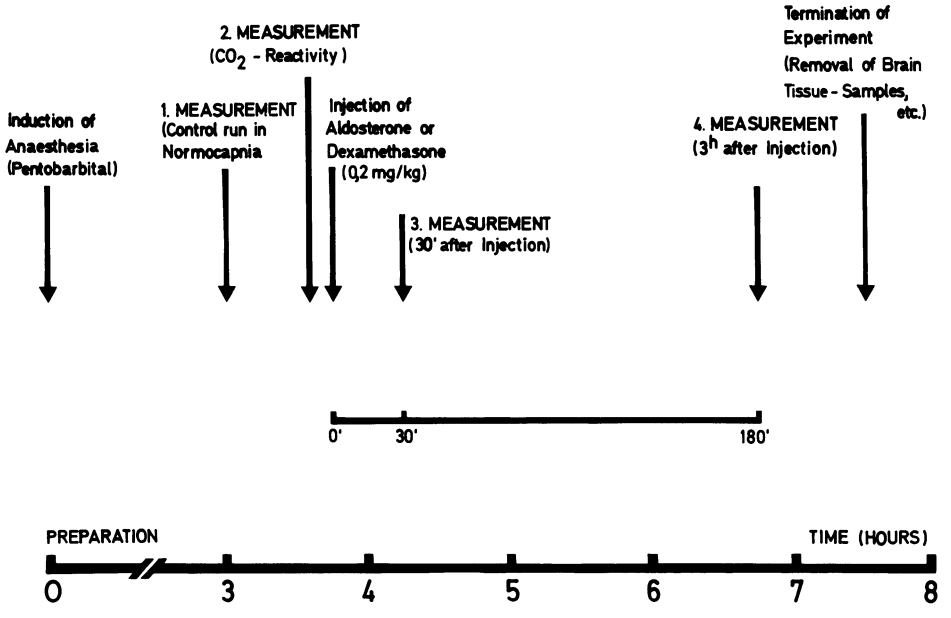


Fig. 1. Schematic diagram of the experimental protocol. Following induction of anesthesia, the animal was prepared for measurement of CBF and metabolism. About 2-3 h later the first control measurement was made, followed by testing of the $CO_2$-response. Thereafter, either aldosterone or dexamethasone were injected, followed 30 min later, by an additional CBF measurement and a final measurement 180 min after injection, both under normocapnic conditions. Subsequently, a craniotomy was performed in order to obtain tissue samples under freeze-stop conditions or for electron microscopy. The brain was then removed in order to measure the water, sodium and potassium content in cortex and white matter as well as in the nucleus caudatus

# Critical Intracranial Effects of Osmotherapy

M. GAAB, K. W. PFLUGHAUPT, M. RATZKA, R. WODARZ, and P. GRUSS

## Introduction

Shortly following the discovery, by WEED and McKIBBEN in 1919 (27), that intracranial pressure was reduced by intravenous hyperosmolar infusions, this treatment gained clinical application. BÜRGER (5) termed this procedure "*osmotherapy*." Nowadays mannitol and sorbitol, introduced by WISE and CHATER in 1961 (28), are regarded as being the most effective conservative measure for reducing intracranial pressure (ICP) (2, 12, 25). Even in the absence of a verified ICP elevation, treatment with regular doses of 1 to 2 g/kg body weight is part of the basic therapy of acute intracranial disorders with edema or ischemia in many hospitals (19). At the last intensive care meeting of German neurosurgeons (in Giessen, 1977), more than half of the clinics present reported its regular use in severe head injuries.

Whereas the *systemic side effects* of osmotherapy on the circulation and kidneys are known (2, 4, 14, 21), *direct negative actions* on the *intracranial space*, as revealed by our measurements, are hardly ever described (15, 19). The value of regular osmotherapy has been studied experimentally and clinically with the aid of the recording of several parameters.

## Material and Approach

### *1. Experimental Studies*

#### *a) Polygraphic Recordings in Cats With Experimental Brain Edema*

In artificially ventilated cats severe cold injury brain edema was produced through a right parietal trepanation (for methods, 9, 10, 16). Epidural pressure (EP) was recorded biparietally and over the posterior cranial fossa. Additionally, ventricular fluid pressure (VP), systemic arterial (aP), and central venous pressure were recorded. The EEG over both hemispheres (bipolar fronto-occipital leads) was evaluated continuously by Fourier analysis (FFT) with the aid of a computer (Plurimat S).

Elevated ICP values (VP at least 35 mmHg, 1-2 h after injection) were reached, and treated with 30 ml/30 min 40% sorbitol or Mannisorb (= 15% mannitol + 30% sorbitol) intravenously.

#### *b) Tissue Edema Parameters in Rats With Experimental Head Injury*

A standardized cold trauma was produced stereotactically (without opening the cranium) in the right hemisphere of rats (8). The $H_2O$, $Na^+$ and $K^+$ contents of both hemispheres was determined separately 24 h after injury ($H_2O$ by loss of weight, $Na^+$ and $K^+$ after dry ashing (8)). The rats were treated with Mannisorb, Glycerol and Dexamethasone (for dosages and routes of application see Table 2). Tissue edema parameters were compared statistically with the values of untreated control animals.

## 2. Clinical Studies

*Extradural ICP* was continuously measured in neurosurgical patients with miniaturized implanted pressure transducers (11), and correlated to the clinical course. In most patients, the EEG was recorded at intervals before, during, and after osmotherapy. In some patients the EEG was analysed by a computer as specified above (off-line). In a few patients a CT scan was obtained before, as well as 30 min to 2 h and 12-24 h after infusion was performed. Blood osmolality, $Na^+$, $K^+$, glucose, and hemoglobin and hematocrit were measured at regular intervals during osmotherapy. The daily fluid balance was calculated. Intravenous infusions of Mannisorb (see above), in a dosage of 1 to 2 g/kg body weight were made within 20-30 min i.v. (Infusomat). Only the results in patients with *head injuries* are reported here.

# Results

## 1. Experimental Studies

### a) Polygraphic Recordings in Cats

All intracranial pressures rise progressively following the trauma. The right epidural pressure (over the lesion) exceeds the left epidural and the ventricular pressure, indicating the occurrence of considerable intracranial *pressure gradients* (8, 10, 16). EEG power decreases (especially on the right), after an initial post-traumatic activation, corresponding to the rise in ICP (9, 10, 16). Osmotherapy rapidly lowers ICP values (9, 10). Nevertheless, the EEG recovers only for a short time, then rapidly becoming isoelectric. Thereafter circulatory regulation fails (10). This was a constant result (9, 10) in 13 animals. Despite normalization of cerebral perfusion pressure by reduction of the ICP, *survival time is only slightly increased* by osmotherapy, in contrast to what happens with other treatment methods (Table 1).

### b) Tissue Edema Parameters in Rats

Various doses of Mannisorb *do not lead to any improvement* of edema as compared to untreated controls. $H_2O$ and $Na^+$ content of the hemispheres

Table 1. Survival times after 4 min cold brain injury in cats. Despite normalization of ICP by osmotherapy, this treatment prolongs the cat's life only slightly. Note the more marked effects of Tris-buffer therapy and of operative edema resection

| Treatment | n | $\bar{}_a$ (min) | $s_a$ (min) | Signif. ther. group against controls |
|---|---|---|---|---|
| Controls (without therapy) | 15 | 160 | 57 | - |
| Osmotherapy (40%, 8 ml/kg b.w.) | 13 | 205 | 33 | $p < 0.05 > 0.02$ |
| Tris-buffer-therapy (Trometamol 5 mmol/kg) | 5 | 234 | 49 | $p < 0.01$ |
| Operative therapy (Resection of edematous brain tissue) | 5 | 354[a] | (33)[a] | $p < 0.0005$ |

[a] 4 of the 5 animals were still alive and spontaneously respirating 6 h after the injury (end of experiment with KCl i.v.).

remain elevated (Table 2). The only osmotherapeutic agent which shows a significant effect is glycerol (8).

## 2. Clinical Studies

*ICP can be raised acutely and substantially* by osmotherapy (Fig. 1). In this patient, with a severe head injury, CT showed an edema of the right hemisphere and a small left temporobasal hemorrhage. The rise in ICP during the first administration of Mannisorb was appreciable and led to *severe clinical consequences*: dilatation of the right pupil and extensor spasms (brain stem compression). This "*paradocixal effect*" (= increase of ICP by osmotherapy) was observed in 23% of the infusions (Table 3). Its duration exceeded the time of infusion and it was accompanied by a deterioration of the clinical condition or of the EEG. Moreover, the appearance of a *rebound* was striking in 12% (Table 3). Here, a "paradoxical effect" occurs mainly in the early post-traumatic phase. Finally, a more marked lowering of the ICP may occur without rise after infusion.

However, a "paradoxical effect" may still occur days after the injury (Fig. 2). In this form of late traumatic edema, the rise in ICP is accompanied by a marked deterioration of the EEG. In this case, we were only able to lower the pressure eventually by a decompressive trepanation.

Craniocerebral injuries may have a *special course in children*. In two cases we have seen more frequent plateau waves (Fig. 3). The number and height of pressure waves *increase* during and after osmotherapy, the initial pressure and the mean pressure also being substantially raised (Table 4).

## Discussion

The quesion arises as to why we have observed a *paradoxical rise of ICP in osmotherapy* in contrast to others (12, 13). Similar observations have been made only by MARSHALL et al. (19), who found a marked elevation in ICP induced by mannitol three times in eight patients. A possible explanation might be the "negative choice" of severe head injuries (CT finding of edema and/or intracranial bleeding) in our patients, application of a relatively high dose of osmotherapy only during critical pressure phases (19), and in particular, the very early beginning of ICP monitoring, often less than 2 h after the trauma. During the early post-traumatic stages, the rise in ICP is apparently due to an *intracranial congestion* by vasoparalysis (7, 8, 18) rather than to brain edema. The substantial increase in circulating volume and cardiac output by osmotherapy (4, 22, 23) may lead to a further increase in intracranial blood volume ("*false autoregulation*" (7)), and, thus, also to a rise in ICP. Also, edema drainage itself may be impaired by a BBB disturbance (3, 6, 20). When the lesion is extensive, besides a "false autoregulation" there may also be a *reversal of the osmotic gradient* after the infusion (mannitol and sorbitol remain within the barrier-disturbed edema tissue) which explains the "rebound" in 12% of the cases.

A further cause of unfavorable effect of osmotherapy is the *pathological blood osmolality* in cases of severe head injury (Fig. 4). In contrast to controls (Fig. 4a), initial osmolality in cases of severe trauma (Fig. 4b) is already above the "critical osmolality" (dotted line) at which the blood-brain-barrier becomes permeable to mannitol (24, 26).

Table 2a and b. Tissue edema parameters after cold brain injury in rats. Mannisorb at various doses does not lead to any improvement of the $H_2O$, $Na^+$ and $K^+$ content. The only osmotherapeutic agent which lowers water and sodium content significantly is glycerol

*a) 3-min cold brain injury edema in rats*
Tissue edema parameters ($\bar{x} \pm s_x$) sep. for both hemisphres 24 h after injury.
*Therapy*: Mannisorb, glycerol 0.5 ml 15 min after inj., then 4.5 ml within 5 h (0.9 ml/h).
(i.v.) Dexamethasone 0.5 mg 15 min bef. (= 1) or after inj. (= 2), then 0.5 mg 8 and 16 h after inj.

| Groups | n | $H_2O$ (% of weight) | | $Na^a$ (mval/kg dry w.) | | $K^a$ (mval/kg dry w.) | |
|---|---|---|---|---|---|---|---|
| | | Right | Left | Right | Left | Right | Left |
| Controls | 10 | 77.15 ± 0.92 | 77.05 ± 1.00 | 204 ± 22 | 204 ± 30 | 417 ± 58 | 427 ± 58 |
| Untreated edema[a] | 14 | 80.13 ± 2.28 | 78.14 ± 1.16 | 336 ± 143 | 230 ± 42 | 424 ± 56 | 447 ± 45 |
| Mannisorb | 14 | 80.15 ± 1.18 | 78.10 ± 1.02 | 312 ± 114 | 225 ± 71 | 391 ± 58 | 420 ± 54 |
| Dexamethasone 1 | 11 | 79.65 ± 1.34 | 77.89 ± 0.72 | 324 ± 78 | 227 ± 33 | 370 ± 56 | 404 ± 33 |
| Dexamethasone 2 | 14 | 79.90 ± 2.02 | 78.11 ± 0.80 | 330 ± 118 | 225 ± 136 | 420 ± 62 | 452 ± 36 |
| Glycerol | 16 | 79.69 ± 1.86 | 77.82 ± 0.98 | 286 ± 79 | 206 ± 24 | 370 ± 32 | 398 ± 30 |

The values showing a *statistically significant effect of therapy* (comp. with untr. edema) are *underlined* ($p < 0.05$).

[a] Untreated edema: Infusion of 5 ml of a 2.5% glucose + 0.45% NcCl-sol. for fluid balance (within 5 h after inj.)

*b) 4-min cold brain injury edema in rats*
Tissue edema parameters ($\bar{x} \pm s_x$) sep. for both hemispheres 24 h after injury.
*Therapy*: Mannisorb: 3 doses of 1.5 ml each 1 h, 9 h and 17 h after inj. i.v. within 20 min.
(i.v.) Glycerol: 0.5 ml immed. after inj., then 4.5 ml within 5 h (0.9 ml/h).
Dexamethasone: 0.4 mg 15 min after inj., 0.2 mg 6 h after inj.

| Groups | n | $H_2O$ (% of weight) | | $Na^a$ (mval/kg dry w.) | | $K^a$ (mval/kg dry w.) | |
|---|---|---|---|---|---|---|---|
| | | Right | Left | Right | Left | Right | Left |
| Controls | 16 | 76.66 ± 0.89 | 76.52 ± 0.87 | 181 ± 19 | 183 ± 15 | 395 ± 16 | 397 ± 24 |
| Untreated edema[a] | 14 | 80.39 ± 1.17 | 78.25 ± 1.36 | 311 ± 51 | 219 ± 37 | 357 ± 44 | 395 ± 31 |
| Mannisorb | 13 | 79.99 ± 1.44 | 78.09 ± 1.00 | 305 ± 61 | 220 ± 35 | 361 ± 27 | 393 ± 26 |
| Dexamethasone | 13 | 80.20 ± 1.38 | 77.88 ± 0.84 | 313 ± 72 | 219 ± 30 | 360 ± 40 | 407 ± 44 |
| Glycerol | 17 | 79.53 ± 2.19 | 77.62 ± 1.62 | 285 ± 75 | 205 ± 36 | 361 ± 45 | 390 ± 60 |

The values showing a *statistically significant effect of therapy* (comp. with untr. edema) are *underlined* ($p < 0.05$).

[a] Untreated edema: Infusion of 5 ml of a 2.5% glucose + 0.45% NaCl-sol. for fluid balance (within 5 h after inj.)

Table 3. Effect of osmotherapy on ICP controlled by continuous pressure monitoring with extradural transducers. A "*paradoxical reaction*" with marked elevation of ICP by osmotherapy was observed in 23%. In 12% the initial pressure decrease was followed by a secondary pressure overshoot ("*rebound*")

ICP-Monitoring with miniaturized intracranial pressure transducers, 15. Jan. 1977-24. March 1978

Total no. of patients monitored: 115 (100%)
Severe brain inj.: 43 (38%)
(no eye opening to pain; beg. ICP-monit. within few h after inj.)
Osmotherapy: 15 (33% of inj.)
(Mannisorb 40%, 1-2 g/kg, only with ICP reaching 35-40 mmHg)
At least one "paradoxal ICP.increase": In 7 (= 45% of 15) patients

Related to single doses of Mannisorb given in these 15 patients:
(correct infusion always controlled by blood osmolality!)
Total no. of single doses (100-250 ml/15-30 min, mean 250 ml/30 min): 48 = 100%

| ICP foll. inf.: (course) | Doses n | % | Mean Δ ICP ($\bar{x} \pm s_x$, mmHg) | Max. (mmHg) | Min. | Period of ICP ($\bar{x} \pm s_x$, min) |
|---|---|---|---|---|---|---|
| Increasing (↗) | 11 | 23 | 14 ± 10.5 | 40 | 5 | 42 ± 26 |
| No effect (→) | 8 | 16 | (2 cases in final state with dil. pupills) | | | |
| Decreasing (↘) | 29 | 60.5 | 14 ± 8.5 | 40 | 5 | 110 ± 67 |
| Decreasing (↘ ↗) but *rebound* | 6 | 12 | 18 ± 14 | 50 | 8 | |

Osmotherapy then leads to osmolalities which may themselves result in further neurological disorders (1.25); at 390 mosmol death may occur experimentally (1.25). Moreover sodium retention (2, 4, 14) leads to an *increasing hyperosmolality*, even in the interval (Fig. 5), and even when fluid balance is compensated. Osmotherapy is contraindicated here and the values normalize after its withdrawal.

## Conclusion

Osmotherapy at *regular* intervals in craniocerebral trauma does *not* have a favorable effect either in experiments or in clinical trials (15). Osmotherapy should, therefore, *only be used in cases with increased* ICP. The clinical picture should be observed carefully and, if possible, ICP should be recorded continuously during osmotherapy in order to prevent a "paradoxical reaction." Serum *osmolality* and serum *sodium* should be measured regularly in order to avoid toxic osmolalities and progressive hypernatremia. The risk of side effects can probably be diminished by a *lower dosage* of mannitol; 0.25 k/kg are already sufficient as a single dose for effective lowering of ICP (17, 19).

Table 4. Osmotherapy in an 8-year-old child with severe closed head injury (diffuse edema). For numeric data see Figure 3. The number and height of pressure waves, as well as the initial ICP are *increased* by Mannisorb infusion

ICP-pressure waves (plateau-like) before, during and after osmotherapy.
3rd day after injury, therapy: 2 x 100 ml Mannisorb/4h.

| Period | Mean ICP ($mmHg \pm s_x$) | Plateau-waves of 5 mmHg, 5 min n | ICP, $x \pm s_x$ | max. | min. | Base ICP between waves ($\bar{x} \pm s_x$ mmHg) |
|---|---|---|---|---|---|---|
| Within 4 h before osmotherapy | 30 ± 19 | 6 | 39 ± 12 | 70 | 25 | 18 ± 11 |
| Within 4-h-period of osmotherapy | 28.5 ± 20.5 | 8 | 49.5 ± 17 | 80 | 35 | 15 ± 12.5 |
| Within 4 h after osmotherapy | 41.5 ± 17 | 9 | 55 ± 15.5 | 85 | 45 | 29 ± 7.5 |

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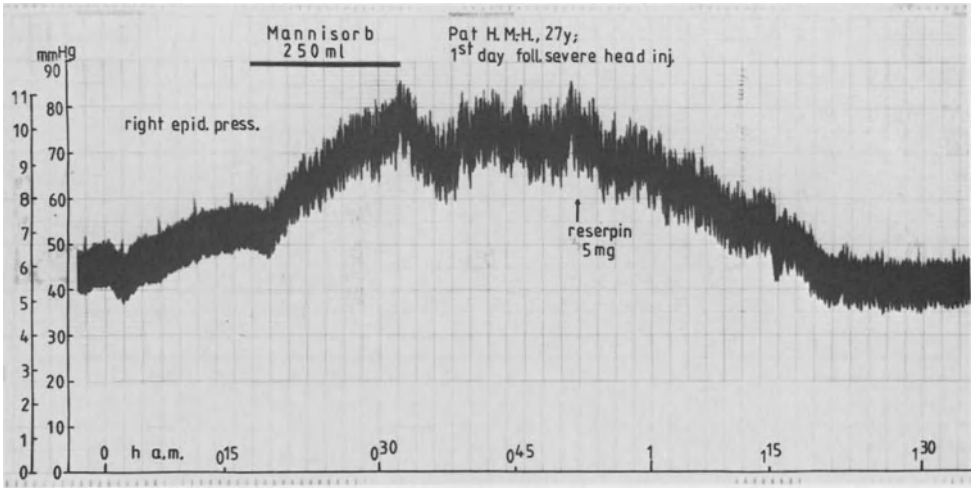


Fig. 1. Osmotherapy in a patient during the first day after severe closed head injury (right hemispheric edema) leading to an acute and marked increase in ICP (wave-like) accompanied by clinical deterioration (mydriasis on the right side, extensor spasms)

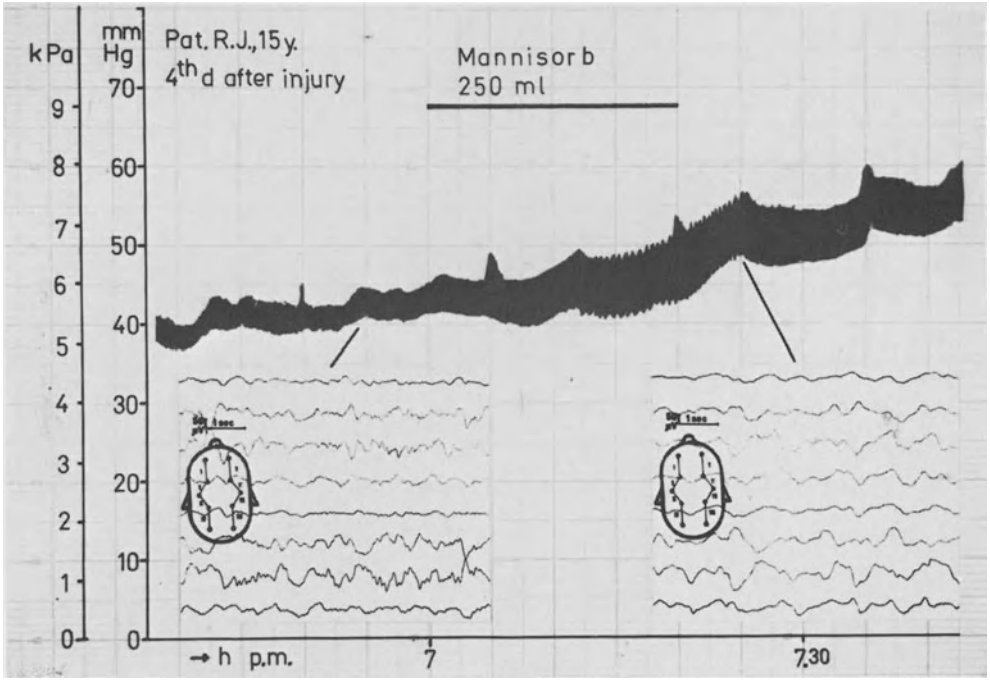


Fig. 2. Paradoxical ICP-increase following osmotherapy at a later post-traumatic stage. Note EEG deterioration after Mannisorb infusion

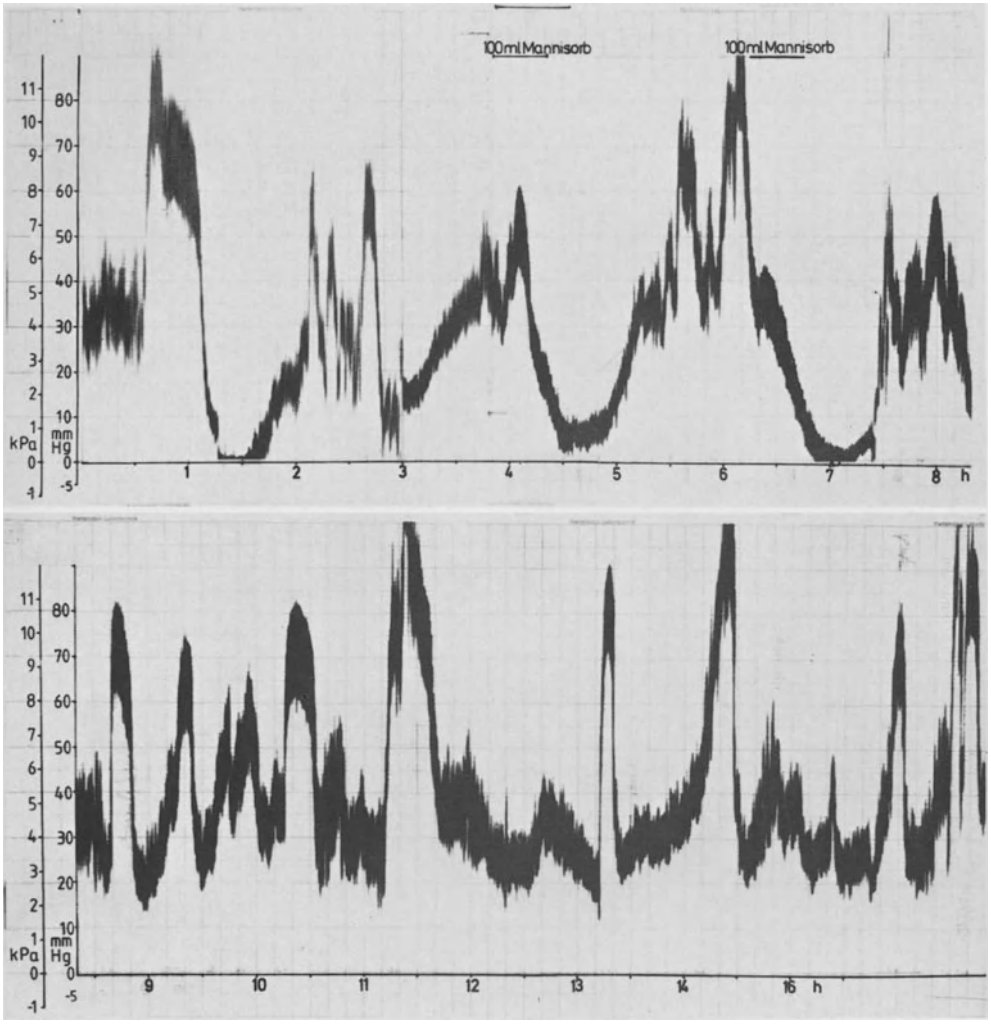


Fig. 3. Osmotherapy in an 8-year-old child with severe closed head injury (diffuse edema). The plateau (B-)waves are *increased* in number and height during and after Mannisorb infusion. ICP between waves is also elevated by osmotherapy. (For numeric data see Table 4)

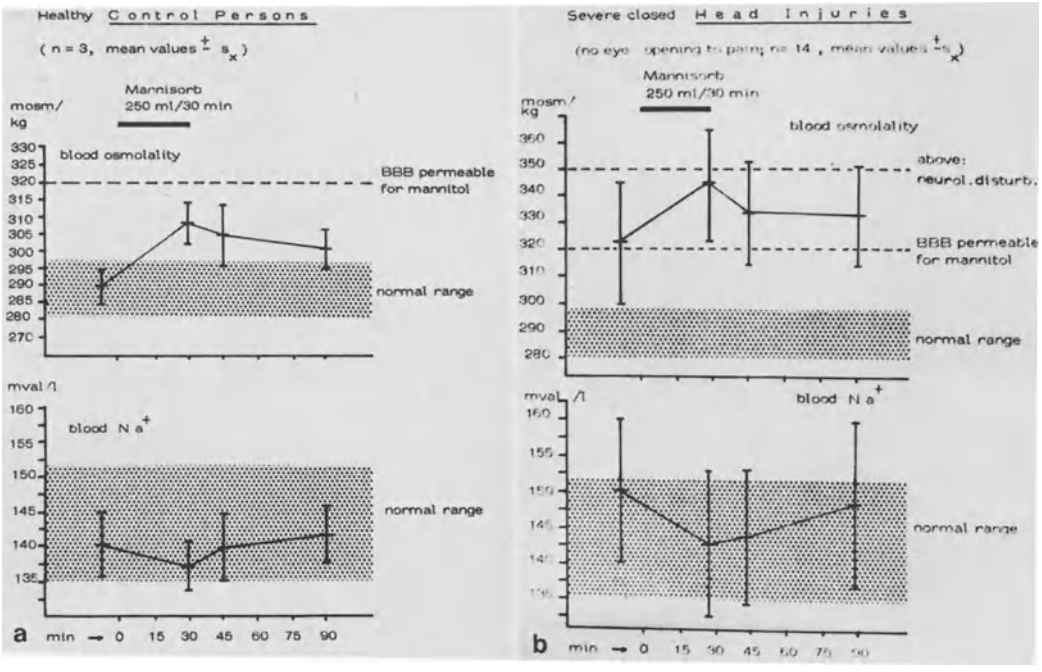


Fig. 4a, b. Blood osmolality and blood *sodium* values during osmotherapy in controls (a) and in patients with severe head injury. Following head injury, osmolality is always increased prior to osmotherapy. Mannisorb then leads to critical osmolalities, which may cause mannitol outflow through the BBB and additional neurological deficit by hyperosmolality

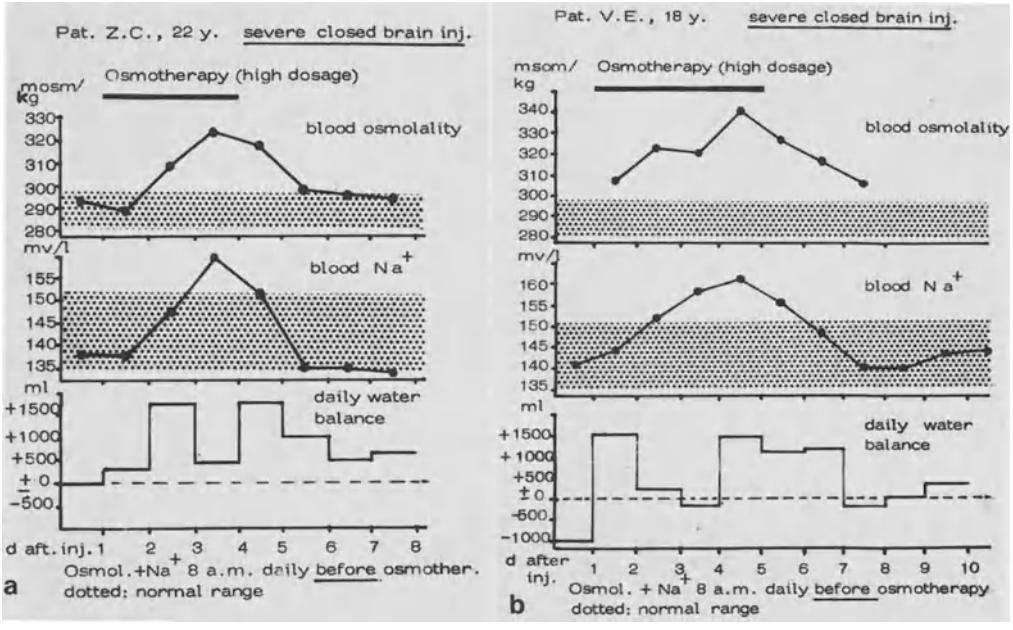


Fig. 5a, b. Long term osmotherapy with repeated regular doses of Mannisorb. Initial daily blood osmolality and blood sodium values prior to hyperosmolar therapy progressively increase to values which contraindicate a further osmotherapy. Values normalize when Mannisorb is discontinued

# Experimental and Morphologic Investigations on Reversible and Irreversible Hypothalamic Compression in the Rabbit

H. E. CLAR, L. GERHARD, and V. REINHARDT

Intracranial space-occupying lesions causing direct or indirect compression of the hypothalamic structures are apt to produce severe disturbances of endocrine and general hypothalamic regulations. Neurosurgical experience confirms the important role dysregulations play for operative and postoperative survival and care. This is true also for procedures performed on the hypothalamic structures proper. Therefore an experimental study of this clinical and morphologic diencephalic syndrome appeared to be of value. Since previous investigations (4, 6, 8, 10) did not deal with clinical problems, we tried to develop an experimental model which imitates a hypothalamic space occupying lesion in the rabbit, so as to study acute and chronical hypothalamic dysfunction.

## Method

Silicon steel balls (weight: 70 mg; diameter: 3.5 mm) were implanted by microsurgical technique in the chiasm and diencephalic region in 11 rabbits (weight 2.8-4.4 kg) under intubation anesthesia (Nembutal 30 mg/kg + 0.05 mg Atropin; $O_2/N_2O$ 1:3 + 0.4-0.5 Vol% Halothan. During and after the procedure body temperature was recorded. Location of the silicon steel ball was controlled by x-ray and necropsy (Fig. 1a, b).

In seven control animals only the bone and subarachnoid space was opened. Anatomic alterations were studied following acute (5 h) and chronic lesions (3 weeks) by perfusing the brain with 6% glutaraldehyd or 9% formalin.

## Results

The following results were obtained:

1. Following the end of anesthesia an increase in temperature occurred as a dominant reaction in all cases with inserted ball but not in the control group (Fig. 2).
2. Aphagia and adipsia could be observed immediately after surgery and lasted for 3-6 days.
3. In the acute cases, a diffuse edema was found in the anterior hypothalamus, mainly unilateral, but with some contralateral involvement. Small perivascular hemorrhages were present in the vicinity of the chiasm as a sign of vascular damage (Fig. 3a, b).
4. The chronic lesion was restricted to the anterior hypothalamus and to the region of the chiasm. These areas showed very small perivascular hemorrhages and cell proliferation. No sign of persistent edema were found (Fig. 4a, b).

## Discussion

By inserting a silicon steel ball in the hypothalamic area of the rabbit we were able to produce reversible and irreversible hypothalamic damage. This experiemental model could imitate the situation of a hypothalamic

space occupying process such as a suprasellar tumor. The main effect appears to be a disturbance of blood brain barrier function in the hypothalamus as seen in the acute lesion. This is the apparent cause of most of the hypothalamic dysregulations. Since the lesion is restricted mainly to the frontal hypothalamus, functional disturbances of the nuclei of this area may be expected. Temperature dysregulation, mainly temperature increase after the end of anesthesia, adipsia and aphagia are signs of anterior hypothalamic dysfunction, and are in agreement with the morphologic findings.

In the acute stage diffuse edema of the anterior hypothalamus predominates apparently caused by compression of this area, and as result of a localized blood brain barrier dysfunction. It leads to a breakdown of the regulatory system. Neuronal functions can no longer be regulated by peripheral feed back or central receptors. Thus a disturbance of the input may be the reason for this dysregulation (5). Since hypothalamic hemeostasis depends on a summation of neuronal inputs (3), a localized disturbance may be followed by a reaction of the entire system. In contrast, animals with a chronic lesion, showed a much smaller area of scar-like alterations and cell proliferation. No edema was found after 3 weeks. At the same time, signs of hypothalamic disturbances had ceased. In these cases, restitution had taken place, so that intact areas have compensated for the dysfunction. This type of reaction is typical for bilateral and symmetric areas, which only decompensate in cases of bilateral lesions (1, 9). In case of an asymmetric lesion maintainance of function may be ensured by the contralateral structure (2).

Comparing the acute and chronic findings one sees that reversible lesions predominate if the experimental procedure has been performed carefully.

These results should be considered as a first step in the investigation of reversible and irreversible hypothalamic compression. The next step, already started, is the production of different phases of hypothalamic compression in a single animal. This would imitate the situation of tumor growth. These steps will be followed by endocrinologic as well as by therapeutic studies.


## Summary

Reversible and irreversible hypothalamic lesions have been produced in rabbits by inserting a silicon steel ball into the hypothalamic region. The main result appears to be a disturbance in blood brain barrier function within the hypothalamus, causing most of the diencephalic dysregulations. These disturbances prove to be mainly reversible. The only permanent morphologic sign of previous blood brain barrier disturbances is proliferation of perivascular astrocytes.


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Fig. 2. Temperature course in 7 control rabbits (*A-C*) and in 11 rabbits with insertion of a silicon steel ball (*1-4*). *A* and *1*: Beginning of anesthesia; *2*: Insertion of the silicon steel ball; *B* and *3*: End of anesthesia; *C* and *4*: End of recording

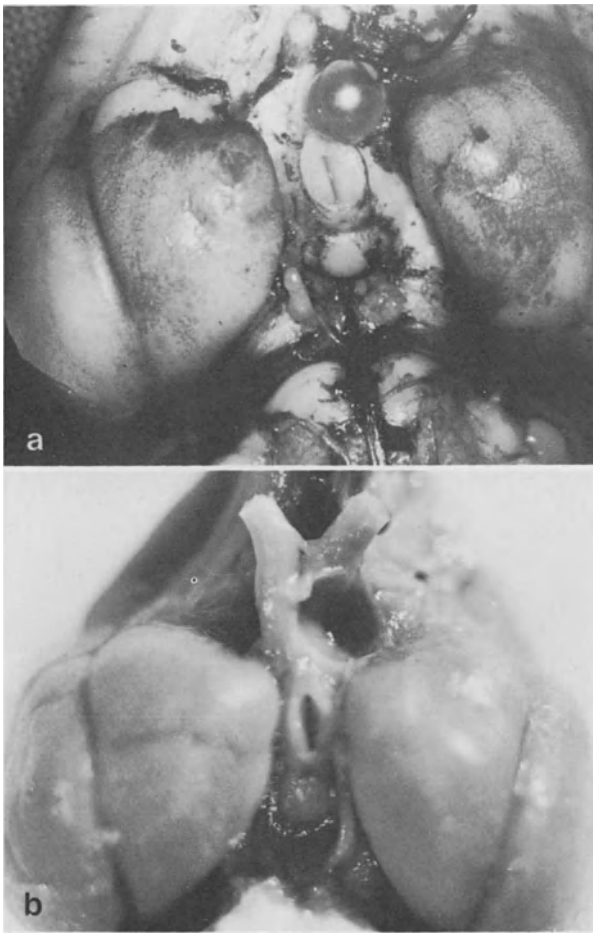
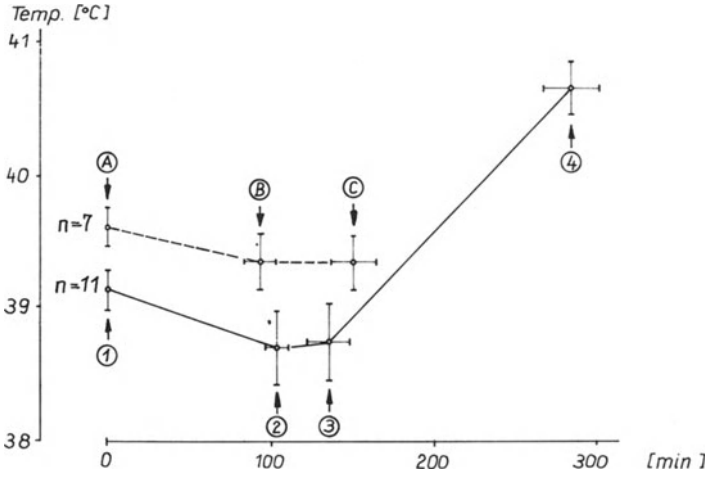



Fig. 1. a Basal view of the brain of a rabbit following the introduction of a silicon steel ball. b Basal view of the brain of a rabbit after removing the silicon steel ball. Impression of the rostral hypothalamus



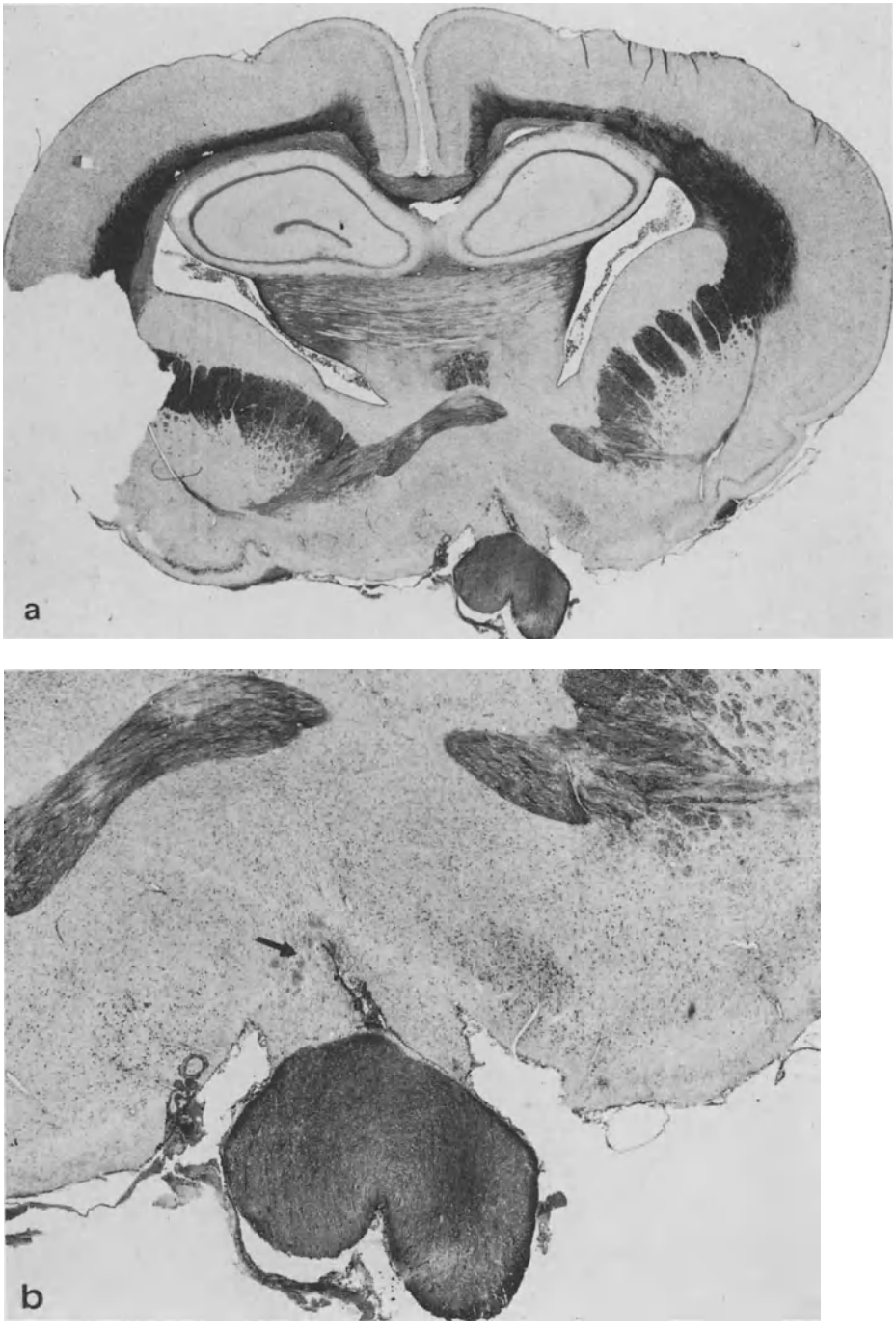



Fig. 3. a Frontal section through the brain of rabbit no. 10 at the level of commissura anterior 5 h after implantation of the silicon steel ball (Luxol-fast-blue - Cresyl violet 10:1). b Detail of the diencephalon and chiasm region (20:1); small acute hemorrhages in the region of the recessus supraopticus

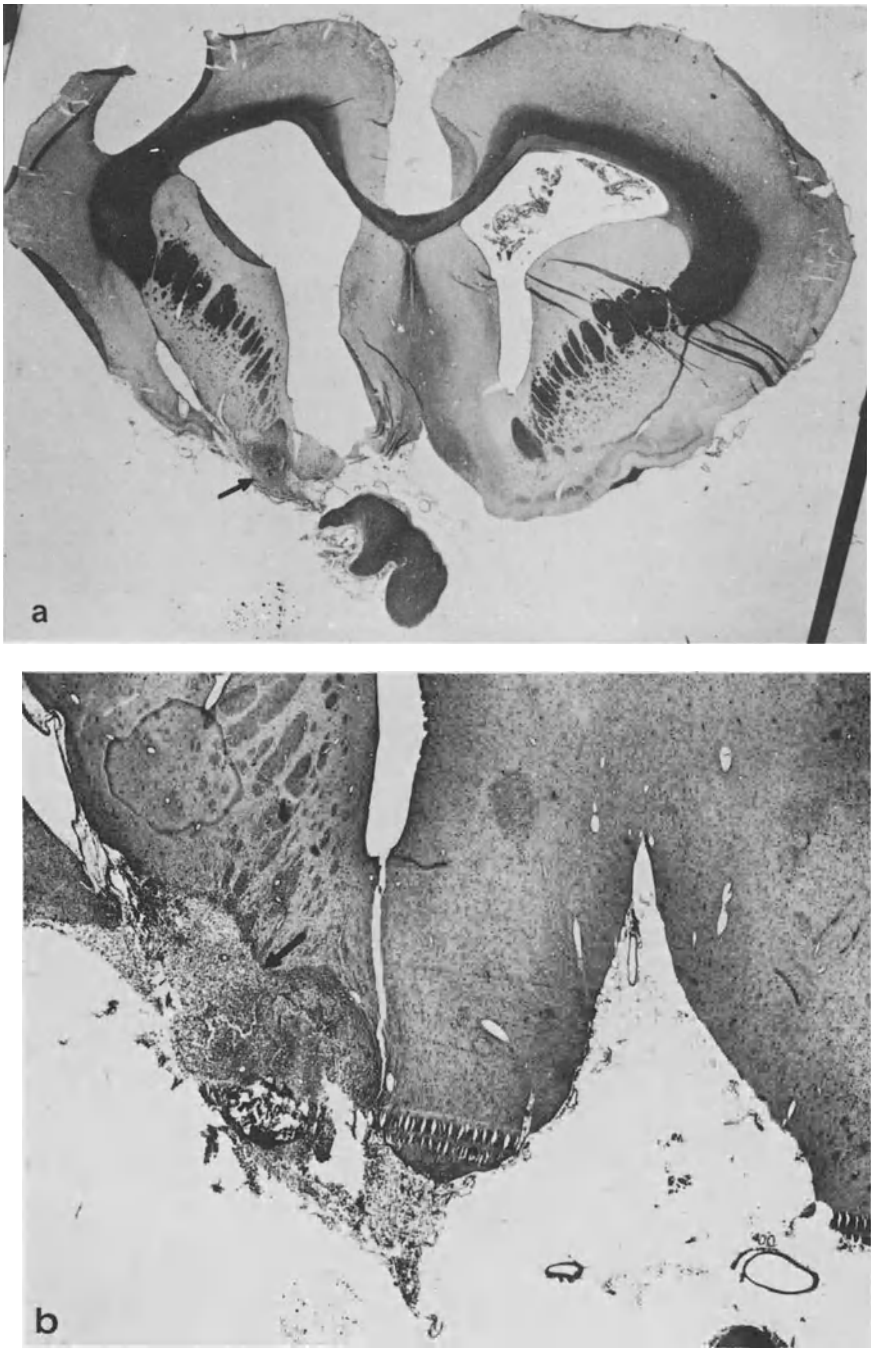



Fig. 4. a Frontal section through the brain of rabbit no.9 at the level of the nuclei septales 3 weeks after implantation of the silicon steel ball (Luxol-fast-blue - Cresyl violet 10:1). Chronic defect at the base of the lateral ventricle. b Detail of the anterior hypothalamus (Elastica - van Gieson 20:1), necrosis with demarcation and small perivascular hemorrhages

# Treatment of Peritumoral Brain Edema

G. MEINIG, H. J. REULEN, K. DEI-ANANG, and K. SCHÜRMANN

## Introduction

Apart from particular clinical situations which may require additional provision, the treatment of peritumoral brain edema is substantially based on two groups of drugs: corticosteroids and diuretics. The aim of this study is to quantify the therapeutic effect of different forms of treatment (including a continued therapy), and to increase understanding of the dynamic process of formation and resolution of brain edema.

## Material and Methods

The following parameters were examined to assess the antiedematous drug effect:

*a) Course of neurologic condition*, scored daily in 42 patients with brain tumor, treated with dexamethasone (4 x 4 mg i.m./die) using a special evaluation and documentation sheet (10, 11, in modification).

*b) Reduction of the area of peritumoral edema* and of mass displacement with the aid of CT in patients receiving a combined dexamethasone and furosemide therapy. As described earlier (3, 4), an attempt was made to determine the area of edema by planimetry and to measure the reduction of midline structure shifting.

*c) Changes in water and electrolyte content* in the peritumoral edema of 146 patients with brain tumor. Following groups were compared:

1. Sixty-one patients who did not receive any antiedematous treatment served as controls. This group contains 28 patients investigated by BAETHMANN and SCHMIEDEK (Munich).
2. Twenty-nine patients received dexamethasone in a dosage of 4 x 4 mg i.m./die for 4-6 days.
3. Eleven patients receiving 3 x 8 mg i.m. for 4-6 days
4. Eleven patients receiving 4 x 4 mg i.m. for a period of 2-4 weeks
5. Thirty-four patients treated with an association of dexamethasone (4 x 4 mg i.m./die for 4-6 days) and furosemide (3 x 40 mg p.o./die for 4-6 days).

## Results

In order to obtain comparable results, grading of neurologic dysfunction was performed by means of frequency distribution analysis before and after therapy with dexamethasone (Table 1 and 2). Despite the relatively short time of treatment (6-9 days), the results were surprisingly favorable. A small group of patients with impending herniation had to be operated on immediately and was excluded from the subsequent study. No single symptom worsened under dexamethasone treatment. Only in a few instances did an initial pathological finding persist, and also in only a few cases did we observe a secondary worsening following an initial improvement.

Documentation Sheet

Family Name First Name Date of Birth Age

Day of Examination

Time of Examination

Grade of Consciousness

1. Normal
2. Responsive (slow in reaction)
3. Responsive (extremely slow in reaction)
4. Responsive to voice call, visual contact
5. Waking response to pain
6. Direct defense reation to pain
7. Indirect defense reation to pain
8. Flexion upon painful stimulation
9. Extensor spasm to painful stimulation
10. No response to painful stimulation

Orientation:

*A. Temporary Orientation*

1. Normal
2. Slightly impaired
3. Severely impaired
4. Lack of orientation

*B. Local Orientation*

1. Normal
2. Slightly impaired
3. Severely impaired
4. Lack of orientation

Memory (recollection for recent events)

1. Normal
2. Slightly impaired
3. Severely impaired
4. Total amnesia

Day of Examination

Time of Examination

Memory (Recollection for ancient events)

1. Normal
2. Slightly impaired
3. Severely impaired
4. Total anamnesia

Strength

1. Normal
2. Able to move against moderate resistance (slightly reduced function)
3. Only able to overcome gravity
4. Movement possible only when gravity counteracted
5. Pronounced paresis; contractions can only be seen or felt
6. Paralysis

Pupillary Size (Contralateral to tumor)

1. Extremely small
2. Small
3. Medium
4. Dilated
5. Maximally dilated

Pupillary Reaction (Homolateral to tumor)

1. Normal
2. Minimal
3. None

Babinski (Contralateral to tumor)

1. None
2. Suspicious
3. Positive

Babinski (Homolateral to tumor)

1. None
2. Suspicious
3. Positive

Tables 1 and 2 show the extent and time course of regression of two different symptoms. Although it is not possible to compare the grade of disturbance of different symptoms, it is, however, obvious that some symptoms show quicker and more complete regression. Out of 26 patients with severe initial disorders of consciousness, only 4 failed to show quick improvement at the end. On the other hand, among 21 patients with paresis of the leg, 8 showed some paresis at the end of therapy. While disorders in consciousness improved already after a few hours of treatment, paresis persisted for days in most cases.

CT studies were made before and during the combined therapy with dexamethasone and furosemide at intervals of 2-4 days. As reported earlier (3, 4), this new technique permits direct visualization of the extension of the area of edema and probably also the dynamic process of its formation and resolution.

Secondary alterations due to edema, such as shifting of midline structures or ventriclar displacement and compression can be measured by this method. The CT following combined therapy often shows an impressive reduction of peritumoral brain edema (Figs. 2, 3). The problem, however, is to quantitate the drug effect and to find reproduceable criteria. The planimetric measurement of the area of edema may be difficult if the edema is not well delineated. Moreover, com-

Table 1. Frequency distribution analysis before and after treatment with dexamethasone

| State of consciousness | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | | (n) |
| Grade of disorder | 1 | 2 | 3 | 4 | 5 | 6 | 7 | - |
| Initial Finding | 17 | 5 | 3 | 9 | 5 | 2 | 1 | 42 |
| Finaly Result | 38 | 4 | - | - | - | - | - | 42 |

| State of orientation | | | | | |
|---|---|---|---|---|---|
| | | | | | (n) |
| Grade of disorder | 1 | 2 | 3 | 4 | |
| Initial finding | 19 | 1 | 8 | 14 | 42 |
| Final result | 35 | 6 | 1 | - | 42 |

| Memory (Recollection for ancient events) | | | | | |
|---|---|---|---|---|---|
| | | | | | (n) |
| Grade of disorder | 1 | 2 | 3 | 4 | |
| Initial finding | 23 | 6 | 6 | 7 | 42 |
| Final result | 38 | 4 | - | - | 42 |

| Memory (Recollection for recent events) | | | | | |
|---|---|---|---|---|---|
| | | | | | (n) |
| Grade of disorder | 1 | 2 | 3 | 4 | |
| Initial finding | 19 | 2 | 10 | 11 | 42 |
| Final result | 28 | 14 | - | - | 42 |

Table 2. Frequency distribution analysis before and after treatment with dexamethasone

| Paresis of the arm | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | | (n) |
| Grade of disorder | 1 | 2 | 3 | 4 | 5 | 6 | |
| Initial finding | 20 | 3 | 9 | 8 | 2 | - | 42 |
| Final result | 35 | 5 | 1 | 1 | - | - | 42 |

| Paresis of the leg | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | | | | (n) |
| Grade of disorder | 1 | 2 | 3 | 4 | 5 | 6 | |
| Initial finding | 20 | 4 | 9 | 7 | 2 | - | 42 |
| Final result | 34 | 5 | 2 | 1 | - | - | 42 |

| Babinski | | | | |
|---|---|---|---|---|
| | | | | (n) |
| Grade of disorder | Negative | Suspicious | Positive | |
| Initial finding | 29 | 2 | 11 | 42 |
| Final result | 35 | 5 | 2 | 42 |

parison of repeated CT pictures depends on the obtaining identical planes. In contrast, determination of midline shiftings is less problematic. Planimetric measurement of the area of the edema as well as determination of the dislocation of the septum pellucidum and pineal body showed a reduction following the combined therapy in 15 out of 16 cases. However, the time delay until the resolution of brain edema as well as its extent varied considerable. The different response to therapy might be related to the nature of the brain tumor.

The normal water content of white matter is about 70% (9). As shown in Table 3, the average water content of the peritumoral white matter of

Table 3. Water and electrolyte content following different forms of antiedematous therapy

| Antiedematous therapy | None | Dexamethasone 4x4 mg i.m./die 4-6 days | Dexamethasone 3x8 mg i.m./die 4-6 days | Dexamethasone 4x4 mg i.m./die 2-4 weeks | Dexamethasone 4x4 mg i.m./die 4-6 days and Furosemide 120 mg/p.o. /die, 2-4 days |
|---|---|---|---|---|---|
| $H_2O$ % | 79.62 ± 0.56 (61) | 76.54 ± 0.83 (29) | 76.38 ± 0.88 (11) | 75.39 ± 1.56 (11) | 75.32 ± 0.65 (34) |
| Na (mEq/kg dry wt) | 389.3 ± 18.8 (47) | 343.1 ± 23.4 (29) | 352.7 ± 25.9 (11) | 323.6 ± 36.5 (11) | 306.9 ± 18.9 (34) |
| K (mEq/kg dry wt) | 272.3 ± 9.0 (48) | 259.3 ± 14.0 (29) | 259.0 ± 17.5 (11) | 224.8 ± 9.5 (11) | 212.5 ± 6.1 (34) |

Water and electrolyte content in peritumoral white matter.
Means ± SEM; number of patients are shown in parentheses.

patients not treated with dexamethasone and furosemide preoperatively was increased by about 10%. Following the treatment with dexamethasone, water content decreased significantly by about 3%. However, patients treated with the combined therapy showed a further significant reduction of peritumoral edema. The long term treatment with dexamethasone alone showed nearly the same therapeutic effect as the short-time combined therapy.

## Discussion

In addition to the well known antiedematous effect of dexamethasone (1, 2, 3, 4, 5) and furosemide (6, 7, 8) an additive effect of the combined therapy with dexamethasone and furosemide has been found. This may be explained by a different mode of action of the two drugs. Although the mechanism is not completely understood, dexamethasone might decrease the formation of brain edema. Diuretics such as furosemide and ethacrynic acid, known to reduce the CAF production rate and intracranial pressure, might increase the clearance of edema fluid from tissue to CSF (7, 8). The reopening of the ventricles and, probably, of the subarachnoid space may be important factors in improving the clearance of edema fluid (7, 8).

The cause of the improvement of the neurological deficit during dexamethasone treatment is still unknown. Reduction of neurological symptoms parallels the reduction of edema. On the other hand, the extent of peritumoral edema apparently does not correspond directly to the neurological condition. While resolution of brain edema appears to be clearly improved following combined therapy, neurological improvement seems not to be significantly accelerated, as confirmed experimentally (5). It is concluded that dexamethasone might have different modes of action, especially on cell metabolism and function, additionally it affects the prevention and resolution of brain edema.

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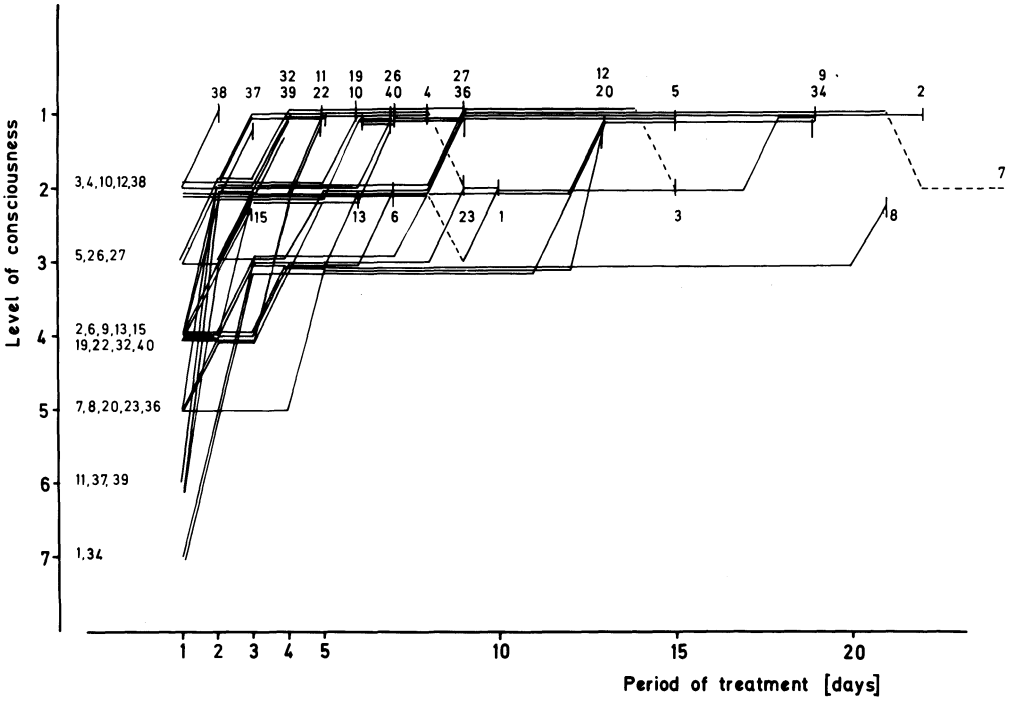


Fig. 1a. Improvement of depressed sensorium during treatment with Dexamethasone

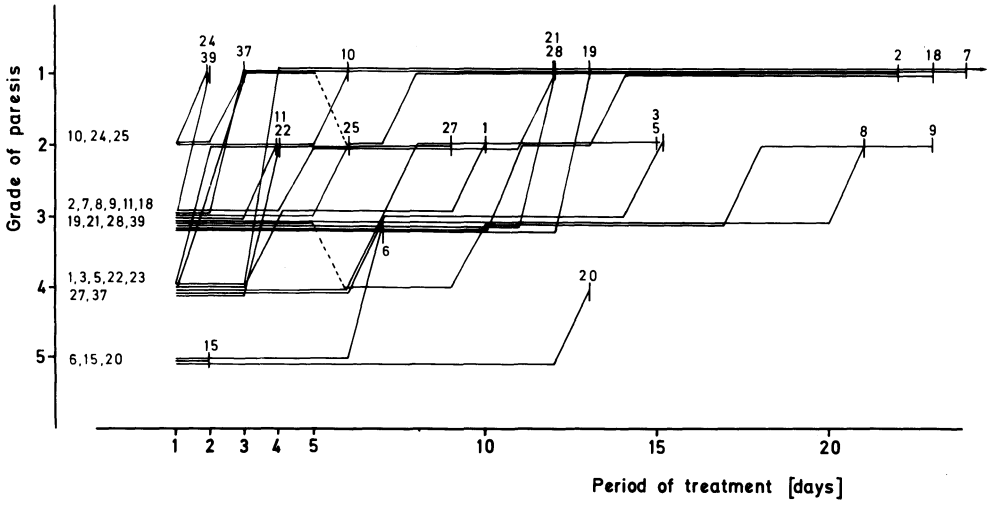


Fig. 1b. Improvement of paresis of the leg during treatment with Dexamethasone

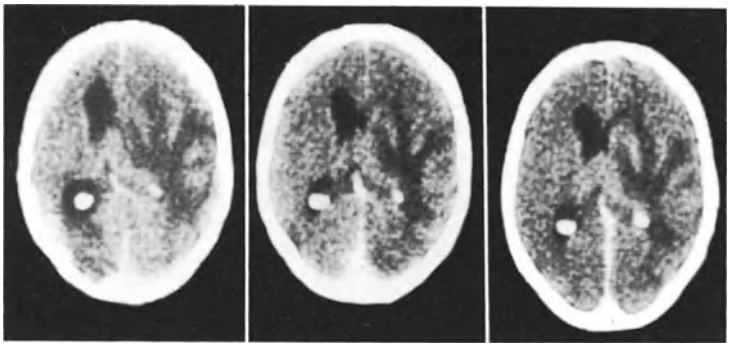

Fig. 2. CT showing decrease of peritumoral brain edema and reopening of right lateral ventricle following combined therapy with Dexamethasone and Furosemide

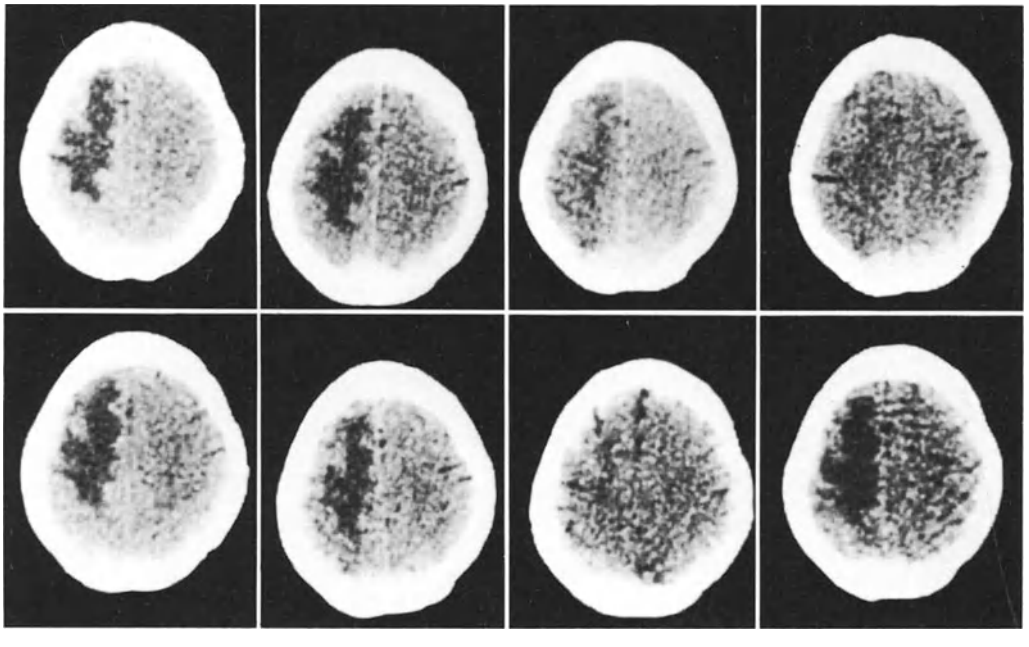

Fig. 3. CT demonstrating resolution and recurrence of peritumoral brain edema parallel to the onset and termination of treatment with Dexamethasone

# Pinealoma With Initial Spinal Manifestation

H. SCHLARB and M. SCHIRMER

Three factors are of major importance in the treatment of pinealomas:

1. Histologic classification (1, 2, 12)
2. Formation of metastases along the CSF pathways or ectopic tumor growth (3, 9, 12)
3. High radiosensitivity of these tumors (3, 4, 5, 6, 9, 11, 13)

In this paper the term "pinealoma" is used for the most common type of tumor, the "seminoma-like" tumor, sometimes called "anisomorphic pinealoma" or "pinael germinoma."

Since 1974 there is a remarkable increase in the number of reports on pineal tumors, possibly due to the use of computerized tomography (CCT) in clinical diagnosis (9, 10). Prior to this time diagnosis was much more difficult since the symptoms are not always clear and neuroradiologic detection of midline tumors is still defective. The tumor could only be demonstrated when intruding into the third ventricle or shifting the vena magna cerebri. However, in most cases only an occlusive hydrocephalus was diagnosed.

In the literature (8) we found the case of 14-year-old boy who had been operated on for an intramedullary anisomorphic pinealoma ($Th_8$-$Th_{10}$) in 1967; 4 years later he still showed no symptoms of intracranial tumor. By publishing the following case we hope to emphasize this observation.

## Case Report

An 18-year-old patient complained about increasing backache and sciatica in both legs for the last 6 months, especially while walking, later also while resting. A 4 week conservative treatment (lumbar disc) in an orthopedic hospital did not improve his condition. Myelography was not attempted since the cerebrospinal fluid showed an excessive amount of protein and the patient suffered from excruciating radicular pain. On October 21, 1977 the patient was transferred to our department for further treatment.

The patient's physical and mental development was as normal as that of his five brothers and sisters. According to his parents, he recovered from pneumonia and pleurisy when he was 5 years old, and he never suffered from any other disease. The patient was 176 cm tall, and weighed 60 kg. The lumbar vertebral column was stiff and straightened, making ventroflexion of the body impossible. He walked by short steps to avoid pain in both legs. The Lasègue sign was positive on both sides at 40°. All tendon reflexes, especially those of the lower extremities were present on both sides. There was no paresis, except for a weakness of the extensor hallucis longus on the left. A hypalgesia in the area of the buttocks was found to be inconstant. X-ray studies of the entire spinal column did not show any sign of bone affection. Myelography with Duroliopaque revealed a complete block at the level of $Th_{12}$.

A laminectomy beginning at $Th_{12}$ was performed. Upon incision of the dura, brownish tumor masses pressed through. The very vascular tumor, 15 cm in length, started at the top of the conus medullaris and reached down to the third lumbar vertebra. Despite its atypical appearance, it was thought to be a neurinoma (because a nerve root traversed it), or an ependymoma of the filum terminale. Postoperatively the patient suffered only from postoperative pain. He was relieved of sciatica. Two days later he was able to get up. There was no further neurological deficit.

The histological diagnosis of "seminoma" by the pathologist, and, later, of "pineal germinoma" (Fig. 1) by the neuropathologist, led us to study attentively the pineal gland and the midbrain as well as the region of the infundibulum. Detailed examination revealed no paralysis of the upward gaze, no chiasma syndrome, and no diencephalic disorder. The patient did not suffer from headache, transient unconsciousness, constant tiredness, or increasing disability to concentrate. X-rays of the skull showed only two tiny globules of contrast medium in the posterior cranial fossa. Within 6 weeks brain scans showed an increased activity in the region of the frontal basis. This diagnosis was confirmed by CCT. Both anterior horns showed areas of marked tumor-hyperdensity already extending into the lateral ventricles, and having increased very much within one month. In the area of the pineal region, or rather in the area of the diencephalon, a tumor could be excluded. A betatron radiotherapy with opposite fields followed.

During the following two weeks the patient's condition worsened: he became somnolent and had to be temporarily treated by infusion therapy. After interruption of the radiotherapy for one week the patient felt better. He received further radiotherapy as an outpatient. Since the control scan (after application o 4000 rad) showed nothing extraordinary, radiotherapy was stopped. Due to technical failure of the instrument, the CT could not be controlled until March 30, 1978 (Fig. 2). Now the tumor was smaller, but had not disappeared. At present, further 2000 rads are being given.

## Discussion

Histological examination of the tumor removed from the cauda equina induced the search for the primary intracranial pinealoma. It was found immediately, despite of the lack of any clinical symptoms. Both, brain scan and CT detected the tumor in the anterior horns, whereas the pineal region seemed to be free of tumor. CT revealed extension of the tumor into the cerebral white matter. Without the histological diagnosis of the caudal tumor, the intracranial pinealoma would not have been suspected. A spongioblastoma or an ependymoma might have caused differential diagnostic difficulties in this case because of their ability for intraventricular growth. Due to its location and extension, however, the tumor would have been thought to be inoperable anyhow. If radiotherapy had been tried, even without histologic diagnosis (4, 6, 9, 11), its striking effect might have led to a correct diagnosis (7).

In the present case, the possibility of an undetected tumor in the pineal region remains open, since the tumor infiltrates may show a different CT density from that of the original, even isodense tumor. We have found no evidence in the literature of a primary lumbar pinealoma proven by autopsy.

*Acknowledgement*. The histological identification of the tumor was made by Prof. Dr. K. KÖHN (Director of the Pathological Institute of the Krankenhaus Neukölln in Berlin) and Prof. Dr. G. ULE (Director of the Neuropathological Institute of the University of Heidelberg). CTs were made at the Clinic Charlottenburg of the Free University of Berlin. Radiotherapy took place at the Radiological Department of the Krankenhaus Neukölln (Director: Prof. Dr. P. SCHAEFER).

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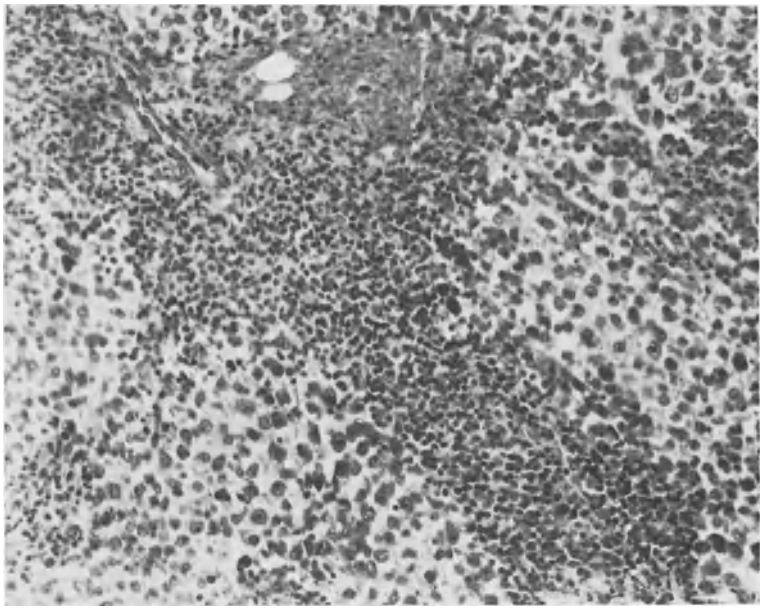

Fig. 1. Typical microscopic appearance of a germinoma showing two cell-types: large spheroidal cells with central vesicular nuclei, and lymphocytic cells. Haematoxylin-eosin-stainig. x 160

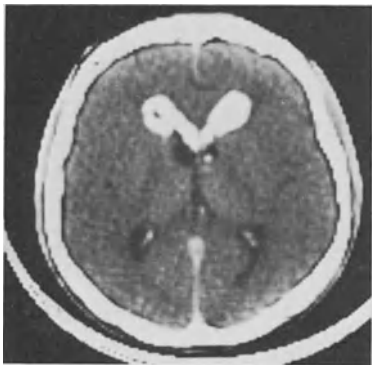

Fig. 2. CCT after application of 4000 rad. Both lateral ventricles are still filled with tumor tissue. Normal density in the pineal region and midbrain (with contrast enhancement)

# Subject Index

Advances in Neurosurgery

Volume 5

# Head Injuries Tumors of the Cerebellar Region

Proceedings of the 28th Annual Meeting of the Deutsche Gesellschaft für Neurochirurgie, Köln, September 18–21, 1977

Editors: R. A. Frowein, O. Wilcke, A. Karimi-Nejad, M. Brock, M. Klinger

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This volume represents the proceedings of the 28th Annual Meeting of the German Society of Neurosurgery 1977. Areas featured are the progress in research and clinical study of head injuries, tumors of the posterior cranial fossa (with the exception of acusticus neurinomas), and other related topics.
The section on head injuries reports on test results concerning the prognostic classification, computertomographic examination, symptomology and results of therapy of traumatic hematomas, frontobasal injuries, and general intracranial pressure increase.
In the section on tumors of the posterior cranial fossa, advantages and disadvantages to the use of the computer tomogram are weighed, and questions are examined concerning cytotatic therapy of medulloblastomas.



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# Cranial Computerized Tomography

**Editors:** W. Lanksch, E. Kazner, University of Munich, Munich, Germany
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1976. 620 figures. XIV, 468 pages
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Cranial computerized tomography (CT) has already, within a few years of its introduction by the English physicists G. N. Hounsfield in the early seventies, led to the revolution in the diagnosis of brain disorders. In addition, new dimensions have been opened up in the evaluation of orbital lesions. This volume provides possibilities of cranial CT in all of the important intracranial disorders.
The data are based on the extensive experiences of well-known authors in the diagnosis of brain tumors, cerebrovascular processes, intracranial hemorrhage, inflammatory and degenerative brain disorders, cranial and brain injuries and in brain edema and neuropediatric diseases processes, as well as in orbital tumors.
The book is a richly illustrated compendium od indispensable source material for all physicians and other scientists involved in cranial computerized tomography.

**The topical importance of this book is reflected by the following subject headings, comprising 64 individual contributions.**

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IV. Brain Atrophy – Epilepsy – Demyelinating – Regenerative Diseases of the CNS
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VI. Head Injuries
VII. Brain Edema – Inflammatory Diseases of the CNS
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