Advances in Neurosurgery 17

Head Injuries

Prognosis Evoked Potentials Microsurgery Brain Death

Edited by
R. A. Frowein M. Brock M. Klinger

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Preface

This 17th Volume of **Advances in Neurosurgery** contains a selection of the scientific reports of the 39th Annual Meeting of the Deutsche Gesellschaft für Neurochirurgie, which was held in Cologne on 8-11 May 1988.

The meeting commenced with the FEDOR KRAUSE MEMORIAL LECTURE, in which Prof. Dr. Dr. Kurt Schürmann presented a universal report on his long excellent clinical and operative experience with tumors of the orbit.

The first section of the subsequent contributions dealt in particular with the **long-term results of severe head injuries**, as well as with problems of acute traumatic hematomas and brain edema.

The second section contained up-to-date papers on **microsurgical experiences**, especially on the anatomy and operative technique for lesions in and around the **jugular foramen** and the craniospinal transition.

In the third section the special new results of **brain death determination** were described.

Beyond this, numerous contributions on clinical and research results were presented in a poster exhibition which was systematically organized in order to give younger neurosurgeons the opportunity for extensive discussion.

I want to express my special thanks to all the authors and organizers as well as to Prof. Klinger and Prof. Brock for their rapid editing of the manuscripts and Springer-Verlag for their renewed acceptance and excellent production of this volume.

R.A. Frowein

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The Development of Orbital Surgery from a Neurosurgeon's Viewpoint

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FEDOR KRAUSE MEMORIAL LECTURE

Mr. President, dear Reinhold Frowein, ladies and gentlemen,

First of all, let me thank you for the great honor you have accorded me. Surely you could have found a more worthy person; nevertheless, I am very happy, especially since I may assume that this honor extends to all the members of my team, who have worked for 30 years with remarkable loyalty towards the goal of achieving a respectable standard of neurosurgery in Mainz in the field of patient care as well as in research. Therefore my gratitude goes first of all to them on this day.

Allow me now to dwell on FEDOR KRAUSE (1857-1937), who, along with OTFRID FOERSTER (1873-1941), is the greatest of our ancestors in Germany. It was WILHELM TÖNNIS, the youngest in the series of great German neurosurgeons, who suggested the creation of the FEDOR KRAUSE MEDAL and the OTFRID FOERSTER MEDAL in 1950, the founding year of the Deutsche Gesellschaft für Neurochirurgie, in order to honor these two remarkable men. He did so using the words of HANS SACHS:

> "Ehret Eure großen Meister,
> dann bannt Ihr gute Geister".[1]

He could not have justified the creation of the medals in a better way, and we too should act in accordance with this motto, since we owe so much to these old masters.

Who was FEDOR KRAUSE? Well, he was born in 1857, the same year as that important Briton VICTOR HORSLEY. HORSLEY and KRAUSE - two very great men who were nevertheless worlds apart. HORSLEY was a very strong, energetic almost fanatical personality who was exposed to strong criticism and animosity due to his often very aggressive form of surgery and his high rate of fatal results. HORSLEY devoted his studies to the reaction of the brain to electrical stimulation. He made very important contributions to clinical neurophysiology. In contrast FEDOR KRAUSE was a quiet, self-contained, and more modest personality. He had deep rooted artistic tendencies and in his youth had difficulty in deciding whether to pursue a career as a pianist or a physician. Both the playing of the piano and his work as a sen-

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[1] Honor your great masters and good spirits will be with you.

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Table 1. Well-known contemporaries of FEDOR KRAUSE (1857-1937)

In Germany		Outside of Germany	
Hermann HELMHOLTZ	1821-1894	Victor HORSLEY	1857-1916
Robert KOCH	1843-1910	Charles Scott SHERRINGTON	1857-1952
		Hughling JACKSON	1835-1911
Eduard HITZIG	1838-1907		
Wilhelm ERB	1840-1921	Jules Joseph DÉJÉRINE	1849-1917
Carl WERNICKE	1848-1905	Pierre MARIE	1853-1940
Hermann OPPENHEIM	1858-1919	Joseph BABINSKI	1857-1932
		Sergej KORSAKOW	1854-1900
Rudolf VIRCHOW	1821-1902		
Ludwig EDINGER	1855-1918	Camillo GOLGI	1843-1926
Franz NISSL	1860-1919	Santiago Ramon y CAJAL	1852-1934
Korbinian BRODMANN	1868-1918		
Wilhelm Conrad RÖNTGEN	1845-1923		

sitive physician were to follow him throughout his life. It is remarkable that after his retirement he gave piano concerts in Rome, where he then lived with his daughter. These concerts were highly praised by Roman critics.

When we look at the contemporaries of FEDOR KRAUSE, we see that he was born into a period of great discoveries and great discoverers. Naturally this did not fail to have an influence on his personality (Table 1).

Table 2. Chronological table of FEDOR KRAUSE'S main contributions to the development of neurosurgery

1892 - Extradural temporal approach to the ganglion Gasseri over the base of the middle cranial fossa (independent of HARTLEY, USA, also 1892)

1894 - Laminectomy in intraspinal tumors

1898 - Unilateral approach to the cerebellopontine angle over a homolateral opening of the posterior cranial fossa

1900 - Transfrontal approach to the optic chiasm and to the pituitary gland over the base of the anterior cranial fossa

1909 - Excision of a lumbar disc prolapse (cauda equina-compression by a "ENCHONDROMA")

1911 - Intraoperative topographical studies on the human cerebral cortex of the central region

1913 - Suboccipital infratentorial approach to the quadrigeminal region (OP **in the sitting position** of the patient!)

One should not for a moment forget that neurosurgery at the time of FEDOR KRAUSE was performed only by a few general surgeons who had recognized the importance of surgery of the brain and the spinal cord and who therefore took great risks upon themselves in order to devote their innovative intelligence and their mental powers to this new area. Today we view their work with admiration and amazement.

There is almost no operative approach in modern neurosurgery which FEDOR KRAUSE did not already mention using with success (Table 2). The approach to the orbit, however, is an exception to this rule. It was the prerogative of KRÖNLEIN to perform the first procedure to remove an intraorbital tumor to maintain visual function - he removed a dermoid cyst in a 21-year-old man. The year 1886 witnessed the birth of lateral osseous orbitotomy, later known as KRÖNLEIN'S operation after its inaugurator. Until then **enucleation** or total orbital exenteration had been the method of choice for the treatment of intraorbital and retrobulbar tumors (Figs. 1-3)

The great merit of KRÖNLEIN'S operation lay in the fact that his procedure carried little operative risk and permitted the maintenance of visual function for the first time, although his method initially remained limited to lateral and upper ventral tumors, which were usually those of the tear gland. It is evident that the KRÖNLEIN operation retains its importance provided it is correctly used and is necessary modified. Let me now describe to you in what manner the orbit and its contents wakened my special interest right from the beginning of my time in Mainz.

The beginning was much simpler than one would think. It was a key experience which led to the later development. This experience occurred in 1955, the first year of my independent work as a young neurosurgeon.

The professor of ophthalmology at that time, Professor JESS - lovingly and with respect and admiration called "Papa JESS" (he was almost 70 years old) - asked me, the 35-year-old neurosurgeon with little experience, to see a pretty 15-year-old girl. She was almost blind on the right and was found to have optic atrophy and a slight prominence of the eye, while the X-rays according to RHESE showed a homolateral dilatation of the optic canal. According to our knowledge at that time, this triad was almost pathognomonic for an optic glioma (usually called a spongioblastoma of the optic nerve in our tumor classification of the time). The glioma extended from the rostral end of the eye to the optic canal, thereby leading to isolated dilatation of the optic canal.

Apart from the test of visual acuity, inspection (exophthalmos), palpation (resistance), and X-rays according to RHESE, no methods for demonstrating the optic nerve canal were available at that time. Above all there was no orbitophlebography, no computer tomography, and no magnetic resonance imaging. "Have courage," I said to myself, "you must show what you are capable of." Now one must know that my respected teacher WILHELM TÖNNIS had taught me that these optic gliomas must be operated on in two stages, a technique he had developed with the Berlin ophthalmologist LÖHLEIN and reported in 1948. According to this method, the first part of the procedure is intracranial operation by the neurosurgeon, who separates the optic nerve at the chiasm, dissects it out of the optic canal, and displaces the stump towards the orbit. The optic canal is then separated from the brain by a long-lapped dura flap. A week later the ophthalmologist removes the eyeball together with the optic glioma. That was the way I had seen the method performed in Cologne. But now, being confronted with

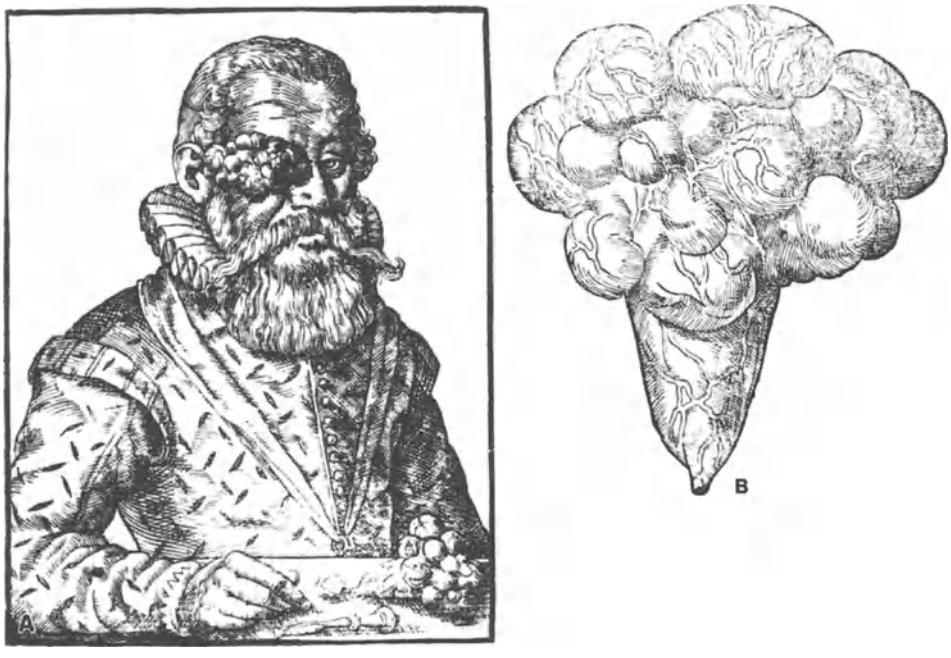


Fig. 1 A, B. Fifty-six year old patient with a huge tumor of the right orbit, treated by orbital exenteration in 1652. The patient survived. **A** Illustration of the patient. **B** Sketch of the removed tumor. (From Joh. Beyers)

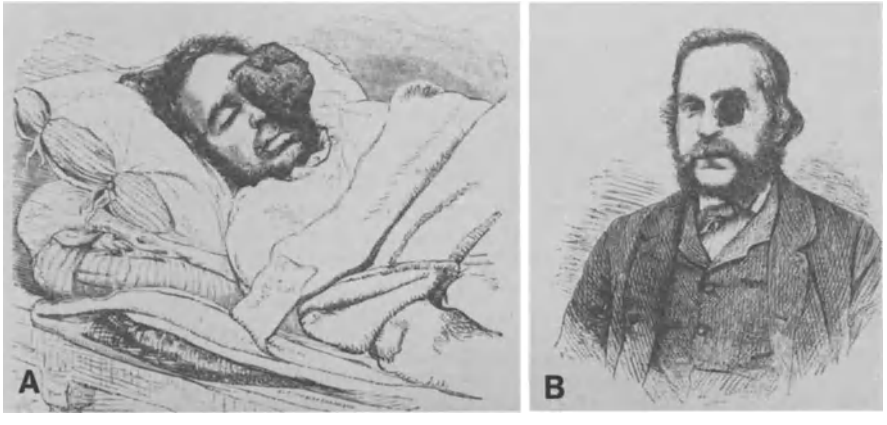


Fig. 2 A, B. Patient with a huge tumor of the left orbit and a large extraorbital mass, a so-called medullary carcinoma. The tumor was removed by orbital exenteration in 1869. **A** patient before surgery. **B** patient 15 months after surgery. Patient died later, because of intracranial invasion of a tumor recurrence

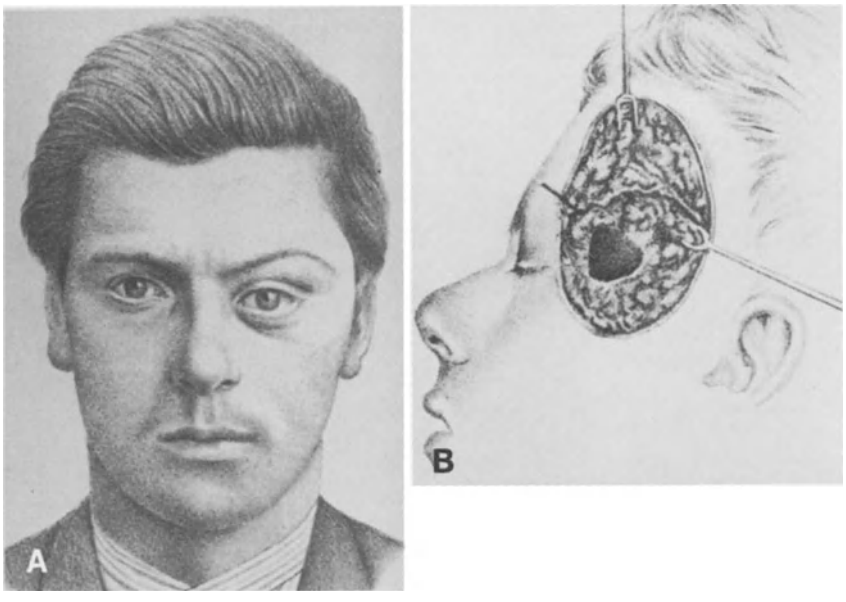


Fig. 3 A, B. KRÖNLEIN's original patient at the age of 21 years, in whom in 1886 he performed the first osteoplastic resection of the exterior orbital wall for the removal of a dermoid cyst in the left superior ventrolateral orbit. A Original sketch of the patient from KRÖNLEIN's paper, published in 1888. B Original sketch of KRÖNLEIN's operation, which shows his classical lateral approach to the orbit, later called ORBITOTOMIA OSSEA LATERALIS

a pretty young girl, I thought that a single intracranial procedure without subsequent enucleation of the eyeball would produce a very much better cosmetic and functional result. In order to achieve this, the optic nerve had to be served at the chiasm and removed from the optic canal; upon removing the roof of the orbit temporarily the optic nerve could be pursued to the anterior end of the tumor and then removed. In this way the total removal of the tumor should be possible, I felt. In fact by operating carefully it was possible to preserve not only the eyeball itself but also the ocular muscles and supplying nerves. To put it briefly: the operation was a success.

Since the skin incision was placed behind the delineation between forehead and scalp, the result was very satisfying. The mobility of the eyeball remained undisturbed, the pupil of the blind eye remained narrow due to consensual light reaction, and the exophthalmos was gone. The girl showed no signs of an operation (Fig. 4). Now, why am I relating this? Actually the only reason is to encourage the younger neurosurgeons to reflect intensely on the problems facing them and to have the courage to draw conclusions for action.

To continue my narrative, "Papa JESS" propagated our successful operation in ophthalmological circles with the result that the number of such operations on these and other tumors of the orbit increased steadily, until we are now surveying a series of 436 cases of space-occupying lesions of the orbit (Table 3).

In the development of this branch of neurosurgery there were further milestones in Mainz. During the 1950s we operated almost exclusively

Table 3. Space occupying lesions of the orbit

Meningeomas	- 74
Cavernous Hemangiomas	- 62
Hemangiopericytoma	- 1
Optic nerve gliomas	- 21
Hamartoma	- 1
Lacrimal glandula tumors (mixed tumors, cylindromas, ca. etc.)	- 35
Mesenchymal tumors (fibromas, myxomas, lipomas)	- 33
Neurofibromas	- 12
Lymphomas (9 primary low grade non-hodgkin's b-cell lymphomas after KIELER classification)	- 21
(EPI-)Dermoids	- 20
Osteomas	- 7
Rhabdomyosarcomas (children)	- 9
Malignant neoplasms (ca, sa, meta, melanomas)	- 34
Granulomas	- 12
Chronic inflammatory fibrous so-called "PSEUDOTUMORS"	- 45
Fibrous dysplasia	- 2
Aneurysmal bone cyst	- 1
Leucemic resp. leucoblastic tumors	- 2
Unclassified tumors	- 30
Inflammation processes and mucoceles	- 14
	436

from the transcranial approach in order to obtain a good view of the contents of the orbit, especially after we learned to remove more and more of the adjoining bony structures, such as the lateral wall of the orbit, the lesser sphenoid wing, the clinoid process, the floor of the middle fossa, and parts of the floor of the orbit (Fig. 5).

The operative view continued to improve steadily and the possibility of maintaining the function of important structures was also much improved over time. The removed bony parts could be reconstructed from adequate materials without difficulty. We used bone (from the

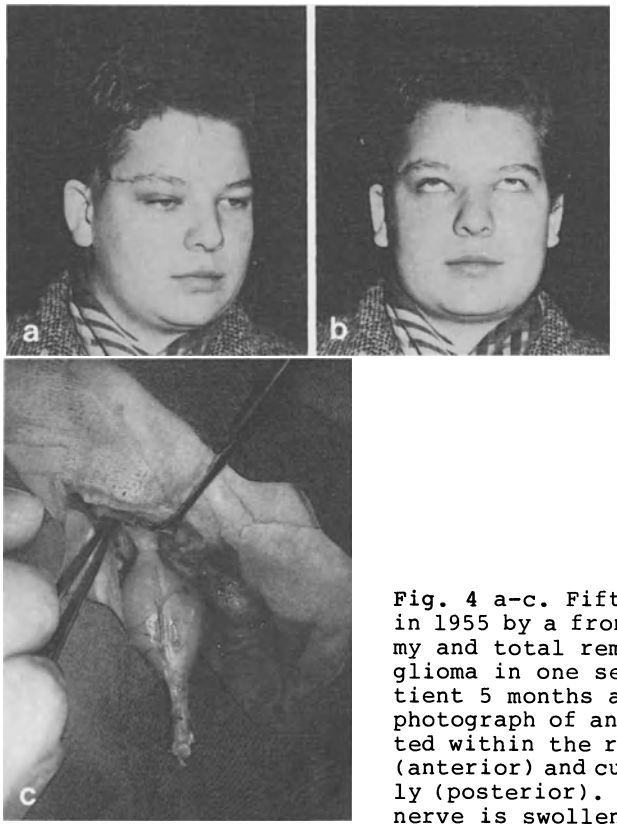


Fig. 4 a-c. Fifteen year old girl, treated in 1955 by a frontal osteoplastic craniotomy and total removal of an optic nerve glioma in one session (see text). a, b Patient 5 months after surgery. c Operative photograph of an optic nerve glioma, isolated within the right orbit between the bulb (anterior) and cut through intracanalicularly (posterior). In its middle part the optic nerve is swollen by the tumor

osteoplastic bony flap) and Palacos, which can be formed in the desired fashion.

The ophthalmologists invited me to present papers at their medical meetings and I was able to present our patient material, which increased steadily.

The nestor of ophthalmology at that time, Prof. THIEL, who was a well-versed surgeon himself, even went so far as to discourage the

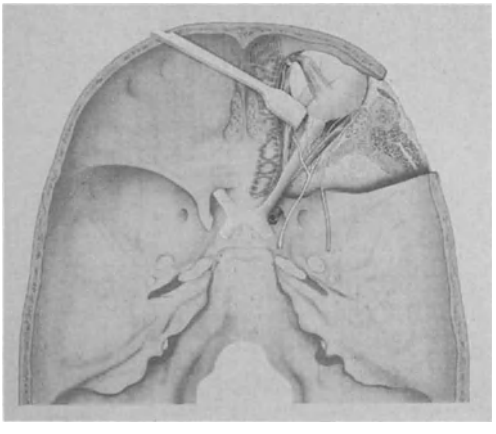


Fig. 5. Operative sketch showing the large extent of bone resection in order to obtain an excellent view of the whole orbit and its contents. This facilitates the prevention of damage to functionally important elements and structures (see text)

lateral approach by KRÖNLEIN, since the transcranial approach with all the additional resection of bony walls offered a better view for the surgeon and thus permitted a better functional result. This was very important because it left this entire area to the neurosurgeons. However, the great value of lateral osseous orbitotomý, the KRÖNLEIN operation, was not realized, even by me, until the operation was reintroduced in a modified form at a later date.

On studying the relevant literature I found the classical paper by WALTER DANDY, published in 1921, where the transfrontal approach is described in detail. Using this approach, DANDY removed an intracranial bilateral meningioma which had already broken through the orbital roof by following the tumor in the direction of the orbit after an osteoplastic craniotomy.

Although this transcranial approach to the orbit was already described in 1921 for the removal of retrobulbar tumors, a number of serious complications, such as purulent meningitis, cavernous sinus thrombosis, and (to quote literally) "lethal hazards" ruled out use of this elegant method for a long time. Only with the beginning of the antibiotic era after the Second World War was the transcranial

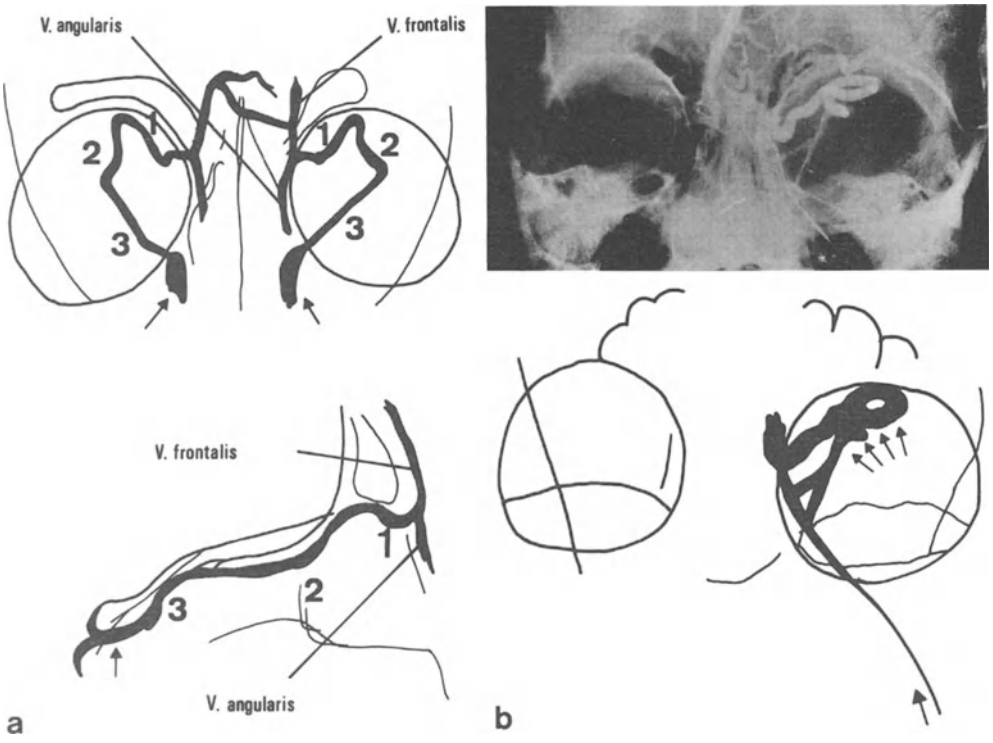
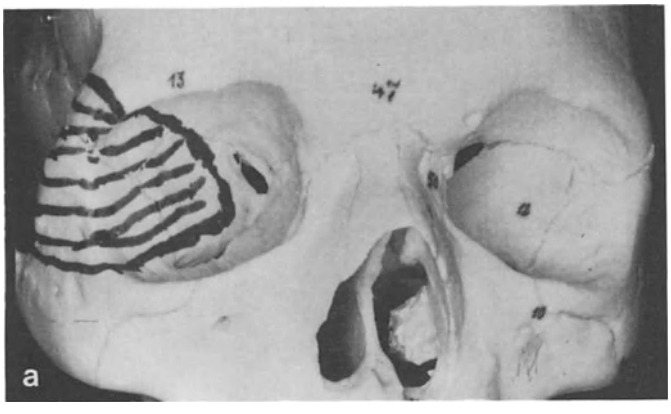


Fig. 6 a, b. Orbitophlebography by puncture of the vena angularis or vena frontalis, in order to visualize the course of the superior orbital vein: a The normal course of the superior orbital vein (a.p. view above, lateral view below). b Space-occupying lesion within the dorsal tip of the left orbit, causing compression, dislocation, and accumulative dilatation of the posterior part 3 of the vein near the entry zone into the cavernous sinus

approach occasionally used. In Mainz we did not have the above-mentioned difficulties because we used an adherent galea-periosteal flap to cover the sphenoid cells or the frontal sinus if they were opened during the procedure.

A further step forward on the road of orbit surgery was placed at our disposal by the neuroradiologists - in Mainz by Professor WENDE - and that was the introduction of phlebography of the orbit, because it permitted the visualization of the superior ophthalmic vein, whose specific displacements permitted us to determine the position of intraorbital lesions much more exactly than before. (Fig. 6). Through this method it became possible to group the intraorbital lesions according to quadrants and thus to modify the operative approaches accordingly. This also led to reconsideration and reintroduction of old and almost forgotten procedures, including orbitotomia simplex, introduced by KNAPP in 1874 (beginning with an eyebrow incision to approach lesions in the anterior part of the orbit), and orbitotomia ossea lateralis according to KRÖNLEIN, which was first performed in 1886.

Depending on the location and extent of a lesion, these approaches were then modified and enlarged. We ourselves were subsequently surprised by what these low-risk procedures offer in comparison to craniotomy if they are sensibly employed and adjusted to the individual case (Fig. 7).



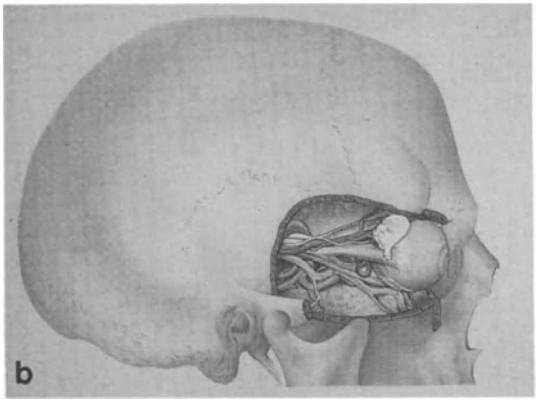
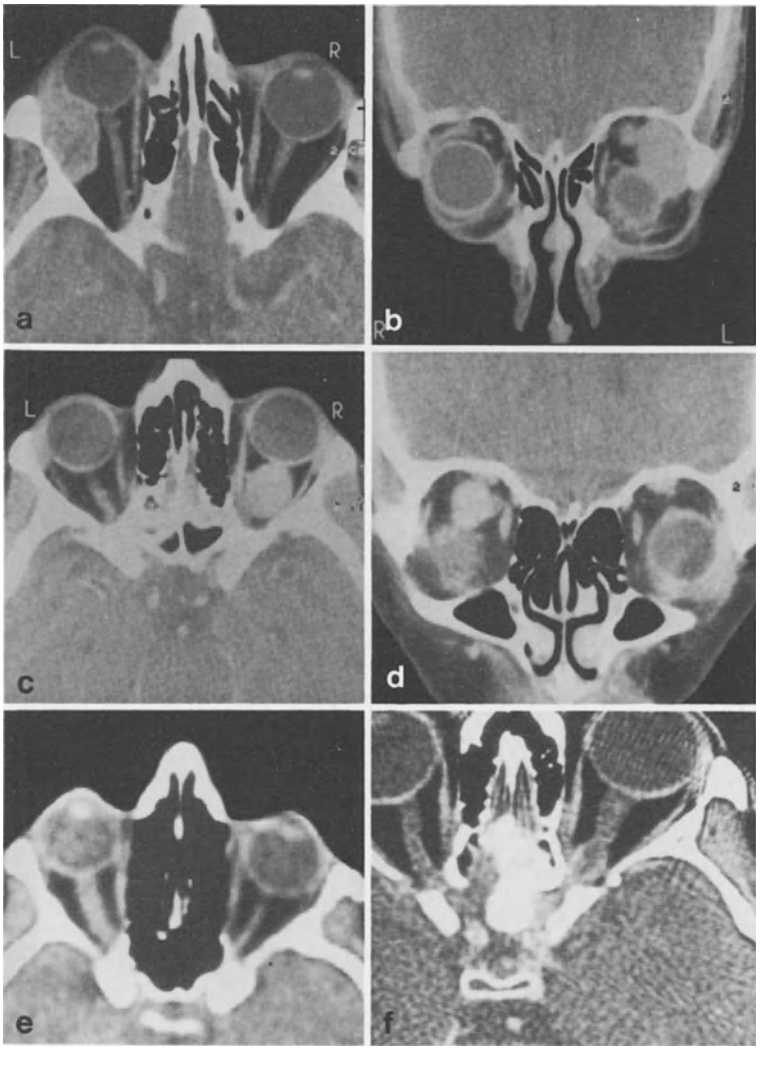


Fig. 7 a, b. Extended lateral osseous orbitotomy in order to reach the dorsal orbital cone. a Stripped bone has to be resected. b Not only the lateral wall of the orbit but also the roof and in part the sphenoid ridge can be resected, if necessary

A further "key experience" - at least for me personally - was the introduction of computer tomography in the mid 1970s. From this point in time the location and extent of the lesion, extension beyond boundaries, bony invasion, and other factors were so clearly delineated - especially with the use of the axial and coronary sections, as well as so-called bone resolution - that it became to possible to combine all the possible approaches to the orbital contents very simply. A few examples will serve to demonstrate this (Fig. 8): This method made it possible to plan the approach to the orbit completely individually depending on the circumstances of each case. The strategy and technique of operations received a new, hitherto impossible stimulus! Furthermore, this development was promoted by constant anatomic and microanatomic studies by and together with our good friend Prof. JOHANNES LANG in Würzburg, so that we were able to improve our operative results even further in respect of radical removemal with complete healing and maintenance of function.



The details of these studies cannot be described here, but they dealt with topographic relationships of the optic nerve and its coverings, its vascularization, and many other aspects. At any rate, they were extremely helpful in our efforts to maintain the function of the orbital contents at surgery. Figure 9 shows an example of such a lovely anatomic study which JOHANNES LANG provided us with (Fig. 9).

Let me now present our patient material and the operative techniques, which were employed according to the specific requirements of each case (Figs. 10, 11).

Ladies and gentlemen, in closing let me emphasize once more that the goal of our efforts in orbital surgery is and will continue to remain the complete removal of a space-occupying lesion and the maintenance of the important nerves and structures at the same time. We have come closer to this goal and yet we are at the beginning of a development which looks most promising.

With this brief address, I hope to have demonstrated that it is most worthwhile to concentrate on a small area of neurosurgery - surgery of the orbit - which was long considered to be of minor significance. You will forgive me for doing this from a subjective viewpoint, but I think my personal experience should be made available so that the younger ones among you may be motivated to draw conclusions for your own activities.

Fig. 8 a-f. Space-occupying lesions of the orbit, identified by cranial CT. a 71 year old male with a mixed lacrimal gland tumor on the left side. b The same patient; the tumor is to be seen in the superior ventrolateral quadrant, coronal section. In this case an orbitotomia ossea lateralis after KRÖNLEIN would be the optimal approach. c 40 year old female patient with a well delimited cavernous hemangioma in the right orbit, located retrobulbar within the muscle cone. The m. rectus medialis and m. rectus lateralis are well demonstrated. d The same patient in whome the coronal section shows very well that the tumor mass is located in the superior part of the orbit. The mm. rectus superior and levator palpebrae are to be seen superior to the tumor mass; the m. rectus lateralis is to be identified laterally, and the m. rectus medialis medially from the tumor mass. In this case a frontal osteoplastic craniotomy was the preferential approach, after which the patient recovered without any functional deficit. e 51 year old female patient with an optic nerve sheath meningioma on the left, which has an extension from the bulb to the optic nerve channel. Visual function less than 50%; therefore total tumor removal by an osteoplastic craniotomy with opening of the optic nerve channel was performed (see also Fig. 13). f 48 year old female patient with an optic nerve sheath meningioma on the right, located within the dorsal orbital tip and intracanalicularly. Visual function less than 20%. Total removal of the tumor was performed by a frontal osteoplastic craniotomy with opening of the optic canal, resection of the clinoid process, and prechiasmal resection of the tumor-bearing optic nerve

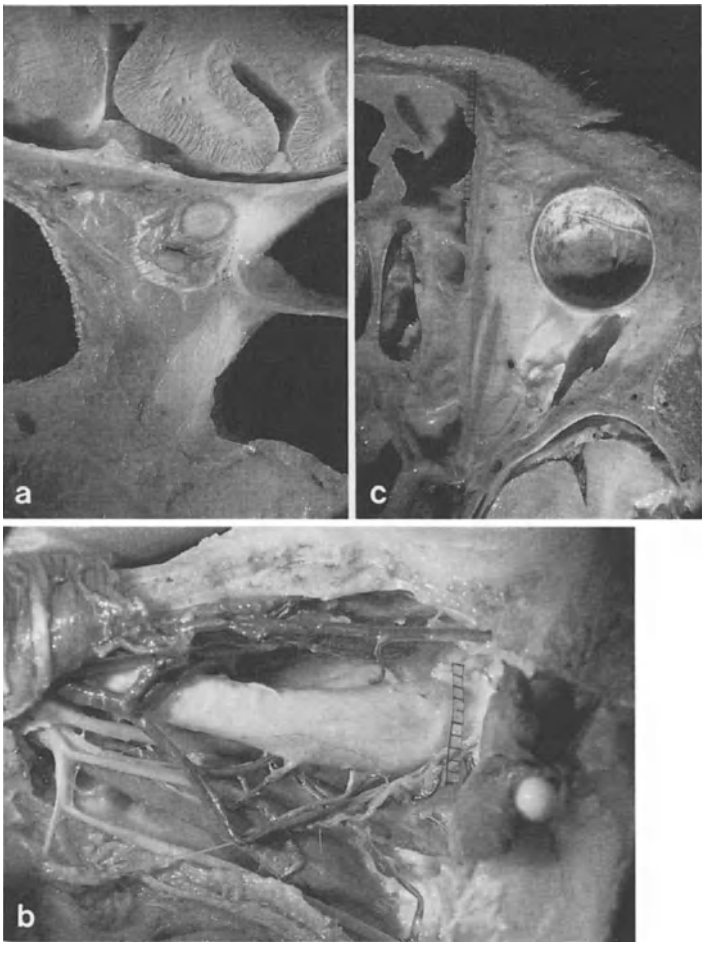


Fig. 9 a-c. Anatomical preparations by JOHANNES LANG. a Coronal cut through the dorsal tip of the right orbit, which shows the compact and close relations between the optic nerve (above), the ophthalmic artery (middle), the oculomotor nerve (below), the trochlear nerve (medial), and the muscles. b Lateral view of optic nerve from the right side. From the left the ophthalmic artery comes out of the canal and some of its branches enter the optic nerve from below. c Horizontal cut of the right orbit, which shows the compact location of the optic nerve and muscle cone within the dorsal orbital tip. Ventromedial to the bulb the trochlea is to be seen. In the case of an ORBITOTOMIA SIMPLEX SUPERIOR MEDIALIS the trochlea is temporarily detached, and refixed after tumor removal (see Fig. 10 b)

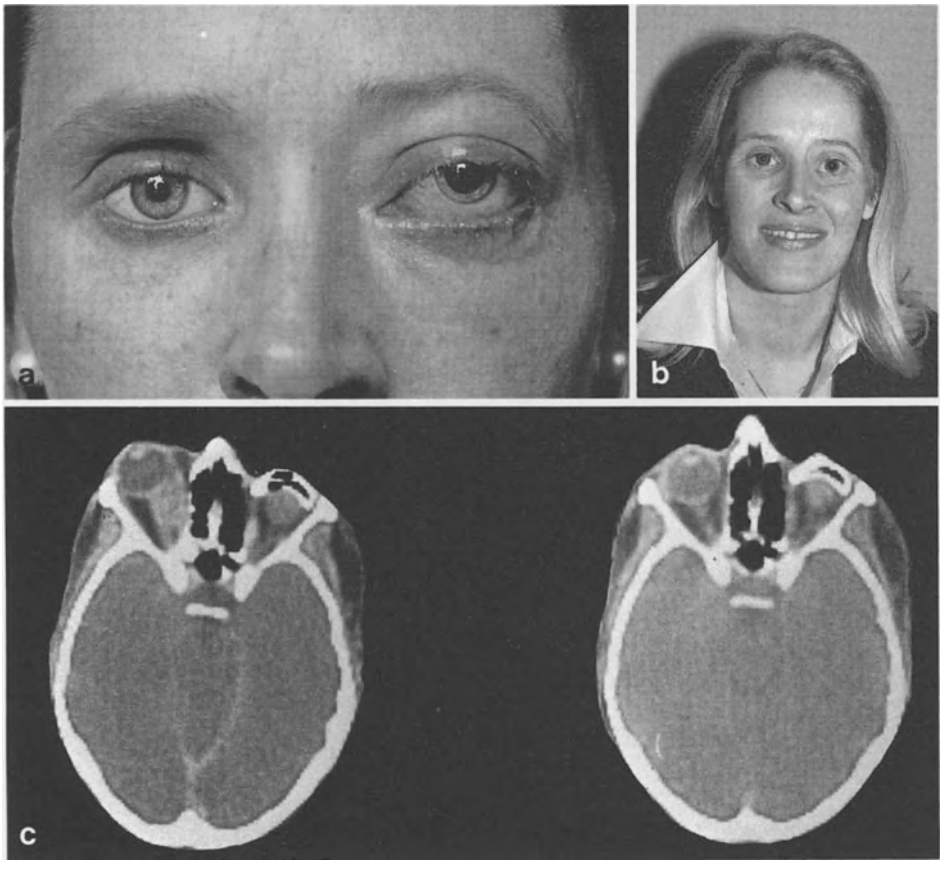


Fig. 10 a-c. Thirty-nine year old female a metastasis of a melanoblastoma within the left orbit, localized in the medial and basal part up to the dorsal cone. Nine years previously enucleation of the right bulb was performed because of a retinomelanoblastoma. **a** Patient before operation: right = ocular prosthesis; left = exophthalmos, superior and lateral dislocation of the bulb. Visual and motility almost normal. **b** Patient 6 months after total removal of the left intraorbital metastasis by an ORBITOTOMIA SIMPLEX MEDIALIS BASALIS. No dislocation of the bulb; visual and motility function almost normal. Patient survived approximately 4 years. **c** Preoperative and postoperative axial CT scans demonstrate the good surgical result

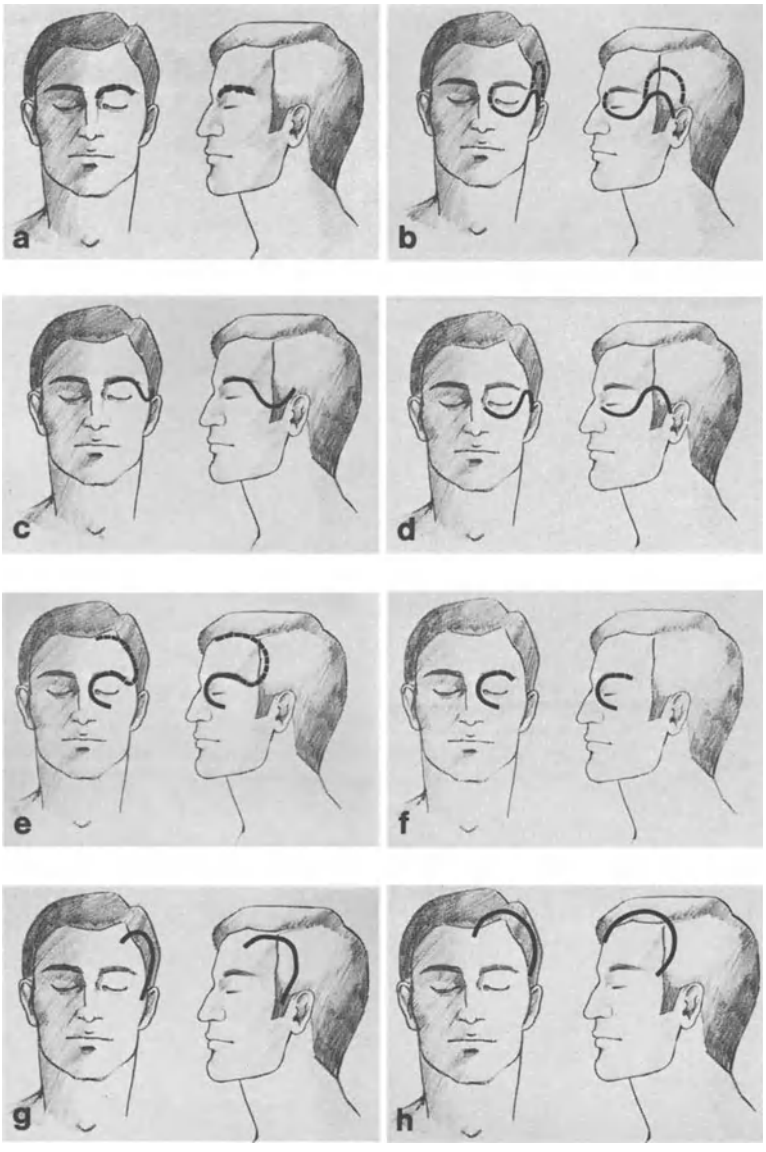


Fig. 11 a-d. Various approaches to the orbit; a orbitotomia simplex superior (extension to lateral or medial is possible); b orbitotomia simplex inferior (basalis), extended to orbitotomia ossea lateralis, and/or lateral craniotomy; c orbitotomia simplex superior, extended to orbitotomia ossea lateralis; d orbitotomia simplex inferior (basalis), extended to orbitotomia ossea lateralis. e-h Various osteoplastic approaches to the orbit; e orbitotomia superior medialis, extended to orbitotomia ossea lateralis and craniotomy; f orbitotomia simplex medialis, extended to superior; g orbitotomia transfrontalis lateralis; h orbitotomia transfrontalis medialis

Head Injuries – Long-Term Results – Prognosis

Long-Term Outcome After Severe Head Injury in Children and Young Adults

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Introduction

Accidents continue to be the major cause of childhood mortality and morbidity. Between 1000 and 1500 children are victims of fatal traffic accidents in Germany every year [9]. On the other hand clinical experience indicates that children recover from head injury better than do adults. They also show an advantage in terms of survial rate and long-term outcome. The following study reflects the experiences of our clinic in the management of head injuries in children.

Patients and Methods

This series consisted of 105 children under 16 years old who had sustained a severe head injury with posttraumatic coma lasting at least 24 h. To fulfil the conditions of coma the patient had to show the following pattern of symptoms: The patient's eyes were continuously closed and opened neither on command nor on application of nociceptive stimuli [3]. For the evaluation of severity of clinical

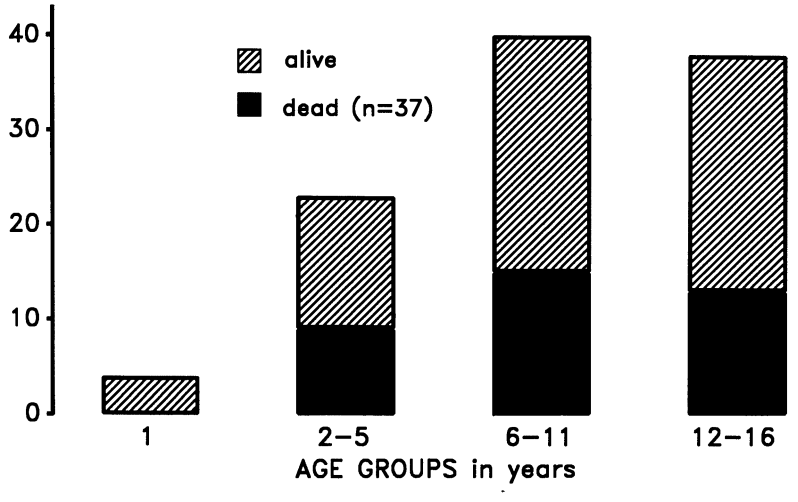

Fig. 1. Distribution of 105 children with severe head injury according to age group

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coma we used the classification proposed by the Neuro-traumatology Committee of the WFNS in Brussels in 1976 [2]. Over a period of 8 years we treated 74 male and 31 female children with a coma corresponding to the conditions mentioned above.

Figure 1 shows the sizes of the age groups. As is to be expected, the majority of victims of the 93 traffic accidents were older children. In ten cases a fall was the cause of the head trauma. Fourteen patients presented open head injuries.

Evaluation of long-term outcome was performed using the Glasgow Outcome scale, which was proposed for the first time in 1975 [5]. The nonsurvivors were classified as stage 1, and patients in a vegetative state as stage 2. Stage 3 referred to severely disabled patients who needed support for daily performances.

Stage 4 signified disabled patients who, in contrast to the former group, were independent and able to provide for themselves. The condition for the assignment of stage 5 was fulfilled if the patient reached the same level of efficiency as before the trauma. Minimal neurological deficits were no reason for excluding children with these symptoms from the best group, stage 5.

Of the 68 survivors, 46 could be followed up over a period of 1 to almost 8 years (Fig. 2). The remaining 22 surviving patients were lost to follow-up. The frequency of severe coma states and the duration of post-traumatic unconsciousness were similar in these two groups.

Results

Of the 105 patients, 37 did not survive their head injury; almost 50% of the latter group died within the first 48 h after admission. The primary damage to the central nervous system remained the limiting factor in the further course of the 19 patients dying at a later time.

Secondary complications occurring in patients with a stable vegetative state, lasting between 3 weeks and 19 months, significantly affected the longer courses only. Sepsis, failure of the rigt heart, and peracute pneumonia were the most frequent causes of delayed death.

There was a proportional relationship between the primary damage to the central nervous system and the duration of posttraumatic unconsciousness. Thirty-one patients or 46% of the survivors were

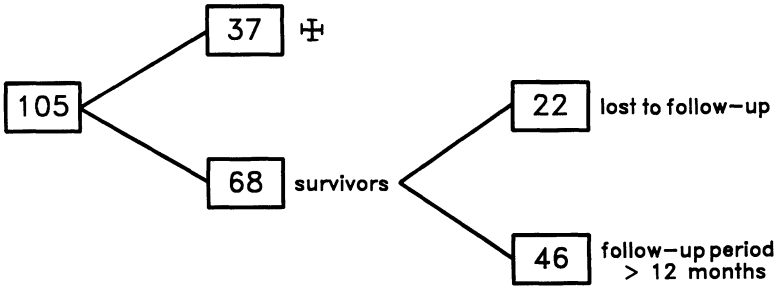

Fig. 2. Long-term outcome in the 105 comatose head-injured children

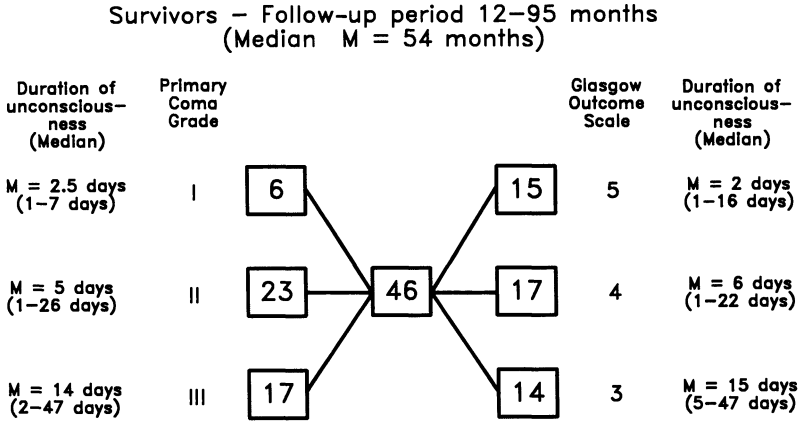

Fig. 3. Left: Primary coma grade of the follow-up group with corresponding duration of unconsciousness. Right: Long-term outcome (Glasgow Outcome Scale) at least 12 months after trauma with corresponding duration of unconsciousness

unconscious for more than 7 days. With worsening state of coma there was a decreasing number of short-term comatose patients and an increasing rate of long-term unconsciousness and death. No children admitted in coma stage IV with flaccidity of muscle tone and wide, nonreacting pupils survived their injuries.

The more extensive the trauma, the higher was the incidence of associated injuries. There were almost twice as many fractures of limbs and five times as many abdominal injuries in coma stage III/IV as in coma stage I/II. But such injuries never definitively determined the course or the fate of the injured patient.

As in the nonsurvivors, the recovery and the long-term outcome of the surviving children were mainly influenced by the initial or secondary damage to the central nervous system. In Fig. 3 primary coma grade and long-term outcome are shown in relation to duration of unconsciousness. The median duration of unconsciousness in the 23 patients in coma grade II was only 5 days, while that in the 17 patients with an initial coma grade III was more than twice as long (14 days). Correspondingly, 14 severely disabled children (Glasgow Outcome Scale, stage 3) had survived coma lasting more than twice as long (median, 15 days) as that in the 17 patients classified as stage 4 on the Glasgow Outcome Scale (median, 6 days).

Discussion

No children with a primary coma grade IV (Table 1) for more than 4 h survived. This corresponded to the results in series of head-injured adults [3]. The majority of these severely injured children died in the first 48 h. Almost half the children with coma grade III died, but the courses were generally longer. Only one patient in coma grade II did not survive. At the time of the follow-up examination no patient was in a vegetative state.

All head-injured children with a primary coma grade I or II who were not unconscious for longer than 48 h recovered very well and reached the pretraumatic level of efficiency or were only slightly disabled

Table 1. Dependence of Glasgow Outcome Scale on duration of unconsciousness and primary coma grade. Survey of all 46 analyzed patients

Coma grade, Brussels	Glasgow Outcome Scale 1	2	3	4	5
I				▲▲	■■■ ▲
II	▲		▲ ●●●	■■■ ▲▲▲ ●	■■■■■ ▲▲ ●
III	■■■■■ ■■ ▲▲▲▲▲ ●●●●		▲▲ ●●●●●	▲▲ ●●	■ ▲ ●
IV	■■■■■ ■■■■■ ■■■■■ ▲ ●●●●				

Duration of unconsciousness:

■ < 2 days
▲ 2-7 days
● > 7 days

(Glasgow Outcome Scale, stages 4 and 5). With increasing duration of coma proceeding from the same coma grade (I or II), the long-term outcome worsened. Most of the children who had an initial coma grade II and only reached a long-term outcome of stage 3 were unconscious for more than 7 days. This was emphasized in the children with coma grade III, of whom 80% remained in coma for more than 7 days. On the other hand an exceptionally good recovery - reaching stage 5 - was possible despite an initial coma grade III and unconsciousness for 15 days.

Posttraumatic long-term epilepsy was described in 17% of the patients with good recovery and in 30% of the severely disabled ones [6]. In our series only 5 of 32 children with stage 4 or 5 outcome showed this posttraumatic sequel.

For the evaluation of the long-term outcome, the pretraumatic neurological state has, of course, to be taken into consideration. In other series 20% of the injured children showed pretraumatic behavioral disturbances and up to 30% presented mild or more extensive cerebral impairments before trauma [7]. Of the 42 children in our follow-up group, only five presented pretraumatic disturbances like the above mentioned. On the other hand the validity of our findings is restricted because they are based on catamnesis only.

In our series the mortality was 38%. On the other hand the long-term outcome was more favorable in the children than in older patients. Of adults over 50 years old, 95% died after unconsciousness lasting 7 days following a head injury [3]. Moreover, only 5% of patients aged 20-30 years survived a coma of 15 days', duration [3], whereas 21% of the children in our study did so. Of the survivors, 69% reached the same level as before the trauma or were only slightly disabled. The remaining 31% were severely disabled, needing daily support.

However, differences in pathophysiological mechanisms and in the site of lesions may explain the better results of head-injured children. This is true for both the survival rate and the long-term outcome. Thus brain stem disorders and diffuse brain swelling are more frequent in children, while parenchymal lesions, particularly superficial contusions, are rare in children compared to adults [2]. The extent of cerebral dysfunction is distinctly reflected by the depth of initial coma following a head injury [1,4].

It is important to predict the outcome of comatose head-injured patients as early as possible, and this is especially true for children. For the rare courses of good recovery in children who initially present a poor coma grade and a long duration of coma, application of all reasonable measures of nursing and medical care is justified.

Summary

One hundred and five head-injured children up to 16 years old were considered; they were admitted to our clinic over a period of 8 years and remained in a comatose state for at least 24 h. According to the severity of the initial injury, 37 patients died, mainly in the first 48 h after trauma. Of the 68 survivors, 46 could be examined over a follow-up period ranging from 12 to 95 months (median: 54 months). We could reveal that there was a distinct relationship between the long-term outcome on the one hand and both the extent of primary coma and the duration of posttraumatic unconsciousness on the other. One-third of the follow-up group recovered well and reached the same level of efficiency as before the injury.

Furthermore 36% were able to provide for themselves and were independent despite their slight disability. Of the examined patients, 31% presented such severe mental and physical deficits that they were dependent on daily support. This study proved that permanent neurological and physical disabilities following severe head injuries are influenced by the initial coma state, the duration of posttraumatic unconsciousness, and the age of the young patients.

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Outcome After Severe Head Injury with Midbrain Syndrome in the Acute Stage

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The acute midbrain syndrome (GCS 4+5) [18] (WFNS coma scale III) [4] is part of the most severe form of central dysregulation from which recovery is possible [9]. In patients with a midbrain syndrome (MS) due to severe head injury we were able to work out prognostic factors by correlating clinical course and computer tomographic findings over 7 years [13,14]. The aim of the present study was to investigate the long-term morbidity and functional recovery of patients who suffered from an MS during the acute stage of a severe brain injury.

Material and Methods

Between 1978 and 1983 1053 patients with cerebral trauma were treated in the Department of Neurosurgery, Gießen. Two hundred and thirty-two presented signs of decerebrate postures, of whom 80 (35%) survived. In collaboration with the family doctors, the health insurance companies, and the workmen's compensation insurance scheme, as well as the patients themselves, we managed to reinvestigate 30 of them clinically.

In another five cases adequate information could be obtained from other departments, including rehabilitation centers. Besides a physical examination, electroencephalography (EEG), measurement of evoked brain stem potentials and brain stem reflexes, computerized tomography (CT), and magnetic resonance tomography (MRT) were performed. In 32 patients we employed a psychometric test battery including the HAWIE test [10], the d_2-**Aufmerksamkeitsbelastungstest** [3], the **Freiburger Persönlichkeitsinventar** [6], and the Benton test [2].

Results

Patients

The average age of the 35 patients (28 male) was 21.7 ± 10.9 years with a range from 8 to 57 years. In four cases a compound head injury was found, while 31 revealed a closed head trauma. On admission 28 patients were graded GCS 4+5, five GCS 6+7, and two better than GCS 10. Seven patients who presented in a rather good neurological status on admission developed an MS within the first 3 days of their clinical course. The initial CT revealed a contusion in ten cases, an epidural hematoma in six, and a subdural hematoma in

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Table 1. Duration of mesencephalic syndrome in relation to outcome (GOS)

	<6 h	7-24 h	2-9 days	>9 days
No.	8	6	7	14
GOS 2 PVS				
3 SD		2	1	6
4 MD		1	3	5
5 GR	8	3	3	3

<1 day/>1 day: P = 0.0133
PVS, persistent vegetative state; SD/MD, severe/moderate disability; GR, good recovery

two. Ten patients showed combined lesions, and in seven no space-occupying lesion could be detected.

The mean follow-up period was 5.7 ± 2.2 years (range 1-9 years). Recovery was graded as good (GOS 5) in 17 cases; nine patients presented as moderately and nine as severely disabled. Eighteen patients regained working ability, six of them in their own profession.

Correlations

There was no correlation between GOS and CT lesions. On the other hand those patients who showed decerebrate postures for less than 24 h had a significantly ($P < 0.05$) better outcome (Table 1).

Performance IQ was significantly better ($P = 0.02$) when the duration of MS was less than 9 days. The short memory capacity was not related to the duration of the MS.

The intellectual capacity and ability to understand complex situations were significantly ($P < 0.05$) better in those patients with better recovery. GOS was correlated to FSIQ ($P = 0.0152$), PIQ ($P = 0.0081$), and d_2-Aufmerksamkeitsbelastungstest ($P = 0.0287$). It was not correlated to the VIQ and the Benton test (Table 2).

Discussion

Our results showing no correlation between outcome after severe head injury and detection of lesions by CT scan were confirmed by the data of BUSCH [5]. MRT, however, seems a lot more sensitive in terms of unmasking minor abnormalities, so it must be taken into account in further investigations [15]. In terms of survival, our data are comparable to those in studies performed by FROWEIN [7,8] and Mahapatra [16]. Nevertheless, the proportion of cases presenting a more favorable outcome seems slightly higher in our group. This might be due to a longer time interval between trauma and reexamination. In addition, less than 50% of the surviving patients could be investigated.

Table 2. Correlation between GOS and psychometric tests

	HAWIE			d_2	Benton
	PIQ	VIQ	FSIQ	GZ-FSW	ERW-L-C
GOS 3 SD	79.7	95.1	87	79.5	-2.3
4 MD	92.3	95.3	93.6	95.1	-2.6
5 GR	105.9	103.5	105.2	100.1	-1.7
P	0.0081		0.0152	0.0287	

PIQ, performance IQ; VIQ, verbal IQ; FSIQ, full scale IQ; GZ-FSW, total number - fault-standard; ERW-L-C, solution expectation form C

The value of using a psychometric test battery in patients who have survived a severe head injury is still controversial [11,17,19]. However, we believe it to be an important tool to detect dysfunctions which might be underestimated in a general clinical examination.

Summary

In this study we found correlations between the duration of MS and outcome. Outcome evaluated with the GOS was significantly correlated with FSIQ, PIQ, and the d_2 test. PIQ was correlated with the duration of MS.

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Comparison of Magnetic Resonance Imaging, X-ray Computed Tomography, Electroencephalography, and Long-Term Outcome After Head Injury: A Prospective Reexamination of 55 Patients[1]

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Introduction

Some authors judge magnetic resonance imaging (MRI) to be a more sensitive method than high resolution X-ray computed tomography (CT) for the assessment of brain pathology after head injury [1,4,5,10-15, 19-24,27,29,31,33-36]. Literature dealing with the artifacts and shortcomings of MRI remains an exception [9]. These publications lead to several questions: Which correlations exist between MRI and CT in the posttraumatic pathology of the brain? What kind of artifacts should one be acquainted with? Does a restitutio integrum after any posttraumatic coma still exist? And is the concept of "commotio" [3,18,25] of further use? Therefore, we reexamined some of our patients by a modified MRI technique [6].

Patients and Methods

During December 1987 we reexamined 55 slightly to severely head-injured patients (44 men, 11 women). The mean follow-up at that time was 16 months (range: 0.5-120). Criteria for Selection were: history of posttraumatic coma or deep drowsiness with inpatient treatment, initial CT, willingness to participate, and no contraindications to MRI [31]. The mean age at the time of accident was 30 years (range: 10-66), and the mean duration of coma, 7 days (seconds/minutes to 2 months; one patient who had been in a persistent vegetative state for 10 years was not taken into account, cf. Fig. 3 [32]. Twenty-eight of the patients had been treated conservatively; four had been trephined for intracranial pressure monitoring, 16 had been treated by large craniotomies; and 13 had been operated on by maxillary surgeons, four of them by teams of the two disciplines.

Both MRI (Gyroscan S15: 1.5 Tesla) and CT (Somatom DR) were performed without contrast media (scans parallel to the orbitomeatal line; thickness: infratentorial = 4 mm, above = 8 mm). For MRI a modified multislice 12-weighted gradient echo sequence with cardiac gating was used, as described elsewhere [6]. The electroencephalogram (EEG) was monitored for 20 min with hyperventilation. Clinical outcome was classified according to the Glasgow Outcome Scale [16].

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1 Dedicated to the nurses and physiotherapists who have cared for them.

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Table 1. Pathological brain imaging after head injury

No.	First CT	Control, 12/87	
		CT	MRI
23	+	+	+
6	-	-	-
9	+	-	+
6	+	+	Artifact
3	+	-	Artifact
5	+	-	-
3	-	-	+

Results

The follow-up was 100% for clinical and CI data, and 84% for EEG data. MRI was now performed for the first time, and was utilizable in 95% of patients (three of whom were claustrophobics).

The clinical outcome was as follows: good recovery, 22; independent but disabled, 28; conscious but dependent, 4; vegetative state, 1 [32]. Three patients still suffered from posttraumatic epilepsy. The development of EEG alterations was as follows: normalization, 14; improvement, 23; unchanged, 6; worsening, 2; not comparable, 9. An estimated comparison of the parenchymal pathology (yes/no/artifact = +/-/?) in CT (on admission/early controls = A, current = B) and MRI (= C) by one of us (DM, cf. Table 1) yielded: 23 A+B+C+; 6 A-B-C-; 9 A+B-C+; 6 A+B+C?; 3 A+B-C?; 5 A+B-C-; 3 A-B-C+ (very early initial CI and comparatively late control, cf. Fig. 1). With the MRI sequences used, artifacts (mainly "black spots") were seen in regions of extreme air/bone contrast (frontobasal, temporobasal, paramastoidal), in the neighborhood of calcifications, and as a result of abrasions of paramagnetic material (cf. Fig. 2) mainly but not exclusively from trephines and saws.

Discussion

As compared to the cited literature, this study reexamined more patients after a longer mean posttraumatic period, including a precedent case of one patient with a hitherto rarely found persistent vegetative state of 10 years' duration [17,26] (cf. Fig. 3). For the rest of the patients, the clinical and electrophysiological outcome was relatively fair, especially for single patients (e.g., independence after an 18-day coma with fixed bilateral mydriasis for 3.5 days, and independence after 2 months of a transient "vegetative state"). The pure technical and morphological data of the MRI of this study are described elsewhere [7]. As a rough guideline for interpreting the MRI scans, a bright appearance in these sequences disclosed cerebrospinal fluid, demyelination, or parenchymal cicatrices, while darkness represented regions of lowered or extinguished signals. This is the case for air (e.g., paranasal sinuses), calcifications (e.g., pineal gland, choroid plexus; cave: upper tangential cuts of the petrous bone), rapidly flowing blood ("flow phenomenon") [31], and different metallic compounds. Because of the amounts of paramagnetic substances (esp. ferric iron, Fe^{3+}) typically present in the neurons of the extrapyramidal motoric system [8,28,30], particularly the pallidum, the red nucleus, and substantia nigra appear

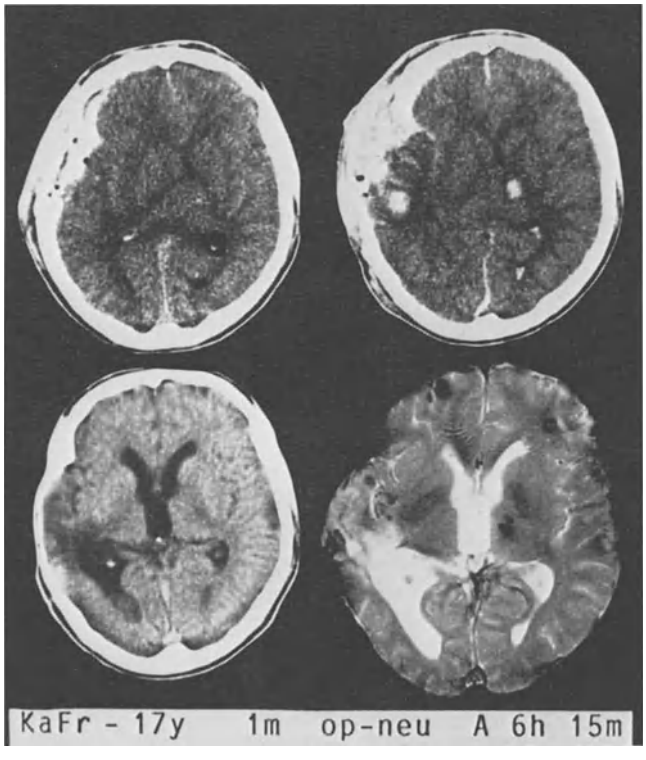

Fig. 1. Imaging of posttraumatic intracranial bleedings: acute subdural, subarachnoid, intracerebral (intraventricular). Follow-up with CT compared to MRI after 15 months. The hemorrhage of the right basal ganglia increased during the first posttraumatic hours; after 15 months the residua are less evident in CT than in MRI (**black spots**). Additional reduced signal intensities (**black**) are seen at the frontal left, in front of the right cornu anterius, and at the temporal right (residua of posttraumatic subarachnoid hemorrhage?, contrecoup?). There are no gross rub-off artifacts after craniotomy. **KaFr**, initials; 17 **y**, age at the time of accident: 17 years; **1 m**, 1 month of posttraumatic coma; op-neu, operation because of left-sided space-occupying subdural hematoma; **A**, CT on admission (upper left); **6 h**, postoperative CT after 6 h (upper right); 15 **m**, reexamination after 15 months by CT (lower left) and MRI (lower right). Clinical outcome: disabled but independent. EEG: theta-delta focus, temporal left. Note: Here, as in other figures, "patient's left" = viewer's left

dark. However, it is not yet clear under which circumstances metallic rub-off from the neurosurgical tools causes huge artifacts (cf. Fig. 2), sometimes impeding any diagnosis in the most interesting neurosurgical region [9]. At least 7 of 16 patients who underwent extensive trephination had had no abrasion artifacts, without correlation to the postoperative timespan. (Correlation with the operator's attitude toward irrigation/suction? Trials with blunt/sharp saws etc. are underway.)

The most interesting regions of altered signal intensity are those representing posthemorrhagic residua (diamagnetic: oxyhemoglobin; paramagnetic: deoxyhemoglobin, methemoglobin, hemichromes, ferritin,

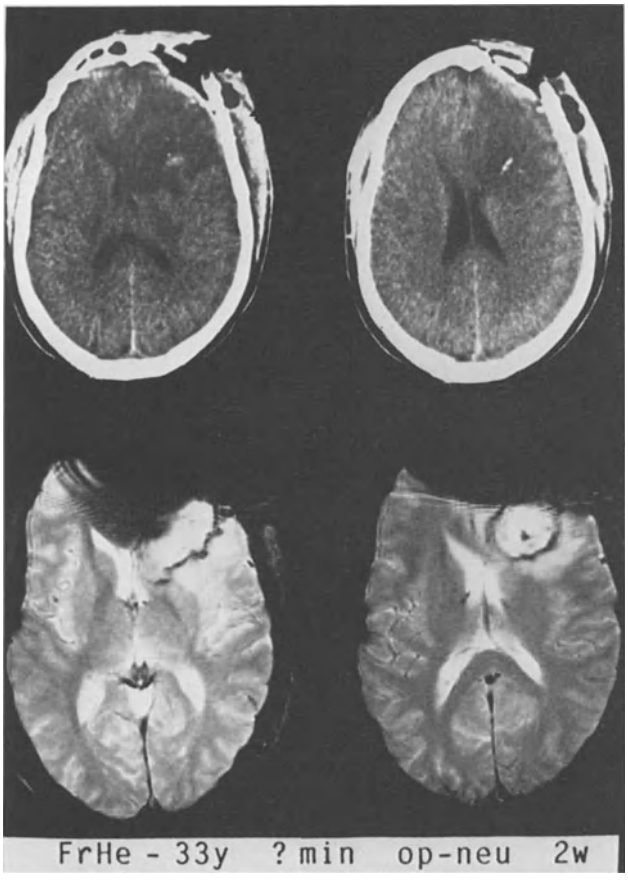

Fig. 2. CT (**above**) and MRI (**below**) 2 weeks after operative removal of a bullet (muzzle-loader accident). CT: Postoperative state. Air artifacts (sinus frontalis opened by the bullet) and subgaleal swelling (partial volume effect) are seen. The hypodensity (frontal right) may correspond to a mixture of brain edema and isodense blood residua. One bone particle (hyperdense) is left in situ. **MRI**: Despite huge bifrontal artifacts (missile lesion and trephination), a part of the CT hypodensity can be identified as a bright area including a dark spot (bone) surrounded by a dark garland. The MRI scan alone could have been misinterpreted as showing space-occupying rebleeding. **FrHe**, initials; **33 y**, age at the time of accident: 33 years; **? min**, very short altered consciousness, probably seconds; **apnea**, neurosurgical emergency removal of the bullet by flap craniotomy; **2 w**, outcome without neurological deficit at 2 weeks. EEG: pathological (theta-delta focus, frontal right)

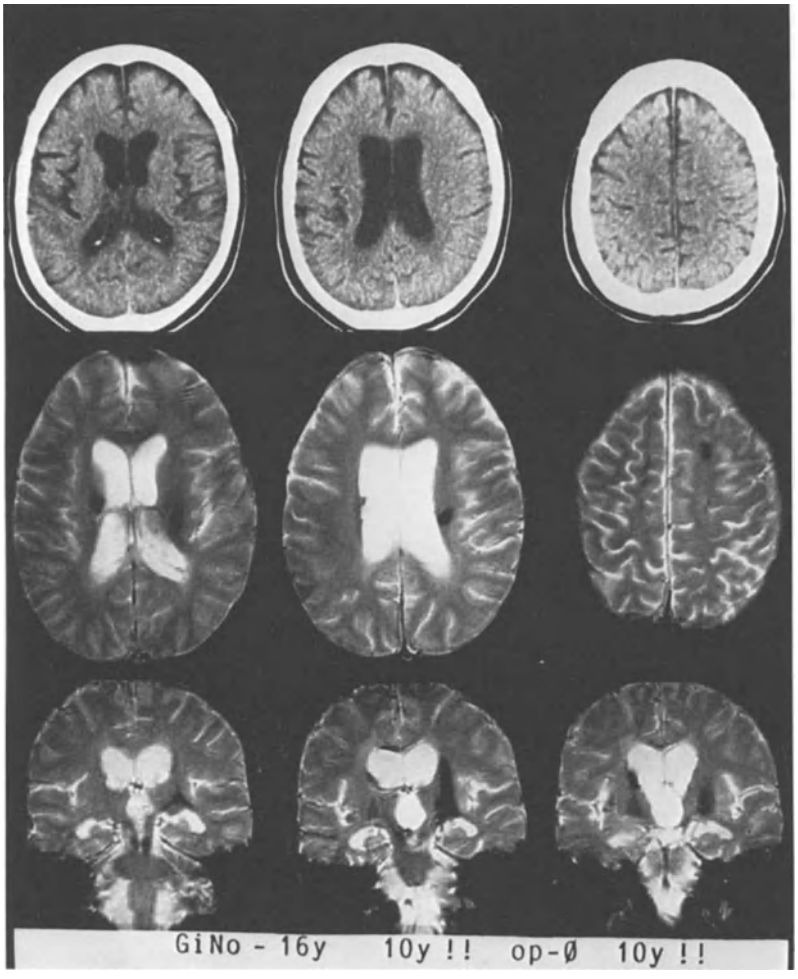

Fig. 3. CT (above) and MRI (middle/below) 10 years after closed head injury with a persistent vegetative state. CT: Widening of external and internal cerebrospinal fluid space; doubtful hypodensity of the upper left thalamus (lower scans disclose the atrophy of the brain stem). MRI: Bilateral reduced intensities: periventricular, frontal right, basal ganglia, and brain stem (coronal sections). GiNo, initials; 16 y, age at the time of accident: 16 years; 10 y, duration of apallic syndrome; op-Ø, no neurosurgical operation; 10 y, clinical outcome after 10 years: vegetative state. EEG: since March 1978 flat beta-wave variant. (From [32])

porobasal contusions). This is one of the reasons why a standardized comparison of CT and MRI has not yet been completely possible (cf. Table 1, cum grano salis). Another problem relates to the follow-up and is documented by Fig. 1: The first posttraumatic CT could have been done very early, and if the course was uneventful the first CT control may have been performed after the reabsorption of minute bleedings. The residua of these hemorrhages remain visible in the MRI, but theoretically they could have been documented at a certain

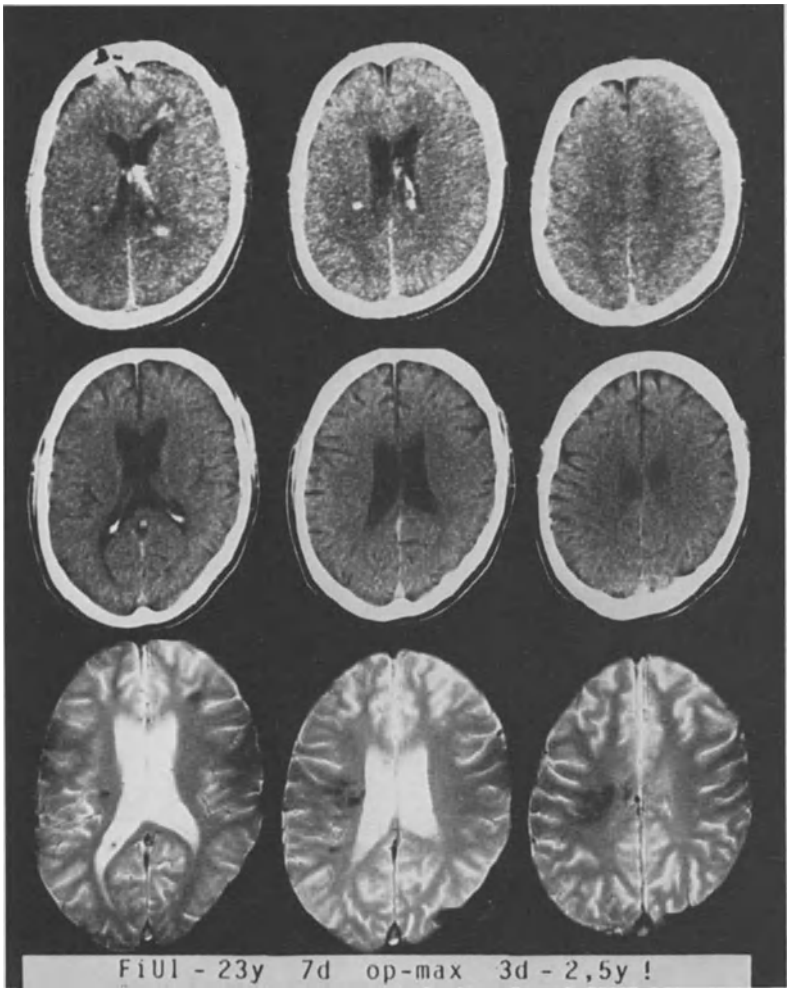

Fig. 4. Imaging of posttraumatic intracranial bleedings (intracerebral, intraventricular, subarachnoid): follow-up with CT compared with MRI after 30 months. Residua of the initial bilateral periventricular hemorrhages remain more clearly visible in the MRI than in the CT scans. Bleedings with direct contact to the cerebrospinal fluid seem to behave differently (washed out effect). **FiUl**, initials; **23 y**, age at the time of accident: 23 years; **7 d**, 1 week of posttraumatic coma; **op-max**, maxillofacial surgery because of mandibular fractures; **3 d**, CT control after 3 days (**above**); **2.5 y**, reexamination after 30 months by CT (**middle**) and MRI (**below**). Clinical outcome: good recovery. EEG: pathological with diffuse dysrhythmia

time with the CT as well. This should be kept in mind with regard to the above-mentioned categorization of "follow-up of CT: negative and MRI: positive." Overall, there is no doubt that at least nine times the MRI was able to preserve the morphology of initially CT-visible lesions which had become CT-invisible with time (cf. Fig. 4). Sometimes MRI was less specific: in our series this was especially the case for one partial infarction of the right posterior cerebral artery,

and for the morphology of CT hypodensities after emergency operations, which could have been misinterpreted as space-occupying rebleeding on the basis of the MRI scan alone (cf. Fig. 2).

Conclusion

Rather than being competitive, MRI and CT hold their own advantages in imaging brain pathology after head injury: CT remains the procedure of choice in identifying neurosurgically relevant space-occupying lesions in the acute phase in critically ill patients [2,13] and is unrivalled in the diagnosis of bony structures (especially the middle ear) as well as for claustrophobic or very agitated patients. MRI discloses a somewhat higher sensitivity with the sequence used here, though it was sometimes less specific and had a greater susceptibility to artifacts. From this study it has become evident that whenever one has to assess the outcome after head injury it is advisable to perform MRI complementary to CT. The concept of "commotio" may serve as a preliminary working hypothesis. The diagnosis of "healing without morphological residua" should be reserved for those head-injured patients with utilizable and negative posttraumatic CT and MRI.

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Longlasting Coma After Head Injury: Late Results

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In 490 survivors of severe head injury with longlasting coma, the maximal duration of coma beyond which survival was seen in only 5% of the cases decreased with age, ranging from 18 days among 10- to 20-year-olds in coma grade III to 6 days in 70-year-olds with coma grade II. Thus there was no change in the likelihood of recovery as compared to our first report in 1977, when the above-mentioned range was 20 to 5 days (FROWEIN, 1977, 1986).

In 1977 and in our current report, the maximal duration of coma allowing for complete recovery of the ability to work within 1-2 years was 4-8 days, which was distinctly shorter than for a 5% survival for the 40- to 60-year-olds. In adolescents up to 20 and young adults up to 30 years of age, the maximal duration of coma allowing for complete recovery was 12 days in 1977, while now the 5% survival limit is approached at 13-17 days of coma. When the duration of coma exceeded this limit of complete recovery, only partial or complete disability to work was achieved.

These late results are based on 339 long-term follow-ups (Fig. 1), but only a few patients recovered completely after a maximally long coma. The question is: Are these results due to variable age-related factors or to variables of the treatment? Does the term "coma" always mean the same thing?

Ability to Work and Duration of Coma

Among 339 patients with long-term follow-up, the number of survivors decreased with age and duration of coma. We therefore analyzed various degrees of recovery in relation to age and duration of coma. Several age groups and various durations of coma were distinguished to achieve sufficiently large numbers to be related to the degree of recovery (Table 1). The graphic presentation is easier to understand (Figs. 2-4). Within the various age groups the frequency of complete, partial, or no recovery again depended on age and duration of coma.

Frequency of Complete Recovery

Of 78 children between 6 and 12 years old, complete ability to attend school was achieved in 45%-65% after coma lasting up to 12 days and in 20% after coma up to 15 days (Fig. 2). Of 121 adolescents between 10 and 20 years old, complete recovery of the ability to work was achieved in 46%-50% after coma lasting up to 7 days and 15%-20% after coma up to 15 days. Of 91 adults between 20 and 40 years of age,

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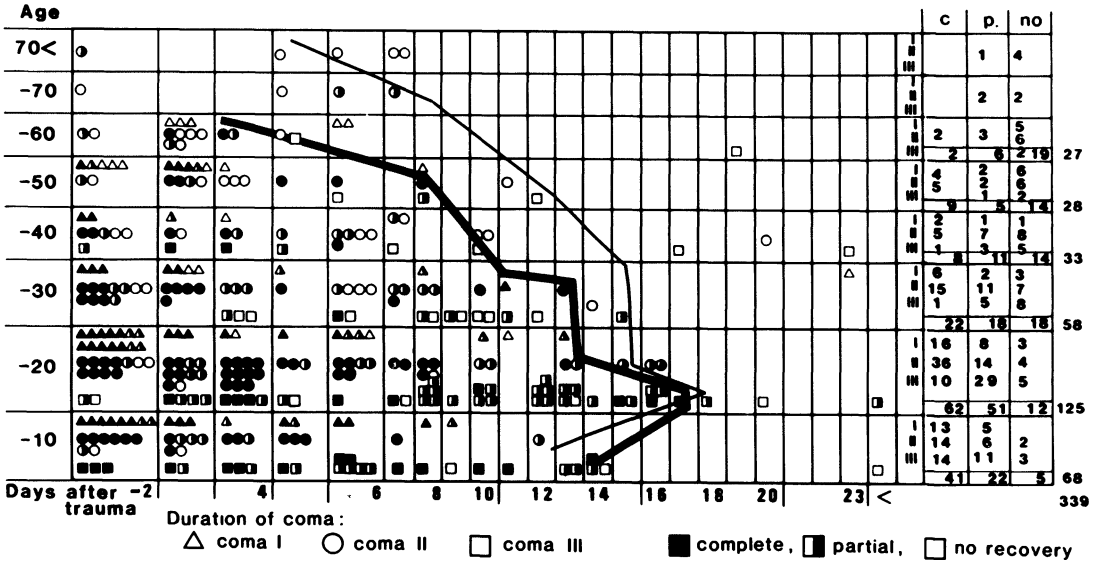

Fig. 1. Outcome of 339 survivors of longlasting coma: degree of recovery of ability to work

Table 1. Late results in 339 survivors of longlasting coma

		-2		-4		-7		-12		-15		<	
		S	%	S	%	S	%	S	%	S	%	S	%
		27		41		18		8		0		3	
	c	1	4	7	17	2	11	1	13	-	-	-	-
-50	p	4	15	4	10	2	11	1	13	-	-	-	-
	no	6	22	13	32	10	56	3	38	-	-	1	33
		S	%	S	%	S	%	S	%	S	%	S	%
		42		43		32		25		6		5	
	c	13	31	10	23	4	13	2	8	1	17	-	-
-40	p	5	12	8	17	9	28	6	24	1	17	5	100
	no	5	12	5	12	8	25	8	32	1	17	-	-
		S	%	S	%	S	%	S	%	S	%	S	%
		36		52		24		27		12		10	
	c	18	50	24	46	12	50	4	15	2	17	2	20
-20	p	5	3	8	15	6	25	16	59	7	58	6	60
	no	3	8	2	4	3	13	1	4	1	8	1	10
		S	%	S	%	S	%	S	%	S	%	S	%
		23		22		17		9		5		2	
	c	15	65	10	46	11	65	4	45	1	20	-	-
-10	p	3	13	6	27	-	35	2	22	3	60	-	-
	no	1	4	1	5	-	-	1	11	1	20	1	50

S, sum of all survivors in the particular group of age and duration of coma, comprising patients with complete recovery of ability to work (c), partial recovery (p), no recovery (no), and patients without follow-up. The patients not followed up are not listed

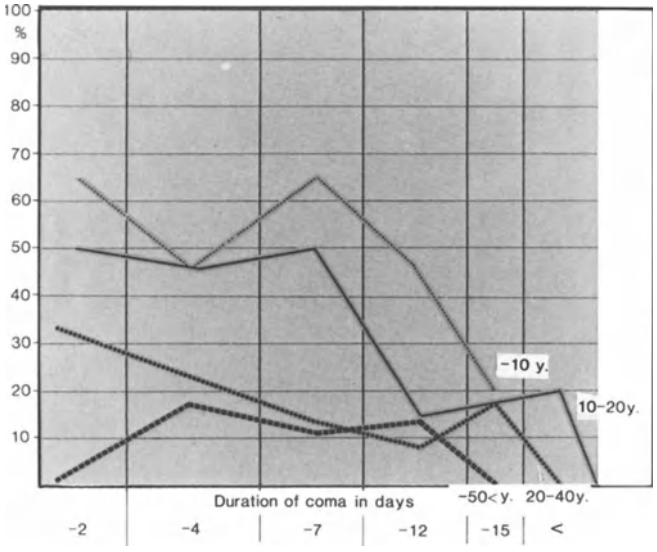

Fig. 2. Longlasting coma: complete recovery of ability to work in different age groups. The lines correspond to the percentages of cases calculated in Table 1

complete recovery of the ability to work was achieved in 31% after coma lasting up to 2 days and in 8%-17% after coma up to 15 days. Follow-up was possible in only 55 of 97 patients between the ages of 40 and 50 years; thus the statistical value was rather limited in this group. It may be noted, however, that complete recovery of the ability to work was achieved in 4%-17% after coma lasting up to 4 days and in 13% after coma up to 12 days. Thus, a distinct difference in the likelihood of complete recovery was found (a) in children and in adolescents up to 20 years of age with a maximal duration of coma of 7 days and (b) in adults. After a coma exceeding 7 days - with the exception of children - the likelihood of complete recovery amounted to 15%; it decreased with age and coma grade.

Frequency of Partial Recovery

As expected, the frequency of partial disability to work followed a reversed pattern (Fig. 3). After coma lasting up to 2 days, all age groups had similar frequencies of partial recovery, e.g., 12%-15% of the 40- to 50-year-olds and 3% of the 10- to 20-year-olds. With increasing duration of coma, the percentage of partial disability to work increased except for the highest age group, but their number was small.

Frequency of Complete Disability to Work (CDW)

As expected, the percentage of CDW was highest after the age of 40 years. CDW was found in 22% of survivors with 2 days of coma. The frequency increased with the duration of coma (Fig. 4). In the 20- to 40-year-olds, the likelihood of definite CDW (12%-32%) was somewhat lower. After coma lasting longer than 12 days CDW decreased due to the small number of cases. The frequency of persistent CDW in chil-

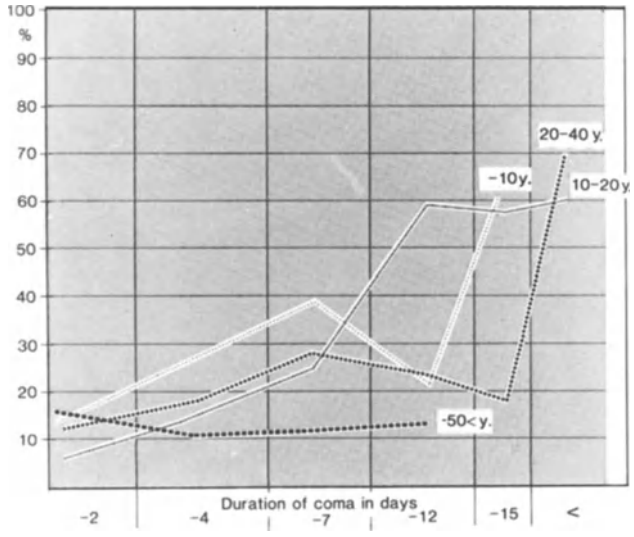

Fig. 3. Longlasting coma: partial recovery of ability to work

dren and adolescents in long-term follow-ups was remarkably low, at 4%-8%.

Thus, the frequency of no recovery, partial recovery, and complete recovery of the ability to work confirms the relation of duration of coma and age to the 5% survival limit. Although these relations appear logical, they have not yet been reported in such a consistent way in the literature.

But why is the duration of coma allowing for complete recovery so close to the 5% survival limit? Like ROBERTS et al. (1979) and JENNETT and TEASDALE (1981), we found that recovery was mainly achieved within the 1st year after the injury. Only 20% showed some

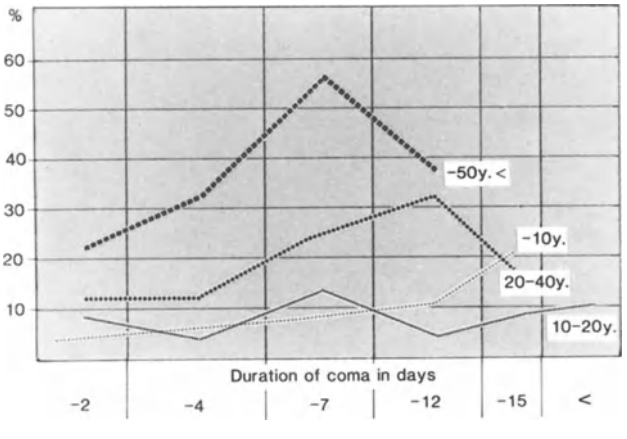

Fig. 4. Longlasting coma: no recovery of ability to work

recovery after the 1st year. Complete recovery was faster after coma grade I than after coma grades II and III (FROWEIN et al., 1986).

Variable Age; Same Duration of Coma; Good Recovery

In 35 patients with a duration of coma of 4-6 days, the time needed for recovery was related to age. The definition of the age groups was arbitrary: 1 of 7 patients between 21 and 56 years old were in coma grade III, 3 of 19 between 11 and 20 years old were in coma grade III, and 5 of 9 between 4 and 10 years old were in coma grade III (Fig. 5).

The interval between injury and complete recovery was 6 months in 48% (16 of 35) and 1 year in 31% (11 of 35). Complete recovery from coma grades I and II after 1 year was seen in 33% (2 of 6) of the 21- to 56-year-olds, 14% (2 of 14) of the 11- to 20-year-olds, and 50% (2 of 4) of the 4- to 10-year-olds. Disregarding coma grade, recovery after 1 year was seen in 29% (2 of 7) of the 21- to 56-year-olds, 21% (4 of 11) of the 11- to 20-year-olds, and 56% (5 of 9) of the 4- to 10-year-olds.

There were fewer adults than children and adolescents with complete recovery; however, if interpreted carefully, it seems their recovery

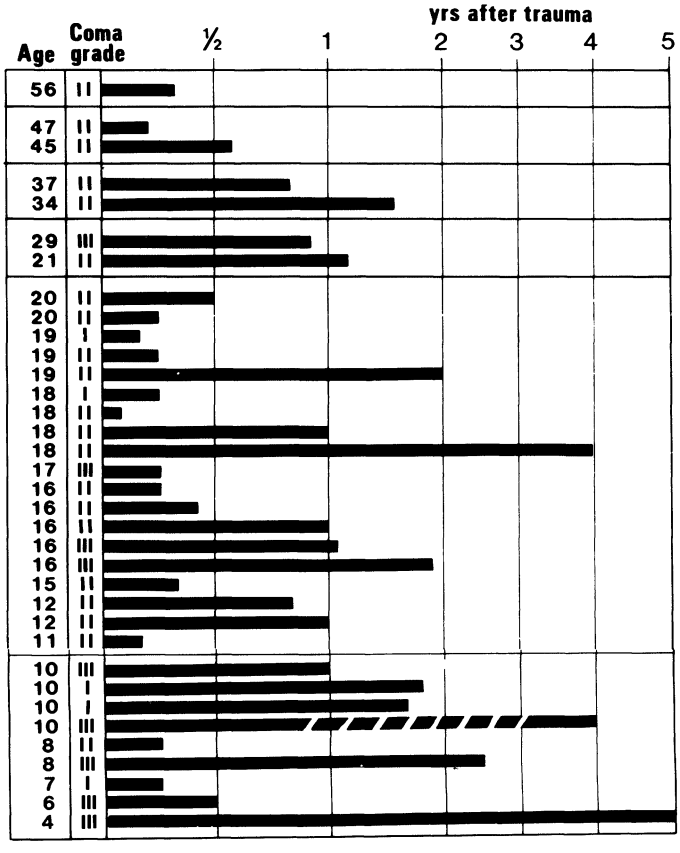

Fig. 5. Period up to complete recovery of patients of all age groups with a duration of coma of 4-6 days

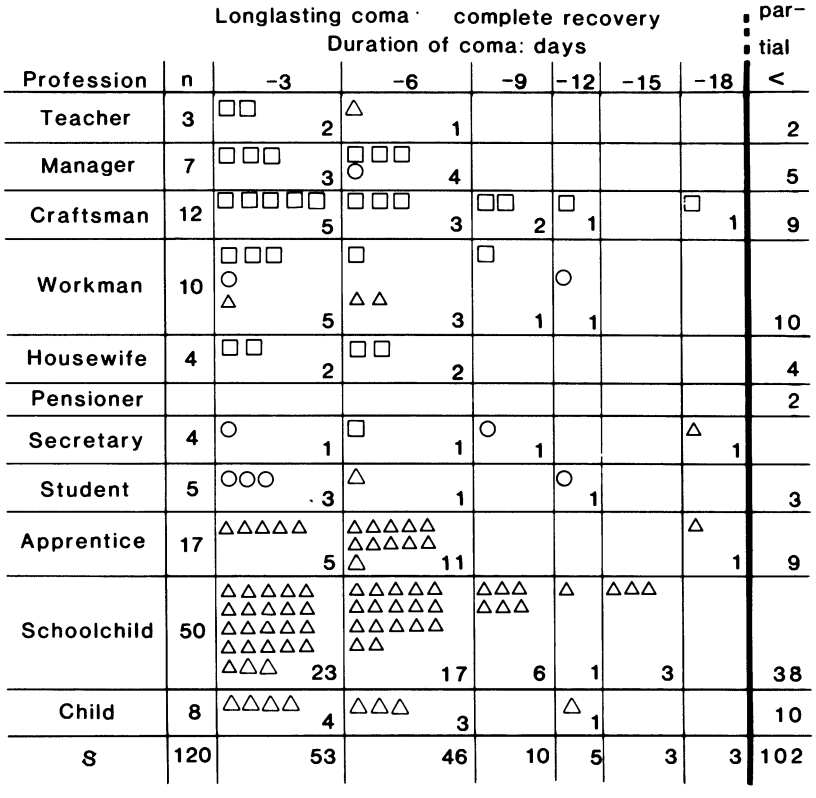

Fig. 6. Professions of 120 survivors of longlasting coma

time was not longer. The clinical finding "coma" therefore seems to have a different meaning in different age groups.

Profession

In Fig. 6, 120 patients with complete recovery are listed according to their pretraumatic profession and duration of coma. This is one-third of all the courses we analyzed.

Almost half of them (58) were small children or schoolchildren. Complete recovery after coma lasting longer than 10 days was achieved in five cases (10%); obviously, this is not very many in absolute figures. Therefore, a comparison with selected patients from rehabilitation centers is not possible.

One-third of all patients came from small groups of students, secretaries, housewives, workers, and craftsmen. Only ten patients with intellectual professions recovered completely after a maximal duration of coma of 6 days. The distribution of professions with partial recovery was similar: 7 of 102 now work in jobs with intellectual demands.

Summary

Long-term follow-up of 339 patients after longlasting posttraumatic coma confirmed the relation of age and duration of coma to recovery. Consistent with the limits of treatment reported previously, the number of patients with complete recovery decreased with age and duration of coma. The paramount importance of intensive care treatment within the first few days after injury is stressed. Children may require more than 1 year for recovery.

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Traumatic Intracranial Hemorrhages in Elderly People

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Introduction

Life expectancy has clearly increased in recent decades and with it that percentage of the population of the FRG who are aged 60 years and older. At present women at the age of 70 have a life expectancy of about 11.6 years and men an expectancy of about 9 years. At the age of 80 years women have a life expectancy of about 6.2 years and men an expectancy of about 5.2 years. In the FRG people older than 60 years constitute 19% of the total population, whereas in Berlin they account for 27%. Of them 70% are women and 30% are men.

Higher age doubtless has an important influence on the outcome of severe head injury [1,2,3,4,5,6,7,8]. In a retrospective trial, KARIMI in 1983 came to the result that the mortality of patients suffering a moderate head injury increases by about 16%-20% because of the age factor. The situation of the inhabitants of Berlin and the area serviced by our hospital, which includes Kreuzberg and Neukölln, is the reason why we are frequently confronted with patients of a higher or very high age who have sustained a severe head injury.

Methods

We reviewed the hospital records and neurological examinations of 64 patients aged over 60 who had been admitted to hospital and submitted to surgery in the years 1978-1987 because of head injury. Forty-six patients had a preoperative CT and 18 had an angiographic examination. All the patients who survived 5 days had a postoperative CT.

Results

The 64 patients were aged 60-91 years and had acute and subacute traumatic intracranial hematomas. Of the patients, 66% were older than 70 years and 23% older than 80 years.

The operative diagnosis was epidural hematoma in nine cases, acute subdural hematoma in 37, subacute subdural hematoma in 16, and intracerebral hematoma in two. Associated injuries of the limbs or chest were found in only 12 of the patients. These injuries led to hypovolemic shock in only two cases.

In 50% of the patients data about past medical history were obtained. Before the injury nearly one-sixth of the patients were under medical care because of diseases of the cardiovascular system, arterial hyper-

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tension, or diabetes mellitus. Another one-sixth of the patients were chronic alcoholics.

With respect of the postoperative course we isolated three groups of patients:

Group 1 (27 patients): These patients had an average GCS score of 4.8 at admission and died within 1-20 days (on average 4.3 days) as a result of the traumatic brain damage. They had a nearly constant coma, with a GCS score always below 9.

Group 2 (22 patients): The state of consciousness in these patients, with an average GCS score of 8.7 at admission, improved within 1-16 days (on average 3 days) to a GCS score of 9-14 (median value: 10.3). All these patients died after secondary deterioration within 4-57 days (on average 18.7 days).

Group 3 (15 patients): These patients improved within 1-11 days (median 3.5 days) after the operation and survived the injury. In this group there were four patients with a GCS score below 8 on admission.

In group 1 the mortality was due to the severity of the injury, and the clinical course was very short. Only one patient died because of extracerebral complications. In group 2 the mortality was due mainly to extracerebral complications. The clinical course was significantly longer than in group 1, and the number of complications increased in older age groups. Group 3 was characterized by a distinctly better GCS score on admission.

The overall mortality of patients aged over 60 was 76%, and that of patients aged over 70 was 86%. The highest mortality was found among patients with acute subdural hematoma (86%).

There was a close relationship between the GCS score on admission and the mortality. Out of 47 patients with a GCS score below 8, 87% died. This was twice as high as the mortality among 17 patients with a GCS score above 9.

The number of complications increased in older age groups. Complications which did not influence the mortality were relatively easy to treat and included diabetes mellitus, arterial hypertension, and delirium. On the other hand the appearance of pulmonary or circulatory complications, infections, or epileptic fits was associated with high mortality. The overall mortality of patients in whom these complications occurred was 90%.

CT Study

The analysis of CT scans showed that the patency of the perimesencephalic cisterns was an important factor for survival. Out of 15 patients with patent perimesencephalic cisterns, seven - all aged above 70 years - died due to extracerebral complications. Out of 19 patients with completely obstructed perimesencephalic cisterns, only two survived (Table 1).

Case 1, 91 years old, a previously healthy and independent woman, presented with subacute subdural hematoma due to a fall from the

Table 1. Patency of the perimesencephalic cisterns and mortality in 46 patients aged over 60 years

Perimesencephalic cisterns	No.	Group 1 Death	Group 2 Death	Group 3 Survival
Free	15	1	7	7
Incompletely obstructed	12	7	1	4
Completely obstructed	19	13	4	2
Total	46	21	12	13

stairs, and with a GCS score of 9. The hematoma was evacuated, but a partial infarction within the a.c. media territory occurred. This patient survived with a neurological deficit and became dependent on external help (Figs. 1, 2).

Case 2 was a 61-year-old woman with subacute subdural hematoma and a GCS score of 6 on admission. This patient recovered without deficits and was subjected to cranioplasty 1 year after the injury.

Analysis of mortality and the shift of the midline as demonstrated in CT examination shows that among 23 patients with a shift of more than 4 mm, only one survived (Table 2).

Out of 42 patients aged over 70, six survived. All these patients had a GCS score on admission above 9. None of them had extracerebral injuries. Four of them had diabetes mellitus, arterial hypertension, or cardiac disease in their past medical history, and two were chronic alcoholics. The clinical course in these patients was subacute, e.g., two were admitted more than 24 h after the injury with slowly progressive deterioration. One patient had epidural hematoma with a

Table 2. Midline shift in CT and mortality in 46 patients aged over 60 years

Diagnosis (hematoma)	No shift		Midline shift			
			Below 4 mm		Above 4 mm	
	No.	Death	No.	Death	No.	Death
Acute subdural	7	5	3	2	17	17
Subacute subdural	6	2	2	1	5	4
Epidural	4	0	0	0	1	1
Intracerebral	0	0	1	0	0	0
Total	17	7	6	3	23	22

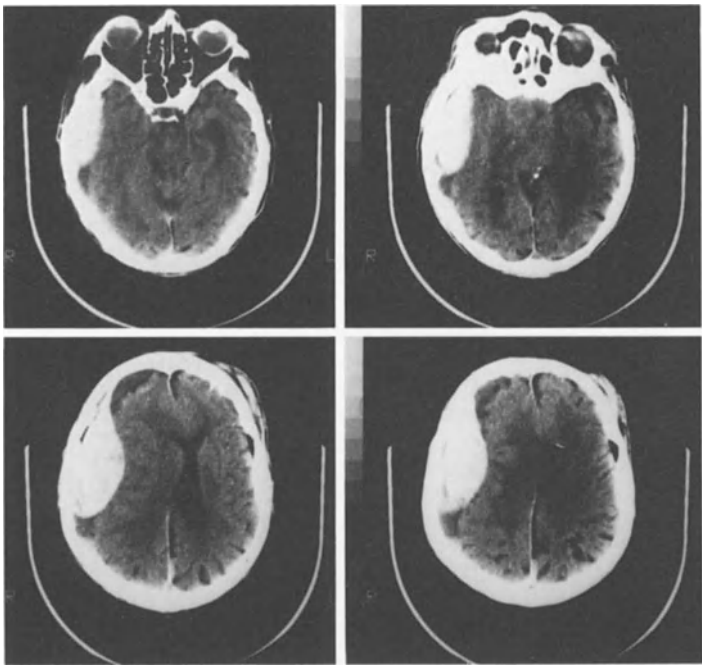

Fig. 1. K.M., 91 years old, preoperative CT: Subdural hematoma with occluded perimesencephalic cisterns and midline shift more than 4 mm

GCS score of 12, and three patients with acute subdural hematoma had GCS scores of 11, 12, and 13 respectively.

With the exception of the above-described two cases, none of the patients who survived had a midline shift in CT or completely occluded perimesencephalic cisterns. The surviving patients showed no signs of brain swelling at the time of craniotomy, and all of them had a GCS score above 10 on the first postoperative day.

Conclusion

Availability of means of diagnosis of intracranial hematoma in a patient who is very old puts the neurosurgeon in a difficult situation in which he has to decide about treatment of radiologically demonstrable surgical lesion knowing that the mortality of these patients will be extremely high in spite of removal of hematoma. In favorable circumstances, i.e., a high GCS score and no signs of tentorial herniation, as encountered among 6 of 42 patients aged over 70, surgery should be performed.

Summary

Surgical treatment of severe head injury in patients in the 7th to 9th decades of life is confronted by difficulties due to the age itself and to the complications of the injuries encountered. In our group of patients aged over 60 who had a GCS score below 8 on admis-

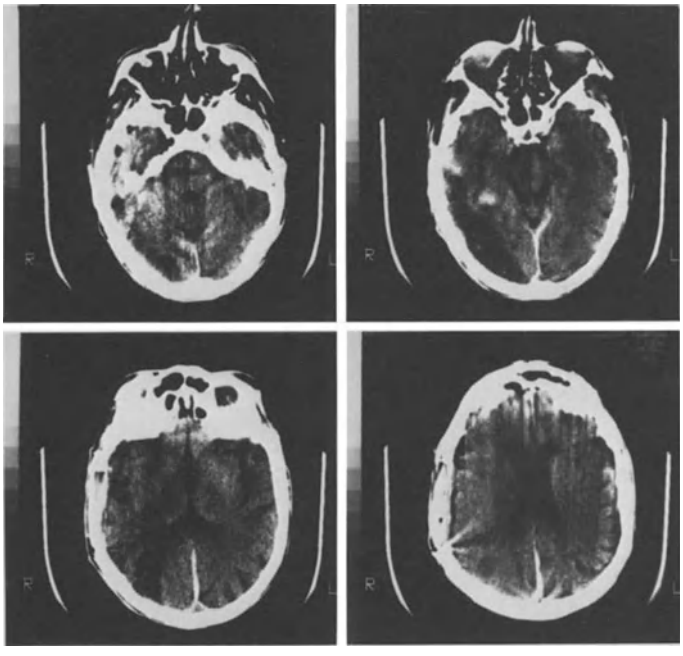

Fig. 2. K.M., 91 years old, postoperative CT

sion and who presented with a surgically treatable intracranial hematoma, the mortality was 87%. Among patients aged over 60 with a moderate degree of head injury (intracranial hematoma and GCS score of 9-13), there was a mortality of 50%.

The surgical treatment of traumatic intracranial hematoma in a patient aged over 70 should be considered when the GCS score on admission is above 9 and if there is no major midline displacement or occlusion of perimesencephalic cisterns on CT examination. It has to be stressed that among our patients aged over 70 who had a shift of the midline structures above 4 mm and occluded cisternes, none survived in spite of removal of the hematoma. The two surviving patients in whom these features were present in CT study had a subacute course with slowly progressive deterioration.

It can be concluded that in patients aged over 70 who harbor an intracranial traumatic hematoma and who have a potential chance of survival (no major midline shift and no signs of tentorial herniation in CT), approximately 50% will die due to extracerebral complications. Pneumonia, sepsis, pulmonary or cardiac complications, and epileptic fits developing during the postoperative course worsen the prognosis and are connected with an 80%-90% mortality.

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Outcome of Patients with an Acute Traumatic Subdural Hematoma

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Introduction

Reviewing the literature of the last 50 years for survival rates of more than 1300 patients suffering from acute traumatic subdural hematoma, one must be surprised by the fact that survival rates have not increased significantly due to better diagnostic tools and progress in intensive care medicine.

Survival rates between 4% and 81% were reported while a correlation between survival rate and the time interval from cerebral trauma to neurosurgical treatment was judged to be uncertain [1-5,7-8,10-20].

In 1980 an air rescue facility was installed under the auspices of the Department of Anesthesiology in our hospital. This gave rise to this study to investigate the influence of rapid hospital admission of patients with acute subdural hematoma on survival rates.

Material and Methods

Between 1980 and 1986 188 patients were treated in our hospital after severe cerebral trauma. Of these 188 patients, 156 underwent surgery for decompression from acute subdural hematoma. Ten of these 156 showed no certain relevant history of trauma and had to be excluded from survival rate calculations. The remaining group was split into five subdivisions depending on the time interval between trauma and neurosurgical treatment as follows: less than 3 h, 3-6 h, 6-12 h, 12-24 h, and 24-48 h. As a measure for the clinical course of these patients the Glasgow Outcome Scale was used [6]. Furthermore we looked for symptoms and associated lesions. Follow-up examinations were performed at least over a period of 1 year after injury. Data for survival rate calculations and Glasgow Outcome Scaling were supplied by our outpatient department and other hospitals or general practitioners. Additionally information was drawn from a questionnaire which was sent to the patients.

Results

Mortality and survival rate: Out of 188 patients, 156 were operated on, and of these, 65 survived. Of the 32 patients treated conservatively, 11 survived. The overall survival rate was 40.4%.

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Mode of admission: 73 patients were transferred by air ambulance, 55 were transported to our hospital in a surface ambulance under primary anesthesiological service, and 50 were admitted without first aid service by an anesthesiologist. No certain information was available from ten patients.

Time delay from cerebral trauma to surgery: Results from 146 patients regarding survival rates are listed in Table 1.

Clinical symptoms: In the group of patients with primary coma, lethality was found to be 66%. Those victims with secondary coma had a lethality of 49%. Decerebrate rigidity was lethal in 75%. All patients with a lack of light reflexes of their medium sized pupils died. Lethality in patients with mydriasis bilaterally was 84%; with mydriasis contralaterally it was 70%, and with mydriasis on the same side it was 59%. Only 29% of the patients with light-reactive pupils died.

Type of cerebral trauma: Survival rates of victims suffering from isolated acute subdural hematoma (n = 50), from an additional contusion (n = 65), and from a second epi- or subdural hematoma contralaterally (n = 73) showed no significant differences (38%, 44%, and 38% respectively).

Associated lesions: 100 patients had acquired additional injuries involving the abdominal region (survival rate 25%), the extremities (survival rate 38%), the thoracic region (survival rate 46%), or the spine (survival rate 58%). Survival rates decreased with the number of combined lesions - from 59% in those with one extracerebral lesion down to 39% in those with two more lesions and 27% in those with three more lesions.

Age: The overall survival rate in the group of patients younger than 60 years was found to be 45%, while it was 29% among those older than 60 years.

Glasgow Outcome Scale (GOS): One-fifth of the surviving group were in a vegetative state or severely disabled, while four-fifths were moderately disabled or in good recovery (GOS 1 = 60%, GOS 2 = 4%, GOS 3 = 4%, GOS 4 = 6%, GOS 5 = 26%). The clinical outcome according to the different time intervals between trauma and neurosurgical treatment is presented in Table 1.

Conclusions

Unfortunately this clinical study confirmed the mean survival rate of 40% in patients suffering from acute subdural hematoma. This figure agrees with the average results published in literature over the last 50 years.

At least it can be stated that immediate referral enables quick cerebral decompression. We were able to show that if neurosurgical therapy could be initiated within 6 h after cerebral trauma, 73% of the survivors recovered well. If treatment was only possible later

Table 1. (a) Survival rates and time intervals from cerebral trauma to neurosurgical treatment (n = 146), and clinical outcome (Glasgow Outcome Scale) of 73 survivors (b)

a			b			
Time from trauma to neurosurgical treatment (h)	Patients (n)	Survival rate (% per group)	Survivors (n)	GOS (% per group) 5	4	3/2
3	54	37	24	71	12	17
3-6	44	48	24	75	8	17
6-12	14	29	7	43	28	29
12-24	18	50	10	50	20	30
24-48	16	50	8	50	25	25
Total	146		73			

than 6 h but not exceeding 48 h after injury, 25% less of the survivors achieved good recovery.

Definition of acute subdural hematoma should be limited to those cases having developed within 48 h, because the early signs of reabsorption and progressive changes appeared 24-36 h after cerebral trauma [9].

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Acute Subdural Hematoma - An Unsolved Neurosurgical Problem

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Introduction

Acute subdural hematoma is one of the most common neurosurgical diseases and at the same time one with the highest mortality. It is difficult to imagine that any neurosurgeon would be willing to operate upon a condition with a mortality of 60% or higher, but in the case of subdural hematoma we do precisely this. It is also surprising that subdural hematoma has gained so little attention in literature and research; it seems that efforts have been directed to more attractive and "modern" topics, even if these often represent rare and exotic diseases. The achievements of modern neurosurgery, like new techniques in anesthesia, microsurgery, and intensive care, have greatly changed the results and indications in many fields of neurosurgery and resulted in an impressive lowering of mortality, but in respect of acute subdural hematoma no such trends are visible.

Different series have produced variable mortality figures, mostly in the 60%-70% bracket [1,2,3]. Lower figures are exceptions or due to selected cases [6].

It seems that the changing pattern of brain trauma, due in great part to high-speed vehicles, has greatly altered the pathology of intracranial hematomas. At the beginning of this century, the prevailing bleeding was epidural; today just the opposite is the case. In addition to more severe trauma, other factors contributing to the high mortality are faster transportation and better on the spot resuscitation, which increases the percentage case load brought alive into hospital, but of whom few will survive the first few hours.

This paper discusses the influence of therapeutic efforts in treating acute subdural hematomas during the last two decades and seeks an explanation for the unsatisfactory results.

Results

A comparison of mortality in subdural and epidural bleeding in the years from 1965 to 1986 is displayed in Fig. 1. The first group includes 435 subdural hematomas and the second one 248 epidural hematomas. Excluded were all firearm injuries and subdural hematomas in children; in both groups multiple injuries were included. It can be seen that the mortality curve in subdural hematoma remains high and unchanged, with slight variations in some years. On the other hand, the curve for epidural hematoma shows a slow but clear downward trend. Mortality in this group is normally low and further lowering

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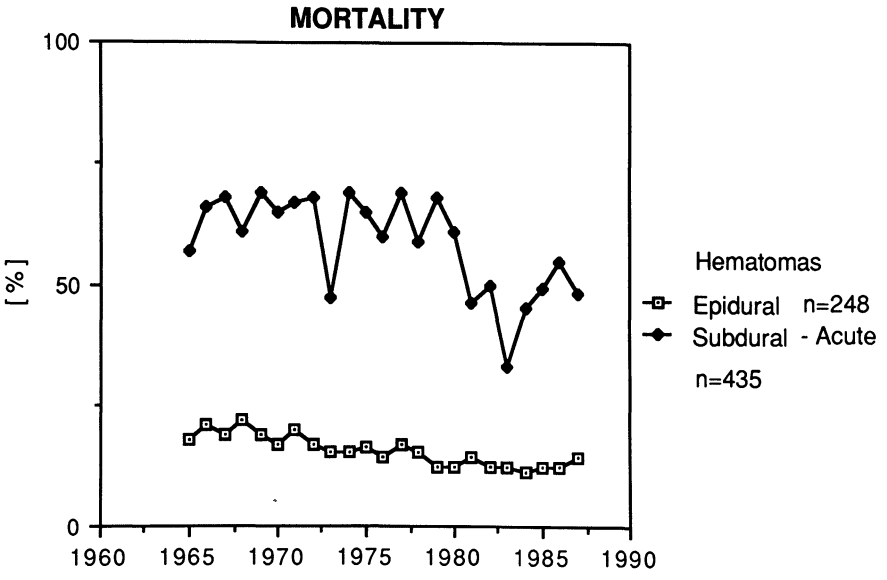

Fig. 1. Mortality in the patients with acute subdural hematomas (435 cases) and epidural hematomas (248 cases) in the last two decades. A downward trend is visible for epidural hematomas, but the mortality in the subdural group remains virtually unchanged

would be difficult to achieve. It is important to stress that the relatively short transport routes in the area covered by our Department (most patients are admitted within 2 h) lower the mortality in the group of epidural bleedings, but seem to raise the mortality in subdural bleedings.

Figure 2 shows the mortality and morbidity in the same period for acute subdural hematoma. Owing to the extensive overlapping of the curves, the percentage on the ordinate is "stretched." The different methods of treatment are cited according to their date of introduction. This should be considered just as an orientation, since many of these methods were combined, or used under changing regimes. However, the figure proves that none of the treatments has changed the prognosis as regards either the mortality or the sequelae. The only clearly visible change occurred with the introduction of computerized tomography (CT), meaning that the prognosis was changed not by a therapeutic but by a diagnostic tool. The reason is obvious. Some patients were not operated on at all, either because of the large extent of the concomitant brain injuries, or because it was decided that the subdural bleeding was too small to be responsible for the clinical picture. In addition, intracerebral hematomas were easily diagnosed. On the other hand, the number of patients with considerable neurological deficit increased with the introduction of CT for obvious reasons.

Discussion

The explanation for the unsuccessful therapeutic efforts probably lies in the poor understanding of the real pathology of subdural hematoma. Acute subdural hematoma is not just a simple collection of

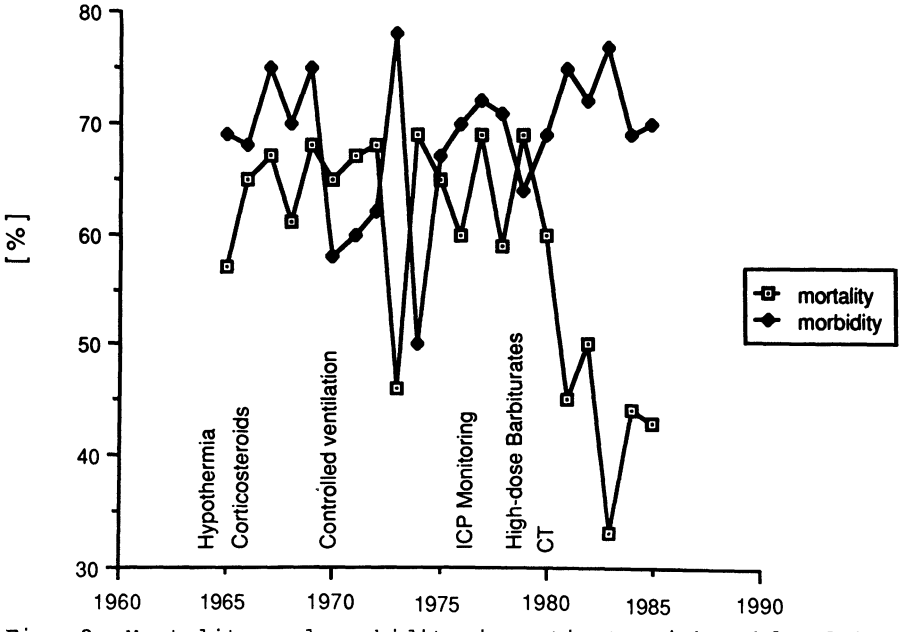

Fig. 2. Mortality and morbidity in patients with subdural hematomas, related to the introduction and use of different therapeutic and diagnostic procedures

blood, comparable to epidural hematoma or distinguished from it just by anatomical landmarks, but part of a syndrome of the severely injured brain [4,5]. In other words, **acute subdural hematoma is just a sign of multiple injuries of the brain.**

Figure 3 shows the dilemma very clearly. This patient has the most severe contusions and destruction of both frontal lobes. The small subdural collection plays an unimportant role, if any, in the severe clinical picture. In the period before the introduction of CT, however, this bleeding would probably have been the only pathology seen on the angiogram. In most such cases, an immediate operation did not help, and often caused the condition to deteriorate because of the prolapsing brain, an almost invariable complication of extensive brain injuries.

Looking at subdural hematoma from this point of view, it is easy to understand why our therapeutic efforts have helped so little in improving the prognosis. If the brain injury represents the main pathology, it is difficult to believe that it will be influenced by evacuation of a blood collection, especially a small one. This also explains why large subdural hematomas have a better prognosis - in cases in which they are not combined with too extensive brain injuries - than the small ones.

The prognosis in acute subdural hematoma therefore remains gloomy with the current therapeutic possibilities and is one of the great unsolved problems in neurosurgery.

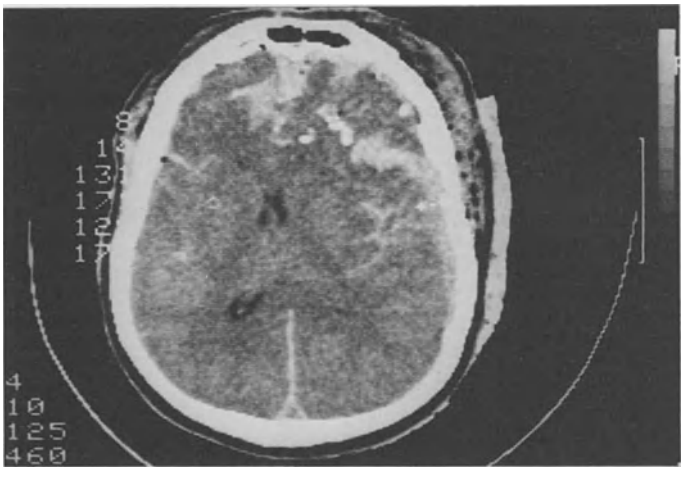

Fig. 3. A CT scan showing severe destruction of both frontal lobes and a small, unimportant subdural hematoma. CT allows a good evaluation of the total brain injury in contrast to angiography, which would most likely show the subdural bleeding only, which in turn could lead to an unnecessary or unsuccessful operation

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Regeneration of Intellectual Functions Following Closed Brain Injury: Follow-up Study on a Pair of Twins using the Co-twin as Control

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Introduction

Neurological and especially intellectual sequelae of brain injuries in children are difficult to evaluate even in prospective studies as pretraumatic personality features and psychoreactive consequences interact with direct effects of the traumatic insult. Uniformity of genetic, of social, and in the ideal case of educational influences in monozygotic twins offers a chance to visualize these traumatic effects on a child's development. Following these considerations, a surprising concordance among both twins, brain-damaged and control, was demonstrated by DENCKER [4] in a retrospective study on 36 monozygotic pairs. EEG, psychometric intelligence, and social behavior agreed to such an extent that most of the remaining differences proved pretraumatic.

Our findings in a pair of female monozygotic twins, one of whom suffered a severe closed brain injury, demonstrate, besides the cited concordant aspects, significant differences according to complex intellectual functions.

Case Report

The pair of twins studied was described anamnestically as inconspicuous until the 8th year of age. Their progress at school was above average. The firstborn twin was characterized as being a little quieter than her sister. This girl was hit by a car on her way from school. The primary neurological status was described by the following symptoms: unconsciousness and extension cramps of all extremities, and two generalized convulsions during admission to our clinic 1 h later. Check-ups indicated a left-sided hemiparesis and a 4-point Glasgow Coma Scale score.

Besides severe brain damage and a contusion of the left maxilla, no other injuries were found. Computerized tomography showed contusions in the left frontal and right temporal lobes.

Under epidural ICP monitoring we administered high-dose antiedematous therapy in the intubated and artificially respirated patient. During the 9th day she opened her eyes for the first time. At the end of the 3rd week, visual contact and emotional reactions were achieved. After 1.5 months she verbalized a few words spontaneously and after 3 months understanding was almost restituted though her vocabulary and psychoreactivity were still retarded. Another 3 months later she was able to start school again together with her twin sister.

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Methods

One and 2 years after the accident a neurological, psychiatric, and psychological investigation was carried out on the brain-damaged child and her twin sister as control. Each time EEG records were obtained from both children, including hyperventilation records. Their parents were thoroughly questioned about behavioral abnormalities.

The following test battery was applied:

- HAWIK-R, the German version of the WISC intelligence test
- PET, the German version of the Illinois Test of Psycholinguistic Abilities (ITPA) [1]
- GFT, the German version of the BENDER-Gestalt Test
- Trials for visual and visuomotoric discrimination ability
- Trials for working capacity and velocity.

Results

Physical appearance revealed an obvious acceleration of our patient 1 year after the traumatic insult as compared with the co-twin. We attributed this to a long-term stress-induced release of growth hormone. After 2 years this difference of acceleration was nearly equalized (Figs. 1, 2).

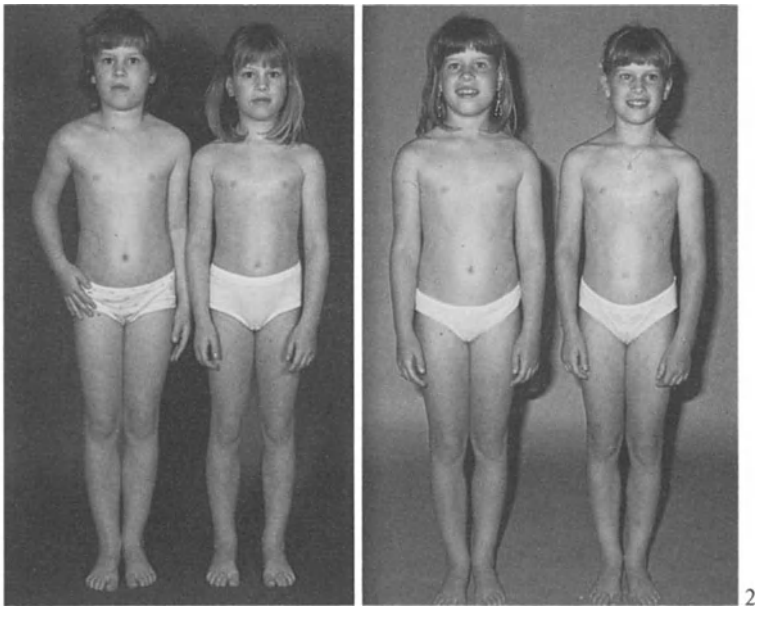

Fig. 1. The twins at the age of 8 years and 8 months; 1 year after brain injury, **patient to the left**
Fig. 2. The twins at the age of 9 years and 8 months; 2 years after brain injury, **patient to the left**

In the **neurological investigation** we still found a latent left-sided hemiparesis and a reduced automatization of complex movement skills with only slight improvement during the 2nd year. Regarding her **behavior**, it was remarkable that her sound sister seemed to be a kind of model she used for orientating herself when she was exposed to unknown situations. In addition she was more quiet, slower, and made less attempt to attract attention.

There was no remarkable impairment in terms of progress of work, concentration, or earlier exhaustion. Two years after the trauma she was still doing well in working behavior but was more self-confident and optimistic.

Although her twin sister showed a high achievement motivation herself, she remained prudent and considerate towards our patient. After the 2nd year she worked more for herself and was more vivacious and verbally active owing to the reduction of emotional stress.

Psychometric testing was performed several times with our patient during the 1st year of convalescence outside our clinic. In accordance with the literature, verbal and knowledge skills were less affected by the brain damage than were action-bound skills, especially those involving visual or visuomotoric processing velocity. Depending on the severity of the initial brain injury, the latter also normalize during the 1st year.

The HAWIK-R evaluated the IQ of our patient to be 120 (= T-score 63) 1 year after the accident while that of her twin sister was 118 (= T-score 62), and other tests confirmed these findings. However, for evaluating the development during the 2nd year after the brain trauma the tests generally used in prospective studies were not meaningful. The only thing we proved by these was an increase of working velocity in our patient after the 2nd year. For this purpose the PET was successfully applied.

Testing the Hypothesis of No Difference concerning intellectual development in the twins without the intercurrent accidental event, we noticed proceeding impairments in our patient compared with her twin sister at the first investigation using the PET:

- On the Integration Level they concerned velocity of perception and the level of acoustic-linguistic automatic and sequential functions.
- On the Representation Level impairment of complex receptive, associative, and expressive function was verified.

Two years after traumatic insult our patient reached significantly better scores on acoustic-linguistic automatic function. Related to the PET T-scores a positive development from a lower average to an average value was remarkable during the 2nd year (Table 1). Her sound sister reached scores above average each time.

Discussion

From long-term studies of adults [2] and children [3] with head injuries it was known that timed measures of visuospatial and visuomotor skills tend to show more impairment than do verbal skills. As there was no suggestion of a specific pattern of cognitive and

Table 1. Comparison between patient (P) and her sound twin sister (S) 1 year (T1) and 2 years (T2) after brain injury. The numbers represent T-scores achieved in the HAWIK-R and PET

Applied test		T1		T2	
		P	S	P	S
HAWIK-R		63	62	-	-
PET	Integration Level (automatic, sequences)	44	55	49	57
	Representation Level (complex functions)	52	60	53	60

intellectual deficit following brain damage after those studies, these sequelae were considered to be individual.

Our investigations on a pair of monozygotic twins demonstrated a specific pattern of impairments in the brain-injured child compared with the co-twin 1 and 2 years after traumatic insult using the PET. The impairments concerned complex cortical functions such as perception velocity, automatic and serial acoustic-linguistic skills, verbal perception, and visuomotoric associations. Although the accuracy of neuropsychological tests is said to be only 70% [5], our results agreed with detailed statements of the parents of our patient about improvement in both complex motoric and cognitive functions.

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The Influence of Independent Parameters on the Evaluation of Patients with Craniocerebral Trauma and Their Occupational Reintegration

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Neurosurgery, often seen as a smaller specialty among other clinical disciplines, has acquired considerable sociomedical significance on account of the steadily rising number of craniocerebral traumas. In particular, the clinical neurosurgeon is frequently called upon to evaluate damage to the nervous system resulting from craniocerebral trauma. The present study, focusing on the potential sociomedical impact or consequences of the current modes and practices of evaluation, involves a catamnestic investigation of 60 patients who, after isolated craniocerebral trauma, were primarily treated in the Münster Clinic of Neurosurgery, with subsequent medicolegal evaluation of the damage resulting from the trauma.

BLOHMKE et al. [1] have distinguished various subfields of social medicine:

1. Medical sociology
2. Epidemiology of chronic diseases, definition of risk factors and noxae
3. Theory of prevention
4. Theory of rehabilitation
5. Theory and practice of social security from a medical point of view
6. Health information to the general public
7. Public health system

Regarding the type of institutions requiring sociomedical or sociolegal certification, and also the type of certificates wanted, we have to realize that for a neurosurgeon the majority of certificates will be called for by cooperative trade associations, i.e., by the legal bearers of industrial insurance against occupational injuries. Thus, our main interest in the following focuses on evaluations on behalf of statutory insurance corporations.

Within the framework of neurosurgical expertise, the most frequent problem is the assessment of "reduction of earning capacity" or reduced fitness for work after an accident or traumatic injury. In defining the exact percentage of reduction, the evaluation must be oriented not so much toward the actual earnings but rather the theoretical reduction of earning capacity in the respective occupational field, and working (life) conditions in general, disregarding the possible reduction of salary or income resulting from the accident. Statistics about the correlation of this reduction of capacity with the percentage of income reduction after an accident will as a rule show that in most cases the accident causing permanent phy-

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sical damage does not lead to a material decrease of income, and may even occasionally involve a rise in salary.

Several studies on the rehabilitation and prognosis of patients with craniocerebral trauma were published in the 1960s and 1970s. FROWEIN [2] reported in 1961 on the prognosis after craniocerebral trauma with prolonged unconsciousness. Favorable survival expectancy was found only in those traumatized patients whose reactions improved within a maximum of 5 days after trauma. In another investigation the same author [3] presented data about the prospects for social rehabilitation of 100 patients with craniocerebral trauma surviving a period of complete unconsciousness lasting between 2 and 27 days, who had been regularly followed up over 2-13 years. Of these, 21% regained normal working capacity and 17% had a slightly reduced capacity, which means an overall adequate recovery in 38%. The study confirmed strong correlations of prognosis with the duration of unconsciousness and also with age. Essential recovery was achieved, on average, 2 years after the accident, but 3 years later in 10%.

Pampus [4] reported a catamnestic study of patients treated in the Rehabilitation Center of Cologne University between 1970 and 1972, and between 1974 and 1976. The material for the second period comprised 114 questionnaires. The patterns of remaining damage after rehabilitation were:

- Motoric disorders in 52%
- Vasovegetative dysregulation in 66.9%
- Mental symptoms in about 94%, comprising:
  a) motivation disorders in 91%
  b) impairment of intellectual capacity in 61%

Of 110 traumatized patients, one-third were reintegrated into free employment while two-thirds were not working. The author compared the results of the second period with those of a previous study on the

Table 1. Interdependence of previous work level and reduced working capacity (from [4])

	In 48 patients with cerebral trauma (1970-1972)	In 110 patients with cerebral trauma (1979)
Reduced working capacity		
40%		
Same level of work	37.5%	9.1%
Inferior level of work	10.4%	7.3%
Without work	6.3%	4.5%
60%		
Same level of work	4.2%	5.5%
Inferior level of work	6.3%	9.1%
Without work	4.2%	10.0%
100%		
Same level of work	0 %	0 %
Inferior level of work	8.3%	4.5%
Without work	14.6%	44.5%

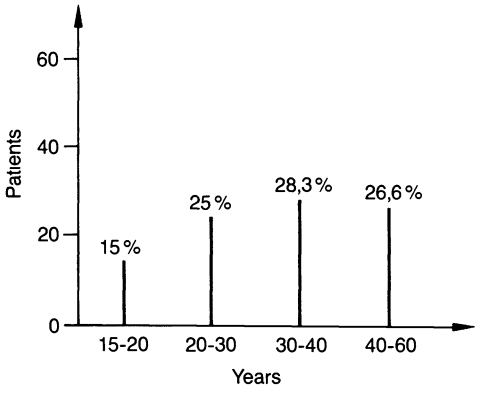

Fig. 1. Age distribution of 60 patients with cerebrocranial trauma

period 1970-1972 (Table 1). Comparable with regard to the severity of damage, this group had been much better reintegrated, i.e., two-thirds of the patients had been able to find a situation in free employment. According to the author, the difference was due to altered employment conditions in the second period, and to a general elevation of requirements in the labor market.

Figure 1 shows the age distribution in our group of 60 patients with craniocerebral trauma. Table 2 gives a correlation of professional activities and trauma-related reduction of earning capacity in the 60 patients we had to evaluate and certify between 1982 and 1985. This period of 3 years corresponds to that recorded by Pampus. In the subgroup with a reduction of 40%, reintegration into their previous jobs was possible in all cases: five students, two soldiers (evaluated according to the military pensions legislation), and two patients from skilled trades. The students suffered no impairment in their further professional training. In the subgroup of patients with 60% reduction, 4% were able to take up a simple employment after rehabilitation training, but the rest remained without employment in the free labor market, as did all patients with a reduction of earning capacity up to 100%. It should be mentioned that our study

Table 2. Authors' data on the interdependence of work level and reduced capacity in 60 patients with cerebral trauma between 1982 and 1985

Reduced capacity		% of total
40%	Same level of work	15%
	Inferior level of work	0%
	Without work	0%
60%	Same level of work	0%
	Inferior level of work	4%
	Without work	52.8%
100%	Same level of work	0%
	Inferior level of work	0%
	Without work	23.2%

included six patients with a reduction of 50-70% who all remained active, namely on their own farms. As they are hardly comparable to the patients previously in employment, they are registered separately.

The pattern of damage observed in our patients at the time of evaluation after rehabilitation is largely comparable to that described by Pampus. Psychic problems such as low motivation and impaired concentration or memory were reported by about 90% of all patients.

The classification of craniocerebral traumas by TÖNNIS and LÖW (cited in [5]), adopted for our study, has established the often confirmed correlation between initial symptoms (main parameter: duration of unconsciousness) and the subsequent grade of deficiencies. Predicting professional reintegration or retraining potential on the basis of initial symptoms is hardly possible from the data gathered in our group of patients.

PAMPUS [4] stated in 1979: "A reduction of earning capacity by more than 60% is practically prohibitive for reintegration into the free labor market under current conditions." According to our investigations, the limit for reintegration tends to settle on a 40% reduction of earning power.

Investigation of the social situation of patients with cerebral trauma may rely on several different parameters which, however, are all hampered by a high degree of subjectivity and multifactorial influence. Questioning about familial status and housing conditions will elicit important aspects of social rehabilitation, but any answers about personal well-being or changes in social environment are subject to highly personal estimation. They depend not only on the effect of organic psychosyndromes, but also ultimately on the success of occupational reintegration. Accordingly, such estimates were always negative in an unexpectedly high number of non-reintegrated patients.

When we asked whether non-reintegrated patients received any pension, 39 of the answers were statistically valid, though highly divergent. In some cases applications were still under way but as yet unapproved, while in others the pension had been denied and the patients received unemployment compensation or social security payment. A third group was receiving pensions.

Other criteria could not be evaluated on account of the high proportion of permanently unemployed patients. When we asked for the patient's personal opinion on the adequacy of recompenses, i.e., according to his own feeling of "justice," answers were mostly negative.

Another query concerned the change of real income suffered at the time of catamnestic evaluation, i.e., some 3 years after the accident. The resulting data were equally insecure, being subject to strong personal influences. Moreover, it is impossible to ascertain whether and to what extent private insurances or other sources have been successfully approached for compensation.

Among the trauma-independent variables (Table 3) responsible for the ultimately unsatisfactory reintegration, the persistently unfavorable situation in the employment market, high levels of unemployment, and increasing requirements for classified jobs were certainly in the foreground. Guidelines for evaluation and certification issued by accident and labor injury insurance corporations have not been

Table 3. Trauma-independent variables

1. Conditions and regulations of insurance
2. Labor market
3. Protection against dismissal
4. Practice of expertise

altered during recent years. Another point is that an employee with a reduction of earning capacity of up to 50% is practically exempt from normal dismissal. Consequently, any employer is apt to be somewhat reticent in engaging such a person even if adequately rehabilitated. Last but not least, the practice of evaluation and certification is liable to certain individual variations. Psychosyndromes of cerebro-organic origin, as well as mental symptoms in general, are often of borderline significance; thus, the mere decision to require additional psychiatric expertise may influence the "reduction of earning capacity" ultimately certified in terms of grade or percentage.

It may be concluded that a "reduction of earning capacity or fitness" by 40% or more will practically render impossible any reintegration into the free employment market. We have to ask whether the practice of evaluation and certification should not be changed in the long run in order to prevent severe social iniquities, or whether the guidelines of statutory insurance corporations should be changed to allow for individual-oriented pensioning or compensation in parallel with the criteria adopted by private accident insurance companies. The results reported above seem incompatible with the fulfilment of the original intentions and aims of statutory insurance against industrial injuries.

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Prognostic Parameters in Severe Head Injury: A Multivariate Analysis

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Introduction

In spite of decreasing numbers of traffic accidents and injuries, severe head trauma still plays a major role in neurosurgical practice. Notwithstanding important developments in the diagnosis of head injury, e.g., cranial CT and MRT, we have not so far been able to achieve a decisive improvement in our patients' prognosis. Primary brain damage, the quality of initial treatment at the site of the accident, safe and quick transport to the hospital, immediate operative treatment, and rehabilitation are still more important for the patient's outcome than the kind of conservative intensive care in the neurosurgical intensive care unit [2,12,16].

Whereas we can scarcely influence the course from the occurrence of traumatic brain damage to admission to the neurosurgical unit, the management in our clinic has to be reconsidered regularly in order to improve the prognosis by preventing or minimizing the secondary brain damage. A variety of authors have examined and described a vast plurality of prognostic factors in severe head injury, such as different clinical data, laboratory blood and CSF parameters, and the results of electrophysiological, morphological, and functional investigations of the injured brain [1,2,3,4,5,19].

Stimulated by the modern imaging techniques like cranial CT and MRT, new models of brain damage have been developed and discussed, e.g., "diffuse axonal injury," "white matter shear trauma," and primary brain stem lesion" [10,13]. The development of practicable methods for continuous measurement of intracranial pressure (ICP) allows early recognition of brain swelling and intracranial hemorrhage and thus monitoring and control of operative and conservative intensive therapy after initial treatment [7,9,15].

Patients and Methods

To evaluate these monitoring methods we investigated for more than 12 years the clinical state, course, and outcome in severely head injured patients by means of computerized analysis. We measured and evaluated the kind of injury, age and sex, the intensity and duration of coma, and the height and duration of elevated ICP according to the following protocol:

- Classification of initial coma (WFNS Coma Scale 0-IV)
- Precise follow-up of the clinical course at least twice daily with a modified, extended Glasgow Coma Scale [18]

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- Immediate CT scan after admission and at repeated intervals
- Start of ICP monitoring within 6 h of trauma using a low invasive epidural method with miniaturized transducers (Gaab)
- Continuous EEG record (two bipolar channels, computer analysis by FFT)
- Classification of outcome according to the Glasgow Outcome Scale by control examination 8-14 months posttrauma according to the Glasgow Outcome Scale [11]

Recently introduced methods such as regional cerebral blood flow investigation by stable xenon CT and transcranial Doppler sonography or neurophysiological monitoring using EEG, SEP, and BAEP were not referred to for this evaluation. The results of our neurophysiological monitoring are described by Lorenz in this volume.

All patients were treated according to the degree of coma: patients with coma III were respirated with mild hyperventilation. High doses of dexamethasone were given up to Nov. 1979, moderate doses of pentobarbital up to 1982. Critical rises in ICP were treated by osmotherapy (mannitol, sorbitol, glycerol), oncotherapy, or antiacidotic therapy. All data were evaluated by computer. ICP and EEG were analyzed on- or off-line with an Intertechnique IN 110 computer calculating mean ICP, max. ICP >30 min, max. ICP >5 min (effective peak ICP), and the occurrence (frequency, amplitude, duration) of A, B, C, and other ICP waves. EEG was evaluated by FFT or Berg transformation.

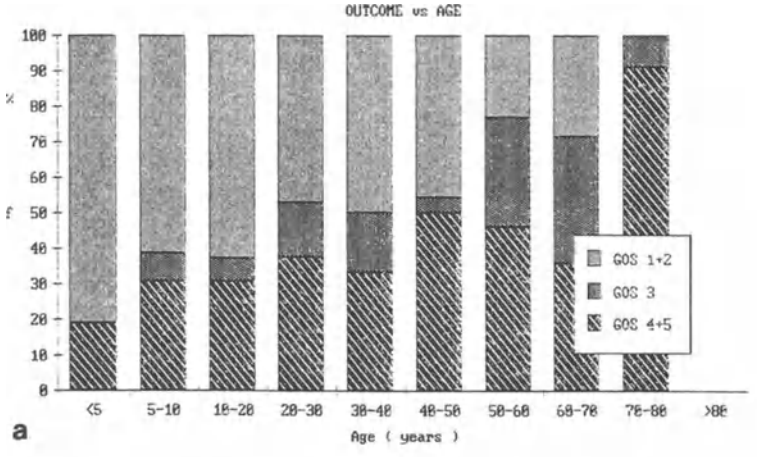
Results

Complete data of 379 severely head injured patients (297 men and 82 women) could be evaluated. The distribution of age and sex corresponds to that of all head trauma patients treated in our departments. ICP monitoring was dependent on the availability of intensive care resources and ICP measuring equipment and was employed in severely head injured patients with coma expected to last for more than 24 h and/or significant pathological findings in the CT scan. Therefore young patients were somewhat overrepresented and these young patients often had more serious traumata.

We could confirm the well-known relation between age and prognosis [6,14]. With the exception of infants, who are especially susceptible to water-electrolyte imbalances and malignant brain swelling, we found an almost linear correlation between age and prognosis. There was also a significant relationship between different stages of the initial coma, the duration of coma, and prognosis (Fig. 1).

The statistically most important factor for our patient's prognosis was the epidurally measured ICP (mean ICP as well as duration and maximum peak of intracranial hypertension) (Fig. 2). However, children and adolescents may have a surprisingly good outcome despite a massively elevated ICP of up to and even over 70 mmHg. Adults and especially people aged over 50 years hardly tolerated even a briefly elevated ICP of more than 35 mmHg.

Surprisingly a coma of long duration may have a good prognosis if there is no pathological rise in ICP of over 20 mmHg, e.g., in "traumatic normal pressure coma" (126 survivors = 33.2%). Coma associated with elevated ICP over 30 mmHg, which we call "high pressure coma," has a significantly worse prognosis (39 survivors = 10.3%) (Fig. 3). This important differential diagnosis between patients with


OUTCOME vs AGE
%
100
90
80
70
60
50
40
30
20
10
0
<5
5-10
10-20
20-30
30-40
40-50
50-60
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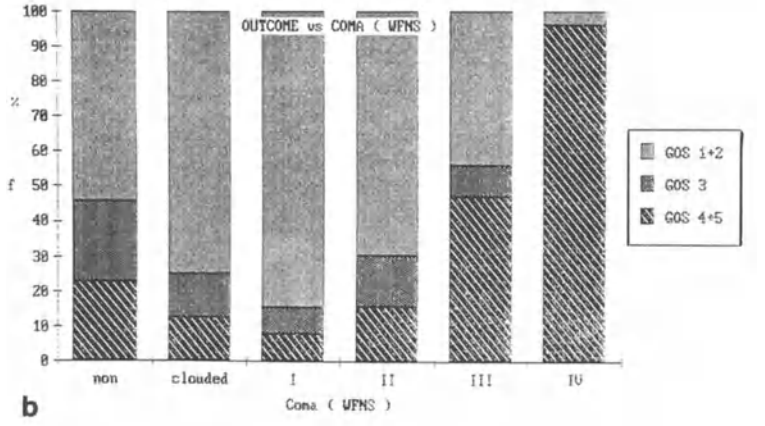
70-80
>80
Age (years)
GOS 1+2
GOS 3
GOS 4+5

a


OUTCOME vs COMA (WFNS)
%
100
90
80
70
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50
40
30
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I
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IV
Coma (WFNS)
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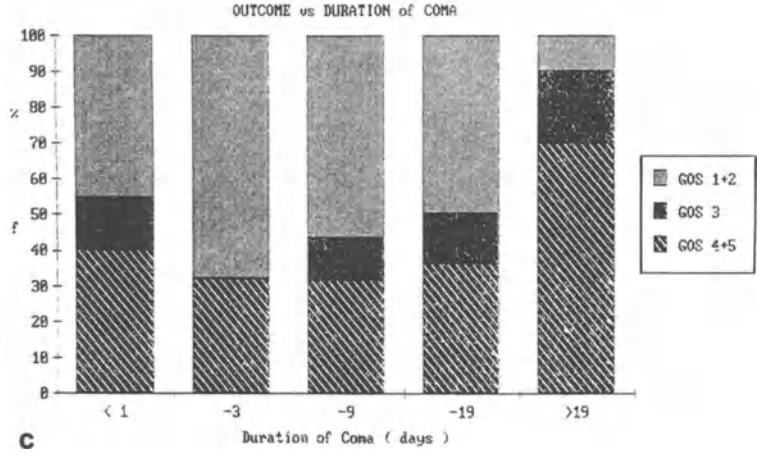
GOS 3
GOS 4+5

b


OUTCOME vs DURATION of COMA
%
100
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40
30
20
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-3
-9
-19
>19
Duration of Coma (days)
GOS 1+2
GOS 3
GOS 4+5

c

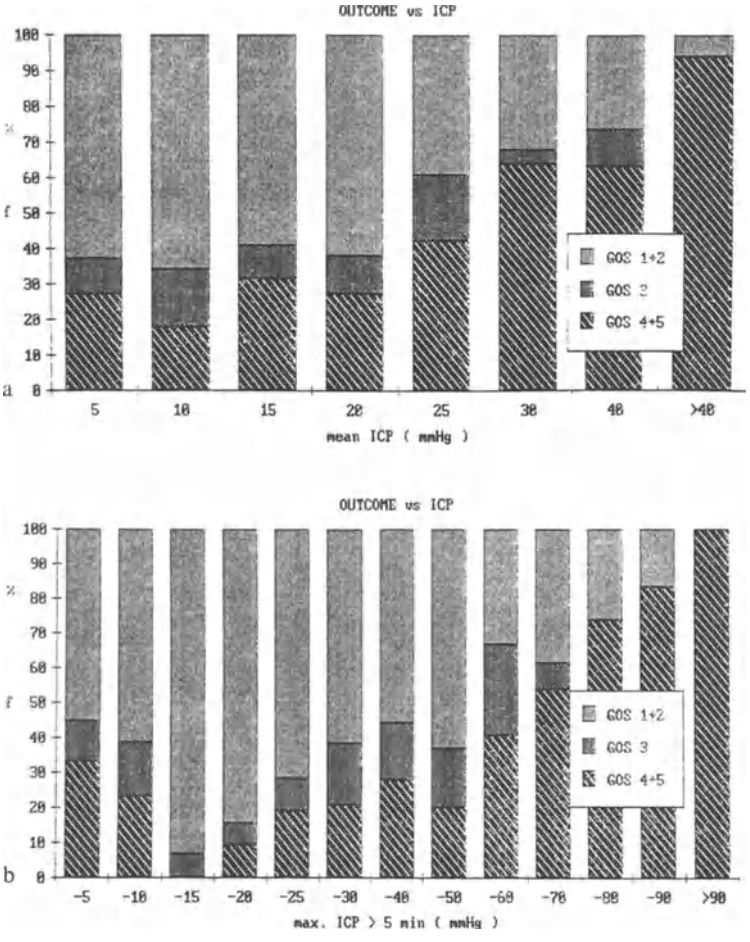

Fig. 2 a, b. Relationship between outcome and ICP. The relative frequency of three outcome groups (Glasgow Outcome Scale) is shown in dependency on a mean and b maximum ICP for over 5 min (mmHg)

coma due to brain stem lesions and patients with coma and supratentorial brain swelling is further justified by ICP wave analysis and SEP measurements [8]. In normal pressure coma there are no plateau waves and only rare uncritical B-waves. Using SEP measurements an increase in central conduction time gives a good parameter for depth of coma and prognosis, provided this increase is not caused by primary brain stem concussion in patients with normal pressure coma.

Fig. 1. Relationship between outcome and a age and b depth and c duration of coma. The relative frequency of three outcome groups (Glasgow Outcome Scale) is shown in dependency on age (years) and depth (WFNS Coma Scale) and duration (days) of coma. [Glasgow Outcome Scale: 1 = good recovery, 2 = moderate disability, 3 = severe disability, 4 = persistent vegetative, 5 = dead]

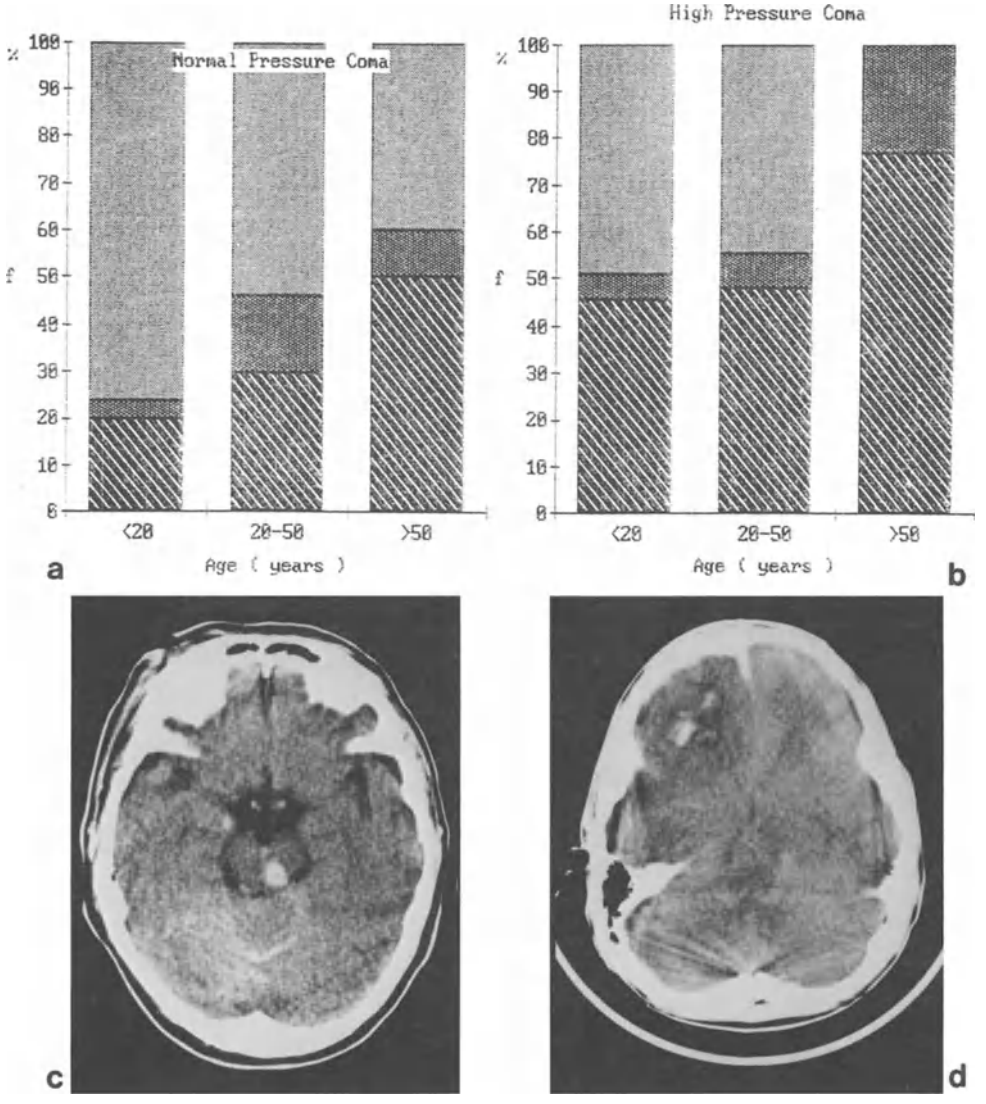

Fig. 3 a-d. Traumatic normal pressure coma and high pressure coma. Typical CT scans of (c) normal pressure coma (primary concussive brain stem lesion without hypertensive brain swelling) and (d) high pressure coma (frontal contusion with massive brain swelling) are shown together with the relationship of these two pathophysiologically different states to three age (years) and outcome [Glasgow Outcome Scale (GOS)] groups (a, b)

Discussion

Two pathophysiological groups can clearly be differentiated after head injury: patients without any significant increase in ICP and patients with coma due to intracranial hypertension. In patients with traumatic normal pressure coma no space-occupying lesions are to be

found in CT scans. The disturbance of consciousness is therefore not a result of brain stem compression but of a primary brain stem lesion or diffuse axonal injury. These lesions are quite often found by careful examination. Contrary to previous opinion, primary brain stem lesions and/or white matter trauma are often functional with good recovery and better statistical prognosis. The mechanism underlying such lesions may be a tractional, shearing, or rotational stress of the nerve fibers in the brain stem during acute acceleration of the head. The prognosis of these injuries depends on their morphological extension and not on the ICP.

Patients with elevated ICP, too, often show an initial coma followed by a delayed rise in ICP. This may be a sign of a "diffuse brain injury" combining a primary brain stem lesion/axonal injury with subsequent midbrain compression after the delayed development of brain swelling.

The close relation between raised ICP and prognosis underlines the clinical importance of ICP monitoring. As stated above adults hardly tolerate even short increases in ICP of more than 35 mmHg. Therefore such critical elevations must immediately be recognized and appropriately treated [17]. In children and adolescents a much better prognosis can be achieved even when high pressure coma is present, as long as adequate and appropriate therapy is administered.

Summary

Over a period of 12 years we prospectively investigated patients with traumatic coma by means of continuous ICP monitoring, repeated CT scans, and clinical follow-up. Even when clinical midbrain syndromes were present, over 30% of our patients never had any rise in ICP or space-occupying lesions. This traumatic normal pressure coma is attributed to a primary brain stem lesion or a diffuse axonal injury caused by tractional, shearing, or rotational stress in acceleration trauma. Its prognosis depends on the initial morphological damage and not on the ICP. Diffuse brain injury leading to high pressure coma, however, results in secondary brain stem compression by brain swelling; therefore the ICP is its most important prognostic factor.

ICP monitoring is therefore considered as indispensable in the management of severe head injury, in addition to clinical observation and repeated radiological investigations.

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Prognostic Value of Factors Affecting Outcome After Severe Head Injury

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Introduction

For the prognosis of patients with severe head injury, up to now mostly signs of impaired cerebral function, such as state of consciousness and verbal or motor response, have been considered. The usefulness of other regularly available data, i.e., intracranial pressure (ICP), serum osmolality, and serum urea has been demonstrated [1,3,5,6].

Patient Material

To examine the value and ranking order of these variables for an early prognosis of patients with severe head injury we studied 112 patients by discriminant analysis [4]. All of these patients were comatose for more than 6 h after trauma (GCS $\leq$7).

Results

Looking at the degree of coma (according to WFNS, [2], 30% of the coma II patients died, 40% of the coma III patients, and 100% of the coma IV patients. The influence of age on prognosis is well known, as is the influence of diagnosis and additional injury to the chest or abdomen. These influences were confirmed in our study.

In all patients ICP was measured intraventricularly or with an epidural sensor during the 1st week after trauma.

Irrespective of age, increases in mean ICP up to an extent of 40-60 mmHg were survived. The tolerance towards ICP peaks varied: children and adolescents in some cases survived peaks over 90 mmHg, young adults peaks up to 70 mmHg, and elderly patients peaks up to 50 mmHg. One patient over 50 years of age survived pressure peaks up to 90 mmHg.

Regarding the highest values of serum osmolality, we found that there were steep increases in mortality above a level of 320 mosmol and that a mortality of 100% was reached above 340 mosmol. A similar tendency was observable as regards the highest values of serum urea. Mortality reached 92% at urea values above 100 mg/dl.

If one examines the worst values of the variables used, i.e., the state of consciousness, ICP, serum osmolality, and serum urea, it seems possible to attach 78% of the patients to the groups of sur-

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vivors or nonsurvivors by considering only the state of consciousness. However, this retrospective analysis does include patients with coma IV.

We studied the question of whether and to what extent the above-mentioned factors could be useful for early prognosis based on data from the first days after trauma. As in our clinical material deterioration to coma IV was always fatal, we excluded these cases from the following discriminant analysis of possible prognostic factors.

Under these circumstances the analysis showed that on the day of trauma the state of consciousness was not useful for a prognostic statement. With the help of mean or maximum ICP values not more than 50% of the patients could be correctly classified. On the day of trauma no other variable qualified as having a prognostic value.

On the 1st day after trauma the state of consciousness did not reach a prognostic value either. Maximum ICP, serum osmolality, and serum urea qualified for a prognostic statement with a reliability between 50% and 63% (Fig. 1). Considering these variables together, the percentage of cases correctly classified increased to 76%. With the additional use of diagnosis and age, correct classification of cases reached 82%. For this day, the variables with prognostic value ranked as follows: serum osmolality, aspiration, maximum ICP, state of consciousness, serum urea, and diagnosis.

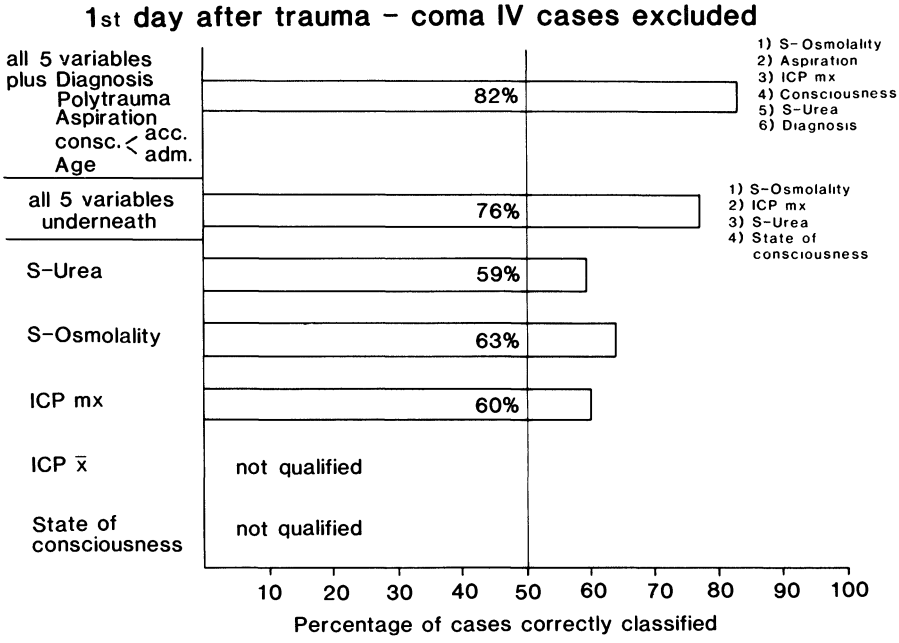

Fig. 1. Percentage of correct prognostic classifications on the basis of the used variables (state of consciousness, ICP $\bar{x}$, etc) and ranking order of the qualified variables on the 1st day after trauma. acc, accident; adm, admission; S, serum; mx, maximum; $\bar{x}$, mean

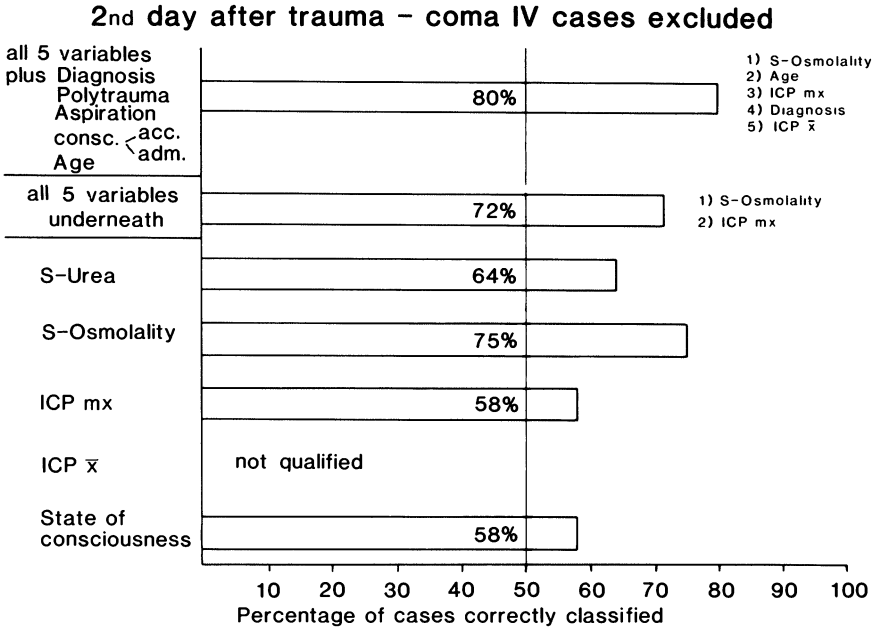

Fig. 2. Percentage of correct prognostic classifications on the basis of the used variables and ranking order of the qualified variables on the 2nd day after trauma. Abbreviations as in Fig. 1

On the 2nd day after trauma, state of consciousness or ICP made a correct classification possible in 58% (Fig. 2). The classification result was better on the basis of serum osmolality, reaching 75%. Regarding these variables together, 72%, or, with additional consideration of diagnosis and age, 80% of the patients could be classified correctly. Again, serum osmolality was in first place.

Considering data from the 3rd day after trauma, the percentage of cases correctly classified on the basis of osmolality decreased to 64%, while with additional use of age and diagnosis the percentage was 71% (Fig. 3). In addition to age and serum osmolality, prognostic value was reached by positive or absent primary pulmonary aspiration. Patients without pulmonary aspiration had normal ICP values in 45% of cases. In contrast, only 24% of the patients with pulmonary aspiration had normal ICP values.

Conclusion

Considering classification results for the first 7 days after trauma based on the given variables, the following conclusions can be drawn (Table 1): excluding patients with coma IV, state of consciousness and ICP during the first days after trauma do not qualify as making possible a sound prognosis. Serum osmolality, the five variables together, and additional data, i.e., age, diagnosis, and pulmonary state, do seem to have prognostic value.

We found that during the 1st week after trauma all variables regarded together allow a prognostic statement whose accuracy reaches the

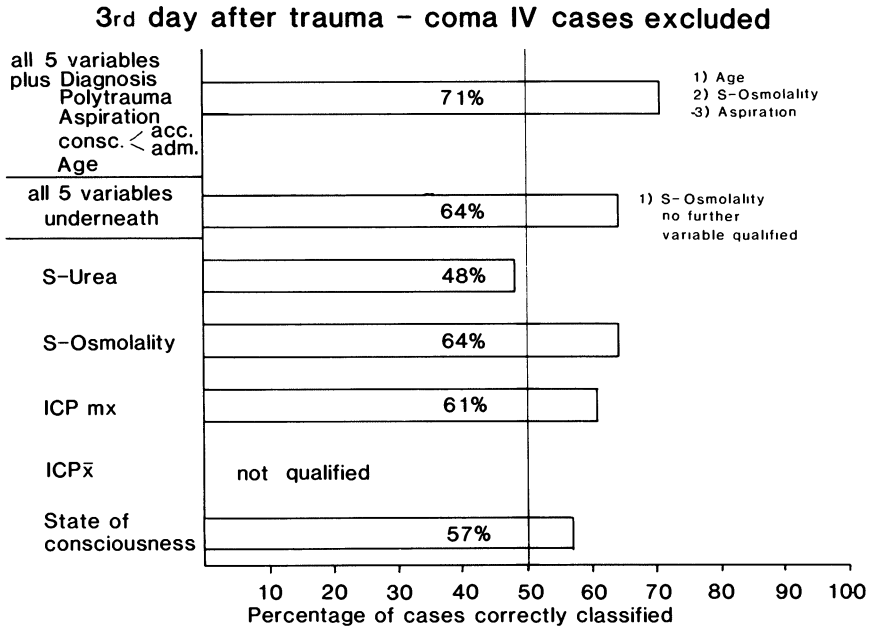

Fig. 3. Percentage of correct prognostic classifications on the basis of the used variables and ranking order of the qualified variables on the 3rd day after trauma. Abbreviations as in Fig. 1

range of 70%-80%. Therefore, the chances for an early prognosis are still limited.

Only continuous observation of the patient during a longer period after severe head injury allows for a prognostic statement with sufficient reliability. As has been shown before [3], the necessary duration of these observations is inversely proportional to the patient's age.

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Table 1. Summary of correct prognostic classification on the basis of the used variables during the 1st week after head injury. On days 4/5 and 6/7 the worst values during both days were taken

	Day of trauma	Day 1	Day 2	Day 3	Days 4/5	Days 6/7
1 State of consciousness	-	-	58%	57%	-	59%
2 ICP $\bar{x}$	50%	-	-	-	-	-
3 ICP mx	48%	60%	58%	61%	-	-
4 S osmolality	-	63%	75%	64%	62%	-
5 S urea	-	59%	64%	48%	67%	62%
Variables 1-5	-	76%	72%	64%	64%	67%
Variables 1-5 ± Diagnosis Multiple injury Aspiration Consciousness Age	- accident / admission	82%	80%	71%	75%	73%

$\bar{x}$, mean; mx, maximum; S, serum

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Organizational Model for the Diagnosis and Treatment of Skull and Brain Injuries at a Neurosurgical Clinic with an Integrated Neuroradiological Department

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The place of accident, the quality of the initial care, and the transportation to the nearest regional neurosurgical department are often decisive for survival of patients with severe skull and brain injuries. Furthermore, it is very important how diagnostic and therapeutic procedures are organized (Fig. 1).

The time factor is very important in the planning of therapy. Therefore we tried to establish a model for interdisciplinary cooperation between neurosurgeons, neuroradiologists, and anesthetists so as to ensure optimal conditions for diagnostic procedures and surgical treatment. It is necessary to win minutes by organizing transport and diagnostic procedures and by preparing operative procedures (Fig. 2).

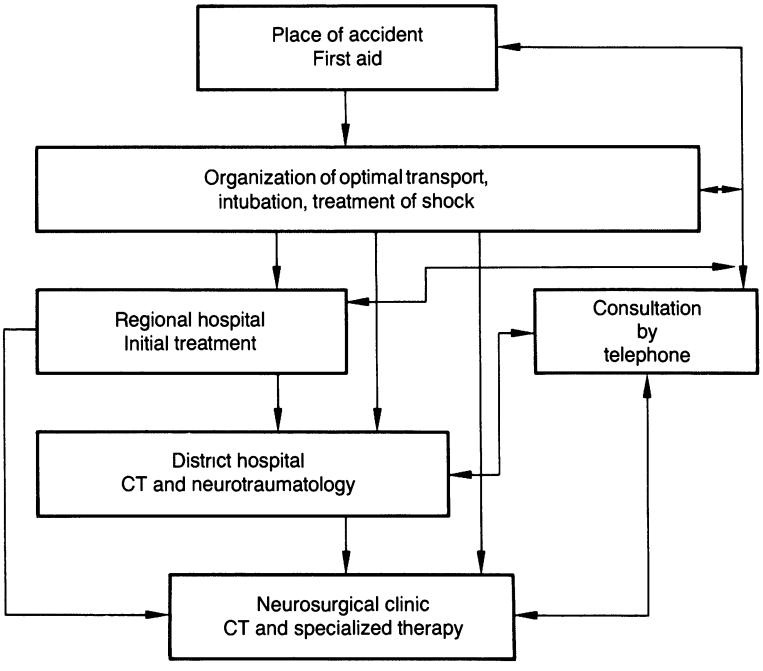

Fig. 1. Model of possible treatment depending on requirement of patients with skull and brain injuries

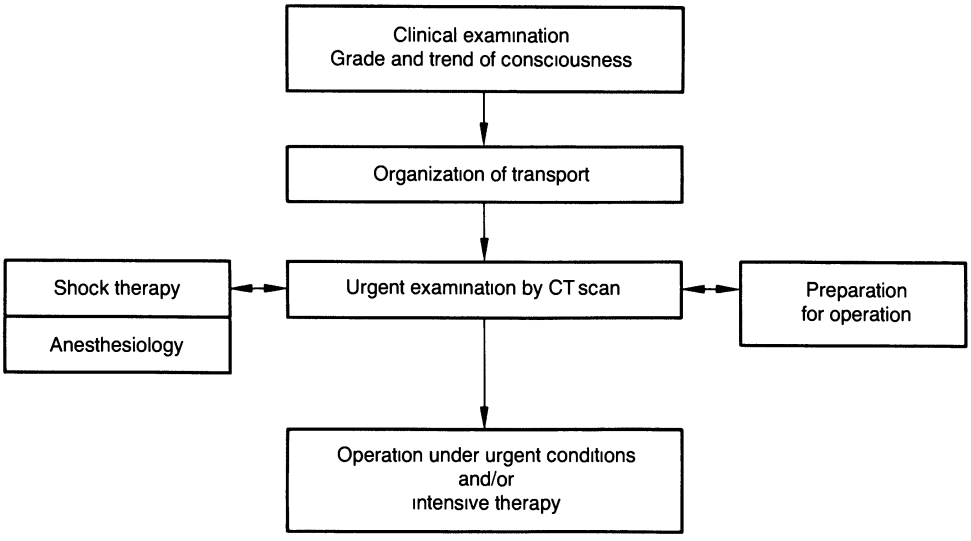

Fig. 2. Interdisciplinary organization of CT and parallel procedures

Neuroradiological procedures are reduced to plain X-ray and CT examination, sometimes with control scans. Sometimes during the intensive care CT scan controls enable adequate surgical therapy or appropriate conservative therapy to be administered (Fig. 3).

Angiography is only necessary in cases of rupture of vessels or pulsating exophthalmos; in the latter context it must be performed as soon as possible. In cases of carotid-sinus cavernosus fistula, CT reveals marked thickening of eye muscles which decreases soon after balloon occlusion of the fistula.

When later complications develop, examinations are usually not as urgent as in acute cases. Neuroradiological procedures include plain

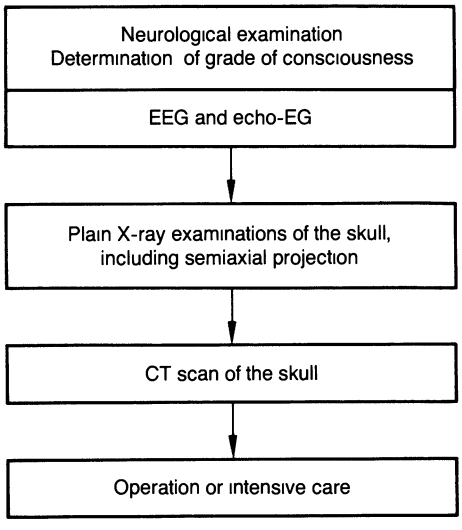

Fig. 3. Strategy of examination (in a specialized clinic) in cases of skull and brain injuries

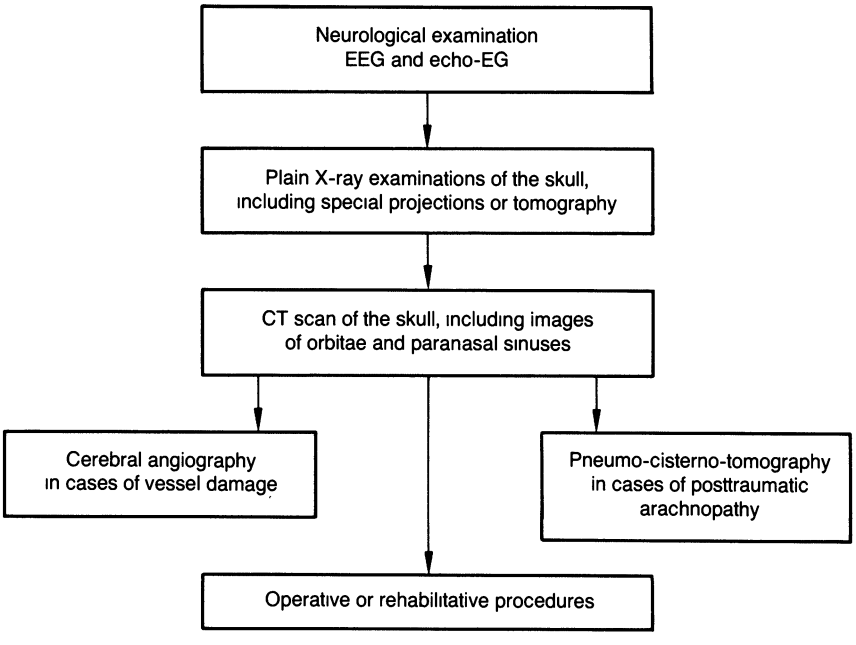

Fig. 4. Course of examinations in later traumatic complications

X-ray examinations of the skull, conventional tomography, and CT. In cases of posttraumatic damage to vessels, angiography is necessary. Only in rare cases is cisternography, combined with tomography, necessary (Fig. 4).

In conclusion, all regional hospitals must be able to perform optimal diagnostic and therapeutic procedures in acute cases of brain injury. A consultation by telephone with neurosurgical clinics may be useful to establish the optimal course and to differentiate between the necessity of acute treatment and the possibility of later treatment in a specialized neurosurgical unit.

Frequency and Prognosis of Traumatic Brain Edema

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Introduction

The limitation of traumatic brain edema has been one of the main aims in the prevention of secondary damage in recent years. "Brain swelling," a term introduced by LANGFITT and co-workers [2,3], is used to describe a pathological increase in cerebral blood volume after head injuries [1,4]. Our study is concerned with the following questions:

- How often do brain swelling and brain edema occur after brain trauma?
- What is the rate of progression of both?
- How do brain swelling and brain edema influence the prognosis?

Patient Material

In 100 consecutive head-injured patients, treated in 1985, yet before the onset of the new randomized double-blind dexamethasone study, neurological course and outcome were correlated with the findings of the CTS of the head.

In the CT scan we differentiated the following findings:

0 : No pathological alteration
1 : Brain swelling
(in accordance with ZIMMERMANN et al. [5], brain swelling was assumed when the perimesencephalic cistern, the ventricles, or the subarachnoid spaces of the brain surface were compressed)
2 : Contusion or hematoma
3 : Contusion or hematoma with signs of brain swelling
4 : Contusion or hematoma with perifocal or local postischemic brain edema
5 : Contusion or hematoma with brain swelling and brain edema

In the majority of the patients (64%) the first CT scan was performed within the first 3 h; in the rest it was performed 4-24 h after trauma.

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Results

Frequency of Brain Swelling and Brain Edema

Without consideration of the time factor, 24% of the CT scans revealed no pathological findings, 17% brain swelling, 4% contusion or hematoma without the signs of brain swelling or brain edema, 29% contusion or hematoma with accompanying brain swelling, 5% contusion or hematoma with brain edema, and 21% contusion or hematoma with brain swelling and brain edema. Thus, the most frequent findings were contusion or hematoma with brain swelling, as well as these findings together with brain edema.

Temporal Development of Brain Swelling and Brain Edema

Brain edema was evident in the CT scans at the earliest after the 3rd posttraumatic hour, whereas signs of brain swelling appeared within the first 3 posttraumatic hours.

Children and Juveniles (Fig. 1). The marked difference in the rate of appearance of brain swelling and brain edema was well demonstrated in the children and juveniles. Remarkably frequent were the early CT findings of brain swelling, locally as a concomitant phenomenon of contusion or hematoma. In half of the patients brain edema developed at a later stage. Earliest recognition of brain edema in the CT scan

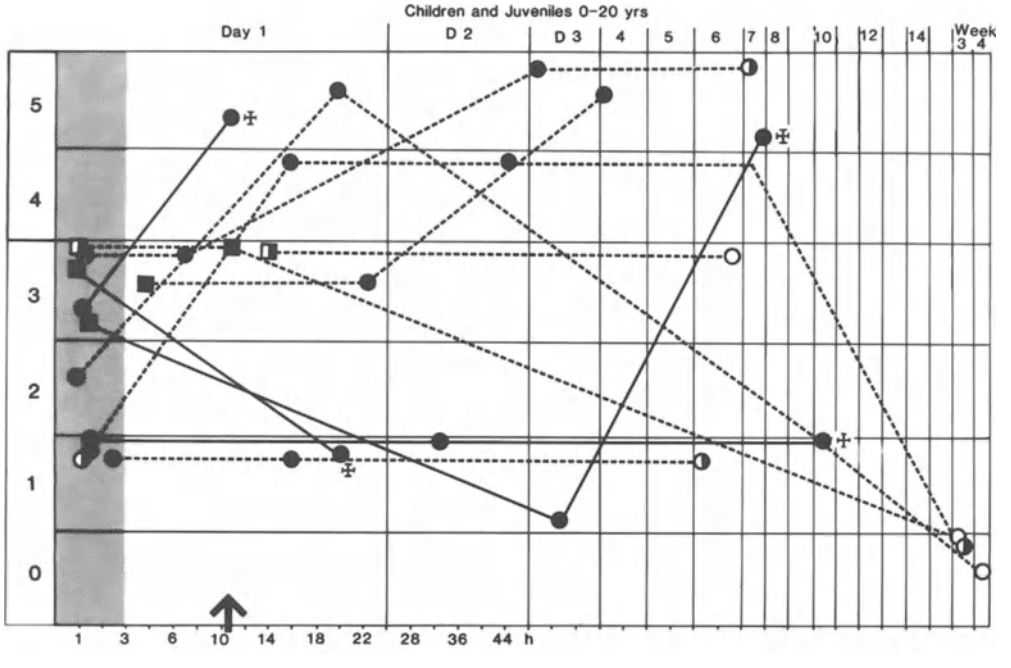

Fig. 1. Temporal and development of brain swelling (1-3) and brain edema (4-5) on the CT scan in children and juveniles after severe head injury. Brain edema never appeared before the 11th hour

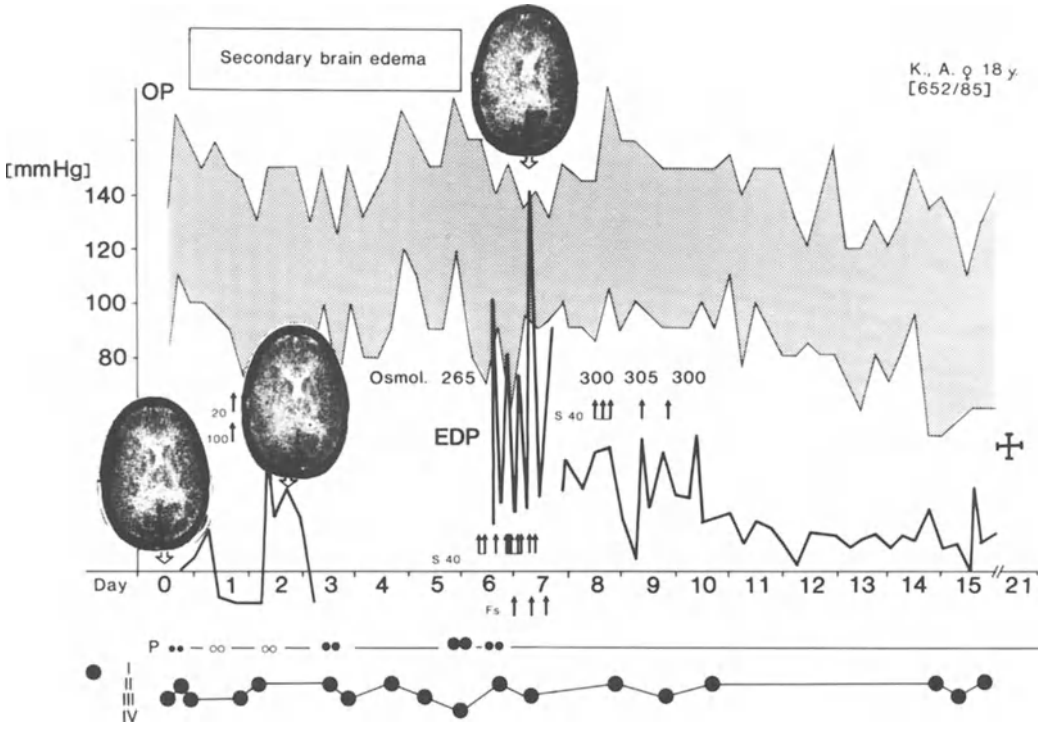

Fig. 2. Typical course of posttraumatic brain edema. Simultaneously with the apex of the cerebral edema, extreme epidural pressure peaks appeared at days 6 and 7 after trauma

was 11 h after trauma, but it was usually recognized between the 2nd and 7th days. Two of the courses were lethal.

Figure 2 represents an example of protracted brain edema in an 18-year-old female. Primarily, the CT revealed a small subdural hematoma with diffuse brain swelling. At day 6 after trauma, a therapy-resistant increase in epidural pressure was monitored simultaneously with edema of the left cerebral hemisphere.

Younger Adults (Fig. 3). In one patient of this group (21- to 40-years-old), who was suffering from AIDS, a hyperdense area was observed in the frontoparietal region at 5 1/2 h after trauma. Apart from this exception, in this age group brain edema was first diagnosed after 30 h using CT scan. Even in this group signs of local or diffuse brain swelling were found remarkably often within the first few hours.

Older Adults (Fig. 4). In the 41- to 65-year-old group, particularly frequent findings in the CT were diffuse or hemispheric brain swelling. At a later stage, in 8 of 15 patients brain edema additionally developed. In one patient with a gunshot wound, this appeared only 5 h after trauma; in the remaining cases, however, it did not appear before the 17th hour. On the whole, signs of brain swelling were already recognizable within the first 3 h.

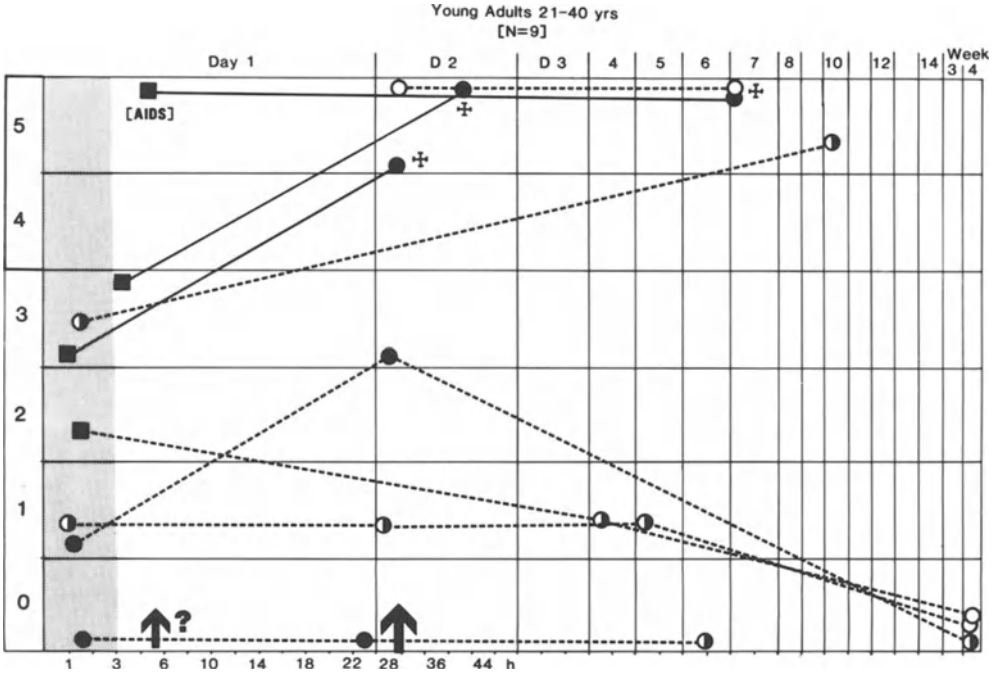

Fig. 3. Temporal development of brain swelling (1-3) and brain edema (4-5) on the CT scan in young adults (21-40 years old). Brain edema did not appear before the 28th hour

In older patients (>65 years), with one exception brain edema did not develop before the 3rd day.

Brain Swelling and Brain Edema in Relation to Patient's Age and Outcome (Table 1)

In 28 of 100 head-injured patients, CT did not reveal any pathological alteration. All of these patients survived. Of 11 patients (18%) with brain swelling, two died; both were juveniles. Contusions or hematomas without brain swelling were seldom seen. Out of three older patients, one died. Of 28 head-injured patients with contusion or hematoma and brain swelling, more than 50% died, independent of age. The mortality in patients with contusion or hematoma and brain edema but without brain swelling was significantly lower: 22%. If, in addition, brain swelling was present, the mortality increased to 48%.

Conclusion

Sequence analysis of the traumatic sequelae in CT of the head makes it evident that brain swelling and brain edema are secondary processes with vastly differing temporal progression. Brain swelling develops very frequently within the first 3 h, not only in children and juveniles, but also in older patients with severe head injury. This leads very rapidly to a life-threatening restriction of the intracranial space capacity, particularly in conjunction with the development of a hematoma or a contusion. Brain edema develops at a

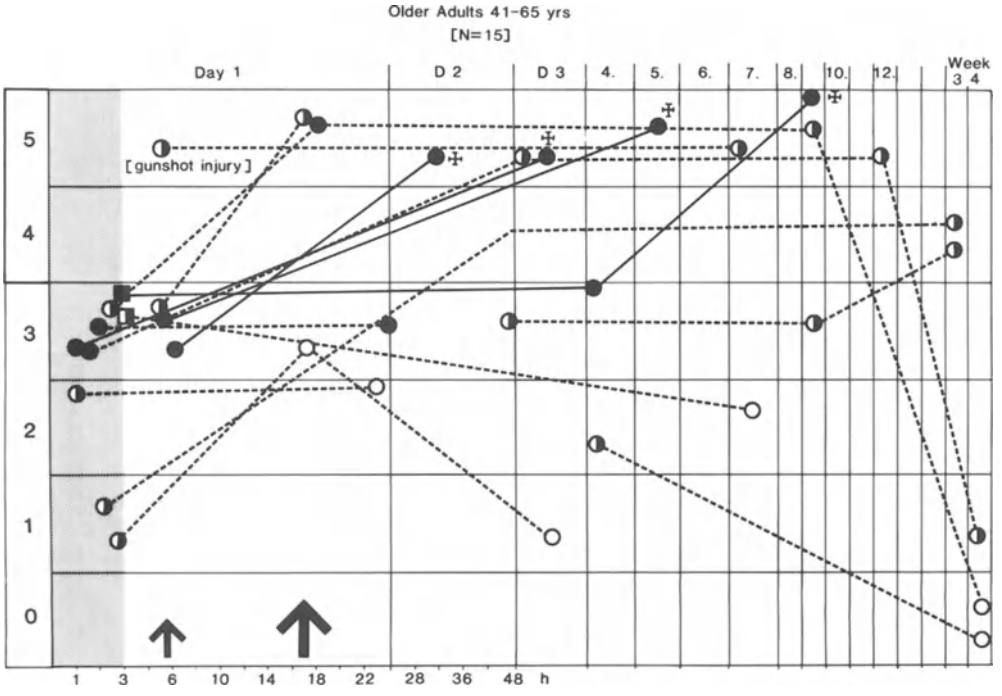

Fig. 4. Temporal development of brain swelling (1-3) and brain edema (4-5) on the CT scan in older adults (41-65 years old). Brain edema did not appear before the 17th hour

later stage, usually not before the 9th hour and quite often as an additional complication of acute brain swelling which is resistant to therapy. Therefore, the main priority should be given to means of preventing posttraumatic brain swelling.

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Table 1. CT findings in relation to outcome and age of the head-injured patients

CT findings	S	Outcome Total (n:100)			Patient's age (years) 0-20 (n:23)			21-40 (n:33)			41-65 (n:29)			>65 (n:15)		
		↑	(↑)	+	↑	(↑)	+	↑	(↑)	+	↑	(↑)	+	↑	(↑)	+
		30	39	31	7	9	7	11	13	9	9	12	8	3	5	7
5 Contusion/hematoma + brain swelling + brain edema	21	1 4%	10 48%	10 48%	-	4	1	1	1	3	-	5	5	-	-	1
4 Contusion/hematoma + brain edema	9	1 11%	6 67%	2 22%	-	-	1	1	-	-	-	2	-	-	4	1
3 Contusion/hematoma + brain swelling	28	6 21%	6 21%	16 58%	3	1	3	1	3	6	2	2	3	-	-	4
2 Contusion/hematoma	3	1 33%	1 33%	1 33%	-	-	-	-	-	-	1	1	-	-	-	1
1 Brain swelling	11	2 18%	7 64%	2 18%	-	4	2	1	3	-	1	-	-	-	-	-
0 None	28	19 68%	9 32%	- -	4	-	-	7	6	-	5	2	-	3	1	-

↑ completely recovered; (↑) slightly disabled; + nonsurvivors

ICP, nrCBF, and Contrast Scan for the Prognosis of Severe Head Injury

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Being capable of reaching an early diagnosis and prognosis of severe head trauma is a prerequisite for the sensible, ethically and economically justifiable employment of our sophisticated means of intensive care. Consequently in this paper we discuss whether prognosis is possible on the basis of initial findings of certain diagnostic methods employing medical equipment, i.e., intracranial pressure (ICP) measurement, cerebral blood flow (CBF) measurement, and contrast scan examination (for the determination of brain death).

ICP

According to our own evaluation, ICP alone provides only limited evidence for use in prognosis. Too often an ICP is seen in the normal or upper region - "normal pressure coma" (A. TROST, preceding essay) - as a result of obviously successful early ICP therapy, whereas the most severe neurological deficit is present. When the ICP approaches the medial arterial blood pressure, i.e., there is an insufficient cerebral perfusion pressure, evaluation of the condition of the patient usually can be achieved without ICP measurement.

nrCBF

In the situation in question (normal pressure coma) CBF measurement provides additional information; this was, incidentally, made clear by the results of earlier experiments by REULEN [3,4]. With focal as well as with global brain edema, each accompanied by a normal ICP, we found highly pathological reduced CBF conditions, presumably as a consequence of narrowing of the capillary and postcapillary bed. Accordingly, in a group of 15 severe head and brain injury patients we found ICP rates in the normal/upper normal range for days, whereas patients suffered brain death within 1-3 days whenever the CBF rates eventually fell to 20 ml/100 g/min or below.

The situation is absolutely clear with CBF rates around 10 ml/100 g/min or below. Such rates, as is common knowledge, are incompatible with life.

BROCK et al. [1], KOHLMEYER [2], and ZWETNOW [5] have already described the xenon-133 clearance curve, which is distinguished by the fact that the highest counting rate after xenon inhalation or intraarterial/intravenous injection remains low. Only a minor dose of xenon reaches the brain, whereas an initial peak may be shown by extracerebral blood flow (external configuration of the clearance

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curve). In the case of brain death, xenon clearance itself is extremely slow, tending towards zero.

Contrast Scan

Proof of lack of cerebral perfusion by means of computer tomography involving intravenous contrast medium application is technically sophisticated. The detector system has a more than hundredfold sensitivity compared to conventional X-ray films, which is why it is possible to make minor differences in contrast visible. As with digital subtraction angiography, it is possible to provide safe intraarterial evidence of contrast medium that has been applied intravenously.

First of all a lateral radiograph needs to be produced. Scans have to be positioned in the region of the heart, the aorta, the lower and upper cervical region, and the base of the brain. Next plain reference scans for these positions have to be performed. While applying 100 ml of a protracted bolus-shaped intravenous injection of a nonionic contrast medium, scans at the same positions as mentioned above are repeated. Safe evidence of the intravenously applied contrast medium in the heart, the aorta, and the neck vessels may hereby be obtained. In the case of brain death, filling and visibility of vessels at the base of the brain does not occur. Brain death can be confirmed by means of a density dispersion curve.

If it is possible to find evidence of contrast medium enhancement in just one of the vessels at the base of the brain, the assumption of lack of cerebral perfusion must be abandoned.

Between December 1984 and April 1988 the method described above was applied to 27 of our patients who showed signs of clinical brain death. In two cases normal enhancement of vessels at the base of the brain was found and in three cases there was partial enhancement, so that in a total of five cases brain death could be definitely established only by clinical diagnosis and repetition of the contrast scan examination the following morning.

While contrast scan examination only answers the question of whether perfusion is still detectable, measurement of cerebral blood supply allows for a more quantitative evaluation, and with increasing brain edema it is possible to provide evidence of increasing deceleration of cerebral blood supply. Thus at an early phase, before brain death occurs, we can obtain an indication of the prognosis.

When measuring CBF the degree of sedation must be taken into account. In principle only measurement of a nonsedated patient is reliable, because studies with different kinds of sedative, like Etomidate, Fentanyl, and Miedazolan, in part have shown a considerable deceleration of CBF which may lie between 20% and 30%. In individual cases an increase in blood flow was also observed.

Summary

In summary it may be stated that criteria for the prognosis of severe head trauma need to be developed in view of overfilled intensive care units, expenditure minimization in health care, organ transplantation, and also the often senseless prolongation of the irreversible process of dying which is made possible by our advanced methods of therapy. The above-mentioned ICP/CBF measurements and contrast scan

examinations, in parallel with other methods, allow for an initial step towards the development of such criteria. But these first steps need to be more firmly established and further developed so that eventually they may become compulsory.

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Head Injuries – Evoked Potentials

Value of Multimodality Evoked Potentials in the Diagnosis of Skull/Brain Injuries in Neurosurgical Intensive Care Units

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Introduction

The value of evoked responses has been investigated in recent years. The initial euphoria has disappeared. Publications concerning the value of evoked responses in neurosurgical intensive care patients are not frequent [4]. Therefore we analyzed the follow-ups of such patients in order to learn something about the advantages of this method.

Patients and Methods

Follow-ups were done in 135 patients subsequently treated in the neurosurgical intensive care unit in the Neurosurgical University Clinic, Essen. Fifty-seven of them suffered from skull/brain injuries. The details of this patient group are shown in Table 1.

Table 1. Characteristics of the analyzed skull/brain injured patients

Total number	57	
Average age (years)	36	
Duration of unconsciousness		
Average (days)	9.5	
Median (days)	8	
Mortality	31	(54.4%)
Coma stages (Brussels) on admission		
0	13	(22.8%)
I	11	(19.3%)
II	17	(29.8%)
III	12	(21.1%)
IV	4	(7 %)
Glasgow Coma Scale (GCS) on admission		
3	11	(19.3%)
4-6	35	(61.4%)
7-10	9	(15.8%)
11-15	2	(3.5%)

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The parameters and settings of evoked potential monitoring have been reported elsewhere [4]. We differentiated between normal potentials (I), slight potential deformities and prolonged latencies (II), marked potential deformities (III), and loss of the response (IV). In this way, we achieved a practicable differentiation for daily work.

Results

Severity of Injury and Alterations of Evoked Responses

There was no connection between the severity of trauma and the degree of alteration of the evoked responses, nor was there a correlation between the coma stage and alterations of electrical phenomena.

Special Clinical Situations and Evoked Potential Monitoring

Crisis of Intracranial Pressure (ICP). In two of four patients with rising ICP we found normal acoustic evoked responses whereas the other modalities showed abnormal configurations. We have pointed to the connections between ICP and visual evoked potentials elsewhere [3]. The beginning of alterations of brain stem acoustic evoked responses (BAERs) and somatosensory evoked potentials (SSEPs) apparently depends on the degree of transtentorial herniation [6]. The follow-up detected the beginning of the pathophysiological mechanisms.

Brain Stem Lesions. Among 135 investigated neurosurgical intensive care patients we found 40 with brain stem disorders with extension mechanisms and disorders of brain stem reflexes. Twenty-one of the cases were due to brain trauma. All patients belonged to coma stage III (corresponding to GCS 4-6). Only seven of them had brain stem lesions displayed by computed tomography. In 12 patients the brain stem disorders were secondary to the trauma. The outcome was poorer than in the rest of the group; their coma lasted longer than in the other patients who survived. All patients with brain stem lesions had alterations of BAERs except one. Thirteen had no acoustic responses, and eight of them died. The alterations were: disappearance of waves III and V always with side differences. Pathological findings in the m-SSEPs (somatosensory responses evoked by median nerve stimulation) were not so frequent (7 of 21).

Apallic Syndrome. Characteristic constellations in the apallic syndrome were missing. We could not recognize any connections between evoked responses and clinical stage.

Brain Death. The value of evoked responses in diagnosing brain death was emphasized by the German Bundesärztekammer [1]. We have commented on this problem elsewhere [2,5]. The difficulties in derivations and interpretation of the results must be carefully considered.

Evoked Responses, Follow-ups, and Prognosis

Follow-ups were necessary in order to update neurophysiological information. The more severe the initial alterations had been, the

Table 2. Correlations between alterations of evoked responses and outcome in skull/brain injured patients

Outcome (GOS)			1	>1
FEPs	Normal	I	20	24
	Patholog.	II/III	10	1
	Loss	IV	0	0
BAERs	Normal	I	11	19
	Patholog.	II/III	9	4
	Loss	IV	11	2
SSEPs	Normal	I	13	17
	Patholog.	II/III	6	6
	Loss	IV	9	0

poorer was the outcome. Of 20 patients with a loss of BAERs, 13 died; all those with a loss of SSEPs (11) did so, too. The relation between evoked response abnormalities and prognosis is shown in Table 2.

Discussion

The results of 57 analyzed follow-ups in brain-injured patients in the intensive care unit (ICU) were surprising, because no connection could be found between the degree of potential abnormalities and coma stage in general. In brain stem lesions, however, BAERs were a sensitive method of diagnosing lesions, even those not visible on CT scans. m-SSEPs were found to be more stable. Whenever there are alterations in the SSEP, the lesions may also be in the pathways to and in the cortex, so that the interpretation can be difficult. Marked alterations of both modalities indicate a fatal prognosis. Though our experience with motoric responses is still small, it shows that they disappear very early and cannot contribute to a differentiation between coma and brain death. The great value of evoked potential monitoring consisted in estimating the outcome at an early stage after admission. Especially in patients with interfering drugs, evoked potential measurements were useful. Even in these patients pathophysiological mechanisms (increasing ICP, herniation) could be recognized.

Summary

Evoked potentials in patients with skull/brain injuries can contribute to staging the patient on admission. The method provides information on the function of the neural pathways and on pathophysiological mechanisms and alterations during therapy (especially in the drug-treated patient). It is of prognostic importance at an early stage, and is also of value in diagnosing brain death.

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Somatosensory Evoked Potentials: Diagnostic and Prognostic Value in Head Injuries

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Introduction

The treatment of severe human head injuries requires investigative methods to assess the clinical status, to monitor the development of the disease, and to facilitate a prediction of outcome. In addition to the neurological examination, to anatomical studies by CT or MRI, and to recordings of EEG and intracranial pressure (ICP), evoked potentials (EPs) offer further possibilities for these requirements after head injury. In nearly all patients somatosensory evoked potentials (SEPs) are of greater prognostic value than brain stem auditory evoked potentials (BAEPs) [1,6] or visual evoked potentials [3]. Also a combination of these EP methods can be used [3].

Patients and Methods

Within the last 30 months 71 patients with severe head injury (Glasgow Coma Score ≤ 8 for at least 24 h after trauma) have been studied. SEPs were recorded from the neck and the contralateral scalp after stimulation of the median nerves over the wrist. From these waves the central conduction time (CCT) and the amplitude ratio of the primary cortical and the cervical response (AR) were calculated (Fig. 1). SEPs were considered to be pathological when CCT was ≥ 6.6 ms or AR <0.3. In several patients continuous ICP and EEG monitoring and repeated transcranial Doppler sonography (TCD) were performed. The outcome was classified according to the five grades of the Glasgow Outcome Scale (GOS). Two patients died of unrelated causes (excluded from this series) and two patients were lost to follow-up.

Results

Patients who suffered from subdural hematoma and those older than 45 years had a significantly worse outcome than others. The CCT and AR showed a good correlation to the coma grade. Especially in the groups of patients with GCS 3-4 and GCS 7-8, a convincing level of significance was seen (Fig. 2). There were only small differences between the electrophysiological potentials from the better and from the worse hemisphere. However, the CCT differences between both hemispheres did not seem to have any correlation to the clinical state or to the later outcome. The comparison between the SEP records and the simultaneously measured ICP and TCD showed no clear correlation either. The calculated CCT and AR were of reliable prognostic value in most cases (Fig. 3) but the predictive value sometimes varied during the course of the disease. When CCT and AR were in the normal

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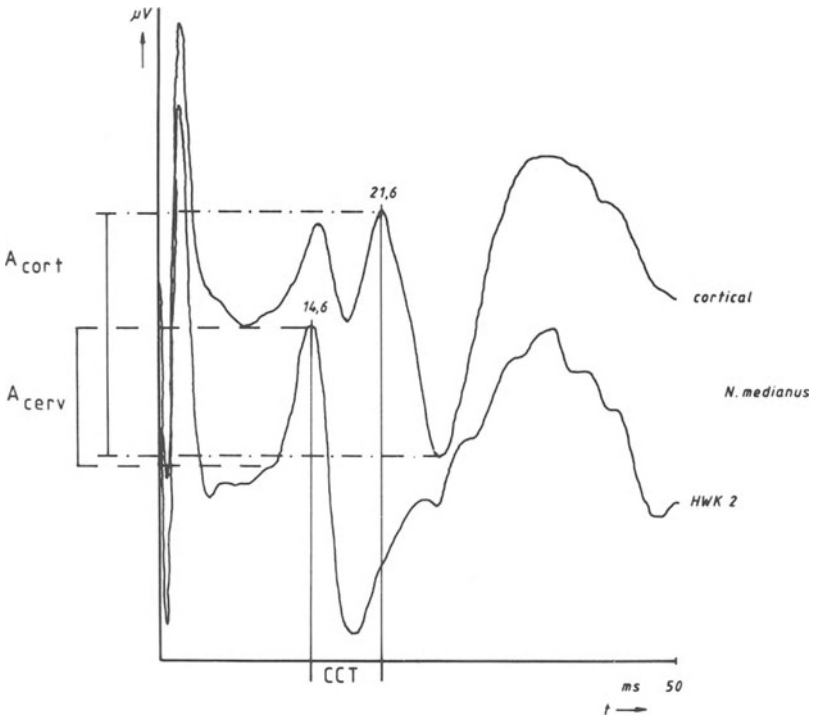

Fig. 1. SEPs after stimulation of the median nerve. Central (somatosensory) conduction time (CCT) = difference of latencies of the cortical N_{20} and the cervical N_{15}. Amplitude ratio = amplitude of the primary cortical response $N_{20}P_{25}$ divided by the amplitude of the cervical N_{15}, recorded over cerv. 2 (= HWK2)

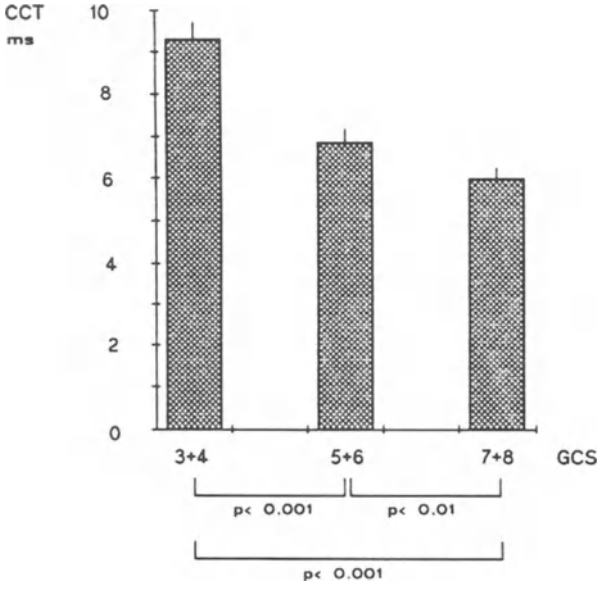

Fig. 2. Glasgow Coma Score (GCS) and CCT at time of recording. The CCT increases with worsening of the clinical state. Mean CCT (ms) and SEM

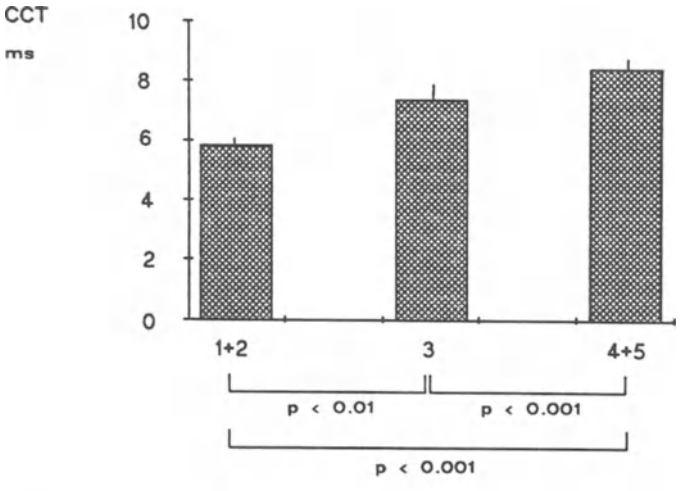

Fig. 3. Outcome (Glasgow Outcome Scale) and CCT. The CCT increases with worsening of outcome. Mean CCT and SEM

range at the last recording, 77.3% had a good outcome (GOS 1-2), 13.6% were disabled (GOS 3), and 9.1% (two patients) died. The reasons were peracute brain swelling in one case and cerebral infection in the other. Of the patients who had pathological CCT and AR, 81% died or remained in a vegetative state (GOS 4-5), 14.3% were disabled, and 4.8% had a good outcome. When only CCT was more than 6.6 ms, 81.1% had a fair or poor outcome. When only AR was less than 0.3 the percentage with a bad prognosis was 91.7% (P <0.001) (Table 1). In most patients repeated recordings were necessary, because in some cases SEPs changed with the clinical state. A secondary deterioration after initially good responses could be seen as well as an improvement of former pathological recordings. Those patients who had a cortical electrical silence of SEPs over one or both hemispheres had a poor clinical course, and most of them died. In these patients the normal cervical response gave evidence of intracranial disturbances and provided exclusion of peripheral lesions. However, in patients who underwent larger osteoclastic craniotomies EPs seemed to lose their prognostic value, i.e., three patients had normalized CCTs and ARs but only one patient had a good outcome 6 months after decompressive trepanation. On the other hand all patients who showed a cortical electrical silence preoperatively died or remained in a vegetative state.

Table 1. Prognostic value of the CCT and AR at the last recording in correlation to outcome (according to the Glasgow Outcome Scale). Highly significant differences are seen between the two groups

		Outcome		P
		1+2	3-5	
CCT	<6.6 ms	85.7%	18.9%	
	>6.6 ms	14.3%	81.1%	<0.001
AR	>0.3	55.9%	44.1%	
	<0.3	8.3%	91.7%	<0.001

Discussion

As previously reported [6], a close correlation does not exist between the CCT or AR and the simultaneously measured ICP in all patients, although a correlation could be shown by rapid inflation of a supratentorial balloon in an animal experiment [5]. The different pathophysiological patterns after brain injury, especially in cases with primary brain stem or midbrain contusions, may be one explanation. Regarding the results of 36 measurements, the differentiation into high pressure and low pressure coma may provide more information. This will be investigated in a larger series. In those patients who underwent decompressive craniotomy SEPs partly lost the correlation to coma grade and outcome. In the diagnostic instrumentation for brain death determination, the BAEPs are of greater interest in combination with the neurological state, EEG, and SEPs. In surviving patients, however, the BAEPs showed less prognostic value than SEPs [1,6]. SEPs are a powerful diagnostic method with a close correlation to the coma grade and with a highly significant prognostic value [1,3,4,6,7,8]. In combination with EEG, ICP monitoring, BAEPs, CT scans, and perhaps TCD, the SEPs provide the most reliable index of the severity of primary brain damage and can be used to assess patients and to monitor the development of the clinical course and of secondary brain damage. It has to be emphasized, however, that SEPs give significant statistical but not safe individual information. So recordings early after the injury may change due to different developments [1,3,6], e.g., brain swelling, and their prognostic value is not as high as that of later records. An early normalization of the SEPs is a favorable sign, while persistent bad responses or a fast deterioration usually predict a poor outcome [1,6,7]. The absence of cortical waves is an extremely bad prognostic sign. Such patients die or remain in a vegetative state [1,6,7].

Especially in deeply sedated and relaxed patients without the possibility of a clinical interpretation the SEPs improve the reliability of monitoring and are helpful in the practical management of patients with critical head injury. In our opinion, monitoring with SEPs should be done more routinely in these patients.

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Prognostic Significance of Somatosensory Evoked Potentials in Traumatic Brain Stem Lesions

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Introduction

Early CT in a case of cerebral trauma often shows no changes of the brain stem, and clinical evaluation is not rarely rendered more difficult by intensive therapy (narcotics, sedatives, relaxants, intubation). The prognostic evaluation of primary or secondary brain stem damage is thus problematic.

An objective function test is represented by the monitoring of evoked potentials. Previous studies [1,6,7,8,11,13,14,15] and our own observations have led us to examine the value of somatosensory evoked potentials (SEPs) on the basis of a large number of patients. In this survey the central somatosensory conduction time (CCT) seems of much importance. CCT is the time that the sensory triggered impulses from the sphenoidal or bulbar nuclear areas need to reach the primary somatosensory cortex. Pathological changes involve a prolonged or nonmeasurable CCT and occur uni- or bilaterally. Such changes can be caused by brain stem damage, as well as by hemisphere damage or a combination of both.

In this paper, SEP changes in patients with brain stem lesions and their prognostic value are examined.

Material and Methods

Eighty-six unconscious patients were examined after acute trauma with neurological signs of a brain stem lesion. The average age was 35.8 years (4-65 years). The patients were divided into two groups:

1. Patients with a primary acute brain stem lesion (n = 24), in most cases with an additional small contusion of cerebral foci. An extracerebral hematoma was evacuated immediately in two patients. In six patients no damage of the cerebrum could be demonstrated radiologically.
2. Patients with a secondary brain stem lesion, with an acute, traumatic, supratentorial space-occupying lesion (n = 62). The majority of these patients (n = 44) had acute or subacute extracerebral hematomas. The rest (n = 18) mainly showed a lateralized, cerebral contusion or hemorrhage with mass displacement and secondary transtentorial herniation. SEPs were monitored on admission in all patients, at the latest 10 h after injury.

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Early and cortical SEPs were evoked by percutaneous bipolar stimulation of the wrist median nerve (right angle impulse, duration 0.2 ms, voltage 40-100 V, stimulating frequency 5-8 Hz). 256-512 potentials were added and averaged (filter 200-1000 Hz, amplification 5-10 x 10^3). The cortical potential was derived transcranially from the contralateral somatosensory cortex (P3/P4), while the early potential was derived from the second cervical spine.

The "indifferent" electrical reference was a centromedian electrode (C_z). The patients' data were compared with those of normal persons (n = 40).

Results

In the control group the standard value varied from 4.5 to 6.3 ms ($\bar{x}$ ± 2 SD). A significant difference between both groups was not found. The cortical SEP was absent bilaterally in seven patients with primary brain stem lesions and unilaterally in three others. A pathological change was found in all cases of demonstrated cortical potentials. There was always a clear bilateral decrease in the amplitude and also a symmetrical latency increase in the cortical signal (Fig. 1). The early SEP was always present and usually normal. Slight asymmetry between the left and right side was found in amplitude and latency in only 15% of the cases. In bilaterally measurable CCT (n = 14) there was no certain asymmetry in the majority of cases (Fig. 2).

In secondary brain stem lesions (n = 62) the cortical SEP was absent bilaterally in 21 patients and unilaterally in seven others. Of the cases with demonstrable cortical potentials, 67% were found to have asymmetrical potential changes of the latency and/or the amplitude between the left and the right side (Fig. 3). In the majority of the remaining patients with a symmetrical cortical SEP there were no pathological findings. The early SEP was always demonstrable. In more than half of the patients (53%) it was normal on both sides. The amplitudes and/or the latency were asymmetrical in 27%. A bilateral symmetrical latency increase existed in most of the remaining 20%. A symmetrical amplitude decrease was rare (n = 2). In measurable CCT (n = 41) a clear time difference was found (Fig. 1). The measured CCT was compared with the clinical course for prognostic evaluation of

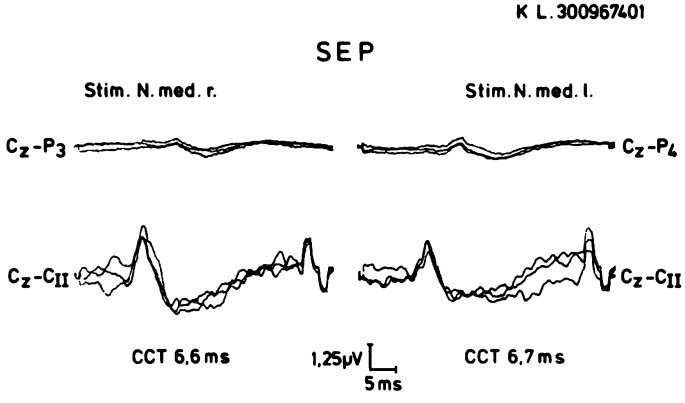

Fig. 1. Typical SEP changes in patients with secondary brain stem lesions

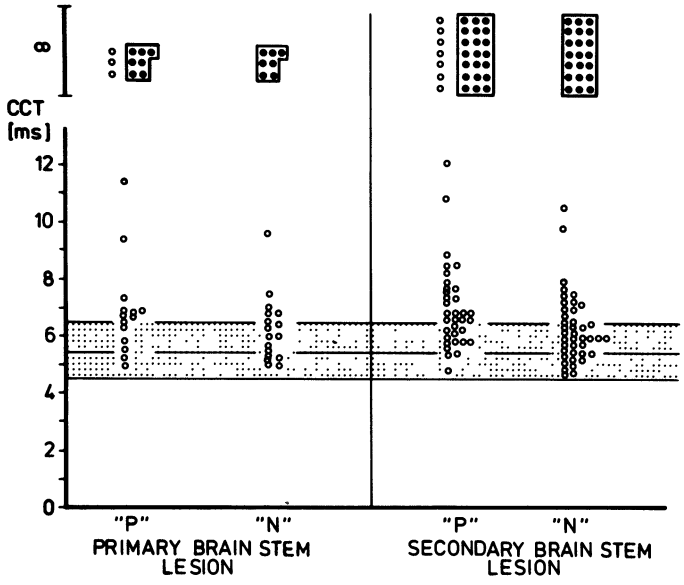

Fig. 2. First CCT findings in 86 patients with primary and secondary brain stem lesions. P, pathologically pronounced side; N, less pathologically pronounced or normal side. The black points stand for patients with a bilaterally absent cortical SEP

the first derived SEP. Concerning morbidity and mortality, only slight differences in SEP changes were found between the primary and secondary brain stem lesions. This is why both groups were chosen for the prognostic evaluation. Thus, the survival rate in patients with bilateral or unilateral normal CCT is higher than that in patients with bilateral pathological or absent CCT (Table 1).

Discussion

Some SEP investigations have been performed in small numbers of comatose patients with midbrain or bulbar syndrome as well as locked-in syndrome and central death [1,6,7,8,9,11,13,14,15]. The cortical SEP is mainly evaluated and the CCT is occasionally considered in comatose patients. All the authors suspect a negative prognosis in severe bilateral changes of the cortical SEP. Systematic SEP investigations with a larger number of patients have not been performed.

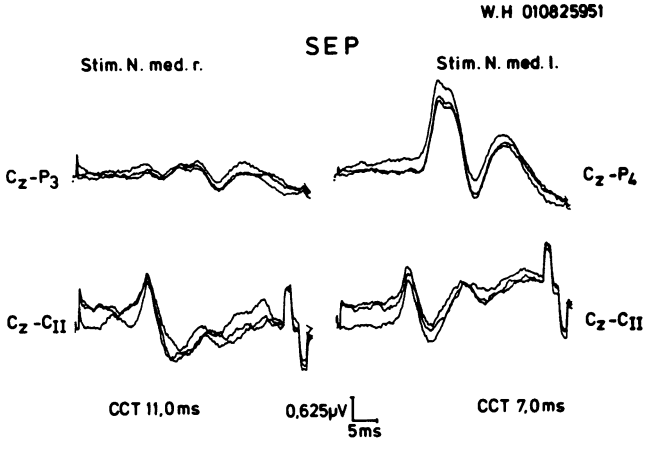

Fig. 3. Typical SEP changes in patients with secondary brain stem lesions

In our investigations of primary traumatic brain stem lesions, we found a symmetrical bilateral change of the CCT and cortical potentials in the majority of cases. In secondary traumatic brain stem lesions we found an asymmetry between the right and left side. One can therefore presume that there is always bilateral damage of the somatosensory tracts in primary traumatic brain stem lesions. On the other hand it seems that an acute progressing supratentorial space occupying lesion can lead to unilateral and in the further course to a bilateral functional impairment of the somatosensory tract due to transtentorial herniation.

The first SEP findings and the clinical outcome of traumatic brain stem lesions are closely correlated. CCT changes are of much significance (Table 1), and the prognostic value of the first findings appears very high. In the case of a bilateral absence of the cortical potential and the CCT, one could presume that the central brain stem region, and especially the reticular formation, is severely damaged, because the somatosensory tracts (lemniscus medialis) lie in the deepest brain stem layers.

Table 1. Comparison between the CCT results and the clinical outcome in traumatic brain stem lesions

CCT	No.	Recovered; moderately disabled	Severely disabled; apallic	Brain death	Other causes of death
N Normal P Normal	5	5	-	-	-
N Normal P Prolonged	25	23	-	-	2
N Prolonged P Prolonged	18	16	-	1	1
N Normal P Absent	6	4	1	-	1
N Prolonged P Absent	4	1	1	1	1
N Absent P Absent	28	-	3	25	-
Total	86	49 (57%)	5 (6%)	27 (31%)	5 (6%)

N, less pathologically pronounced or normal side; P, pathologically pronounced side

Summary

The early and cortical SEPs were monitored in 86 patients with primary and secondary brain stem lesions. The CCT was measured and compared with a healthy control group (n = 40). Changes in or absence of cortical potentials and CCT were compared with the clinical outcome. Of the investigated potentials, 24 belonged to the primary brain stem lesions and 62 to the secondary brain stem lesions. There was mainly a side-different cortical potential and CCT in secondary brain stem lesions, whereas in primary lesions there was no certain side difference in the results. In the primary as well as the secondary brain stem lesions we found a bilateral absence of the cortical signal and CCT in about one-third of cases. The survival rate is higher in bilateral or homolateral normal patients than in patients with primary bilateral pathological or absent CCT.

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On the Prognosis of Severe Head Injury Using Multimodal Evoked Potentials

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Introduction

Determination of the prognosis of patients with severe head injury is a very frequent topic of discussion and is still extremely difficult even after the advent of computer tomography. This is especially true of the first 2 days after injury, when the duration of unconsciousness, an important parameter of clinical progress [1], is not known.

Patients

The investigation was carried out on 93 unconscious patients suffering from severe head injury ranging in age from 3 to 94 years. The clinical course was monitored using multimodal evoked potentials. The first electrophysiological examination (initial findings) took place during the first few hours after arrival in the clinic, but no later than 48 h after the accident. Additional examinations were carried out during the comatose stage, on the average every 2nd day until maximally 48 days after the accident. In total there were 542 monitorings (301 AEP and 241 SEP examinations). SEPs, as well as AEPs, were recorded in 77 patients. The clinical condition was noted during each electrophysiological examination and the degree of coma was scaled according to FROWEIN et al. [1]. Of the 93 patients examined, 40 did not survive the accident; 48% died within the first 24 h after arrival in the clinic. Sixty-two of the 93 traumatized patients had closed head injuries, 27 had intracranial hematomas, and four had open head injuries.

Method

The examinations were routinely carried out with a Basis 8000 apparatus constructed by the Schwarzer Picker company. Contralateral deafening noises were stimulated monoaurally in an alternating pattern of 1500 suction-pressure clicks of 85 db-SL and a frequency of 10/s. The AEPs between the vertex and mastoid process were monitored ipsilaterally and contralaterally simultaneously with platinum needle electrodes. Only reproducible potentials were used in comparing the contralateral sides.

For the SEP monitoring the median nerve and/or the tibial nerve was stimulated with seven 90 grade impulses/s. The strength of the stimulus was always greater than 100 mV and on average 100 stimuli were

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measured. The lead was followed through the stimulated extremity to the appropriate area of the postcentral gyrus.

Results

When the clinical and electrophysiological results were compared it was noted that the normal findings decreased proportionately with increasing grade of coma and at the same time the pathological findings increased. This was true of both modalities. Sixty-six percent of the examined patients in coma I had normal AEPs; this figure was 40% in coma II and III and only 5% in coma IV.

Of the patients in coma I, 78% had normal SEPs; corresponding figures were 33% in coma II, 25% in coma III, and 6% in coma IV. No normal potentials could be obtained in the case of brain death.

We attempted to determine the chance of survival with the aid of evoked potentials during the initial examination. The lethal outcome of normal patients and those without potentials was examined. Figures 1 and 2 show the lethal outcome in the various groups of coma. Lethal outcome in those with initially normal AEPs is shown in Fig. 1. None of the examined 19 injured patients with initially normal AEPs in coma I died. However, as can be seen in Fig. 2, three injured patients of the 18 with initially normal SEPs in coma I did die.

Figures 3 and 4 show the lethal outcome in those patients with an initial lack of potentials. All patients having a lack of AEPs initially died regardless of the grade of coma. In contrast some patients in the group with an initial lack of SEPs did recover: two out of three patients in coma IV recovered from the trauma even though they lacked SEPs initially. In contrast to the initially extinguished AEPs, it appears that the initially extinguished SEPs are still reversible.

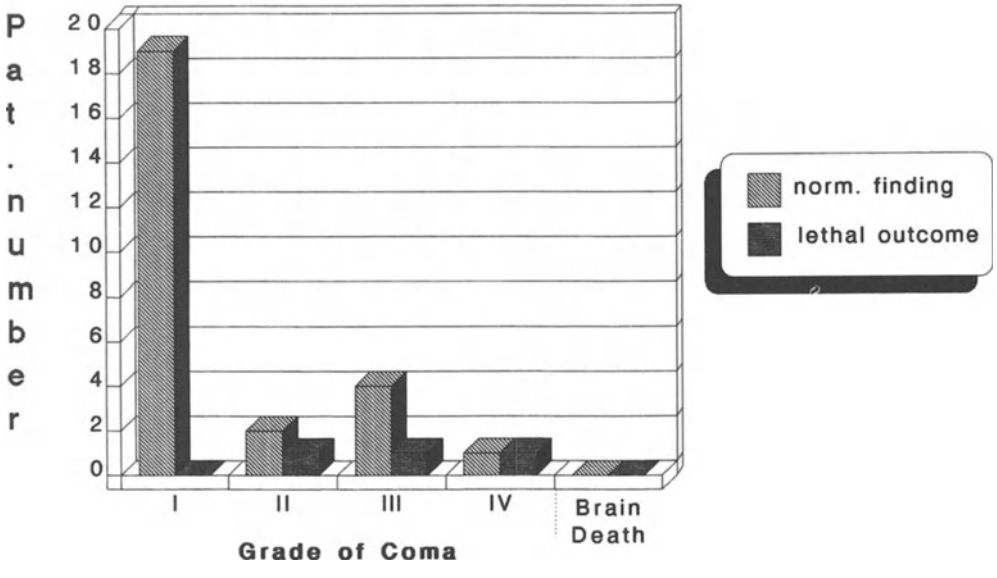

Fig. 1. Lethal outcome with initially normal BAEPs

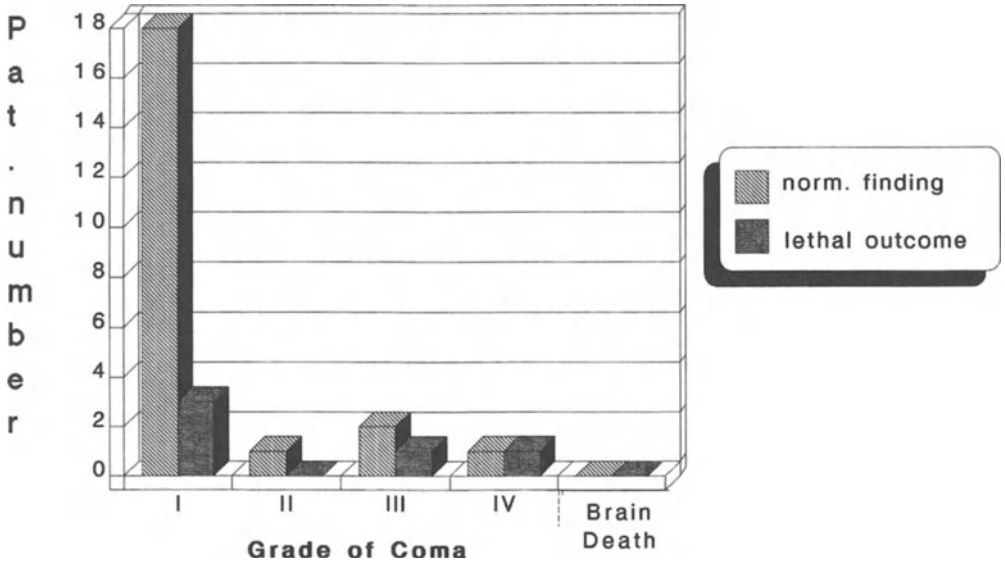

Fig. 2. Lethal outcome with initially normal SEPs

It seems that the prognosis for normal AEPs is quite favorable. Of the 26 patients with normal AEPs, only three died. The prognosis of the patients in coma III who had normal potentials in both modalities did not seem all that unfavorable either, as one might tend to believe when only taking the clinical aspects into account. Two of the six patients in coma III died.

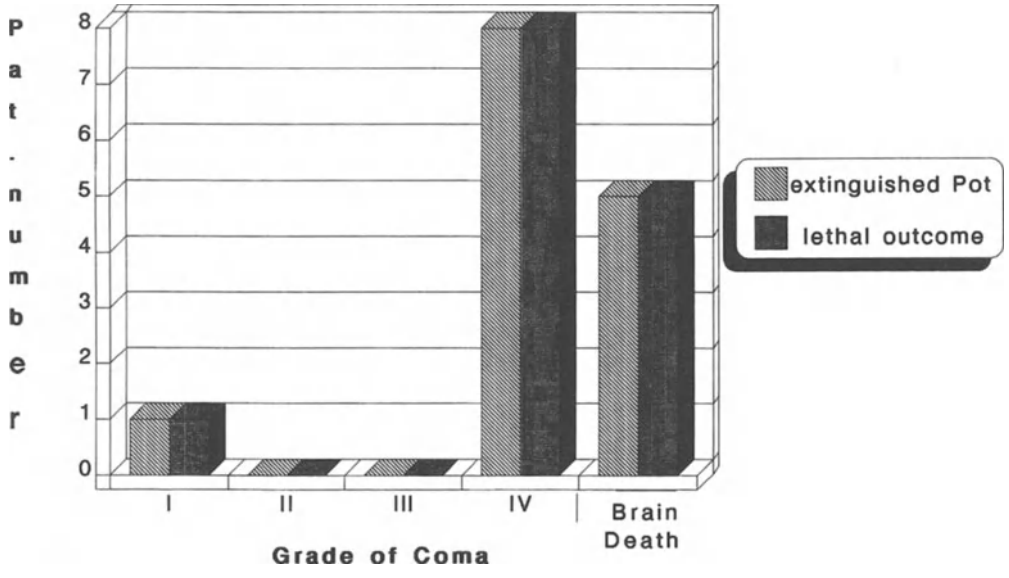

Fig. 3. Lethal outcome with extinguished potentials, BAEPs

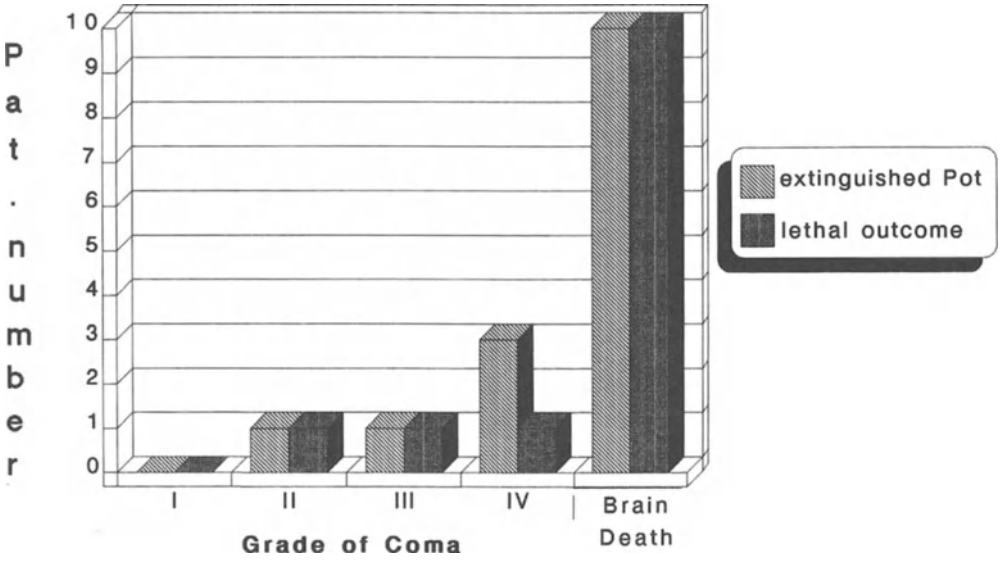

Fig. 4. Lethal outcome with extinguished potentials, SEPs

The potentials from 11 patients in our study could not be used since interference made either a lead or a definite interpretation impossible. The method is limited by the following types of interference:

In obtaining the AEPs it is possible that:

1. A hearing deficiency (because of a fracture of the petrosal bone, hematotympanon, or presbyacusis) hampers the stimulation.
2. Artifacts appear in the leads due to motor excitement or insufficient placement of the electrodes in wounds, drains, etc.

In the case of the SEPs:

1. Encasted or cannulized limbs can impede the stimulation.
2. Artifacts can arise in the same way as with the AEPs. In addition, medications can influence the potentials strongly.

Discussion

A brain stem lesion is responsible for the clinical condition in many posttraumatic comatose patients. According to RUMPL et al. [6], the brain stem lesion determines the outcome of the patient for the most part. The evoked potentials give dependable information as to the brain stem function - each modality respective of its path throughout the brain stem.

Our electrophysiological findings are in accordance with observations by other authors [2,3,4,7] who believe in the prognostic value and the assessment of traumatic injuries by measuring evoked potentials.

Conclusion

The AEPs are, as PAPANICOLAOU et al. [5] called them, a "predictor of survival" and are more dependable than the SEPs. Normal AEPs in coma I give a favorable prognosis in regard to the chance of survival. A total lack of AEPs on both sides indicates a bad prognosis because bilaterally extinguished AEPs cannot be survived. Initially extinguished AEPs seem to be irreversible. In contrast SEPs are reversible and are not to be regarded as so prognostically unfavorable.

In attempting to give a dependable prognosis it is essential that the electrophysiological findings be evaluated together with the clinical findings.

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Isolated Traumatic Lesions of Ventricular and Periventricular Regions and Cerebral Midline Structures: Outcome Prediction by CT Scan, Evoked Potentials, and ICP Monitoring

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Introduction

Identification of reliable prognostic indicators for patients with severe head injury is of importance to match control and treatment. In addition to clinical data the recording of anatomical parameters (CT scan, MRT) and functional data [ICP, evoked potentials (EPs)] is common [1]. We examined the predictive value of these data in the small group of patients with traumatic lesions of ventricular and periventricular regions and cerebral midline structures.

Patients and Methods

Between January 1983 and January 1988 1200 patients with severe head injury were treated. Twenty-three patients (1.9% : 14 males, 9 females; aged 3-66 years, median 28.3 years) showed isolated traumatic lesions of ventricular and periventricular regions and cerebral midline structures, i.e., lesions of the brain stem, basal ganglia (Fig. 1), corpus callosum, thalamus, capsula interna, and the ventricles themselves. Combined lesions of white matter or cortex and midline structures were excluded from the analyzed group. The clini-

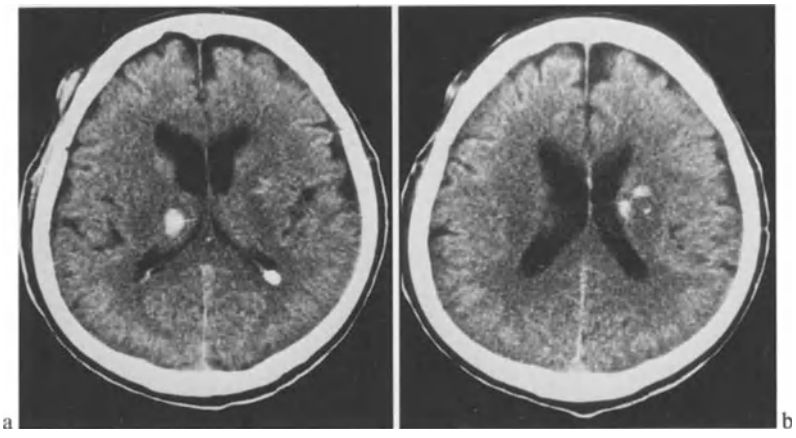

Fig. 1 a, b. CT scans on day of accident in a 66-year-old male with bilateral contusions in the basal ganglia. The patient survived in a vegetative state

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cal conditions were graded by GCS score (group I, 3-6; group II, 7-10; group III, 11-15). The outcome was classified as follows: dead, severe disability or vegetative state, and good recovery. All patients underwent CT scan (Siemens DR 2) and ICP monitoring (Ladd or Gaeltec system) performed on the day of injury. The EPs were recorded by a transportable 4-channel unit (Ca 1000, Nicolet).

The AEP lesion patterns were graded as follows [2]:

Grade I : uni-or bilateral V-waves, III-V or I-V missing = worst prognosis
Grade II : reduced or delayed V-wave bilaterally = uncertain prognosis
Grade III : reduced or delayed V-wave unilaterally = favorable prognosis
Grade IV : normal AEPs = good prognosis

The SEP lesion patterns were graded as follows [2]:

Grade I : bilaterally provoked answers N_{20}/P_{25} missing = bad prognosis
Grade II : N_{20} unilaterally delayed/or reduced, contralaterally reduced or missing = uncertain prognosis
Grade III : N_{20} unilaterally normal, contralaterally reduced, delayed or missing = favorable prognosis
Grade IV : bilaterally normal medianus SEP = good prognosis

Results

The GCS scores at admission, the CT diagnoses, and the clinical outcomes are shown in Table 1.

The traumatic cerebral lesions localized by CT scan are related to the posttraumatic interval before CT diagnosis (Fig. 2). If the CT diagnosis was delayed, all initial CT scans seemed normal. Continuous ICP monitoring in no case showed pathological pressure waves. The mean value achieved was 14.86 mmHg ± 5.29 (min.8, max. 26 mmHg). The ICP could not be used to predict an unfavorable outcome. The correlation between neurological outcome and prediction based on EP data is shown in Table 2. SEP data performed well as a prognostic indicator in predicting an unfavorable as well as a favorable outcome. The patients with a favorable EP-based prognosis who died suffered from extracranial complications.

Discussion

Among the common prognostic parameters of severe head injuries the EPs are of great value in predicting outcome in the small subgroup of cerebral midline lesions. On admission to the clinic there is a great difference between the poor neurological conditions and the often seemingly normal CT scans. In only 6 of 23 patients was a diagnosis of contusional midline lesion made by CT scan on the day of the accident. Space-occupying signs were never present on the CT scan. On this basis there is no development of elevated ICP (mean 14.86 mmHg). CT recording of midline structure lesions could only predict a severe course, but not recovery or death. Compared with predictions based on AEPs, CT prediction of survival seemed of use only in brain stem con-

Table 1. GCS score group[a] at admission, clinical outcome, and CT diagnosis (n = 23 patients)

	Clinical outcome		
	Dead	Severe deficit/ vegetative state	Good recovery
Brain stem (n = 6) (Group 1: 6)	5	1	0
Bilat. basal ganglia (n = 5) (Group 1: 5)	1	4	0
Thalamus (n = 3) (Group 1: 1 Group 2: 2)	1	1	1
Nucleus caudatus (n = 3) (Group 1: 1 Group 2: 1 Group 3: 1)	0	1	2
Intraventricular bleeding (n = 3) (Group 1: 1 Group 2: 2)	2	1	0
Capsula interna (n = 1) (Group 1: 1)	1	0	0
Corpus callosum (n = 1) (Group 2: 1)	0	1	0
Nucleus lentiformis (n = 1) (Group 2: 1)	1	0	0
Total	11 = 47.9%	9 = 39.1%	3 = 13.0%

[a] GCS group 1: score 3-6; GCS group 2: score 7-10; GCS group 3: score 11-15

tusions. In lesions of the nucleus caudatus a relatively favorable prognosis is justified (two of three patients with good recovery).

Combined prognostic prediction by GCS grading and CT scan seems of greater value in midline lesions than in brain contusions in other locations characterized by secondary development of space-occupying edema. It is unnecessary to monitor the ICP; CT scan offers the initial possibility of excluding further mass lesions. There is a significant improvement in outcome prediction when EPs are recorded than when only neurological examination, ICP, and CT scan are used. AEPs are reliable predictors of an unfavorable course but not a favorable

Table 2. SEP/AEP outcome prediction and neurological outcome

AEP prediction
(based on interpeak latency I-VII, III-V, I-V; amplitude ratio V/I)

AEP prediction	No. of patients	Neurological outcome		
		Good recovery	Severe deficit/vegetative state	Dead
Grade 1 (worst)	3	0	0	3
Grade 2 (uncertain)	1	0	1	0
Grade 3 (favorable)	3	0	1	2
Grade 4 (good)	16	3	7(!)	6(!)
Total	23	3	9	11

SEP prediction
(based on central conduction time N13b-20; amplitude ratio $\frac{N20/P25)}{N13b}$

SEP prediction	No. of patients	Neurological outcome		
		Good recovery	Severe deficit/vegetative state	Dead
Grade 1 (bad)	5	0	2	3
Grade 2 (uncertain)	13	2	6	5
Grade 3 (favorable)	2	0	1	1[a]
Grade 4 (good)	3	1	0	2[a]
Total	23	3	9	11

[a] Extracerebral lethal complications

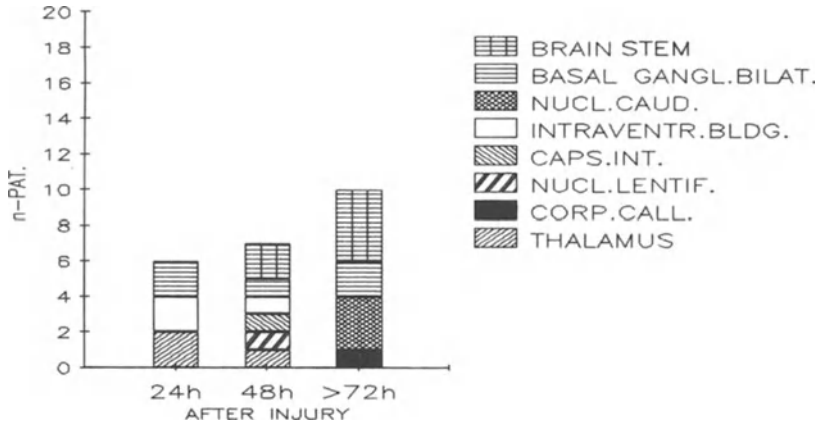

Fig. 2. Posttraumatic time interval before CT diagnosis of cerebral lesions in periventricular regions and midline structures

outcome (all three patients with AEP grade I died). SEP data, however, predicted an unfavorable as well as a favorable outcome. The favorable SEP predictions were nevertheless unrelated to extracerebral complications leading to poor or terminal outcome.

Conclusion

1. The CT identification of isolated cerebral midline lesions often succeeded only after subsequent CT scan controls and does not offer certain prediction of the outcome.
2. ICP monitoring also does not represent a therapeutic or prognostic indicator.
3. EPs partially fill the diagnostic and prognostic gap.

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The Prognostic Importance of Somatosensory Evoked Potentials, Computed Tomography, and Clinical Findings in Severe Head Trauma

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Introduction

Somatosensory evoked potentials are generally thought to be a good predictor of outcome in severe head trauma [2,5,7,10,13,16]. However, in a recently published analysis [17] we stated that this is not valid for the elderly patient, who has a poorer prognosis. This difference to the reports of other investigators may be interpreted as possibly being caused by differences in the studied trauma population, for data about age distribution, lesion type, etc. are often incomplete in electrophysiological papers. However, prognostically favorable or hopeless combinations of age and electrophysiological findings could be outlined, proving that estimating outcome requires a multifactorial approach. Therefore we performed a multiple linear regression to elucidate the relative prognostic importance of clinical, electrophysiological, and CT findings.

Clinical Material and Methods

Analysis was carried out using the data of 64 severely head-injured patients admitted to our hospital between June 1982 and September 1987. The distribution of some clinical data and of the outcome is shown in Table 1. The complete list of clinical, evoked potential, and CT findings assessed after the trauma is depicted in Table 2. They are called "input variables." Some parameters could be measured directly in centimeters or milliseconds (e.g., central conduction time or the size of hematomas); others had to be numerically coded in a grading system. As far as possible existing and widely accepted classification systems are used, for example the Glasgow coma scale [18] and the Greenberg scheme for evoked potentials [6]. The coding of the remaining variables is indicated in the table. Outcome, the dependent variable, was measured according to the Glasgow Outcome Scale [11].

The electrophysiological parameters are drawn from median nerve somatosensory evoked potentials. Stimulation and recording techniques correspond to the recommendations of the German EEG society. The information is reduced to the parameters listed in Table 2. The central conduction time is defined as latency difference between N20 and the main spinal component N13 [9]; in addition the amplitude ratio of the cortical primary complex (N20-P27) vs N13 was calculated. Computed tomography findings reflect the maximal diameter of the pathological processed recognized. No differentiation between intracerebral hematoma and contusion was done. The major part of the

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Table 1. Parameters characterizing the studied trauma population

I. Age distribution

Age	0-10	-20	-30	-40	-50	-60	-70	-80	>80
No.	2	14	14	11	6	10	1	2	0
%	3	22	22	17	9	16	2	3	0

II. Incidence of CT lesions

	EPI	SUB	INT	AXIAL	NONE
No.[a]	17	24	46	10	6
%	27	38	72	16	9

III. Outcome (GOS)

	GR	MD	SD	PVS	D
No.	28	13	15	5	23
%	13	20	23	8	36

CT findings: EPI, epidural hematoma; SUB, subdural hematoma; INT, intracerebral lesion, either hematoma or contusion; AXIAL, lesion in midline structures; NONE, no circumscript lesion detectable on CT

Glasgow outcome scale (GOS) classes: GR, good recovery; MD, moderate disability; SD, severe disability; PVS, persistent vegetative state; D, dead

[a] In patients with combined lesions each type was counted separately so the total number of cases exceeds 64

patient group had combined lesions (e.g., epidural hematoma plus contusion); each lesion type was separately assessed.

Statistics

Multiple linear regression is based upon the assumption that the dependent variable can be described by the addition of weighted input variables (Eq. 1).

$$Y = k_0 + k_1 \cdot X_1 + k_2 \cdot X_2 + ... + k_n \cdot X_n \quad (1)$$

However, the number of observed cases should exceed the number of input variables by at least three times, so in the concrete case a reduction is necessary. The task is facilitated by the bilateral presence of many observations which could be combined within one patient. This is called a linear combination and creates a new variable. Besides the addition of parameters measured on both sides, such linear combinations are done for pupil size and light reaction and for the size of the different types of CT lesions. Because it is well known that for equal sized lesions their location determines

Table 2. Variables assessed after trauma

I. Variables measured initially
A. Clinical parameters
Age
Glasgow coma scale (GCS) value
Pupil size (1 = normal, 2 = narrow, 3 = dilated, 4 = maximally dilated) and reaction to light (1 = present, 2 = missing)
Motor reaction (measured like in GCS)
Accompanying lesions (1 = limb fracture, 2 = chest or abdomen trauma, 3 = multiple extremity fractures, 4 = combination of chest/abdomen/extremity lesions)
B. SEP results
Greenberg's scheme of cortical responses
Central conduction time (CCT)
Amplitude ratio of the primary complex vs N13 (amplitude)
C. CT findings
Hematoma - contusion (epidural, subdural, intracerebral, axial)
Size of the lesion
Midline shift
Cisterns (0 = all present, 1 = cortical cisterns not present, 2 = basal cisterns not present, 3 = neither cortical nor basal cisterns present)
II. Outcome variable
Glasgow outcome scale (GOS) (5 = good recovery, 4 = moderate disability, 3 = severe disability, 2 = persistent vegetative state, 1 = dead)

prognosis, epidural, subdural, intracerebral, and axial processes are differentially weighted prior to combination. The weights are related to the mortality of patients with each lesion type. All data modifications and computation of the regression coefficients are done using SPSS Version X. A stepwise regression was performed excluding redundant variables.

Results

Because the variables entering the regression analysis should be independent, a correlation matrix was calculated showing that only a few parameters correlate with each other. These were the Glasgow Coma Scale value with the motor reaction and the Greenberg scheme value with the central conduction time. Due to the results of a probative run, motor reaction and Greenberg scheme value were removed from our model, too. Table 3 shows the result: only three parameters are necessary to predict outcome. These are ranked according to their relative importance (see BETA value): age, central conduction time, and the recognizable cisterns. The final regression equation is then:

Table 3. Original SPSSX result protocol showing the variables accepted in a stepwise regression procedure. The regression coefficients are listed in column B. The relative importance of the variables for the outcome is, however, better seen in column BETA

Equation number 1 Dependent variable: GOS

		Analysis of variance	df	Sum of squares	Mean square
Multiple r	0.73171				
Square	0.53539				
Adjusted r square	0.50298	Regression	3	43.53780	14.51260
Standard error	0.93736	Residual	43	37.78135	0.87864

F = 16.51719 Signif. F = .0000

Variables in the equation

Variable	B	SE B	Beta	Tolerance	T	Sig. T
Age	-0.050548	0.008138	-0.663294	0.947601	-6.212	0.0000
CCT	-0.093960	0.026631	-0.380704	0.928041	-3.528	0.0010
Cisterns	-0.274138	0.116776	-0.250370	0.949916	-2.348	0.0236
(Constant)	5.654868	0.463046			12.212	0.0000

$$\text{GOS value} = 5.6549 - 0.0505 \cdot \text{age} - 0.0939 \cdot \text{CCT} - 0.2741 \cdot \text{cisterns} \quad (2)$$

Discussion

A lot of initial observation after the trauma has been associated with outcome, but mostly univariate analysis has been performed. Interestingly, a former evaluation of our results [17] using cross tabs, as well as a set of univariate regressions performed as preparation for this study, showed no recognizable relationship between outcome and any input variable except age. Again we can only explain this by the composition of the studied trauma population because rather high correlations to input were achievable if we selected the cases below the age of 30. However, the age distribution and other parameters (see Table 2) are comparable to larger head trauma studies [1,4,12,15], so we feel our populations representative for brain injuries.

The existing multivariate studies [3,8,14] also differ in their judgment of prognostically important variables; however, age is always mentioned. The role of this parameter was again confirmed by our results, which rank it first among outcome predictors. The missing inclusion of other factors does not mean that they have no relationship to prognosis, but that they have only minor importance in the chosen model, which predicts outcome as the weighted sums of age, central conduction time, and the presence or absence of cisterns, which may be an indicator of raised intracranial pressure. For theoretical reasons this model may be criticized for its inclusion of rank scaled parameters (which in a simplification are assumed to be interval scaled). However, practical experience and especially the analysis of residuals, i.e., the difference between computed values using Eq. 2 and the observed values, showed no gross abnormality calling the model in question. The advantage of a linear regression is a more differentiated calculation of outcome which now will be tested prospectively.

Summary

By means of a linear regression analysis we determined clinical, electrophysiological, and radiological findings bearing a strong relationship to outcome in severe head trauma. Age, central conduction time in median nerve somatosensory evoked potentials, and the presence or lack of cisterns on CT scans are found to be the most important predictors. Advantages and disadvantages of the model used are discussed and a regression equation is formulated.

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Microsurgery

Anatomy in and on the Jugular Foramen

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Anatomical Nomenclature

In Roman times the clavicula and the groove above it were known as the "jugulum." "Jugularis" means the veins and lymph vessels which run in this area and also their course through the skull: the foramen jugulare, or jugular foramen.

Bony Structures

The jugular foramen is bordered by the petrous bone anteriorly and laterally and the occipital bone posteriorly and medially. In most skulls an incisura jugularis and a processus intrajugularis of the margo posterior partis petrosae are developed. Medial to this intrajugular process the janua arcuata is seen. Below and anterior to the dura-covered janua arcuata is the exit zone of the perilymphatic duct and in its neighborhood are accompanying vessels and small canals. On the posterior medial side of the foramen an intrajugular process of the occipital bone is present in 11% (LANG and SCHREIBER 1983). The medial border of the jugular foramen is surrounded by a bony spur of the occipital bone which is termed the processus hamatus. The results of our measurements of the foramen are shown in Fig. 1.

Shape

More than 50% of jugular foramina are shaped like a triangle; in about 35% of cases we found an oval type, and in about 12% foramina divided by bony bridges at different levels and different orientations. The entrance area to the jugular foramen is oriented from above and laterally to below and medially. An important borderline is the terminal sigmoid ridge, which has very different orientations and was sometimes found to be doubled. On the medial and anterior edge of the jugular foramen is situated the lower end of the inferior petrosal sinus in most cases. Its bottom area is a groove between the petrous and the occipital bone. In this area is situated the petrobasilar synchondrosis or suture, more laterally or sometimes medially in the groove of the inferior petrosal sinus. In about 6% in this area a canal was found in which the inferior petrosal sinus, the IXth cranial nerve, or both run together. The walls of this canal may be formed of the petrous bone, the occipital bone, or both together (Fig. 1). LYSSENKOW (1926) found canals between the sulcus sinus petrosi inferioris and the outer skull base at a distance of 5-15 mm from the jugular foramen in 2.6%. The postnatal enlargement and shifting are shown in Fig. 2.

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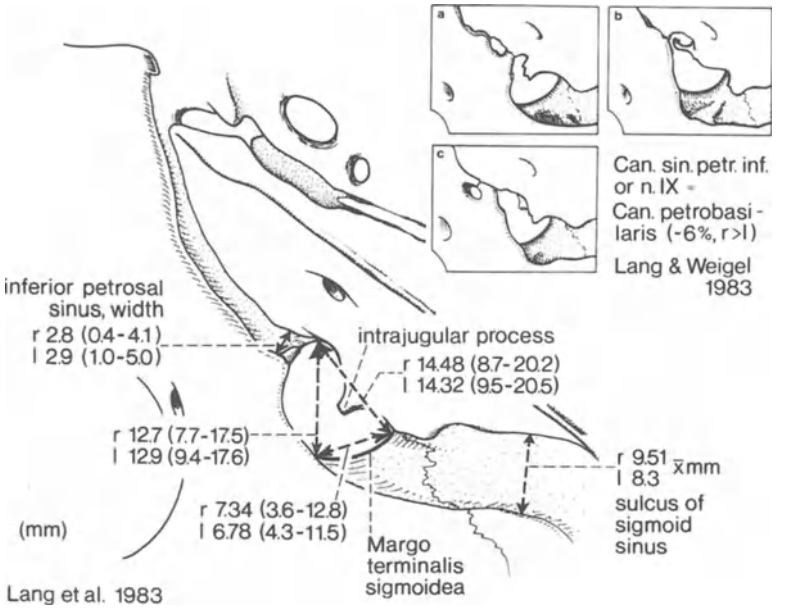

Fig. 1. Jugular foramen: measurements in adults

Dura Mater

The area of the jugular foramen, the sigmoid, and the inferior petrosal sinus is covered by dura mater (see Fig. 172 in KOOS et al. 1985). Medial to the margo terminalis sigmoidea the IXth-XIth cranial nerves reach the dura mater. The superior ganglia of the IXth and Xth cranial nerves are situated in small extensions of the dura mater and are reached by the arachnoid membrane at least on its upper surface. In most of the cases a dura guide plate is developed for the IXth-XIth cranial nerves which extends obliquely from anterolaterally to posteromedially and from the dura mater to the pericranium on the outer inferior skull base. In cases without larger bony spurs three different positions of the dural plate have been observed: The guide plate can be extended obliquely from anterolaterally to posteromedially with an anterior attachment medially to the intrajugular process of the petrous bone (Fig. 3). Less frequently the guide plate can be seen with an attachment on the intrajugular process of the petrous bone or the guide plate is formed in a sagittal direction and emerges from a region medial to the intrajugular process of the petrous bone and reaches the medial border of the margo terminalis sigmoidea.

In cases of completely divided jugular foramina, the bony bridges may be situated between the inferior petrosal sinus and the three nerves of the jugular foramen or between the IXth-XIth cranial nerves (see Fig. 146 in LANG 1983).

IXth-XIth Cranial Nerves and the Jugular Foramen

The exit end entry zone of the glossopharyngeal nerve was measured in our material as 1.8 (1-3) mm lateral to the exit zone of the facial nerve. It has a short central segment (LANG 1982); its lateral fiber

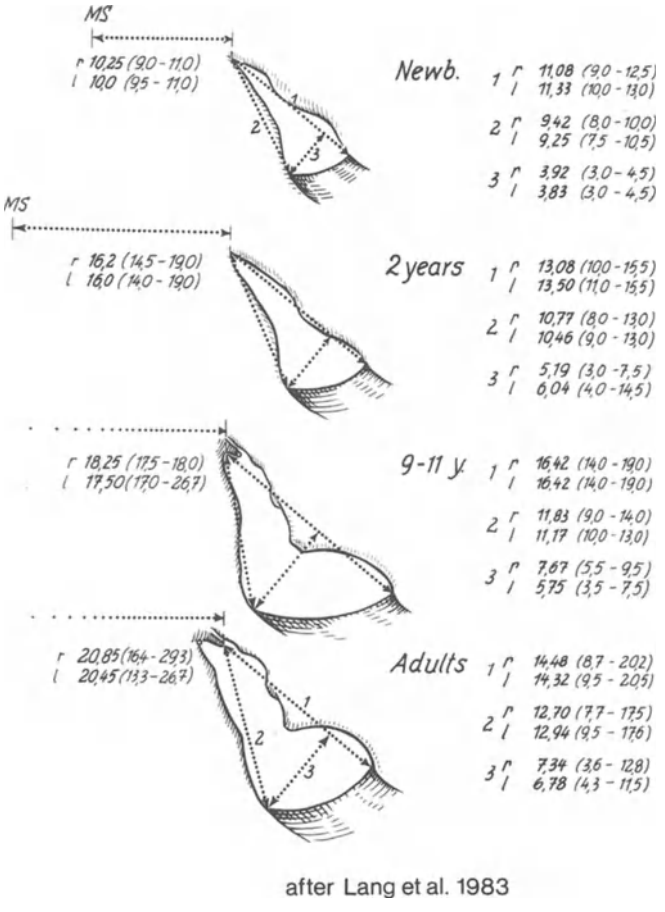

Fig. 2. Jugular foramen; postnatal shifting and enlargement. **MS**, median sagittal plane. (After LANG et al. 1983)

bundles are sensory and the medial ones motoric. The intracisternal length of the IXth cranial nerve was estimated between the brain stem and the dural pore to be 15.65 (10-20) mm (LANG and REITER 1985).

The Xth cranial nerve is built up by a mean of 8.6 (4-15) fiber bundles. Its intracisternal length is 15.5 (10.5-21) mm. On its dural pore is situated the superior ganglion of the vagus nerve.

The accessory nerve is composed of a spinal and a cranial part. We found a mean of 11 (6-16) cranial nerve fiber bundles which in the upper area had a mean length of 16 and in the lower area one of 23 mm between the brain stem and the dural pore and the jugular foramen area (Fig. 4).

Vessels

Arteries

Not infrequently a loop of the PICA is situated on the inner surface of the dura mater. Sometimes we found this vessel between the Xth and XIth cranial nerves or between the IXth and Xth cranial nerves. The

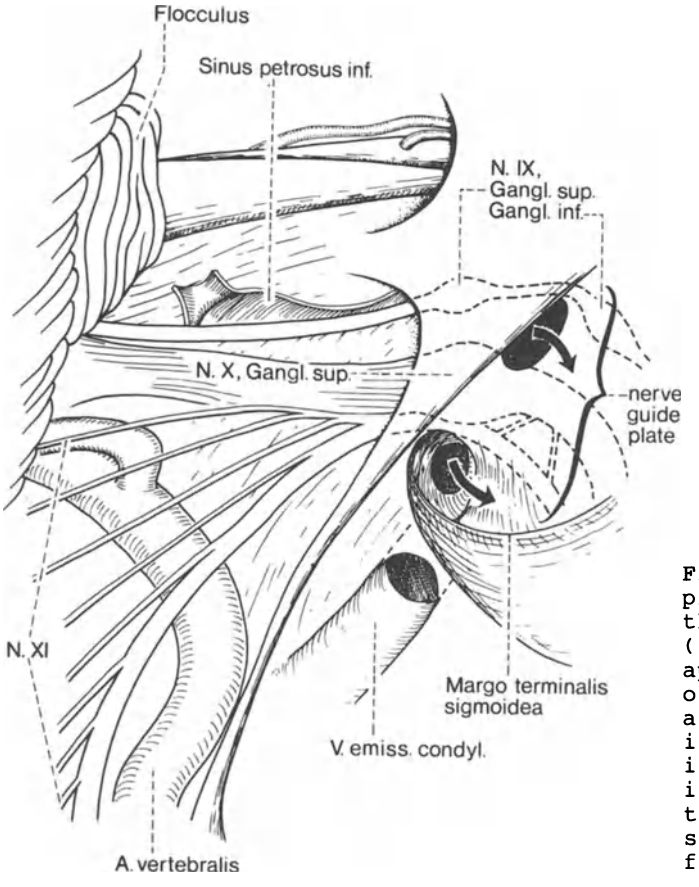

Fig. 3. Nerve guide plate in the area of the jugular foramen (seen by retrosigmoid approach). Dura mater of the sigmoid sinus and partly of the inferior petrosal sinus is removed. The **arrows** indicate the inflow of the inferior petrosal sinus in the jugular foramen

PICA may have 1.5 mm thick anastomoses with meningeal arteries of the bottom area of the posterior fossa (see Fig. 277 in LANG 1981). In 80% of cases the ascending pharyngeal artery has its origin on the external carotid artery or from a trunk (together with another artery), in ~13% directly at or in the immediate neighborhood of the carotid bifurcation (LANG and HEILEK 1984). Sometimes we found two or three ascending pharyngeal arteries. In about 11% of cases the artery has its origin on the occipital artery, in about 5% on the internal carotid artery, and in 1.6% on the facial artery (for details about the course and origins, see LANG and HEILEK 1984). Twigs of the artery were found to the pharynx, to lymphatic nodes, to the ganglion of the sympathetic nerve and IXth-XIIth cranial nerves, to the prevertebral muscles, to the carotid canal and the auditory tube, and to the external cranial base. There was also one twig to the jugular foramen in 50%, two twigs to this foramen in 37.5%, and three twigs in 10.5%. The mean diameter of these jugular foramen branches was 0.63 mm. In about 4% we found twigs of the occipital artery running through the jugular foramen to the inside of the posterior cranial fossa. In about 56% branches of the ascending pharyngeal artery ran through the hypoglossal canal.

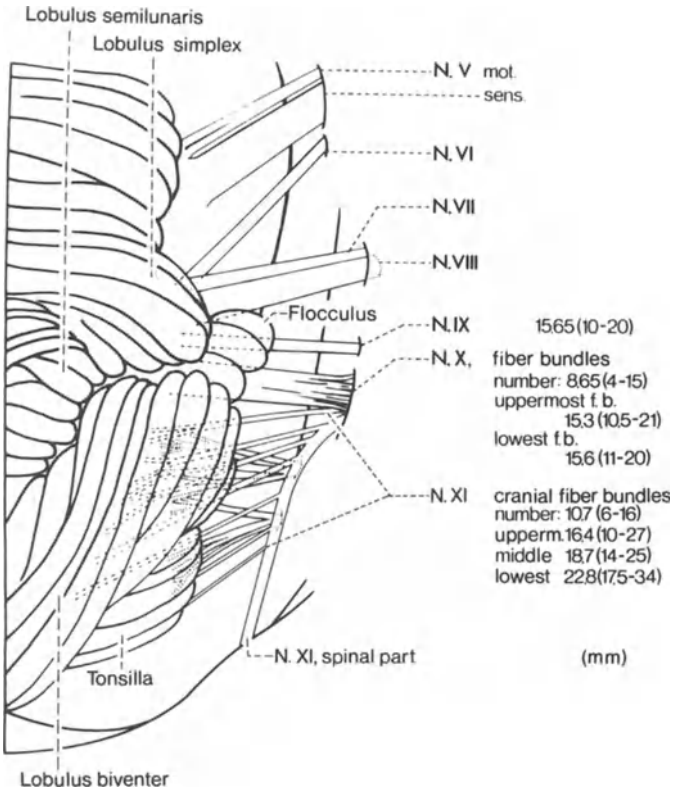

Fig. 4. Caudal cranial nerves in the area of the jugular foramen, seen by retrosigmoid approach. The length and number of the fiber bundles is shown

Sinuses and Nerves

In about 33% we found transcisternal veins to the area of the jugular foramen. In most cases these veins drain lateral to the IXth-XIth cranial nerves. In about 23% veins to the area of the hypoglossal area or the foramen magnum area were found.

The inferior petrosal sinus drains through a hole of the nerve guide plate in the upper area of the jugular bulb in about 9% (more often on the right than on the left), in the upper level of the jugular foramen in 11.5% (more often on the left than on the right), a little below this in about 16%, and below the cranial base in about 13%. In these cases the inferior petrosal sinus forms a vein which may drain in the inferior jugular vein up to 6 cm below the skull base.

In about 50% we found a medial and also in 50% a lateral intrapetrosal vein which connects the venous network around the internal carotid artery with the inferior petrosal sinus or the internal jugular vein (details see LANG and WEIGEL 1983). The inferior petrosal sinus reaches the area of the jugular foramen between the IXth and Xth cranial nerves in about 50%, anterior to the IXth cranial nerve in 30%, around the Xth cranial nerve in 16%, and between the Xth and XIth cranial nerves in 11%. Not rarely we found two pores in the nerve guide plate for the inferior petrosal sinus (see Fig. 3).

Lateral to the jugular foramen is found the bulb of the internal jugular vein. This bulb may be placed medially, below, or in the area

of the internal acoustic pore and meatus or laterally in the bottom area of the cavum tympani. It is placed 5-15 mm above the margo terminalis sigmoidea (higher on the right side than on the left). The venous plexus of the hypoglossal canal communicates in the jugular foramen area in about 18% (more often on the right than on the left), in about 17% with the inferior petrosal sinus inside the jugular foramen area. In the jugular foramen and its transition zone to the internal jugular vein in about 14% on the left side and in about 5% on both sides these veins of the hypoglossal canal and in about 6% on the right side the venous plexus of the hypoglossal canal did not drain in the internal jugular vein, but instead proceeded below the base of the skull backwards to the vertebral plexus.

The condylar emissary vein was found to drain into the superior bulb of the internal jugular vein in about 38% (more often on the left than on the right), in the area of the margo terminalis sigmoidea in 25% (more often on the right than on the left), into the sigmoid sinus in about 14%, and sometimes in the venous plexus of the hypoglossal canal or in the internal jugular vein. It is well-known that the condylar emissary vein is lacking in about 16%.

Dural Portals of the IXth-XIth Cranial Nerves

In our material (DAUSACKER 1974) the mean distance to the lower border of the internal acoustic pore was 4.52 (2.5-6.5) mm. Between the dural portals of the IXth and Xth cranial nerves we found a distance of 2-3 mm, and the mean distance between the portal of the XIth cranial nerve and the upper border of the XIIth cranial nerve was 11.4 mm. As regards the nerve cells, their function, and twigs of the superior and inferior ganglia of the IXth and Xth nerves, see LANG (1985) and Figs. 3 and 5. It should be noted that two to six

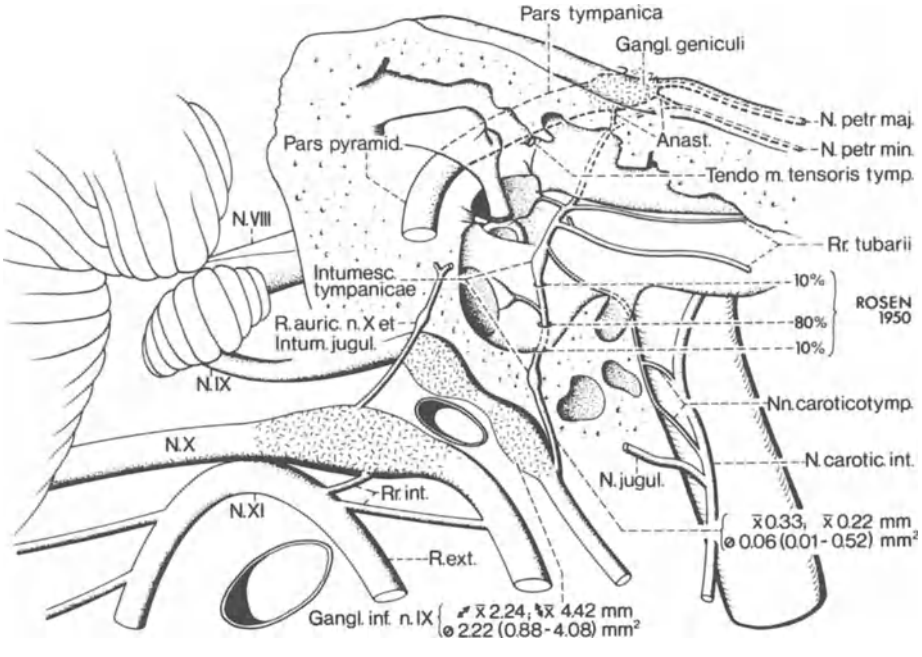

Fig. 5. Intumescences of the IXth and Xth cranial nerves

intumescences were found in the course of the tympanic nerve, the IXth cranial nerve, and the auricular branch of the Xth cranial nerve, and that in these intumescences a so-called chemodectoma (= glomus tumor) may be developed.

Syndromes

Villaret's syndrome (lesion of the IXth-XIIth cranial nerves, including Horner's syndrome) and Vernet's syndrome have been described (for details see LANG 1985).

In cases of intracranial lesions Collet-Sicard syndrome is often found. Jackson's syndrome (also intracranially) is a disturbance of the IXth-XIIth cranial nerves. Vernet's or Siebenmann's syndrome concerns only the jugular foramen area in its intracranial region (IXth-XIth cranial nerves), while Schmidt's syndrome is due to a lesion in the area of the external cranial portal to the Xth and XIth cranial nerves. Tapia's syndrome derives from a lesion of the Xth-XIIth cranial nerves (according to GEJROT 1968; MUMENTHALER 1973).

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Branchial Paragangliomas

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Introduction

Also called chemodectomas, glomic or neurochristopathic tumors, paragangliomas are second in frequency of all neurogenic tumors. They derive embryologically from neural crest cells. Almost half of them occur in the temporal bone, but they are often multicentric or associated with other neural crest tumors.

Paragangliomas best represent the multidisciplinary therapeutic strategy and technical aspects involved in the treatment of benign neoplasms at the base of the skull.

Epidemiology

Paragangliomas may be differentiated by their location: tympanic, jugular (these two being classified as temporal paragangliomas), carotid, vagal, laryngeal, nasopharyngeal, and orbital. Multicentricity is common, vagal and carotid paragangliomas being more likely to be multifocal than those in other locations. This multicentricity can be observed with malignant paragangliomas, as is secretory activity [6]. Vagal, carotid, and laryngeal paragangliomas have a malignant potential estimated at between 10% and 18%, whereas those in the temporal region have a lower incidence, at 3% [4].

Two groups of paragangliomas can be differentiated: those with a sex predominance (F/M ratio 2.5/1: temporal, vagal, nasal, nasopharyngeal lesions), and those without one (carotid, laryngeal, orbit lesions). There may also be a familial distribution with an autosomal dominant inheritance.

Vascular Architecture - Angiographic Findings

Small arteries are present in the capsule and septa of paragangliomas. This pathological consideration explains the compartmental arrangement that exists inside the tumor [5]: angiographically, a feeding artery superselectively injected will strongly opacify a quarter of the lesion with a specific venous drainage, each part representing a "puzzle piece" of the entire tumor. Different types of tumor can therefore be described: unicompartmental masses (with only one puzzle piece) and multicompartmental masses (with at least two puzzle pieces). Furthermore, the existence of a capillary bed and arteriovenous communications explains the rapid venous filling seen on angiographic studies. One must also consider that because they are

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encapsulated, branchial paragangliomas seldom recruit regional arterial feeders from other territories like other types of invasive or nonencapsulated tumor.

Two important points have to be emphasized:

- The ascending pharyngeal artery is the unique link between paragangliomas in the tympanic, jugular, vagal, carotid, and laryngeal locations as each of these territories is supplied by a different branch of this artery (Fig. 1).
- Jugular paragangliomas specifically develop inside the lumen of the jugular vein. Extrinsic jugular or carotid compression is not specific and can be due to other cervical masses.

Clinical Aspects - Semiology

Paragangliomas are slow growing tumors: the mean age of the patients presenting with this pathology is in the fifth decade. Two types of clinical symptoms should be distinguished: those due to the tumor itself and those due to its secretory activity.

The primary symptoms of paragangliomas are related to the location of the tumor. This allows three types of syndrome to be described:

1. **Tympanic paragangliomas:** The most prominent symptom is the presence of pulsatile tinnitus. It is unilateral and related to arteriovenous pulsation. Conductive hearing loss, otorrhagia, VIIth nerve palsy, and a reddish or bluish tympanic membrane complete the typical presentation that will enrich according to the tumoral growth.

2. **Jugular paragangliomas:** These usually present with jugular foramen syndrome. Nonpulsatile tinnitus and retroauricular pain may also be described. It is important to note that venous occlusion of the internal jugular vein usually has no specific clinical manifestations. Further extension occurs towards the tympanic bone or intracranially; the VIIth and lower cranial nerves may be involved [3,7].

3. **Cervical paragangliomas:** These present as expansile tumors often presenting as a cervical mass. They may extend into the parapharyngeal space [9], the oral cavity, or the larynx. Cranial nerve involvement is rare; if present, it affects primarily the XIIth nerve and the superior laryngeal nerve.

The most frequent presenting symptom in vagal paraganglioma is a mass in the cervical (78%) or pharyngeal space (44%); 30% of patients have cranial nerve impairment.

The endocrine activity of some paragangliomas may produce additional symptoms (in 5% of patients). These may be of different types: hypertension, headaches, palpitation, diaphoresis, or anxiety. Malignant vagal, carotid, and laryngeal paragangliomas are more likely to have endocrine manifestations [6]. It is also important to note that theoretically any paraganglioma has the potential to become malignant [1]. Because of this and because spontaneous regression of paragangliomas never happens, these lesions must always be eradicated whenever possible. This strategy, best achieved by surgery, must be

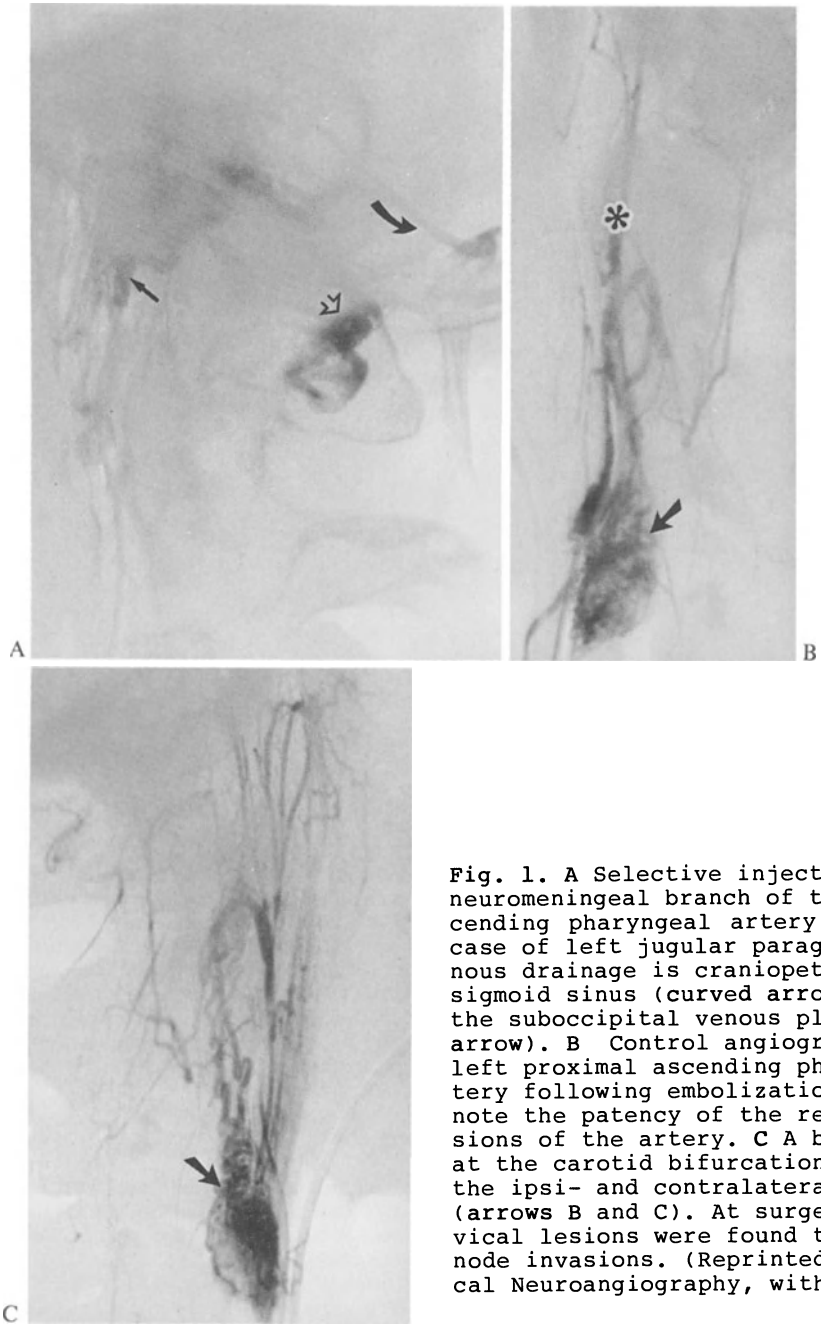

Fig. 1. A Selective injection of the neuromeningeal branch of the left ascending pharyngeal artery (arrow) in a case of left jugular paraganglioma. Venous drainage is craniopetal into the sigmoid sinus (curved arrow) and into the suboccipital venous plexus (open arrow). B Control angiogram in the left proximal ascending pharyngeal artery following embolization (asterisk); note the patency of the remaining divisions of the artery. C A blush is noted at the carotid bifurcation level during the ipsi- and contralateral controls (arrows B and C). At surgery both cervical lesions were found to be lymph node invasions. (Reprinted from Surgical Neuroangiography, with permission)

preceded by a precise morphological mapping that includes CT and angiography (diagnostic and, in the same session, therapeutic).

Supraselective angiography remains the most specific diagnostic technique and should always be performed. Ascending pharyngeal artery

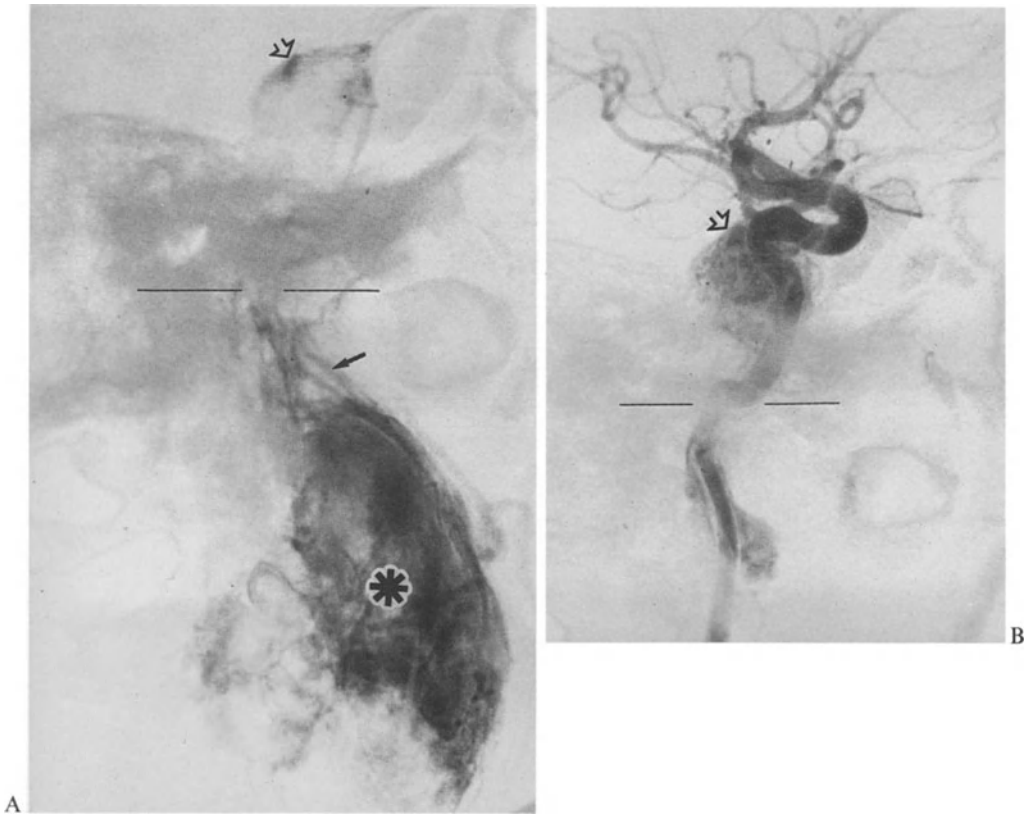

Fig. 2. A Selective injection of the pharyngeal branch of the right ascending pharyngeal artery (APhA) in a case of vagal paraganglioma (asterisk). Note the ascending drainage towards the cavernous sinus (open arrow) and the arterial carotid canal branches of the APhA (arrow). B Internal carotid injection. Note the clival blush (open arrow). The lesion was only embolized; no surgery and no radiation therapy were undertaken. Both lesions were stable after 2 years. Spinal metastasis became symptomatic a few months ago. A similar patient has previously been reported [4]; however, although immediately operated on following embolization, she developed diffuse metastasis 16 months later, from which she died. (Reprinted from Surgical Neuroangiography, with permission)

injection confirms or rules out the diagnosis of a suspected branchial paraganglioma: absence of the typical appearance of the tumor blush excludes that diagnosis. The classical angiographic aspect is constituted by moderately enlarged feeding arteries, intense parenchymal blush, and rapid venous filling (Fig. 1). Although angiography is the diagnostic procedure of choice in cases of paraganglioma, it remains imprecise in differentiating malignant from multicentric disease (Fig. 2). Some specific features may nevertheless be distinguished:

1. **Temporal paragangliomas:** The ipsilateral vertebral, internal carotid, distal external carotid, posterior auricular, and occipital

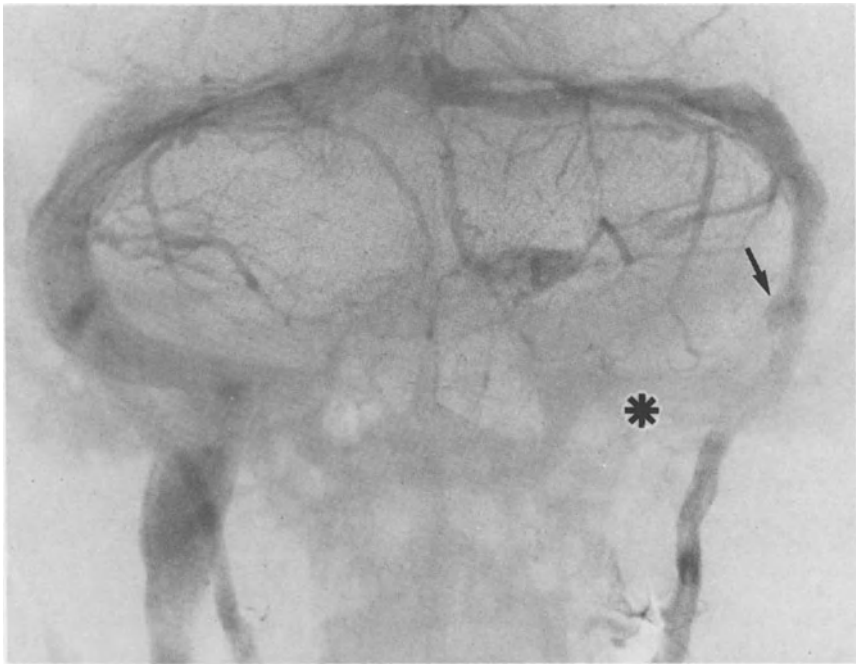

Fig. 3. Venous phase of the dominant vertebral artery in a case of left jugular foramen paraganglioma (asterisk). The arrow points to the sigmoid sinus collateral pathways into the mastoid vein, proximal to venous thrombosis. (Reprinted from Surgical Neuroangiography, with permission)

arteries and the ascending pharyngeal arteries bilaterally (in order to visualize contralateral localization) must be studied. Late phase of the dominant vertebral artery will confirm occlusion or patency of the jugular vein (Fig. 3). In the case of transdural extension of the tumor, blood supply will be demonstrated by ipsilateral opacification of AICA and PICA. Carotid canal invasion is diagnosed by CT examination showing bone destruction, narrowing of the intrapetrous carotid artery, and tumoral blush on angiographic studies.

2. Cervical paragangliomas: The ipsilateral vertebral, proximal internal carotid, facial, lingual, superior laryngeal, carotid body, ascending cervical, inferior laryngeal, and bilateral ascending pharyngeal arteries must be studied. One should note that in these cases the tumors compress the internal jugular vein without invading it; in contrast, true parietal invasion of the adventitia of the internal carotid artery is sometimes noted.

Therapeutic Strategies

As previously stated, treatment of paragangliomas (Table 1) requires a multidisciplinary approach [4]. Because paraganglion tumor cells are not radiosensitive, radiotherapy should be avoided as a primary form of treatment and the therapeutic panel should be constituted by embolization followed by surgery, aiming to remove the tumor entirely [2-4]. Embolization is a safe and efficient method to devascularize

Table 1. Endovascular technical aspects

Arteriography + embolization in a single session
General anesthesia and femoral approach
Presurgical embolization with particles 2-10 days before removal
3 days' hospitalization (1 day prior + 1 day after)
Morbidity of embolization of paragangliomas:
- Transient cranial nerve palsy (VIIth, Xth, or XIIth) in 10% - One case presented a postembolization incomplete VIIth that did not regress before surgery 5 days later
No mortality, no CNS complications in 82 cases (1977-1988)

these tumors, helping in the resection of the lesion and cranial nerve preservation. It can even be used as the sole therapy for inoperable lesions, where the objective is the stabilization of growth [4] (Fig. 4). It is therefore necessary to use an active agent that can be safely delivered into the tumor bed. In our experience, PVA particles of 160/250 μm are the most reliable agent to embolize paragangliomas. Each feeding artery is superselectively catheterized, embolized, and further occluded proximally by strips of Gelfoam, to

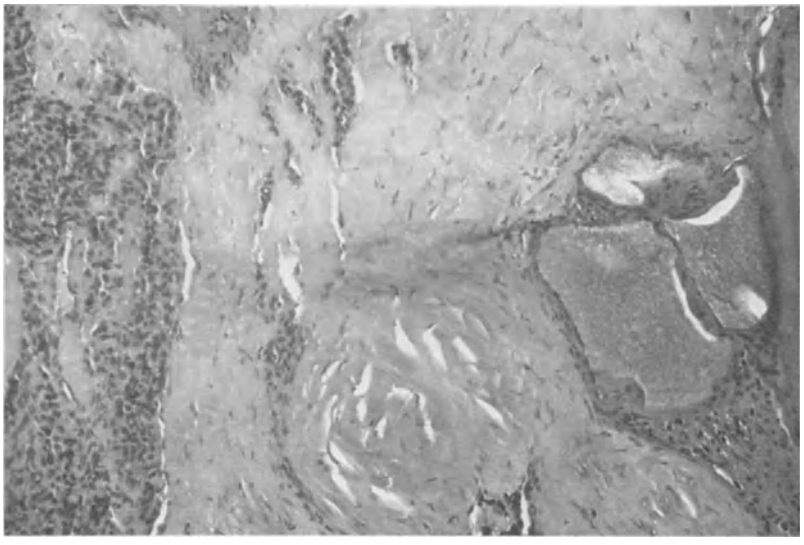

Fig. 4. Pathology specimen of a paraganglioma removed 1 year following conventional presurgical embolization. Immediate postembolization improvement led to cancellation of surgery; 1 year later surgery was felt to be feasible. Histology shows the fibrotic transformation of three-quarters of the lesion, even though the most aggressive agents had not been used for this embolization. (Reprinted from Surgical Neuroangiography, with permission)

Table 2. Multifocal blushes

Multicentric paragangliomas
Metastasis
Lymph node invasion
Perilesional hyperemia (nontumoral)
Pseudotumoral blush following surgery (nontumoral)
Associated neural crest tumors (carcinoid, etc.)

partially devascularize the surgical field and promote further thrombosis within the tumor. Fluoroscopic controls and angiographic series are mandatory during the procedure in order to avoid ectopic embolization through dangerous anastomoses or reflux into cerebral arteries.

The sacrifice of the internal carotid artery is sometimes necessary (especially in the event of petrous apex involvement when extensive base of the skull surgery is foreseen); on the other hand, exceptionally embolization of AICA and PICA needs to be discussed. The rate of recurrence depends on the quality of the resection and spread of the disease (Table 2). Its management includes presurgical embolization even though some postoperative symptomatic blush does not reflect tumor recurrence in all situations.

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Surgery of the Jugular Foramen

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Introduction

Lesions involving the jugular foramen represent a rare group of skull base tumors. Their diagnosis and management present a great challenge to neuroradiologists, otolaryngologists, maxillofacial surgeons, and neurosurgeons, and interdisciplinary cooperation is needed to achieve an optimal outcome of this surgery.

Material

Among over 900 skull base tumors of different etiology operated on at the Neurosurgical Clinic of the City of Hannover, Nordstadt Hospital between October 1978 and April 1988, we found 44 involving the jugular foramen. Their classification is shown in Table 1.

Among the 20 tumors arising within the jugular foramen, we found ten neurinomas, eight glomus jugulare tumors, one paraganglion, and one vascular tumor (hemangioepithelioid endothelioma). The other 24 skull base tumors infiltrating the jugular foramen were 16 meningiomas, four chordomas, three epidermoids, and one adenocarcinoma of the mastoid.

Table 1. Classification of 44 skull base tumors involving the jugular foramen

Tumors originating within the jugular foramen (n = 20)		Tumors secondarily affecting the jugular foramen (n = 24)	
Neurinomas	10	Meningiomas	16
Glomus jugulare tumors	8	Clivus chordomas	4
Paraganglion	1	Epidermoids	3
Vascular tumors (hemangioepithelioid endothelioma)	1	Malignant tumors (adenocarcinoma of the mastoid)	1

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Findings

Neurinomas of the jugular foramen are very rare lesions. So far, there have been reports of approximately 80 cases in the world literature. They are grouped together due to the fact that clinically and even intraoperatively the nerve from which the tumor originates is not always identified. They are slowly growing tumors; the duration of symptoms in our patients varied from 6 months to 10 years.

The clinical picture of jugular foramen neurinomas depends on the site and extension of the tumor. Those extending intracranially present with cerebellopontine angle symptoms such as hearing loss, tinnitus, vertigo, and ataxia, and rarely with facial, trigeminal, or caudal cranial nerve affection. Those extending extracranially present with early deficits of the caudal cranial nerves. Neurinomas originating within the jugular foramen and extending both intra- and extracranially (the so-called dumbbell-shaped neurinomas) present with both types of symptoms and pose the most difficulty for operative management.

The most frequent presenting symptom was hoarseness to vocal cord paralysis (60%). The rest of the symptoms are listed in Table 2, and the clinicosurgical findings are shown in Table 3.

It is interesting to point out that the patient's complaints do not always correspond to the origin of the neurinoma, as, for example in patient No. 7, whose neurinoma originating from the accessory nerve (N.XI) had altered facial sensation and hemiparesthesia as the only symptomatology.

Glomus jugulare tumors (chemodectomas) arise from nonchromaffin paraganglial cells which are located at the promontory of the middle ear, along the tympanic nerve, or at the jugular bulb. Those arising at the jugular bulb show a more invasive and destructive character. Glomus jugulare tumors present with tinnitus, hearing loss, and headache lasting from a few months to over 3 years. Palsies of the lower cranial nerves were usually a late finding in the course of disease. The presenting symptomatology, classification, and clinical findings of glomus jugulare tumors in our patients are presented in Tables 4-6.

Table 2. Presenting symptomatology in neurinoma patients (n = 10; age 19-73 years; 4 females, 6 males)

Main symptoms	No.
Hoarseness	6
Dysphagia	5
Vertigo and unsteady gait	5
Diffuse headache	4
Hearing impairment	4
Tinnitus	2
Decreased taste sensation	2
Shoulder atrophy	2
Facial palsy	1
Altered face sensation	1
Nausea and vomiting	2

Table 3. Clinical findings in neurinoma patients

Patient	Tumor origin	Cranial nerve affection						
		V	VII	VIII	IX	X	XI	XII
1. CC 38 F	N.IX		+	+	+			
2. CE 19 F	N.IX			+	+	+	+	
3. ST 32 M	N.IX			+	+	+	+	+
4. JG 54 M	N.X					+		
5. KJ 45 M	N.X and XII					+		+
6. SI 31 M	N.X			+		+		
7. JS 43 M	N.XI	+						
8. HE 73 F	N.XII							+
9. DE 66 F	N.XII			+	+	+		+
10. HH 38 M	N.IX-XI			+	+	+	+	+

Meningiomas of the posterior skull base are divided into basal, craniocervical, petroclival, and CPA meningiomas. Each of them can invade the jugular foramen; 16 such cases are demonstrated in Table 7.

As a rarity, our material includes a case of a hemangioepithelioid endothelioma with regional lymph node metastasis. The patient was a 38-year-old woman who had as a primary complaint atrophy of the shoulder followed by hearing loss on the same side and dysphagia. At admission, we found Collet-Sicard syndrome (palsy of Nn. IX-XII), a perceptive deafness, and hypesthesia of the 2nd and 3rd branches of the trigeminal nerve (Table 8).

Epidermoids of the posterior cranial fossa usually present with headache, hydrocephalus, and/or gait ataxia. They may extend extracranially through the foramen magnum or the jugular foramen. Of 23 operated epidermoids, three showed involvement of the jugular foramen (Table 9).

Table 4. Presenting symptomatology in patients with glomus jugulare tumors (n = 8; age 19-62 years; 6 females, 2 males)

Main symptoms	No.
Hearing impairment	8
Tinnitus	7
Diffuse headache	6
Facial palsy	3
Altered facial sensation	3
Vertigo and unsteady gait	3
Hoarseness	3
Dysphagia	3
Tongue atrophy	3
Diplopia	1

Table 5. Classification of chemodectomas (glomus jugulare tumors [KEMPE 1971])

Tumor class	Tumor localization and extension	Arterial blood supply
1.	Small chemodectomas, confined to the middle ear and mastoid	1-Ext.carotid a.(mainly ascending pharyngeal a.)
2.	Chemodectomas within the lumen of the jugular bulb, may extend to the sigmoid sinus or to the neck veins	2-Vertebral a. (extracranial portion)
3.	Chemodectomas dilating the jugular foramen, without infiltrating the skull base. The caudal cranial nerves remain intact	(all 4 tumor classes, 1 to 4)
4.	Chemodectomas eroding the jugular foramen, infiltrating the skull base. Caudal cranial nerves involved, but facial n. intact	
5.	Chemodectomas eroding the petrous portion of the temporal bone, affecting N.VII, VIII and occasionally N.V. The tumor reaches the post. aspect of the cavernous sinus and into the parapharyngeal space of the neck	1-Ext.carotid a. 2-Vertebral a. (extracranial portion) 3-Int.carotid a. (petrous and cavernosal branches)
6.	Extended chemodectomas that cross the midline to invade the clivus and the cavernous sinuses on both sides. Could metastasize into lymph nodes, lungs or to the ribs. Without selective tumor embolization, considered inoperable	1-Ext.carotid a. 2-Vertebral a. 3-Internal carotid a. (from both sides)

Clivus chordomas, though benign lesions, are locally very invasive and may infiltrate the caudal cranial nerves at the jugular foramen, as seen in four of our eight operated cases (Table 10).

Malignant tumors of the skull base, as seen in one operated case of papillary adenocarcinoma of the mastoid, may extend and infiltrate the jugular foramen, causing cranial nerve palsy of Nn. VII-XII (Table 11).

Table 6. Clinical findings in patients with glomus jugulare tumors

Patient	Tumor class	Cranial nerve affection						
		V	VII	VIII	IX	X	XI	XII
1. RP 24 M	4			+		+		+
2. BG 62 F	5		+	+				
3. PA 19 F	3			+				
4. MC 30 F	6			+	+	+	+	+
5. PG 51 M	5		+	+	+	+		
6. KM 36 F	3			+				
7. HA 45 F	5		+	+	+	+		
8. CH 31 F	3			+				+
9. BF 41 M	Paraganglion			+	+	+	+	+

Operative Technique (Figs. 1-3)

The choice of the proper surgical approach is determined by tumor location and extension after a thorough preoperative neuroradiological diagnostic workup including CCT, NMR, and panangiography, especially DSA. For glomus jugulare tumors, which are very vascularized, a preoperative selective embolization of the feeding vessels is an important strategy in reducing the intraoperative tumor bleeding, thus significantly facilitating tumor resection.

Tumors arising from the jugular foramen and extending intracranially to the cerebellopontine angle (CPA) are excised via lateral suboccipital craniotomy, and those extending extracranially are removed

Table 7. Meningiomas (n = 16)

Tumor localization	Foramen jugulare syndrome	Other neurological findings
CPA meningiomas (n = 56)	9	Ataxia Vertigo Impaired hearing
Petroclival meningiomas (n = 20)	4	Ataxia Vertigo Impaired hearing
Craniocervical meningiomas (n = 9)	2	Ataxia Hemihypesthesia Babinski's sign
Intraosseous meningiomas (n = 1)	1	N.XII paresis Occipital headache

Table 8. Vascular tumor (hemangioepithelioid endothelioma)

Main symptoms:
1. Shoulder weakness
2. Impaired hearing
3. Dysphagia
4. Tongue atrophy
Clinical findings:
1. Collet-Sicard syndrome (Nn. IX-XII paresis)
2. Perceptive deafness (30 dB)
3. Hypoesthesia V 2 and 3
4. Scotoma, left field of vision

Table 9. Epidermoids

Main complaints:
1. Diffuse headache
2. Vertigo and unsteady gait
3. Visual disturbances
Clinical findings:
1. Cerebellar ataxia
2. Hydrocephalus
3. Papilledema

through a cervical approach. Problematic are those tumors which are located within the jugular foramen and extend both intra- and extracranially. In the past, they have required a two-stage excision. In the early 1970s, a combined approach, retromastoid/transcervical, began to be standardized, allowing total excision in one session. Our modification to that approach differs slightly from those reported by FISCH and PILLSBURY (1979), GLASSCOCK et al. (1979), and KAYE et al. (1984); a retroauricular skin incision is extended along the anterior border of the sternocleidomastoid muscle to the level of the hyoid bone. The mastoid is exposed after the origin of both the sternocleidomastoid and the posterior belly of the digastric muscles have been mobilized. The caudal cranial nerves are identified in the neck and followed cranially to the skull base. The facial nerve is exposed in front of the stylomastoid foramen. Then the otological approach to the superior aspect of the jugular foramen is carried out. First, we drill a cortical mastoidectomy exposing the antrum, lateral semicircular canal, and the vertical portion of the facial nerve. Rerouting the facial nerve is only necessary if the tumor expands to the middle ear cleft or to the carotid canal, as in the case of extended glomus jugulare tumors.

Table 10. Clivus chordoma (n = 14)

Main symptoms:
1. Diplopia
2. Diffuse headache
3. Hoarseness of voice
4. Unsteady gait
Clinical findings:
1. N. VI paresis
2. Foramen jugulare syndrome (Nn. IX-XI paresis)
3. Hydrocephalus
4. Hypoesthesia V 1, 2, and 3

Table 11. Malignant tumors (adenocarcinoma of the mastoid)

Main symptoms:
1. Facial paralysis
2. Purulent ear secretion
3. Impaired hearing
4. Dysphagia
Clinical findings:
1. Peripheral facial paresis
2. Conductive deafness
3. Foramen jugulare syndrome (Nn. IX-XI paresis)
4. Hypoesthesia V 2 and 3

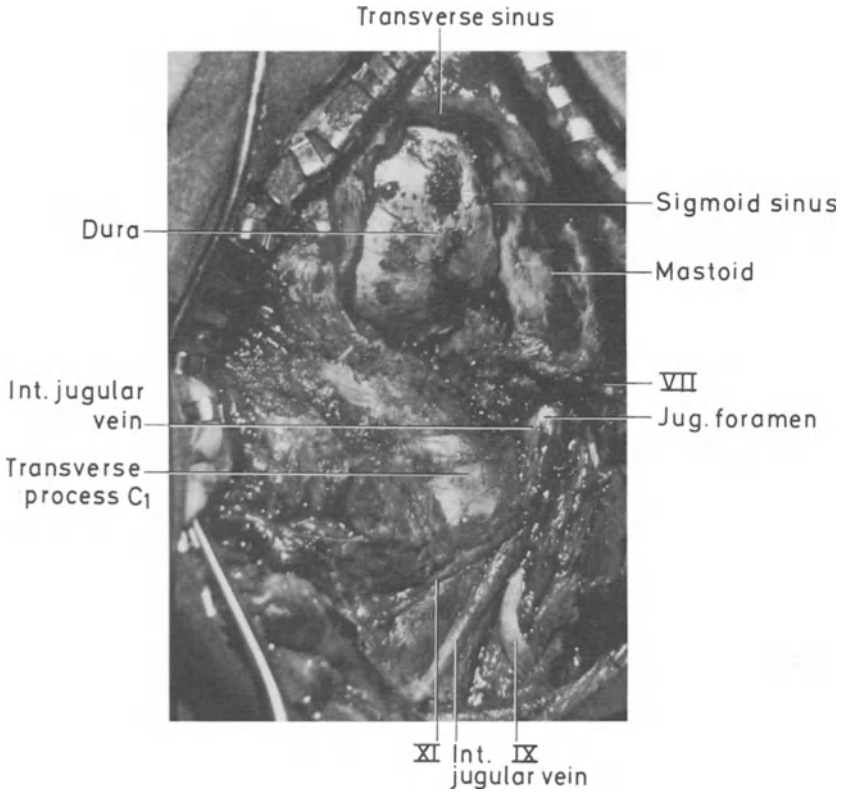


Fig. 1. The combined cervico-oto-neurosurgical approach showing the caudal cranial nerve and the jugular vein in the neck, the partially resected mastoid and the exposed facial nerve. Through a lateral subocciptal craniotomy the dura, transverse and sigmoid sinus are exposed, as well as the jungular foramen, which is opened from dorsolaterally (SAMII et al. 1988)

A lateral suboccipital osteoclastic craniotomy is performed exposing the junction of the transverse and sigmoid sinus, mobilizing the latter caudally down to the jugular foramen. The planum nuchale is further drilled till the lateral edge of the occipital condyle, and the petrous pyramid is drilled till the styloid process, so the jugular foramen is exposed and opened from dorsally. Through this approach, the tumor, sigmoid sinus, jugular bulb, and internal jugular vein (IJV) come into view. Neurinomas displace the jugular bulb dorsally, whereas glomus jugulare tumors arise from the dome of the jugular bulb, extending intraluminally to the sigmoid sinus and the IJV and also causing erosion of the skull base. Medial to the junction of the jugular bulb and the IJV, we identify the lateral process of the atlas with the origin of the oblique atlantis muscle.

The dura mater is opened cranial to the transverse sinus and lateral to the sigmoid sinus down to the jugular foramen. Then we retract the dura, the transverse and the sigmoid sinus, and the jugular bulb medially, allowing gentle traction of the cerebellum, opening the cerebellopontine cistern, and exposing the intracranial tumor portion at the CPA.

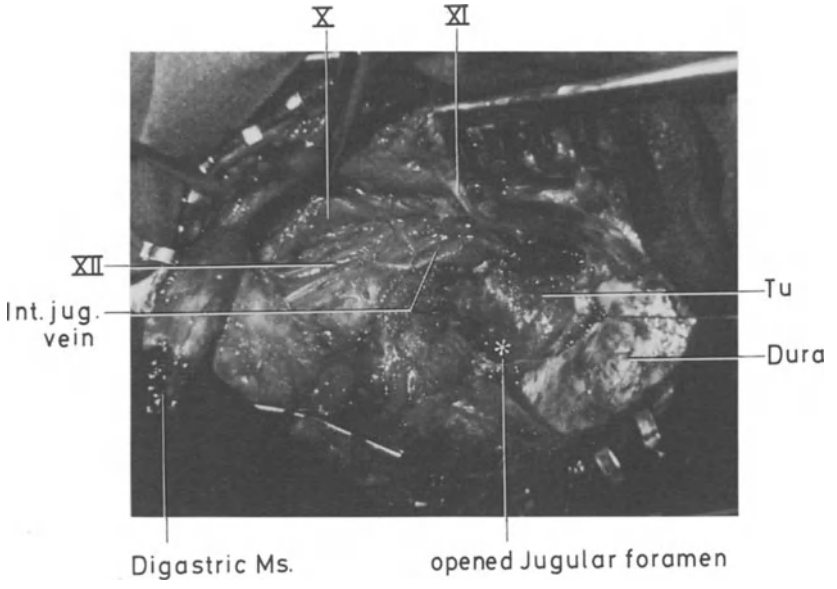


Fig. 2. The dura is opened at the jugular foramen, exposing the huge neurinoma, with its intra- and extracranial extension

We start resecting the extracranial portion of the tumor so that the caudal cranial nerves remain under vision, enucleating the tumor using microsurgical technique. The intracranial portion of the tumor is then resected through the widened jugular foramen, taking care not to damage the neural structures (Nn. IX-XII) ventral to the tumor

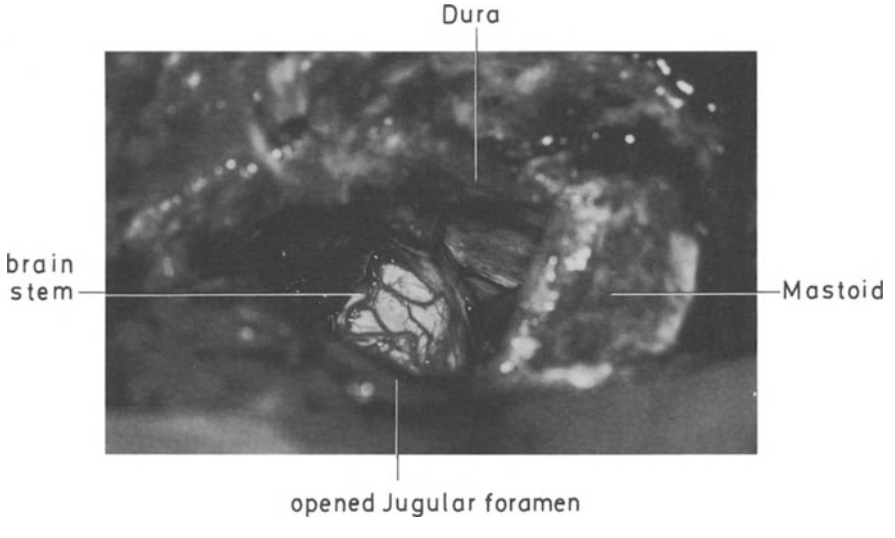


Fig. 3. Operative situation after total tumor removal; the tumor; the brain stem is seen through the opened jugular foramen

mass. In cases of glomus jugulare tumors, the sigmoid sinus is ligated and packed as well as the IJV. After enucleating the tumor intraluminally, an en bloc resection follows. In the presence of neurinomas and other nonvascular lesions we leave the sinus intact.

After meticulous hemostasis we return the jugular bulb to its site within the jugular fossa, and the dura is then sealed. A triangular piece of lyophilized dura is used to seal the defect at the jugular foramen, using fibrin glue and packing with the posterior belly of digastric muscle. Finally the sternocleidomastoid muscle is sutured at its site of origin.

To avoid a CSF leak, lumbar drainage is placed and antibiotics are given during the first 10-14 postoperative days.

Results and Discussion

The outcome of jugular foramen surgery has improved tremendously thanks to the modern neuroradiological investigations, the microsurgical techniques, and the teamwork between ENT, maxillofacial, and neurosurgeons.

Based on the extensive experience and the pioneering works of Cappes in the 1950s, SHAPIRO and NEUSS, GEJIOT, GARDNER et al., WÜLLSTEIN, DENECKE, HOUSE, and PORTMANN in the 1960s, KEMPE, MENZEL, FISCH, JENKINS, and JACKSON in the 1970s, and DRAF and SAMII, KAYE et al., CRUMLEY and WILLIAMS, HORN, HOUSE, HITSELBERGER, and HAKUBA in the 1980s, the combined approach to jugular foramen surgery has been standardized, allowing total tumor removal in one session, with less hazardous complications.

Our modification to the surgical technique showed a very satisfactory outcome (as listed in Tables 12-14). The operative mortality was nil. Two patients died 3 weeks and 6 months postoperatively, due to nonsurgical causes (cardiovascular insufficiency in the first case and leukemia in the second). Total tumor excision was achieved in all but two cases. The subtotal removals were a case of clivus chordoma and an extended petroclival meningioma infiltrating the jugular foramen and cavernous sinus.

Our overall surgical morbidity was 36.4% (n = 16). The 16 cases had new cranial nerve deficits, aspiration pneumonia, and/or CSF leak. How the new cranial nerve deficits evolved is represented in Tables 13 and 14. The five cases of CSF leak were successfully treated using lumbar drainage. This has become a standard postoperative technique in all our operated cases.

Table 12. Foramen jugulare surgery

No. of patients:	44
Total removal:	42 (95.5%)
Subtotal removal:	2 - 1 clivus chordoma 1 petroclival meningioma
Operative mortality:	0 (2 deaths due to nonsurgical causes)
Surgical morbidity:	16 (36.4%)
- cranial nerve lesions:	16 (36.4%)
- aspiration pneumonia:	7 (16%)
- CSF leak:	5 (11.4%)

Table 13. Cranial nerve affection

Nerve	Preop. status	Postop. status
V	1	3
VI	0	1
VII	4	10
VIII	17	20
IX	8	10
X	12	12
XI	6	8
XII	9	9

Table 14. New cranial nerve deficits (n = 16)

Nerve	Temporary	Permanent
V	1	1
VI	1	0
VII	4	2
VIII	0	3
IX	0	2
X	0	0
XI	2	0
XII	0	0
		8 (18.2%)

Conclusion

To summarize our approach: thorough preoperative neuroradiological investigation with selective embolization of vascular lesions, interdisciplinary cooperation using proven, meticulous microsurgical technique, and the modified combined approach; all these factors together lead to an optimal outcome in the surgery of these challenging jugular foramen tumors. Our experience shows that a remission of the symptoms is observed in approximately half of the new cranial nerve deficits which may arise postoperatively. To avoid the appearance of CSF leak, lumbar drainage for the first 10-14 days postoperatively is mandatory. In the immediate postoperative period the danger of aspiration pneumonia is high and careful patient observation is important.

A Clinical Example

A 41-year-old patient had noticed hoarseness as well as weakness of his left shoulder for about 20 years. Six years after the onset he started to complain of impaired hearing in his left ear as well as tinnitus. A few years later he noticed atrophy of the left side of his tongue and started to complain of choking and dysphagia, especially for fluids. A thorough diagnostic check, done at another clinic, could not reveal any cause for the caudal cranial nerve palsies. Headache was sporadic and diffuse and in the last 4 years he became dizzy and drowsy. Most recently he complained of permanent occipitocervical headache with attacks of blurred vision, nausea, and vomiting.

On clinical examination we found a horizontal rotatory nystagmus to both sides, perceptive deafness, and palsy of the left caudal cranial nerves. There was papilledema with slightly blurred vision, but the other cranial nerves were intact. There were no cerebellar signs, no pyramidal signs, and slightly exaggerated muscle reflexes on the left side.

The neuroradiological diagnostic investigations showed a huge, slightly hyperdense, well bordered tumor which extended intracranially to the CPA, reaching the tentorium and extending extracranially to the parapharyngeal space and infratemporal fossa (Fig. 4). The avascular tumor dilated the jugular foramen without destroying the skull base or petrous temporal bone (Fig. 5). The fourth ventri-

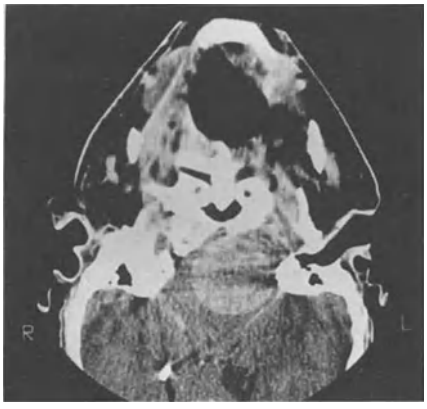


Fig. 4. A coronal CCT reconstruction showing a large neurinoma of the left jugular foramen, extending intracranially to the tentorium and extracranially to the parapharyngeal space

cle was compressed by the tumor, causing an occlusive hydrocephalus (Figs. 6, 7).

Angiography showed a slight tumor blush, while the left vertebral and basilar arteries were shifted by the tumor mass to the opposite side and the superior cerebellar artery was displaced upwards and medially.

First we performed a ventriculoperitoneal shunt to manage the hydrocephalus, after which the headache and blurred vision improved. Two weeks later the huge dumbbell-shaped neurinoma of the jugular foramen was successfully totally removed using the combined cervico-oto-neurosurgical approach, previously mentioned (Figs. 8-10). The intracranial tumor diameter was 5 x 6 cm, the intraforaminal tumor diameter, 2.5 x 2 cm, and the extracranial tumor diameter, 2 x 1.5 cm. Lumbar drainage was placed for 14 days and the postoperative course was uneventful, except for a transient left-sided abducens and one seizure. The facial and vestibulocochlear nerves could be preserved, but the perceptive deafness worsened to 80 - 100 dB. A nasogastric tube was used for 3 weeks postoperatively, until the swallowing (lying to the opposite side) showed a slight improvement. For rehabilitation of the vocal cord paralysis a Teflon injection is planned, and with thorough physiotherapy the shoulder musculature showed a satisfactory improvement.

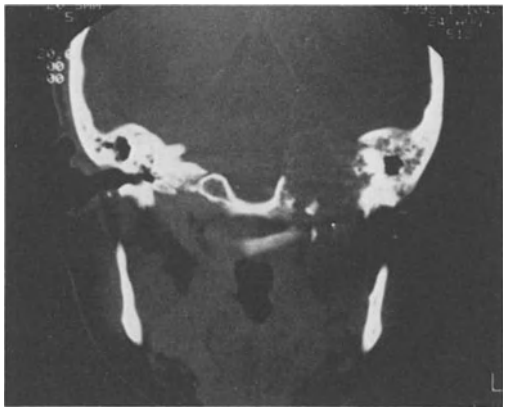
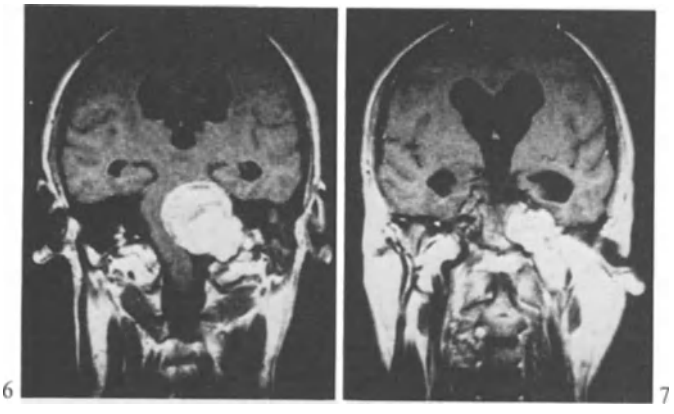


Fig. 5. The left jugular foramen is widened by the neurinoma. Note the sclerotic margins and the absence of bony destruction



Figs. 6, 7. MRI of the same neurinoma as in Figs. 4 and 5, showing its intra- and extracranial extension as well as compression and shifting of the brain stem. Note the dilated lateral and third ventricles, caused by the obstructive hydrocephalus

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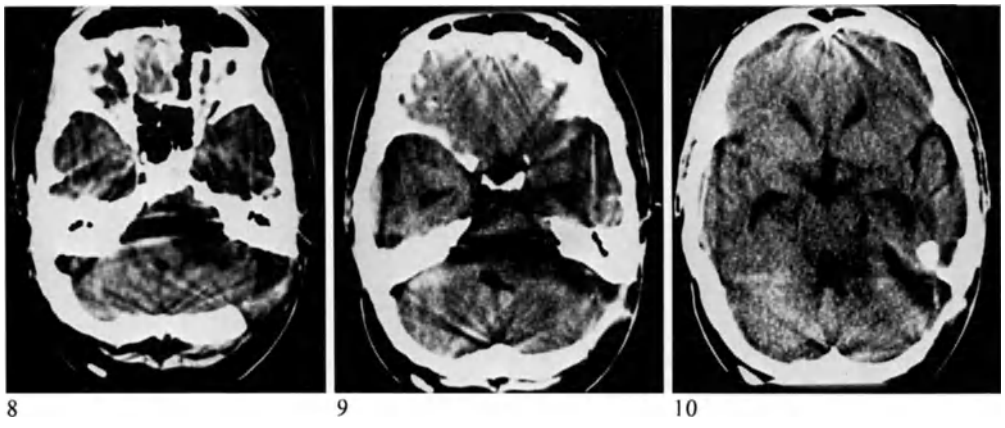

Figs. 8-10. The postoperative CCT of the same patient, showing the lateral suboccipital craniotomy and the partially resected temporal bone. The hydrocephalus subsided and the ventricles normalized in size

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Microsurgical Approaches to the Cavernous Sinus

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Introduction

The explosive development of technical standards in and around neurosurgery has made possible the treatment of previously problematic pathological conditions without too much risk to the patient, one example being lesions involving the cavernous sinus.

From the surgical point of view the cavernous sinus can be divided into three parts (Fig. 1). The **anterior part** corresponds to the parainfraclinoidal portion and is mainly connected with the problems of surgery of para- and infraclinoidal aneurysms [2,3,6,7,8]. The **middle part** represents the field of the lateral sinus wall, consisting of the IIIrd-Vth cranial nerves and the underlying posterior knee of the carotid siphon with the abducent nerve [1,4,5,8,9,10]. The **posterior part** involves the region of the petrous bone tip, including the gasserian ganglion and the canal of the internal carotid artery [1,7,9].

Approaches

Ipsilateral Approach to the Anterior Siphon Knee [6,8]

In patients with para- and infraclinoidal aneurysms, where the aneurysm sac points anterolaterally, posterolaterally, or laterally, we choose an ipsilateral pterional approach (Fig. 2 A).

Steps of the ipsilateral approach are as follows:

1. Unroofing of the ipsilateral optic canal for gentle mobilization of the nerve
2. Removal of the anterior clinoid process
3. Identification of the roof of the cavernous sinus
4. Dissection of the fibrous ring around the carotid artery sharply
5. Blunt dissection along the internal carotid artery up to the anterior siphon knee

Contralateral Approach to the Anterior Siphon Knee [3,6,8]

In cases where the aneurysm points from infraclinoidal to medial the contralateral approach is appropriate. This approach obliquely leads below the optic chiasm to the medial wall of the contralateral inter-

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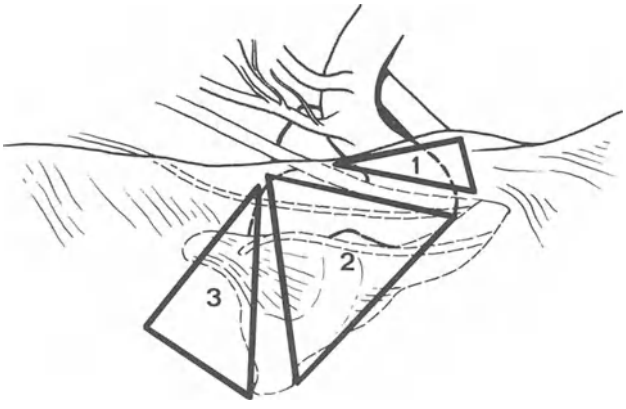

Fig. 1. Three parts of the cavernous sinus to be surgically dissected by different approaches. 1 anterior part; 2 middle part (lateral wall); 3 posterior part

nal carotid artery. The craniotomy is performed fronto-latero-basally (Fig. 2 B).

The steps of the dissection are:

1. Opening of the chiasmatic cistern
2. Removal of the tuberculum sellae: the mucosa of the sphenoid sinus is left intact and retracted
3. Removal of the medial wall of the contralateral optic canal
4. Identification of the contralateral transversal plate (roof of the cavernous sinus) and fibrous ring
5. Sharp dissection of the fibrous ring
6. Blunt dissection along the medial and anterior wall of the carotid artery up to the anterior siphon knee

Approach Through the Lateral Wall of the Cavernous Sinus [1,4,5,8,10]

If the lesion is located more proximally along the intracavernous carotid artery portion and/or involves the lateral wall of the caver-

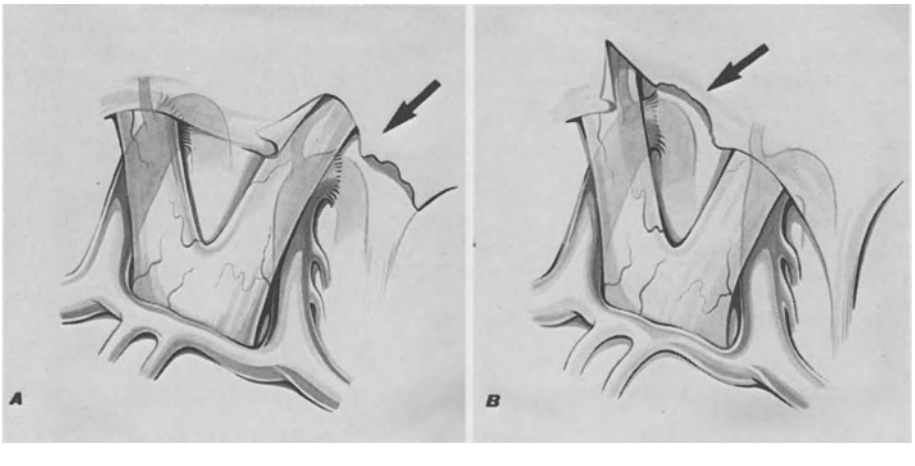

Fig. 2 A, B. Approach to the anterior part of the cavernous sinus. **A** ipsilateral; **B** contralateral

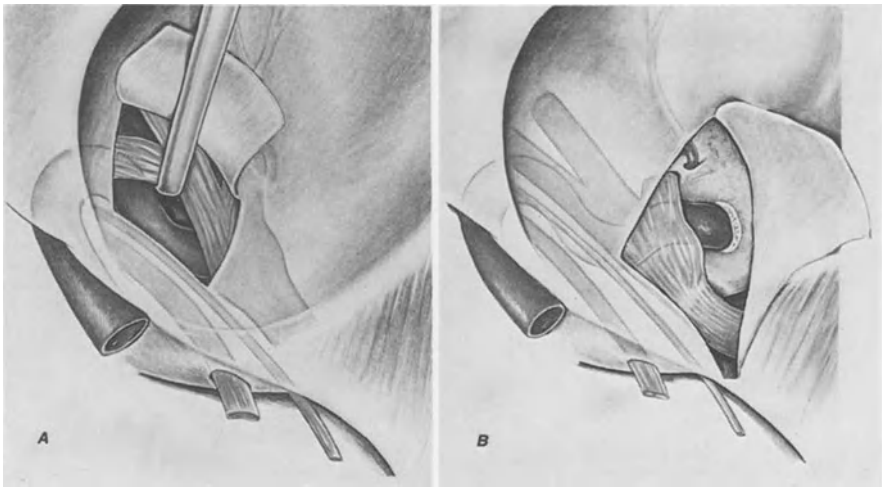

Fig. 3 A, B. Approach through the lateral wall of the cavernous sinus. A modified dissection of Parkinson's triangle; B approach to the petrous portion of the internal carotid artery

nous sinus, we use a modified approach through the lateral wall of the cavernous sinus [8]. The craniotomy is orbitopterional. Since the lateral sinus wall consists of two layers, between which the IIIrd-Vth cranial nerves are located [10], it is possible to open the outer layer like a flap (Fig. 3) without tearing the venous space.

In other cases, however, where the extension of the process reaches more toward the floor of the middle cranial fossa, a subtemporal approach is necessary.

In cases where the intracavernous portion of the internal carotid artery is involved, initially the intrapetrous portion of the internal carotid artery must be exposed. For this exposure, the middle meningeal artery has first to be coagulated and the greater superficial petrosal nerve transected in order to prevent traction of the geniculate ganglion. The dissection of the petrous segment of the carotid artery belongs to the extradural steps of the procedure. The canal of the eustachian tube and the petrous portion of the carotid artery should be covered with a muscle flap in order to prevent possible CSF leak. If the flaplike opening of the lateral sinus wall is used, the lateral wall can be closed by sutures. In cases in which a defect of the lateral wall remains, we cover with oxycellulose and fibrin glue.

Material and Results

Between 1980 and January 1988 we carried out intraoperative dissection of one or more parts of the cavernous sinus in 70 cases. In the anterior cavernous sinus portion the vascular lesions were predominant: there were 25 aneurysms and six patients with tumor [8]. By contrast in the middle part of the sinus tumorous processes were more frequent: there were two aneurysms and 36 tumorous lesions.

Table 1 shows the functional results in the tumor case group. The results of this series speak in favor of the direct medial repair of

Table 1. Functional results of cavernous sinus surgery for tumor lesions (n = 43)

Preop. lesion	Postoperative result			Morbidity[a]	
	Unchanged	Improved	Full recovery	Transient	Persistent
II[b]	4	5	4		
III	2	2	5	4	1
IV	1	1	2		
V	4	7			
VI	2	1	4		
VII	2	1			
VIII	2				
Hemiparesis	1	3	4	2	
Seizures		1	1		
POS			5	2	
Diabetes insipidus				1	
Occlusion of int. car. art.					3

[a] Two patients died of pneumonia; [b] II-VIII, cranial nerves

intra- and pericavernous lesions. This operative strategy has proven to be a safe procedure, feasible without deep hypothermia, extracorporeal circulation, or cardiac arrest. The technique in many cases permits total removal of intracavernous lesions with the preservation of the internal carotid artery and avoidance of operative traumatization of the nerves. It is likely that in the case of lesions invading the cavernous sinus this procedure will be the treatment of choice in the future.

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A Combined Transsylvian-Subtemporal Approach for Management of Tumors Located in the Cavernous Sinus and in Meckel's Cave

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Introduction

Tumors confined to the cavernous sinus and Meckel's cave are of different entities and can have a varied clinical presentation [1]. The most common tumors in this area are meningiomas and neurinomas. The exact location and relation to surrounding structures can be sufficiently determined by computed tomography and magnetic resonance imaging. Various techniques and approaches for the surgical removal of such space-occupying lesions have been presented and described by a number of authors [4,9,10,12].

Normal and pathological anatomies of the parasellar region have been extensively studied and described [9,11,13,14,16,17]. In our institution tumors of the parasellar region are usually removed using either a standard pterional approach [18] or a standard subtemporal approach [6].

Large tumors often require extensive brain retraction. Postoperative complications (e.g., hemiparesis, aphasia) due to compression of the temporal lobe and the peduncle or due to interruption of significant temporal bridging veins are known from the literature [5] and were also observed in our own series [7].

Retraction of the medial part of the temporal lobe during a pterional (transsylvian) procedure can be limited in cases where the temporal lobe is fixed to the sphenoid wing by superficial sylvian veins.

To avoid these postoperative complications and to achieve an adequate view of large tumors originating in or invading the cavernous sinus and Meckel's cave, it seemed appropriate to use a combined transsylvian-subtemporal route which DRAKE suggested [6] for basilar aneurysm surgery.

Clinical Material

Between September 1982 and March 1988, 74 patients who were admitted to the Neurosurgical Department, University of Freiburg, underwent surgical removal of space-occupying lesions of the medial skull base (Table 1). In 10 cases the combined transsylvian-subtemporal approach was applied. These patients included five females and five males, whose ages ranged from 9 to 65 years.

Preoperative symptoms and signs, tumor location, pathological entities, and postoperative complications are listed in Table 2.

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Table 1. Pathological entities and operative approaches in 74 patients with tumors related to the cavernous sinus and/or Meckel's cave

	Pterional approach	Subtemporal approach	Combined approach
Meningioma	29	11	5
Neurinoma	8	3	1
Epidermoid	5	1	2
Pituitary adenoma	4		1
Metastasis	1	1	
Hemangioma		1	
Chordoma			1
Total	47	17	10

Surgical Procedure

A standard pterional craniotomy [18] is performed with the following modifications: the patient's head is turned sidewards so that the squamous portion of the temporal bone forms an angle of approximately 30° with the horizontal plane; the skin incision is extended backwards 3-4 cm behind the hair line to expose a larger portion of the temporal bone; the temporal muscle is incised down to the zygomatic arch, which will also be exposed; the craniotomy is extended to the floor of the middle cranial fossa by removal of the anterior temporal squama to the external auditory canal [6].

The dura is opened in the standard manner. The arachnoid of the sylvian fissure is opened and the fissure is split. By retracting the frontal lobe, optic nerve and carotid artery are exposed and cerebrospinal fluid is aspirated continuously from basal cisterns. Dissection is carried out along the tentorial edge by separating the oculomotor nerve from the tip of the uncus and the crus. If necessary, intraoperative spinal drainage, hyperventilation, and intravenous mannitol can be applied to reduce brain bulk. After the temporobasal dura has been opened, the temporal lobe is gently elevated with special attention given to the temporobasal veins. The previous separation of temporomedial structures from the crus, as well as the early CSF drainage, facilitates the elevation of the temporal lobe without significant compression of the midbrain. Intradural tumor removal is then carried out in the usual way.

Results

In five cases the tumors originated in Meckel's cave and/or in the cavernous sinus (Table 2). The preoperative CT scan of a representative case is shown in Fig. 1.

Two patients each had a large temporomedial epidermoid which spread across the basal surface of the brain and reached as far as the basal cisterns and the posterior fossa. In one patient the tentorium was incised along the petrosal rim, opening a wide surgical field and permitting the removal of the infratentorial part of the epidermoid.

Table 2. Clinical data of patients with combined approach

Case No.	Age/ sex	Location of tumor	Pathology	Initial symptoms and signs	Postoperative complications
1	9/m	Meckel's cave	Chordoma	Generalized seizures; no neurological deficit	-
2	19/f	Cavernous sinus	Meningioma	Incomplete VIth nerve palsy	Complete VIth nerve palsy; trigeminal sensory loss
3	45/m	Meckel's cave	Malignant trigeminal neurinoma	Continuous facial pain; trigeminal sensory loss	Transient IIIrd nerve palsy; surgically removed epidural hematoma on 2nd postop. day
4	46/f	Cavernous sinus	Pituitary adenoma	IIIrd and VIth nerve palsy	IIIrd nerve palsy increased
5	53/m	Meckel's cave	Meningioma	Incomplete palsy of nerves III through VII	-
6	27/m	Cisterna ambiens	Epidermoid	Seizures; no neurological deficit	Transient IIIrd nerve palsy
7	46/m	Cisterna ambiens; post. fossa	Epidermoid	Seizures; no neurological deficit	Transient IIIrd nerve palsy; transient hemiparesis
8	43/f	Tentorial fold	Meningioma	Diplopia; numbness of the face; trigeminal sensory loss	Transient IIIrd nerve palsy
9	56/f	Tentorial fold	Meningioma	IIIrd, IVth, and VIth nerve palsy	Transient aphasia; IIIrd nerve palsy increased
10	65/f	Tentorial fold; posterior fossa	Meningioma	Visual deterioration; dementia; VIth and VIIth nerve palsy	Permanent IIIrd nerve palsy

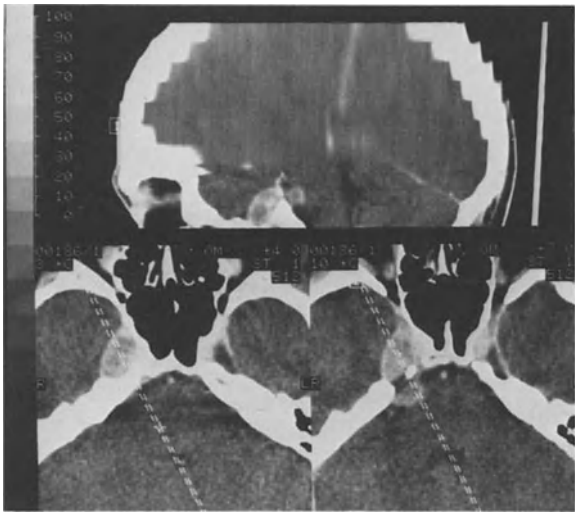

Fig. 1. Preoperative CT scan of case No. 3 (Table 2)

Three patients had large meningiomas arising from the tentorial fold, invading the cavernous sinus and/or Meckel's cave, and extending to the suprasellar region and to the posterior fossa.

Almost complete tumor removal was achieved in seven cases. Three patients with meningioma required a second operation later on, in which a suboccipital approach was taken to remove large infratentorial parts of the tumor. There was no mortality in this series.

Discussion

Most tumors related to the cavernous sinus and to Meckel's cave are accessible via a subtemporal as well as a transsylvian route. The surgical approach depends upon the extent of the tumor as determined by the preoperative diagnostic studies.

KAWASE et al. [10] used a transzygomatic approach to large meningiomas around the cavernous sinus and extending into the orbit. They removed meningiomas arising from clivus or anterior pyramidal bone through a transpetrosal approach. Large frontotemporosphenoid meningiomas were removed using the orbitofrontomolar approach associated with bone reconstruction [12].

Our own previous studies [7] demonstrated that less extensive tumors of the tentorial edge can be removed surgically via a transsylvian route with low morbidity and that the best results were obtained by removing tumors that primarily involved the cavernous sinus.

If the tumor is localized laterally in the peduncle area or extends to the posterior fossa, it can be reached from this approach only by lateral retraction of the uncus, which may cause damage to the sylvian veins. Phlebography is therefore essential prior to surgery to determine the venous drainage pattern of the temporal lobe. However, bony overlap hinders angiography from always supplying sufficient information concerning the venous drainage of the temporal base.

The superficial middle cerebral (sylvian) vein shows the greatest variation of all the superficial cerebral vessels [2]. In 84% of cases this vein drains into the sphenoparietal and/or paracavernous sinus [9]. According to our anatomical study in 81 cadaveric specimens [15], bridging veins between temporal lobe and middle cerebral fossa are present in 96%. These veins may restrict elevation of the temporal lobe. Insufficient visualization of the rostral part of the cisterna ambiens constitutes another disadvantage of the subtemporal approach.

The combination of pterional and subtemporal craniotomy was first described by DRAKE [6] for a combined approach to the anterior circle of Willis and the basilar bifurcation region. DOLENC used a similar combined approach with additional exposure of the intrapetrous part of the ICA for treatment of intracavernous vascular lesions [3], as well as for removal of tumors of the skull base [4].

We chose this combined transsylvian-subtemporal approach on the basis of the following concepts:

- It provides visualization of all anatomical structures in the region of the tentorial fold from two different angles.
- All the anatomical structures in the area can be adequately identified in the surgical field via the shortest route.
- It permits early identification of the oculomotor nerve and perforating vessels of the posterior communicating artery.
- It provides the possibility to take both approaches alternatively.
- Elevation of the temporal lobe is possible without critical compression of the midbrain due to early CSF drainage and prior dissection of medial temporal structures through the sylvian fissure.
- Significant temporal bridging veins can be preserved, even in cases with large tumors.
- Postoperative complications due to compression of the temporal lobe and/or the peduncle, as well as due to venous damage, are avoided.
- If necessary, the approach can be extended to the posterior fossa by splitting the tentorium parallel to the petrous bone.

The surgical results in this small series were satisfactory. There were no major complications such as injury of important temporal bridging veins - the vein of Labbé or sylvian veins, for example. In case 7, however, two smaller temporobasal bridging veins had to be occluded, which was probably the cause of a transient postoperative hemiparesis.

Fixation of the temporal lobe to the sphenoid wing by sylvian veins proved to be of no disadvantage in these cases due to sufficient tumor exposure on each side of the veins (sylvian and subtemporal).

In most of these cases postoperative complications consisted of impairment of the third cranial nerve. Functional disturbance of the masseter and temporal muscle following the procedure was observed in one patient.

Summary

A combined transsylvian-subtemporal approach to the medial skull base is presented and discussed. The purpose of this approach is to reduce compression of the temporal lobe and midbrain, as well as to preserve the sylvian veins. Our experience in ten patients showed several

advantages compared with pterional (transsylvian) and subtemporal approach.

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Microsurgical Resection of Tumors Involving the Cavernous Sinus: Possibilities and Limitations[1]

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Introduction

Thanks to the deeper insight into the topography of the cavernous sinus (CS) and sellar region provided by modern diagnostic procedures [2,12,24,35,38], and on the basis of our growing experience in the operative management of pathological lesions in the region of the tentorial notch [6-9,31,33,39], we were encouraged to treat 28 tumors invading the CS more aggressively by a direct microsurgical approach from different sides, in some cases via combined approaches in two stages.

Patients, Operative Treatment, and Results

Since March 1983, 25 patients have undergone a direct surgical approach to the CS because of tumor pathology. Their age ranged from 28 to 56 years. There were 9 meningiomas (Fig. 1), 16 pituitary adenomas (Fig. 2), 2 malignant tumors (Fig. 3) of the skull base, and 1 fibrous dysplasia. Our cases are listed in Table 1 in respect to the type and extent of the tumor, the operative approaches, and the tumor removal. There was only one meningioma that originated primarily inside the CS (Fig. 1). Tumor invasion into the CS depended on tumor sites at the base, e.g., sellar and parasellar region, sphenoid ridge, petrous apex, and tentorial edge. Different operative approaches were chosen in this respect so as to avoid brain damage and injury to the neurovascular structures outside and inside the CS.

Pituitary adenomas with parasellar invasion of the CS were approached in cooperation with the ENT surgeon in one stage via the transnasal, transsphenoidal, transsellar route (Fig. 2), avoiding infrasellar lateral tumor resection in the inferior-anterior part of the CS because of limited space and view, or via the subfrontal and pterional route, as well as in two stages from inferior and superior.

In 6 cases of subtemporal approach to petrous apex meningiomas the tentorium was partially resected with preservation of the IVth cranial nerve in all but one case, where the recurrent meningioma had invaded the CS along the sheaths of the IIIrd and IVth nerves.

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[1] Dedicated to Prof. Dr. J. Lang on the occasion of his 65th birthday.

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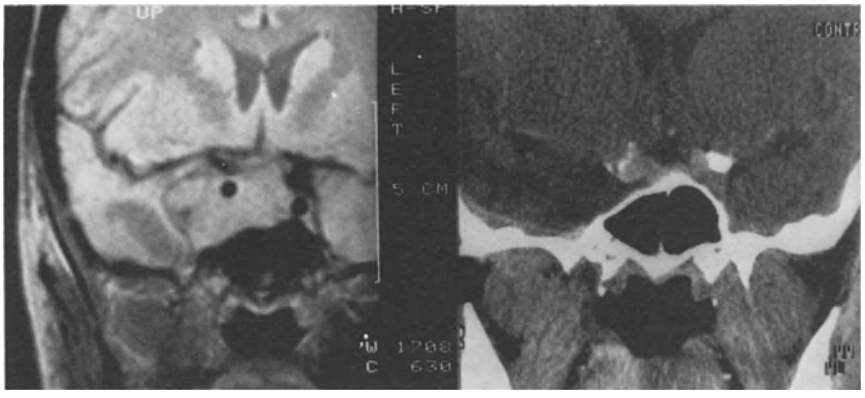

Fig. 1. Nuclear magnetic resonance image of intracavernous meningioma with tumor invasion into the hypophyseal region and suprasellar extension. Notice intracavernous ICA surrounded by tumor tissue 3 years after subtotal tumor removal via the subtemporal lateral approach and radiosurgery of residual intracavernous tumor 1 year later. Postoperative contrast CCT scan in coronary sections 4 weeks after the Dolenc approach demonstrates total removal of the tumor with preservation of the ICA and the IIIrd cranial nerves

The opening of the CS followed microsurgical anatomy:

Inferior medial: seven adenomas, one ICA injury and secondary occlusion due to a false aneurysm after 10 days because of previous chronic parasellar inflammation of the paravascular area
Anterior lateral after resection of the orbital roof and anterior clinoid process with control of extradural and subarachnoid ICA; two cases; one ICA injury, ICA patent after temporary clipping

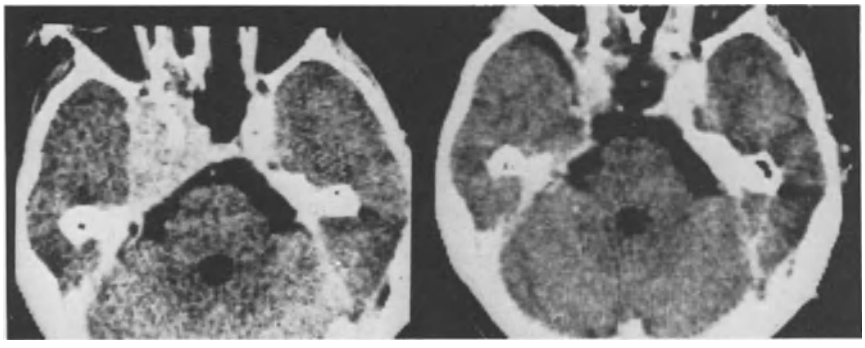

Fig. 2. Postoperative CCT scan of a giant invasive pituitary adenoma with parasellar extension into the left CS and middle cranial fossa as well as supra- and intrasellar tumor growth, which was treated first by the combined transethmoidal-transsphenoidal approach to deal with CSF leakage, and then by the left pterional medial approach with total removal of the tumor, as demonstrated by the postoperative CT scan

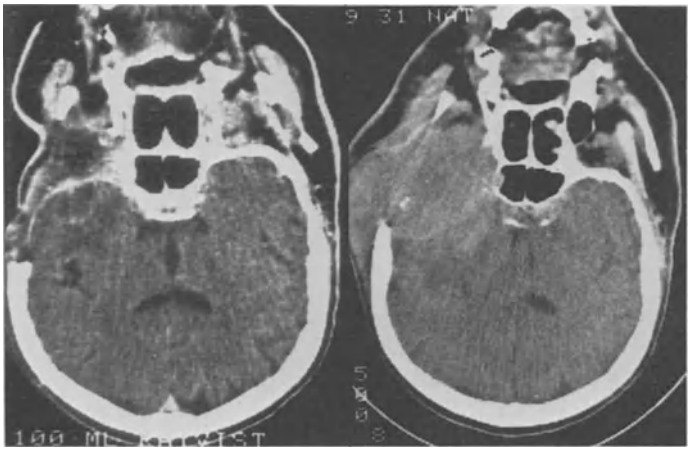

Fig. 3. Pre- and postoperative CT scans in coronal sections of a 55-year-old man with metastatic carcinoma of the left temporal and infratemporal region and invasion of the CS and V 1, 2, and 3 compression causing neuralgic facial pain. Tumor removal was limited because of malignancy; residual tumor tissue was left to ensure preservation of the ICA and the IIIrd and IVth cranial nerves. Plastic reconstruction was undertaken with a muscle flap and free epidermis transplant

Anterior medial via the "medial triangle" of HAKUBA (1982); subchiasmatic transsellar or opticocarotid following along adenoma tissue invasion
Lateral via "Parkinson's triangular space" (1965)
Lateral inferior when V 2 and 3 were displaced in cases of metastatic carcinoma, malignant meningioma, or fibrous dysplasia
Dorsal superior via the "oculomotor trigone" (ONO et al. 1984) in cases of giant adenoma and meningioma of the diaphragma sellae

Total removal was accomplished in 13 of 28 tumors invading the CS. Postoperative computerized tomography (CT) and/or magnetic resonance tomography (MRT), follow-up examinations, and hormone measurements (Fig. 2) demonstrated the tumors in question to be 9 pituitary adenomas and 4 meningiomas.

Only subtotal removal could be achieved in 5 meningiomas because of tumor tissue surrounding the intracavernous ICA, with one ICA injury and temporary clipping during the subtemporal approach, or because of limited space and view in the anterior part of the CS and in respect of tumor malignancy. In two cases resection was limited to preserve the function of the IIIrd cranial nerve (Figs. 1, 3).

Pituitary adenoma resection was subtotal in 7 of 16 cases. There was one invasion of the base, in which intraoperative injury of the ICA occurred. In two hormone-secreting adenomas some tissue had to be left inside the CS because of limited transsphenoidal space and view, although growth hormone levels were brought down to less than 10 ng/ml in one, while in the other serum prolactin levels were in the normal range under dopamine agonist medication. In 4 giant adenomas resections were incomplete because of limited space and view within the CS via the lateral subtemporal and anterior subfrontal approaches. At admission cranial nerve disturbances due to meningiomas

Table 1. Tumors invading the cavernous sinus

Tumor type	No. of cases	Extent of tumor	Cavernous sinus operation
Meningiomas	1	Intracavernous, middle fossa	Pterional- subtemporal lateral subtotal
	2	Petrous apex, tentorial notch	Two stage suboccipital-subtemporal, dorsal total
	1	Petrous apex tentorial edge	Subtemporal, lateral total
Recurrent meningiomas	1	Intracavernous, intrasellar, tentorial edge	Dolenc-approach fronto-subtemporal, anterior lateral patent ICA subtotal
	1	Medial sphenoid wing, middle fossa	Pterional, anterior-lateral patent ICA, subtotal
	1	Petrous apex, tentorial edge	Pterional-subtemporal dorsal lateral total
	2	Diaphragma sellae, tentorial edge petroclival area	Pterional-subtemporal dorsal-medial 1 total dorsal-lateral 1 subtotal
Pituitary adenomas	5	Intra-,para-,supra-sellar	Bilateral subfrontal, medial 1 case total subfrontal, opticocarotid, medial 2 cases total pterional, lateral 2 cases subtotal
	5	and infrasellar	Transethmoidal-transsphenoidal, inferior-medial, occlusion of ICA, 2 subtotal transnasal-transsphenoidal inferior-medial, 3 total
	2	Intra-parasellar	Transsphenoidal 1 total
Recurrent adenomas	1	Intra-,supra-,parasellar and infrasellar	Two stage transethmoidal-transsphenoidal and pterional with inferior-medial, superior opticocarotid and lateral approach total
	1	Intra-,para- and suprasellar	Subfrontal, opticocarotid medial subtotal
	2		pterional, lateral 1 total 1 subtotal
Fibrous dysplasia	1	Sphenoid, temporal, petrous bones and zygomatic	Orbitozygomatico-subtemporal, anterior subtotal
Malignant meningioma	1	Medial sphenoid wing, infratemporal and middle fossae	Two stage embolization and orbito-pterional, anterior and lateral subtotal
Carcinoma metastasis	1	Sphenoid wing, middle and infratemporal fossae	Orbitozygomatico-subtemporal, inferior lateral anterior subtotal

were present as follows: isolated VIth nerve palsy in intracavernous meningioma; neuralgic facial pain and numbness of V 1 in one and of V 2 and 3 in two meningiomas of the petrous apex; numbness of V 1-3 in both malignant tumors, one of them part of a complete ophthalmoplegia. Extension of adenomas into the tentorial notch caused IIIrd nerve paresis in six, in two cases combined with IVth and VIth nerve involvement. Optic nerve compression was evident in all suprasellar adenomas, and in two medial sphenoid wings and one diaphragma sellae.

Follow-up findings demonstrated the following cranial nerve lesions: In the case of complete removal of meningiomas: V 1 same as before in one; additional V 1 numbness in one; IVth nerve palsy in one; incomplete IIIrd nerve paresis in one recurrent tumor. In the case of subtotal removal of malignant meningiomas: VIth cranial nerve same as before with additional V 1 palsy; ophthalmoplegia following anterior lateral approach, although the IIIrd and IVth nerves and V 2 and 3 (Fig. 1) were preserved. In a case of recurrent diaphragma sellae meningioma there was incomplete IIIrd and IVth nerve palsy on the left, and after a second approach 1 year later, partial injury of the IIIrd nerve on the right. Partial injury of V 1-3 occurred in metastatic carcinoma (Fig. 3).

In 4 giant adenomas IIIrd nerve paresis remained unchanged, but it recovered partially in the case of ICA injury. In a case of partial intraoperative injury to Nn. III, IV, V 1, and VI, the IIIrd and IVth nerves showed partial recovery after total tumor removal (Fig. 2).

There were no postoperative deaths; however, one patient died because of myocardial infarction and pulmonary complications 5 months after transsphenoidal surgery with injury of the ICA.

Postoperative extracranial irradiation has not been a routine procedure in either total or subtotal removal except for malignancies. However, radiosurgery was carried out by Prof. Steiner in a case of primarily intracavernous meningioma with further tumor progression.

Illustrative Cases

Invasive Pituitary Adenoma

A 43-year-old woman presented with giant adenoma invading the base and left CS, residual right hemiparesis, paresis of the IIIrd, IVth, and VIth nerves, and impaired visual acuity 2 years after partial removal of the tumor via a left frontotemporal approach. Postoperative irritation and meningitis occurred secondary to CSF leakage, with surgical occlusion of the fistula by an ENT surgeon in another clinic. The tumor was completely resected in two stages via a combined extradural (Prof. W. Draf, Fulda) ENT-neurosurgical, transethmoidal-transsphenoidal approach on 09.07.84 and an intradural pterional approach on 30.01.85 with superior medial transsellar, opticocarotid, and dorsal-superior opening of the CS for tumor removal with injury of Nn. V 1 and VI. The postoperative course was uneventful, with recovery of full capacity.

Intracavernous Meningioma

A 28-year-old woman was admitted because of a 5-year history of headaches and intermittent double vision that had worsened during pregnancy and menstruation. There was partial paresis of the IVth nerve with numbness of V 1 and 2. CCT and angiography demonstrated a typical meningioma of the right CS extending into the middle fossa. After subtotal removal via a right pterional-subtemporal indural approach on 25.03.85, there was injury of V 1 with partial numbness of V 2 and 3, and preservation of the IIIrd and IVth nerves. In May 1986 radiosurgery was performed by Prof. L. Steiner, Stockholm. After 1 year tumor progression occurred with intra- and suprasellar tumor extension. On 18.03.1988 Dolenc (Ljubljana) and the senior author approached the tumor extra-intradurally with total removal of the intracavernous and intrasellar tumor mass (Fig. 1). The intracavernous ICA was injured but kept patent after temporary clipping; the IIIrd and IVth nerves were preserved. The postoperative course was uneventful.

Discussion

Despite the many studies of the anatomy of the parasellar space [19, 21,23,24,32,35,46,47,49], a definite topographical description of the region remains necessary [47]. The anatomy and topography of the CS and its neurovascular structures, including anatomical variants, are described elsewhere in detail [12,15,19,23,24,35].

Tumors originating outside the CS with secondary invasion of the CS most commonly arise from parasellar structures and the hypophyseal region. Tumors originating primarily inside the CS are rare. Because of the arachnoid network inside the CS and the intracavernous intrasellar venous connection [21,49], tumors may cross from one side to the other and may follow the dural sheaths along the IIIrd, to VIth cranial nerves into the CS [48]. Therefore total removal of tumors invading the base is hardly possible. Because radical excision in the region of CS has its limitations when the ICA and IIIrd nerve should be preserved, the neurosurgeon has to focus his efforts on tumor mass reduction without damaging functionally important neurovascular structures in this area.

However, based on recent anatomical studies which have given a better insight into CS topography, and microsurgical experience with different approaches, a number of tumors now can be completely excised without additional severe injury [1,3,10,22,25,26,28,39,40). This was possible in as many as two-thirds of 63 cases in the DOLENC (1987) series via a direct extra-intradural, anterior lateral approach. SEKHAR and MOLLER (1986) reported on total removal of six of seven tumors involving the CS by a lateral, superior, or inferior approach, with temporary clipping and suture of ICA in one patient. LESOIN et al. [27,29] have preferred to approach intracavernous tumors via the pterional route together with an orbitozygomaticomolar flap and have achieved extensive though incomplete resection in 18 of 21 cases. KAWASE et al. (1987) advocate the transzygomatic approach for orbitcranial boundary tumors, and have achieved total removal in 5 of 7 cases. Moreover, these authors used a transpetrosal approach in 12 patients and removed two clival meningiomas invading the CS totally via this route [22,42]. CIOTTI et al. (1987) reported on 11 subtotal and one total removal of CS meningiomas with postoperative radiotherapy for the partially resected tumors. In the case of large lateral and posterior cranial base neoplasms invading the CS, SEKHAR et al. [41] described a subtemporal-preauricular infratemporal fossa

approach that may be combined with an extradural transethmoidal or an intradural frontotemporal or retromastoid approach.

GUIOT and DEROME [16], like JEFFERSON [20], thought certain forms of invasion by adenomas, for example of the CS, to be inaccessible via the inferior medial approach [18]. SEKHAR et al. (1987) did not recommend transsphenoidal approaches for CS-invading tumors because of inadequate exposure, poor proximal and distal control of the ICA, and the danger of CSF leakage.

From our experience we can say that in the case of soft adenomas, tumor tissue can easily be sucked out of the CS, whereas inflammatory tissue contains a high risk of vascular lesion, especially when the medial wall of the CS is destroyed, as happened in a case of erosion of the bone of the sphenoid sinus where the ICA was bulging into it [12,15,24].

Intracavernous meningiomas and craniopharyngiomas are usually very hard to remove due to their type and because they are mixed with the trabeculated network; an ultrasonic surgical aspirator can be helpful but may be dangerous next to the carotid wall. The abducens nerve, running freely in the CS, was found to be most vulnerable, whereas the ophthalmic nerve more often than the V 2 and 3 branches may be sacrificed in order to obtain better access for tumor removal. Intraoperative extracranial monitoring of cranial nerves II through VIII has proven helpful in locating them during microdissection [40,41], as has been described for the ICA with the aid of Doppler sonography [14].

Inside the CS venous bleeding because of tumor infiltration was rare and could be easily controlled by packing with Surgicel. Because tumors surrounding the intracavernous ICA may erode the arterial wall, especially in the case of an inflammatory process, the slightest operative trauma can bring about rupture of the artery. We advocate leaving a part of tumor on the wall in such cases.

Intracavernous tumor surgery is limited because of its duration, which is why it has to be restricted to younger patients, and because of preservation of neurovascular structures that are involved by the tumor. Therefore the question of whether to perform radical tumor excision may involve consideration of the quality of life.

Postoperative radiation therapy for incompletely resected meningiomas and invasive pituitary adenomas is recommended today [4,5,11,13,16, 20,25,36,42,44,45,50] but the number of cases and follow-up period in meningiomas is still too limited to draw final conclusions. Moreover the most important limiting factor in radiation therapy for subtotal resected tumors invading the CS remains the vulnerability to radiation of the surrounding brain, even in radiosurgery [45].

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Anesthesia-Independent Facial Nerve Monitoring with Orthodromic Intra / Extracranial Neurography

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Introduction

Electromyographic [1-4] recording from the facial muscles after intracisternal electrostimulation or mechanical irritation of the nerve is becoming increasingly common for the identification and functional testing of the facial nerve during cerebellopontine angle surgery. Institutions which use anesthesia with muscle relaxants have introduced neurographic recording techniques such as antidromic [5] or orthodromic [6] neurography, because the effect of muscle relaxants can impair the reliability of muscle recordings. This report describes our experience with 31 consecutive cases of cerebellopontine angle surgery in which orthodromic neurography was applied for the electrophysiological localization and functional testing of the facial nerve.

Material and Methods

Surgery was performed in 31 patients aged between 22 and 78 years (mean: 52 years) for removal of cerebellopontine angle tumors (18 neurinomas, eight meningiomas, one epidermoid tumor) or, for microvascular decompression (two cases of trigeminal neuralgia and one of hemifacial spasm); in one case exploration was negative. The diameter of the tumors ranged from 1 to 7 cm (mean: 3.3 cm).

For facial nerve recording, a 3-cm conventional neurography needle was inserted into the stylomastoid segment of the facial nerve. After exposure and during removal of the tumor, the facial nerve was stimulated repeatedly with threshold stimuli, observing the nerve compound action potential on-line on the oscilloscope. In this fashion, tumor areas without recordable facial CAP could be safely removed.

Threshold stimuli were also used for measurements of latencies and amplitudes after stimulation of various intracisternal segments of the facial nerve. Latencies were measured at the peak of the first positive potential deflection, and amplitudes were assessed by peak-to-peak measurement of the first positive-negative potential deflection of the CAP. The use of suprathreshold stimulus intensities increased the risk of stimulating more distant sites of the facial nerve by current loops.

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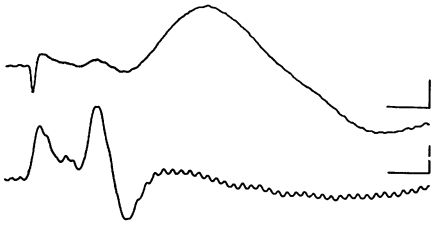

Fig. 1. Orthodromic intra-extracranial facial nerve action potential before relaxation (upper trace, amplification 100 μV/unit) and after relaxation (lower trace, 10μV/unit); time base 1 ms/unit. Stimulation site was the intracisternal segment of the facial nerve; recording site was the stylomastoid segment of the facial nerve

Results

Typical recordings of CAPs of the facial nerve (and - for comparison - a facial muscle) after stimulation of the intracisternal part of the facial nerve are shown in Figs. 1 and 2.

Of the 27 tumors in this series, 25 were removed totally or radically, two subtotally or partially. The facial nerve was preserved in its anatomical continuity in 25 cases, with the nerve as an intact bundle in 17 and split or thinned in eight; the nerve was transected in two cases. After surgery, 20 patients had active voluntary facial movement, with no or only slight facial asymmetry in 16 cases, and with moderate paresis in four. Four patients had severe but subtotal palsy, and three had complete facial palsy; six of these seven cases were acoustic neurinomas 3-5 cm in size, and one was a meningioma.

Prior to the dura closure, all 27 tumor patients had the facial nerve CAP preserved when the nerve was stimulated at its entrance to the internal acoustic meatus; the mean transtemporal latency was 1.34 ms (0.92-1.76 ms), the mean amplitude 99.7 μV (18-360 μV). When the facial nerve was stimulated near its exit zone from the brain stem, a nerve CAP was recordable in 18 patients and was absent in eight (not assessed: two patients); the mean combined transcisternal/transtem-

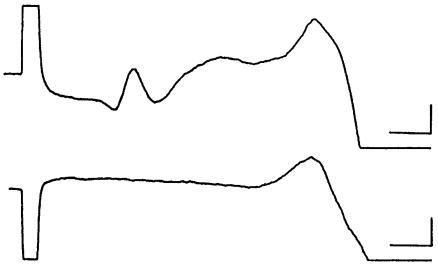

Fig. 2. Orthodromic intra-extracranial facial nerve action potential without relaxation, amplification 30 μV/unit, time base 1 ms/unit. Stimulation site was the intracisternal segment of the facial nerve; recording site was the stylomastoid segment of the facial nerve (uppertrace) and - for comparison - the orbicularis oris muscle (lower trace)

poral latency was 1.71 ms (1.07-2.28 ms), the mean amplitude 95 μV (15-480 μV). Preserved active facial movement was correctly predicted in 15 cases, and was false-negative in two. Subtotal paresis was associated with a negative CAP (stimulation near the brain stem) in four and with a positive CAP in two cases. Both total facial palsies were correctly predicted.

These data indicate that loss of facial nerve function occurred solely by manipulation of the split nerve in the tumor capsule, and not during manipulation of the nerve in the internal acoustic meatus.

Discussion

Orthodromic (intra/extracranial) neurography of the facial nerve [6] is a new and straightforward technique of intraoperative facial nerve monitoring. Carried out with the same electrophysiological equipment as evoked potential neuromonitoring, it allows on-line identification of the facial nerve during surgery in the cerebellopontine angle, independently of the status of neuromuscular blockade. Relaxation, part of so-called "balanced" (neuro)anesthesia, facilitates controlled ventilation and avoids coughing [5]; the reduced need for narcotic agents diminishes the risk of arterial hypotension, shortens the postoperative wake-up phase, and increases the accuracy of intraoperative (cortical) evoked potential monitoring [7]. So far, with correct needle placement in the stylomastoid fossa, we have always obtained a facial nerve CAP after stimulation of the intracisternal facial nerve.

The goal of orthodromic neurography is to localize and preserve the facial nerve during tumor dissection. Distinction between tumor tissue and facial nerve fibers was usually possible, one of the factors responsible for the favorable outcome of the patients in the present series. Nevertheless, the fan-shaped nerve fibers sometimes adhered to the tumor capsule in such a fashion that microsurgical dissection was technically impossible, although the nerve had been localized by electrophysiological means.

In rare cases, however, no facial nerve CAP was obtained after stimulating the nerve near its exit zone from the brain stem. This confused the surgeons, especially when the nerve seemed to be anatomically intact. Reasons for this phenomenon may be neurapraxia when severe facial nerve palsy was observed after surgery, and reversible conduction block when the facial nerve function was partially preserved and improved rapidly during the early postoperative period. This reflects one of the important dangers of any electrophysiological monitoring method: Negative stimulation results do not exclude that nerve fibers have been stimulated, nor do they necessarily predict complete loss of postoperative function.

The opposite case, a preserved facial nerve CAP associated with severe facial palsy postoperatively, occurred occasionally. However, if the latency of the CAP following nerve stimulation near the brain stem was similar to the value obtained following stimulation 1-2 cm distally near the meatus, this phenomenon could always be discovered and explained by "current jump" across the nerve lesion. Thus, latency measurements proved that the anatomical and electrical stimulation sites were not necessarily identical in some patients, a fact to be taken into account during assessment of facial nerve function under the narrow and wet anatomical conditions in the cerebellopontine angle.

If one used threshold stimuli, and provided that the respective nerves were not in direct contact with the facial nerve, distinction from the caudal cranial, the trigeminal, and the vestibulocochlear nerves was mostly easy. Again, the risk of current jumps could be minimized by the use of threshold stimuli, and comparison of amplitudes or of the stimulus threshold proved helpful.

Summary

This report describes our experience in 31 patients with the recently introduced technique of intra/extracranial (orthodromic) neurography of the facial nerve for monitoring facial nerve function during surgery within the cerebellopontine angle. By stimulation of the intracisternal segment of the facial nerve, a compound action potential (CAP) with amplitudes of 10-480 μV could be recorded extracranially from the nerve near the stylomastoid foramen after 0.92-2.28 ms. Usually there is no need for signal averaging, and the method is independent of the effect of muscle relaxants. The technique is useful for immediate and repeated localization of the facial nerve and its discrimination from the trigeminal and the lower cranial nerves during preparation within the tumor capsule.

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Clinical Subtyping of Trigeminal Neuralgia and Its Correlation to the Intraoperative Findings and Surgical Results Following Microvascular Decompression

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Introduction

From the beginning of 1983 through mid-March 1988, 150 microvascular decompressions were performed on a total of 137 patients (13 reoperations). In this period, a different form of treatment for trigeminal neuralgia (thermal coagulation) was adopted only if the patients disagreed with the suboccipital route or if their condition did not permit general anesthesia [3]. The operation was usually performed via a 2.5 x 2.5 cm craniotomy. A self-retaining spatula was introduced above the dorsolateral cerebellum, so that its tip was pointing directly towards the superior petrosal vein [1]. After initial experience with 15 patients, this route was found to avoid the preparation of the cerebral nerve VII and VIII regions. With the exception of two patients, all operations were performed in a sitting position.

Materials and Methods

There were 58 male and 79 female patients. Fifty-nine procedures were performed on the left-hand side, 78 on the right. The average age was 61.3 years; 33 patients were over 70 years of age.

The range of the postoperative observation period is between 1 month and 5 years. The operations were evaluated according to three defined categories: 1) totally pain-free patients, 2) patients with distinct improvement, and 3) patients with no or little improvement. Patients with pain recurrence with or without resurgery were classified as group 3 "not improved." Based on the analyses of clinical pain status, the patients with trigeminal pain were categorized in four groups:

Group I: patients with triggered shooting pains only (n = 85/137, 62%)
Group II: patients with continuous pain plus triggered shooting pains (n = 33/137, 24%)
Group III: continuous pain only (atypical neuralgia; n = 9/137, 7%)
Group IV: patients with burning pain either alone or combined with isolated continuous pain or combined with triggered pain (n = 10/137, 7%)

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Table 1. Results of microvascular decompression (137 patients)

		Pain-free	Improved	Unchanged
Group I	85	72 (84.7%)	8 (9.4%)	5 (5.9%)
Group II	33	23 (70%)	8 (24%)	2 (6%)
Group III	9	4	2	3
Group IV	10	3	2	5

Results

In group I, 67/85 patients were pain-free after the first operation, three had a late recurrence, eight were improved (one late recurrence), and ten remained unimproved (two of these were primarily unchanged, seven shifted from the "pain-free" group to the "improved" group due to an early recurrence). In group II, 23/33 patients were primarily pain-free, eight were improved, and two remained unchanged (Table 2). In group III, four of nine were pain-free, two improved, and three unchanged, while in group IV three of ten patients were pain-free, two improved, and five unchanged.

After 12 reoperations, 72 of 85 patients in group I were pain-free, eight improved, and five unchanged (one of these was reoperated for late recurrence). One reoperation was performed in group II, leaving 23/33 (70%) patients pain-free (Tables 1-3).

Table 2. Results of microvascular decompression in group II (33 patients)

Pain-free	23	(1 recurrence)
Improved	8	of which:
		2 with continuous pain disappeared, shooting pain unchanged
		2 with continuous pain improved, shooting pain disappeared
		1 with continuous pain and shooting pain improved
		2 with continuous pain unchanged, shooting pain disappeared
		1 with continuous pain unchanged, shooting pain improved
Unchanged	2	

Table 3. Relations of each group to the intraoperative finding and the postoperative results (R, recurrence)

		Typical arterial compression	Other vessels, arterial branches, veins	No vessel, arachnoiditis
Group I	(85)			
Pain-free	(72)	64 (9 R)	4	4 (1 R)
Improved	(8)	6 (1 R)	2	-
Poor	(5)	3 (1 R)	-	2
Group II	(33)			
Pain-free		21 (1 R)	1	1
Improved		6	1	1
Poor		2	-	-
Group III	(9)			
Pain-free		2	1	1
Improved		-	1	1
Poor		-	1	2
Group IV	(10)			
Pain-free		3	-	-
Improved		2	-	-
Poor		4	-	1
137		113 = 82.5%	11 = 8%	13 = 9%

A renewed compression was found in 9 of 13 reoperations. In one case, there had been no intraoperative finding the first time. Due to the relatively long pain-free period of 17 months, a reoperation was performed, showing an arterial vessel which had been overlooked during the first operation. With regard to the intraoperative findings, 113/137 patients had typical arterial compression. Of these 113 patients, 80 were provided lasting relief from the time of the first operation until now, and ten after a reoperation. Only 18 of the 24 patients without definite arterial compression or any other vascular finding improved (Table 3). In group I, an arterial vessel was found in 73/85 cases; 64 of these were pain-free and six improved. In group II, a vessel was found in 29 of 33 cases; 21 were pain-free and six improved. Two of the nine group III patients presented with a vessel (both pain-free), and nine group IV patients had a definite intraoperative finding (three were pain-free and two improved). The complications are listed in Table 4.

Discussion

The conclusion from our findings is that patients with trigger and shooting pains have the best postoperative results (1). Group II, with additional continuous pain, still had a good prognosis, with 70% pain-free and 24% improved, so that the continuous pain component does not represent a contraindication for the operation. When a definite arterial compression was found, 88% of group I and 72% of group II patients were pain-free postoperatively. Since renewed vascular compression was noted in 8 of 13 patients at reoperation due to lysis

Table 4. Complications after 150 microvascular decompressions, including 13 reoperations (R)

Fatal outcome due to aspiration (sudden onset, 10th postoperative day)	1
Intracerebellar hemorrhage with severe brain stem damage (died after 5 months)	1 (R)
Acute subdural hematoma (supratentorial) due to a torn small bridging vein (favorable outcome)	1
Postoperatively decompensated cardiac insufficiency (reversed)	1
Impaired coordination (mild)	2
Dizziness (mild)	2
Anacusis	1
Hypoacusis	4 (1R)
Hypo- or paresthesia	6 (1R,4 Ivalon)
Hypo- or paresthesia completely reversible	4 (2R,4 Ivalon)
Trochlear paralysis (reversible)	1
Psychoorganic disorder	1
Cerebrospinal fistula	1
Wound infection	2
Hyperthermia without meningitis for 1 month	1
Meningitis	1
Occipital nerve neuralgia	1
Deterioration of encephalitis disseminata	1

or dislocation of the graft, we are now using Ivalon patches (34 patients). We do, however, have the impression that this relatively firm material leads to sensory deficits in an overproportionately high number of cases (8 of 34, 4 completely reversible). The complications we noted (Table 4) most frequently occurred in patients 70 years of age or older [2].

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Neurovascular Compression as a Cause of Essential Hypertension: A Microanatomical Study

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Introduction

In industrialized countries 10% of the population suffer from elevated blood pressure (diastolic $\geq$ 95 mmHg, systolic $\geq$ 60 mmHg - WHO). Arterio-/arteriolosclerosis, parenchymal changes of the kidney, pheochromocytoma, coarctation of the aorta, and elevated intracranial pressure (Cushing response) are known to be conditions that induce and maintain arterial hypertension (HTN) in about 10% of the cases. Idiopathic arterial HTN or essential HTN is diagnosed in the other 90% of patients with HTN, in whom the etiology remains unclear (WHO).

The possible influence of the central nervous system (CNS) in creating this most frequent type of HTN has been discussed [1,2,6,19,20]. GUYTON and REIS postulated an elevation of the normal level of CNS regulation [3,21].

From the neurosurgical standpoint, JANNETTA (1979 - 1985) introduced his concept of neurovascular compression (NVC) at the root entry zone (REZ) as a cause of essential HTN. On the basis of intraoperative observation, microvascular decompression, and animal experiments, NVC at the ventrolateral medulla (VLM) and the REZ of cranial nerves IX and X on the left is suspected to be a central factor causing HTN [8,10-18,23,24,26,27].

Comparative pathological studies between patients with and patients without HTN had not yet been carried out. We therefore investigated the neurovascular relations at the medulla of deceased patients by microsurgical technique.

Material and Methods

Material

Forty patients aged between 40 and 87 years (mean age 68.2 years) were examined 8-24 h postmortem during autopsy.

Twenty-three patients had a documented history of elevated blood pressure. Of them, 19 had essential HTN, whereas four suffered from renal HTN. Seventeen patients served as controls. They were known to have had normal blood pressure during their lifetime.

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Table 1. Clinical data

	No.	Sex (m / f)	Min-max age	x age
Normotensive pat.	17	8 / 9	40 - 82	63
Renal HTN	4	2 / 2	56 - 79	70.2
Essential HTN	19	12 / 7	48 - 87	71.5
Total	40	22 / 18	40 - 87	68.2

The patients did not have any neurological deficit and did not die from any neurological condition. Patients' history and clinical charts were reviewed carefully (Table 1).

Methods

The calvaria was opened with a circular cut and removed cautiously to avoid any damage to the dura. After opening the dura, cerebrospinal fluid was let out and the adhesions of the hemispheres with the dura of the anterior cranial fossa were loosened. The tentorial edge was exposed. Using a transversal cut through the cerebral peduncle the hemispheres could be removed from the skull, and the brain stem remained in situ below the tentorium in the posterior cranial fossa.

Subsequent examinations were carried out with microsurgical technique using 6- to 25-fold magnification. Cutting the tentorium at its insertion in the petrous bone, the ventral surface of the brain stem was exposed by stepwise dissection of the arachnoid along the clivus. By injecting an autologous blood-H_2O solution into the posterior cerebral artery or basilar artery at a pressure of 100-160 mmHg we were able to simulate the vital neurovascular status. The neurovascular relations between all cranial nerves and vessels and especially the REZ of cranial nerves IX and X and the bilateral VLM were examined carefully. The findings were documented by microphotography.

Results

There were no signs of NVC at the left VLM and REZ of cranial nerves IX and X in 17 patients with normal blood pressure. On the right side, however, we observed signs of compression in two cases. Patients with renal HTN did not have any NVC. In contrast, we observed a distinct NVC at the left VLM in all 19 patients with essential HTN (Table 2).

One patient from the normotensive controls with a longstanding history of left-sided idiopathic hypoacusis and tinnitus had a cross-compressing vein at the acoustic nerve on the same side.

Altogether we observed 24 NVCs at the VLM. The compression appeared 19 times on the left side and only five times on the right. Common to all NVCs at the VLM was a compression of the medulla in the retro-olivary sulcus at the medial surface of the REZ of cranial nerves IX and X.

The posterior inferior cerebellar artery (PICA) caused NVC 12 times (Fig. 1), followed by a combination of PICA and vertebral artery. The

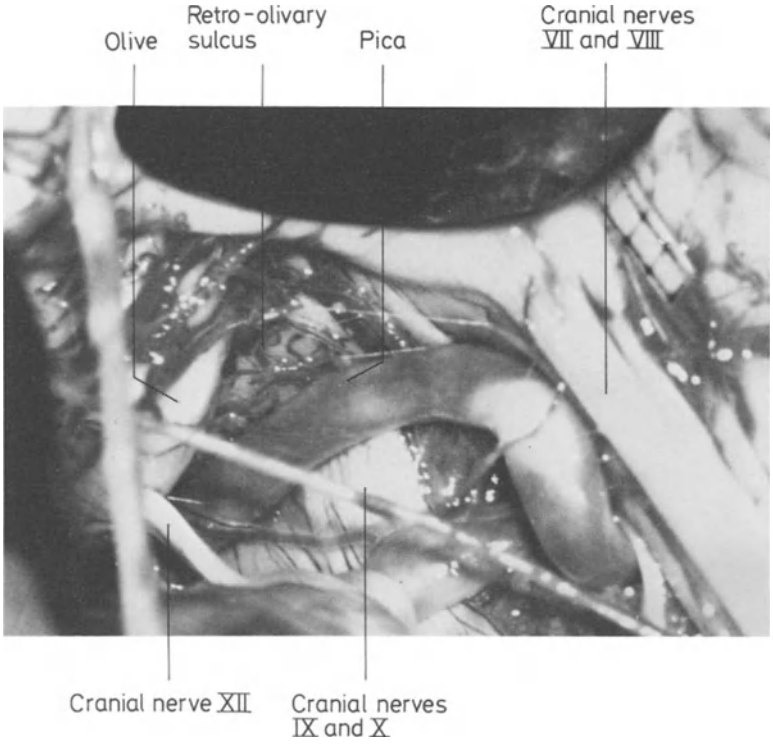

Fig. 1. NVC at the VLM on the left by the PICA as was seen 12 times. Ventral surface of the medulla with the cranial nerves VII-X. The compression appears in the retro-olivary sulcus and involves the medulla and the REZ. X 16

basilar artery, vertebral artery, and anterior inferior cerebellar artery were each observed to cause compression once (Table 3).

Discussion

Our constant finding of a close connection between an NVC at the VLM and essential HTN corresponds to JANNETTA's intraoperative observations. He reported NVC in 51 of 53 patients with essential HTN who underwent left retromastoid craniectomy for trigeminal neuralgia or hemifacial spasm [12,13,15,17].

There were no neurological signs of cranial nerve disturbance in any patient with HTN we examined. From this one may assume that the NVC at the VLM observed by JANNETTA is not to be seen in the light of trigeminal neuralgia or hemifacial spasm, but rather essential HTN.

Older microanatomical studies on cranial nerves from WATT (1932) and SUNDERLAND (1948), together with more recent studies from RHOTON (1975) and FEIN (1980) on caudal cranial nerves, also point in this direction [6-9,22,27]. FEIN (1980) reported signs of NVC in seven of eight patients with arterial HTN.

Table 2. Frequency and distribution of NVC at the VLM in the different groups

	Normotensive pat. (n = 17)	Renal HTN (n = 4)	Essential HTN (n = 19)
Unilateral NVC, right	2	0	0
Unilateral NVC, left	0	0	16
Bilateral NVC	0	0	3
Total NVCs (n = 24)	2	0	22

Table 3. Arteries causing left NVC in essential HTN

A. basilaris	1
A. vertebralis	1
A. cerebell. inf. anterior (AICA)	1
A. cerebell. inf. posterior (PICA)	12
PICA and A. vertebralis	4
Total	23

HTN by itself leads to changes of cerebral vessels and may result in ectasia and elongation. If such neurovascular contacts as are seen in NVC were only the result and not the cause of HTN, one would expect to find NVC on the right side with about the same frequency as on the left side. One would also expect to find an increased vascular looping and NVCs of the cranial nerves III-VIII. At autopsy we did not find any compressing vascular loop at the upper brain stem. Only one patient from the normotensive controls with longstanding left hypoacusis had NVC at the VIIIth nerve by a vein.

There are no clinical data available indicating a higher incidence of cranial nerve dysfunction in arterial HTN.

Compression of the medulla in the retro-olivary sulcus was common to all types of NVC we found in essential HTN. This suggests the compressing vessel loop is not a result of HTN, but rather its direct cause.

Our investigations are now being continued by histological brain stem studies from the cases examined for the present study and by retrospective and prospective evaluation of vertebral angiograms.

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Essential Hypertension in Patients with Hemifacial Spasm or Trigeminal Neuralgia

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Introduction

Elevated arterial pressure is a major public health problem in industrialized countries. When untreated it may lead to lethal complications [3]. Nearly 20% of a Caucasian suburban population, such as that of the Framingham Study, have hypertension (blood pressure over 160/95 mmHg) [14]. Hypertension is essential or idiopathic in at least 90% of cases [4].

Recently, vascular compression of the left ventrolateral medulla (VLM) at the root entry zones of the cranial nerves (CN) IX and X was thought to represent a cause of essential hypertension (EH) [5,8,9,10]. It has been reported that microvascular decompression (MVD) of the left VLM, performed in hypertensive patients during operations devised for cranial nerve vascular syndromes, is associated with postoperative lowering of the blood pressure [9]. However, it is not clear in which way MVD of the left VLM lowers the blood pressure, nor do we know the incidence of EH in patients undergoing MVD for cranial nerve vascular syndromes.

We undertook this study to clarify possible relationships between EH and hemifacial spasm (HFS) or trigeminal neuralgia (TN) and to study the antihypertensive effect of MVD of the left VLM. The present paper reports on the prevalence rates of EH and on the postoperative changes in diastolic blood pressure observed in patients undergoing MVD of the root entry zones of CN V or VII. In addition, results obtained in seven EH patients with left-sided HFS who underwent MVD of the left VLM are presented.

Materials and Methods

Two hundred and twenty-six consecutive patients, presenting either left (L) or right (R) HFS or TN, form the subject of this study. They were treated by MVD of the root entry zones of CN V or VII between 1980 and 1988. Forty-nine of them had HFS-L, 33 HFS-R, 60 TN-L, and 84 TN-R. In HFS the vertebral artery alone or in combination with the anterior or posterior inferior cerebellar artery was responsible for the vascular compression in 30% of the left-sided and 9% of the right-sided cases. In the other cases of HFS the offending vessels

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Table 1. Comparisons between 226 patients with HFS or TN, 49 patients with HFS-L, and a reference population[a]

Characteristics	HFS+TN	HFS-L	Reference	p[a] HFS+TN vs Reference	p[a] HFS-L vs Reference
Sex: male (%)	44	49	-		
Mean age (years)	55.0	55.1	45-64		
Overweight (%)	17	10	19	>0.25	>0.1
Smoking (%)	20	20	32	<0.025	>0.1
Diabetes (%)	1	0	1	>0.25	
EH(%)	22	41	23	>0.25	<0.025

[a] P values calculated using chi-square test

were the anterior or posterior cerebellar artery, arteriovenous malformations, or veins.

The prevalence rate of EH and the distribution of age, sex, and hypertension-related risk factors (overweight, smoking, diabetes) were assessed and compared to reference rates in a matched general population [6,7,11,13] (see Table 1).

- Patients were judged to be **hypertensive** when blood pressure was over 160/95 mmHg or when they were on antihypertensive treatment. Newly diagnosed patients had a thorough checkup to rule out secondary hypertension. In patients with known hypertension, secondary hypertension had already been ruled out.
- **Overweight** was defined as a deviation of 20% or more from the desirable weight according to the Metropolitan Life Insurance Company criteria [2,11].
- **Smoking** more than ten cigarettes a day classified a patient as a smoker.

We did not study the incidence of hyperlipidemia and hyperuricemia, since our patients were not routinely screened for these additional risk factors before 1987.

A mean diastolic blood pressure was calculated from three daily measurements, obtained in three postoperative time periods (2nd-5th days, 6th-9th days, and the last 2 days before discharge). These values were compared to preoperative diastolic blood pressure means, measured on at least 2 preoperative days.

Finally, the efficacy of MVD of the left VLM in seven patients with EH and HFS-L was assessed, observing postoperative changes in anti-

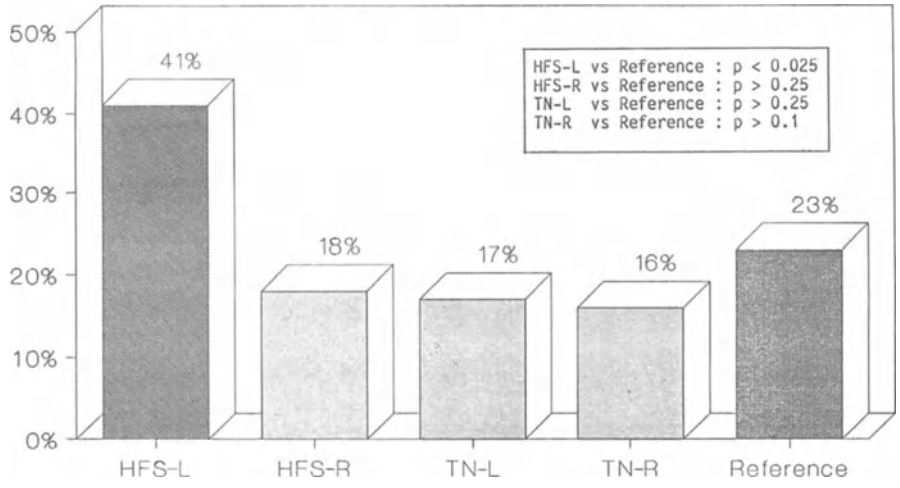

Fig. 1. Prevalence of EH: comparison between a reference population and four surgical subgroups

hypertensive medication and diastolic blood pressure levels during a follow-up period ranging from 3 to 48 months (median 24 months). Patients' response to MVD of the VLM was graded as **good** when they did not require any antihypertensive medication, **fair** when their medications were reduced, and **poor** when there was no change in their antihypertensive therapeutic regimen.

Inspection of the VLM for vascular compression of the root entry zones of CN IX and X was not routinely performed. When carried out it was found to be strongly positive in the seven patients mentioned.

Statistical analysis was done whenever indicated, using the chi-square test or t-test.

Results

Forty-nine (22%) of our patients had EH; 44 (90%) of them were on antihypertensive treatment prior to operation, while five (10%) had newly diagnosed EH.

Even though there was no statistically significant difference in the prevalence of EH between our complete patient group (22%) and a matched reference population (23%) ($P > 0.1$), there was a significant difference ($P < 0.025$) between the HFS-L group (41% prevalence) and the reference population (Table 1). The same did not hold true when the other subgroups (HFS-R, TN-L, TN-R) were compared to the reference population ($P > 0.1$) (Fig. 1).

There was no statistically significant difference between the HFS-L patients and the other patient groups, including the reference group, with regard to age, sex, overweight, smoking, or diabetes ($P > 0.1$) (Table 1).

The postoperative diastolic blood pressure was statistically significantly lower than the preoperative one in all patients, normotensive as well as hypertensive (Table 2).

Table 2. Mean changes in blood pressure, preoperative vs postoperative[a]

	Diastolic blood pressure (mmHg)		Change	
Patients	Preoperative	Postoperative	Value	p[b]
All (n = 226)	80.7	73.7	-7.0	<0.01
EH (n = 49)	90.6	78.9	-11.7	<0.01
Normotension (n = 177)	77.0	70.6	-6.4	<0.01

[a] At the time of discharge
[b] P values calculated using t-test

Six of the seven EH patients with HFS-L in whom MVD of the left VLM was performed did not require any medication in the immediate postoperative period, while one had his medication reduced by half. At a median follow-up of 24 months (range 3-48 months) two patients remained without any medication, two required half their original regimen, and three had to be switched back to their preoperative dose (Table 3).

The main offending vessel found to compress the root entry zone of CN IX and X was the vertebral artery (six of seven cases). In HFS-L the vertebral artery was involved in compression of the root entry zone of CN VII in 30% of cases, whereas the vertebral artery was involved in only 9% of the HFS-R cases.

Discussion

Hypertension is essential in over 90% of cases. The cardiovascular system is controlled by mechanical, chemical, and neural mechanisms. Subtle alterations in the organization of neural control may cause an imbalance at the peripheral and/or central level, resulting in EH

Table 3. Early and late results of MVD of the VLM in 7 patients with EH and HFS-L (all patients were on preoperative antihypertensive treatment)

Grade[a]	Discharge	Follow-up[b]
Good	6	2
Fair	1	2
Poor	0	3

[a] Good: no antihypertensive medication needed; fair: reduction in antihypertensive medication; poor: no change in medication
[b] The follow-up ranged from 3 to 48 months (median 24 months)

[1,12]. Information concerning the blood pressure is mainly mediated by the left vagal and glossopharyngeal nerves and converged to the nucleus tractus solitarius and vasomotor center in the VLM.

Jannetta has demonstrated in animal experiments that pulsatile compression at the root entry zones of CN IX and X can cause transient hypertension. He speculates that a similar mechanism may be operative in humans with increasing age, due to elongation and dilatation of the aging blood vessels as well as to the sagging of the brain [8-10]. Were that to be true, one would expect to find a higher incidence of EH in patients with cranial nerve vascular syndromes. Our data fail to show this, but at the same time they show that patients with HFS-L have a higher incidence of EH, even when adjustment is made for hypertension-related risk factors. It is not possible to state whether this has anything to do with the observation that in HFS-L the vertebral artery was found to be involved in compressing the root entry zone of CN VII more frequently than in the other subgroup with HFS-R (three times more frequently). One could speculate that more proximal alterations in the vertebral artery itself or in its branches could somehow, by compressing the root entry zones of CN IX and X or by compressing the VLM, be responsible for a neural imbalance resulting in a higher incidence of EH in the subgroup of patients with HFS-L. That this relation is more complex than one would expect is shown by the variable response of our seven patients to MVD of the VLM. It was interesting that all of them showed an immediate response to the MVD while within a 1- to 4-month period the response declined or disappeared completely. Jannetta reported relief or improvement of hypertension and reduction in antihypertensive medication in over 80% of the patients he treated with MVD of the left VLM [9]. The issue of the relationship between vascular compression of the left VLM and hypertension is further complicated by the observation that the diastolic blood pressure was significantly lowered in all of our patients, hypertensive and normotensive.

In conclusion, our study indicates:

1. That there is not a higher incidence of EH in patients with cranial nerve vascular syndromes
2. That there is a subgroup of patients with a high incidence of EH (HFS-L)
3. That the response of EH patients of this group to MVD of the left VLM is not predictable
4. That in all patients in whom an MVD for cranial nerve vascular syndromes was performed the diastolic blood pressure at the time of discharge was significantly lowered

Relationships between EH and vascular compression of the VLM are obviously extremely complex and can only be proved by a prospective randomized study in which the VLM is explored in all patients with EH and cranial nerve vascular syndromes. When an offending artery is obvious a decompression should be randomly performed.

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Neurosurgical Topography of the Pyramidal Tract

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Introduction

The pyramidal tract is one of the most important motor fiber systems and must be preserved during surgery in the area of the precentral gyrus. Detailed knowledge of the localization of the pyramidal tract and the craniocerebral topography facilitates its preservation [6-8]. In the present study Klingler's fiber dissection method was applied to dissect the pyramidal tract in the frontal lobe and centrum semiovale, in order to obtain exact data on the topography of the pyramidal tract.

Topographical Anatomy of the Pyramidal Tract

"The pyramidal tract consists by definition of all those fibers which course longitudinally in the pyramid of the medulla oblongata, regardless of their site of origin" [5]. The origin of the pyramidal tract includes wide areas of the frontal and parietal lobes [1].

In order to facilitate the presentation of the pyramidal tract its origin was restricted to the main origin in the area 4 (40%) [1]. The fibers of the pyramidal tract originate from the whole convexity of the precentral gyrus and converge to the posterior part of the posterior limb of the internal capsule [1,2,4,5,10,11,15-17]. Thus the fibers of the face, arm, trunk, and leg show an arrangement like a fan, which opens against the cortex and shuts when approaching the posterior limb of the internal capsule [3,4,16,19]. Hence the course of the pyramidal tract is defined by the localization of its origin, the precentral gyrus, and the topography of the posterior limb of the internal capsule.

Material and Methods

Thirty formalin-fixed hemispheres of normal adult brains of both sexes were investigated. To gain data on the topography and three-dimensional course of the pyramidal tract itself, the fiber dissection method of Klingler was applied [14]. The brains were fixed in formalin for 2-4 weeks. The formalin was then washed out and the brains were frozen at -8° to -10°C for some days [14]. After thawing of the brains, dissection was carried out with a "Schweizer Uhrmacherpinzette" and a Zeiss microscope according to the recommendations of KOMAROMY and HUILTKRANZ [12,13]. On the basis of the known topography of the precentral convolution, the precentral gyrus was identified (Fig. 1) [6,8]. Afterwards the fibers of the pyramidal

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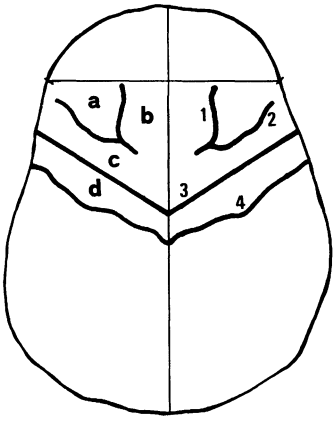

Fig. 1. Schematic drawing of the central region at the vertex. Convolutions (left side): a, middle frontal; b, superior frontal; c, precentral; d, postcentral. Fissures (right side): 1, superior frontal; 2, precentral; 3, central; 4, postcentral

tract, originating from the precentral gyrus, were dissected free after the method of Klingler by removal of the frontal and occipital mass of fibers. Medially the caudate nucleus and thalamus and laterally the insula Reilii with the basal ganglia were removed.

Results and Discussion

Results of Klingler's Fiber Preparation Technique

With this technique it is possible to prepare clearly the course and topography of a fiber tract in the hemisphere. Instead of reconstructing the course of such tracts by numerous sections, the technique allows demonstration of the course of the tract from its origin to its termination, in its three-dimensional and topographical relations. With this technique the pyramidal tract was dissected exactly in the centrum semiovale and internal capsule, from its origin, the precentral convolution, to the cerebral peduncles (Figs. 2, 3). The accurate dissection of the fiber systems, i.e., fibers of projection and association, and especially commissural fibers [10-12,15-18]. The intermingling of the commissural fibers of the splenium with their horizontal course and the vertically running fibers of the pyramidal tract allows only a rough dissection of the pyramidal tract in this area. Nevertheless, a clear specimen of the whole pyramidal tract could be prepared (Fig. 3). Regardless of these anatomical and tech-

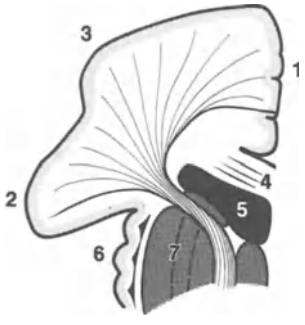

Fig. 2. Schematic drawing of the fan of the pyramidal tract of a left hemisphere (looking from behind). 1, medial border of the precentral convolution; 2, lateral border of the precentral convolution; 3, convexity; 4, splenium; 5, lateral ventricle; 6, insula Reilii; 7, lentiform nucleus

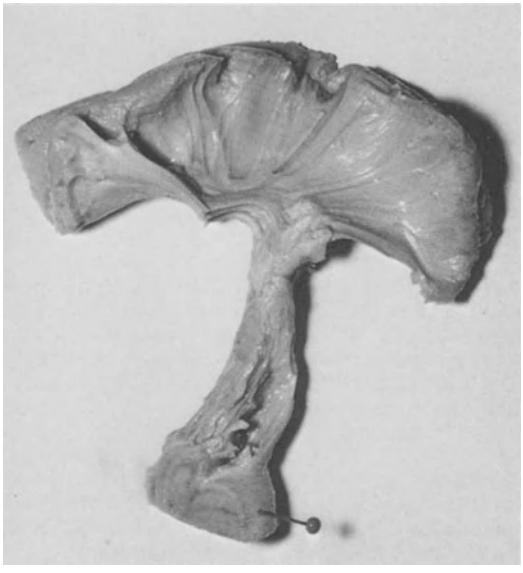

Fig. 3. Photograph of a left specimen of the pyramidal tract, obtained with the preparation method of KLINGLER [14] (looking from behind)

nical problems, the method has great advantages; with training very good preparations can be accomplished, and the fan-like arrangement of the pyramidal tract and its microsurgical topography can be clearly shown [14].

Topography of the Pyramidal Tract in the White Matter of the Frontal Lobe

In the description and definition of specific fiber tracts in the white matter, the problem arises of how to define their course, because in the mass of the white matter no landmarks exist and the fibers in question are mixed with other fiber systems. In some tracts, e.g., the optic radiation, the inferior and posterior horn of the lateral ventricle can be used as a safe landmark [9]. Especially in the centrum semiovale of the frontal lobe the course of the pyramidal tract is not defined by any cerebral landmark and cannot be differentiated from the neighboring fiber systems. This problem can only be resolved by following the pyramidal tract from its well defined main origin, i.e., the precentral gyrus, to its known "termination" in the cerebral peduncle above the pons. The preparation after Klingler's method allowed this approach because the fibers were followed from their origin to the cerebral peduncle.

Thus the configuration and topography of the precentral gyrus (origin) and the fiber system in the internal capsule and cerebral peduncle determine the shape and topography of the pyramidal tract itself. The anterior and posterior limits of the pyramidal tract are defined by the thin layer of fibers originating from the most anterior and posterior borders of the precentral gyrus. The medial and lateral borders of the pyramidal tract are determined in the subcortical area and centrum semiovale by following the most medial originating fibers at the medial end of the precentral gyrus and the most lateral originating fibers of the lateral end of the precentral gyrus. With this technique the fan-like configuration of the pyramidal tract could be prepared clearly (Figs. 2, 3).

Summary

The microsurgical topography and three-dimensional course of the pyramidal tract in the white matter of the frontal lobe can be shown clearly using Klingler's fiber preparation technique. The localization of the pyramidal tract is given by the topography of the precentral convolution and the posterior limb of the internal capsule. Knowledge of the exact topography of the pyramidal tract can facilitate the preservation of this important fiber tract during surgery.

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Postoperative Mortality in the Era of Microneurosurgery

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Introduction

Even with the aid of microneurosurgery, not all neurosurgical diseases can be cured. Further improvements of the surgical results may be possible by detailed analysis of the unfavorable outcomes and unsatisfactory results. In order to assess the major problems limiting microsurgical attempts at curing neurosurgical diseases today, we performed a retrospective reevaluation of the postoperative deaths in our intensive care unit (ICU). We believe that the mortality during hospitalization is an indicator of severe surgical and perioperative failures and complications.

Method

In our department all patients who have undergone intracranial operations, including transsphenoidal ones, are brought to the ICU. Patients with operations on the skull, orbit, and spine are only electively admitted to the ICU. Trauma cases are only admitted for secondary procedures (e.g., CSF fistula repair). Patients whose condition has deteriorated secondarily in other wards are readmitted to the ICU. Patients in poor preoperative or postoperative condition are only transferred to other hospitals when they have been vegetatively stabilized.

Between January 1983 and December 1987, 3355 patients were admitted to the ICU. Brain tumor operations or other intracranial procedures were performed in 75%. Ninety-four patients died in this period, 30 of them without a preceding operation and five after stereotactic procedures. The remaining 59 patients died after neurosurgical procedures (Table 1).

Mortality in Patients Not Operated On

Ten percent of the patients who were not operated on died in the ICU. The most frequent cause of death were bleedings in the subarachnoid space or into the substance. This is a consequence of our policy of investigating early acute bleedings. Four patients died after a recurrence of subarachnoid hemorrhage from aneurysm: one from an unnoticed anterior communicating artery aneurysm, the others while waiting for a decision on whether a giant aneurysm could be treated by balloon. Two of them had not bled until the final event. These patients underline once more the danger of rebleeding of aneurysms during hospitalization (Table 2).

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Table 1. Admissions to the ICU, 1983-1987

Diagnosis	No.
Intracranial lesions[a]	
Tumor	1669
Vascular	475
Other	342
Extracranial and spinal lesions[b]	533
Stereotactic[b]	38
Supervision, no op.	298
Total	3355
Postoperative deaths	64
Nonsurgical deaths	30
Mean postoperative stay: 5 days	

[a] Routinely admitted; [b] Electively admitted

Mortality After Neurosurgical Procedures

The majority of routine operations were followed by a mortality of below 4%. Even difficult procedures in cases of mediobasal meningioma or acoustic neurinoma were followed by a mortality of 1% or less, despite the fact that nearly all of these tumors were removed radically. The outcome in vascular diseases was not as favorable, with

Table 2. Causes of death in nonoperated patients (30/298 = 10%)

Cause of death	Patients	
	No.	%
Intraparenchyma hemorrhage		
No recovery	10	3.4%
Aneurysm		
Initial bleeding	3	1.0%
Lethal "preoperative" rebleeding	4	1.3%
Subarachnoid hemorrhage		
No recovery	4	1.3%
Tumor		
Nontreatable	4	1.3%
Spinal	2	1.0%
Nontreatable quadriparesis		
Trauma	2	1.0%
Nontreatable		
Encephalitis	1	0.5%
Total	30	10.1%

mortality as high as 8%. The results of early aneurysm surgery were not as satisfactory as those of delayed surgery, with 6.3% mortality as opposed to 3.4%. This is associated with the fact that one of the consequences of the policy of early surgical intervention is an increased number of patients in poor preoperative condition. In patients in good preoperative condition the mortality in early surgery equals that in late surgery. Unsatisfactory results were obtained in cerebellopontine angle meningiomas, especially if they were of the petroclival subtype. The results in cases of chronic subdural hematoma which were treated in the ICU (because of their poor condition) were worse than those in the majority of brain tumors. The mortality in patients with subdural hematomas treated in the same period was 2%, which was twice as high as for mediobasal meningiomas, for example (Table 3).

The causes of death could be subdivided roughly into three equal groups: One-third of the patients suffered from lesions which were not effectively treatable by an operation (e.g., patients in grade 5 after an aneurysmal bleeding or incarcerated brain tumors). Another third died of surgical and perioperative failures and problems. Surgical lesions with early postoperative death were rare (5 of 59 patients). Secondary fatal complications following surgery were more frequent: 12 of 59 patients. These secondary fatal outcomes in most cases were caused by pulmonary embolism, pneumonia, and infections following surgical complications with poor recovery. The perioperative management was as important as the direct surgical trauma (e.g., vasospasm which decompensated after inadvertent hypotension), with 5

Table 3. Diagnosis of diseases with postoperative mortality

Diagnosis	Operations	Mortality	
	No.	No.	%
Meningioma, CPA	38	5	13.2%
Abscess/empyema (ICU)	20	2	10.0%
Chronic subdural hematoma (ICU)	77	7	9.1%
Cerebellar hemangioblastoma	25	2	8.0%
AVM	79	6	7.6%
Aneurysm, early	188	13	6.9%
Cerebellar metastasis	34	2	5.9%
Hypophyseal TU, trep.	29	1	3.4%
Aneurysm, late	58	2	3.4%
Hypophyseal TU, oronasal	188	3	1.6%
Cerebellar glioma	62	1	1.6%
Hydrocephalus/shunt (ICU)	162	2	1.2%
Glioma, metastasis, supratentorial	624	8	1.1%
Meningioma, mediobasal	193	2	1.0%
Frontobasal fracture, CSF fistula	106	1	0.9%
Meningioma, convexity + falx	229	2	0.9%
Cerebellopontine angle TU (glomus, acoustic, epidermoid)	140	-	-
Total	2252	59	2.6%

(ICU), lesions treated only electively at ICU

of 59 deaths. Finally, one-third of the patients died from secondary complications after an uneventful operation. In 10 of 59 patients these complications were related to the operation (hemorrhage and infection). In eight cases, complications were not related to the operation (Table 4).

Of 1669 patients operated on for a brain tumor, 24 (1.4%) died in the ICU. The analysis of the surgically induced fatal outcomes shows that with one exception, the patients suffered from inadvertent lesions of large arteries or small perforating vessels. Five patients died of early pulmonary embolism within 48 h after uneventful operations. Since we have preferred a position in which the legs are elevated to the level of the nose, these problems have disappeared, despite the fact that we use no mechanical or pharmacological antithrombotic measures. Only one single patient died of a late pulmonary embolism despite good initial recovery. One patient died due to an unrecognized CSF hyperdrainage during operation with brain stem incarceration, one 80-year-old patient died due to fluid lung caused by hyperinfusion, and four died due to infections after an uneventful operation.

Of 236 patients who underwent aneurysm surgery, 15 (6.1%) died. The cause of death in seven of these patients was the initial bleeding. This fact emphasizes the importance of better prognostic criteria. The remaining eight patients died of surgical or management complications, which reflects the typical problems of (early) aneurysm surgery. These cases included two rebleedings (one after clipping and another after coagulation of a large cavernosus aneurysm), one hypertensive intrapontine hemorrhage due to a high perfusion pressure (lumbar drainage and systemic hypertension), one decompensated vasospasm (after unintended hypotension), and four cases of complicated surgery.

Table 4. Causes of postoperative mortality in intracranial procedures (59/2486 = 2.4%)

Cause of mortality	Patients	
	No.	%
Nontreatable lesion: poor preoperative condition, no recovery	19	32.2%
Indirect surgical lesion: no or incomplete recovery, secondary death	12	20.3%
Direct surgical (brain stem) lesion: no recovery, early death	5	8.5%
Management/wrong indication	5	8.5%
Uneventful operation, secondary lethal complications:		
Postop. hemorrhage	2	3.4%
Postop. infection	8	13.6%
Internal medical	8	13.6%
Total	59	100%

Of 79 patients who underwent surgery for arteriovenous malformations, 6 (7.6%) died. There were two deaths (8%) after the removal of hemangioblastomas of the posterior fossa in 25 patients. The fatal complications in cases of arteriovenous angioma were typical for arteriovenous malformations: twice incomplete removals with rebleedings, once rebleeding after complete removal, and once breakthrough. Two patients operated on in the acute stage after a massive bleeding did not recover.

Discussion

Analysis of the early postoperative mortality during hospitalization supplies only limited information on the late result. Six months' follow-up after early aneurysm surgery revealed that the mortality increased (from 6.9% to 8.0%) due to secondary complications (mostly in patients discharged in poor condition).

The mortality analysis showed that an active policy for vascular diseases is followed by a relatively high incidence of surgical and nonsurgical deaths due to bleedings and surgical complications in arteriovenous malformations and in aneurysms. To overcome or to reduce the fatal problems in arteriovenous malformations, the endovascular neuroradiologist can reduce the volume of the malformation to a volume suited for operation and adapt the brain circulation on a normal flow. In aneurysm surgery, especially for large and giant ones, endovascular procedures may also be helpful. In "normal" aneurysm operations the experience of the surgeon and the preoperative management must be improved in order to obtain better results.

Tumorous lesions, gliomas, metastases, convexity and frontobasal or parasellar meningiomas, as well as acoustic tumors pose no serious problems as concerns mortality. In these tumors, the reduction of the morbidity is more important. The unfavorable results in petroclival meningiomas are partially due to tumors which did not respect the arachnoid membranes and in which dense adhesions existed between the tumor and brain stem or vessels. For these tumors we now prefer partial removal or a staged operation.

Incidence, Management, and Outcome of Patients with Premature Rupture of Cerebral Aneurysms During Surgery

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Introduction

Despite the routine application of microsurgical techniques, the intraoperative rupture of an intracranial arterial aneurysm represents an unexpected and sometimes disastrous event. Although every neurosurgeon has encountered intraoperative rupture, the neurosurgical literature on this topic is surprisingly sparse. Few authors have tried to determine the incidence, the criteria of management, and the impact of intraoperative aneurysm rupture on the subsequent outcome of patients with subarachnoid hemorrhage [2,4,9,10,14]. In this study we report the results of a retrospective investigation of 85 intraoperative aneurysm ruptures occurring among 204 surgically treated aneurysms.

Material and Methods

Within an observation period of 4 years, 85 patients among a group of 204 aneurysm patients presented with intraoperative rupture of their aneurysm during the surgical procedure (41.6%).

When the overall outcome is considered it is obvious that intraoperative aneurysm rupture has an unfavorable impact on the subsequent outcome (Table 1). Whereas in the group without intraoperative rupture 73% of the patients achieved a satisfactory postoperative result, only 61% in the group with intraoperative rupture did so. Consequently the percentage of patients in grades IV and V of the

Table 1. Intraoperative rupture and postoperative outcome (GOC)

119 patients without intraoperative rupture			85 patients with intraoperative rupture		
Postoperative status	No. of patients		Postoperative status	No. of patients	
I	69	(57.9%)	I	40	(47.1%)
II	18	(15.1%)	II	12	(14.1%)
III	8	(6.7%)	III	9	(10.6%)
IV + V	24	(20.1%)	IV + V	24	(28.2%)

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Table 2. Aneurysmal location and intraoperative rupture

119 patients with intraoperative rupture			85 patients with intraoperative rupture		
Localization	No. of patients		Localization	No. of patients	
Anterior comm. artery	46	(38.6%)	Anterior comm. artery	38	(44.7%)
Internal carotid artery	36	(30.2%)	Internal carotid artery	23	(27.0%)
Middle cerebral artery	25	(21.0%)	Middle cerebral artery	23	(27.0%)
Vertebr./basil. artery	12	(10.1%)	Vertebr./basil. artery	1	(1.2%)

Glasgow Outcome Scale [7] is increased from 20.1% in the nonrupture group to 28.2% in the rupture group.

Concerning the relationship between aneurysmal location and incidence of rupture, surgery of aneurysms of the anterior communicating artery has the highest risk of premature rupture, at 45%. The risk in carotid artery and middle cerebral artery aneurysms is equal, at 27% (Table 2).

As proposed by Yasargil, we have divided the surgical procedure into opening of the dura, microsurgical dissection, and finally clipping of the aneurysm (Table 3).

Premature rupture during the craniotomy period occurred in six patients, or 7% of the whole cohort. The grave prognosis of aneurysm rupture during this early operative period is demonstrated by the fact that five of these patients died.

Almost four-fifths of the intraoperative ruptures happened during the dissection period prior to definitive clipping. Of this patient group, 70% reached a satisfactory postoperative result, while one-fourth remained in a vegetative state or died. The high percentage of satisfactory outcome demonstrates that premature rupture during this operative period can usually be managed appropriately with the application of microsurgical techniques.

Table 3. Time frames of aneurysm (intraoperative rupture and outcome)

			Outcome (GOS)				
Surgical period	No. of patients		I	II	III	IV	V
Craniotomy	6	(7%)	0	1	0	0	5
Dissection	66	(78%)	33	10	6	0	17
Clipping	13	(15%)	6	1	3	1	2

Table 4. Intraoperative management (temporary clipping/induced hypotension) and postoperative outcome)

Postoperative status (GOC)	No. of patients	
Temporary clipping (n = 20)		
I	10	(50%)
II	4	(20%)
III	1	(5%)
IV + V	5	(25%)
Induced hypotension (n = 62)		
I	31	(50%)
II	6	(10%)
III	6	(10%)
IV + V	19	(30%)

Rupture during the final period of clip application occurred in only 15%, again with 70% of the patients reaching the postoperative status of I - III (GOS).

In addition to the continuation of microsurgical techniques, both temporary clipping and induced hypotension were used in our patients (Table 4).

In 20 of our 85 patients (23.5%) with intraoperative rupture, temporary clipping of the aneurysm-bearing vessel became necessary. In 17 patients exact data concerning the time of clip application were collected from the operative reports and anesthesiological charts. This interval ranged from 2 to 12 min, with a mean closure time of 3.2 min. Fourteen patients, or 70%, reached a satisfactory postoperative outcome, whereas five patients had aneurysms of the anterior communicating artery.

Assessment of the impact of induced hypotension, which was performed in 62 of our patients (72.9%), on the subsequent outcome is more difficult. The mean time of hypotension was 78 min. The mean systemic blood pressure (SBP) induced was 86 mmHg, with a range from 50-130 mmHg. No absolute threshold of the SBP below which the danger of ischemic sequelae was apparent could be defined. However, a dynamic relationship between the resting blood pressure of the patient prior to surgery and the percentage of the blood pressure decrease during induced hypotension was clearly demonstrable. Whereas a decrease of the SBP of no more than 40% was well tolerated in most of the patients, a decrease of more than 40% from resting parameters was accompanied by significant ischemic sequelae and consequent worsening of the outcome.

Discussion

Intraoperative aneurysm rupture is not a rare event during microsurgery for intracranial aneurysms. According to a literature survey premature rupture occurs in between 19% and 65%, with a mean percentage of 37%. This incidence of intraoperative rupture is slightly lower than that reported from our data. Despite the familiarity and

routine use of microsurgical techniques, the occurrence of premature aneurysm during surgery definitely worsens the postoperative results, as could be demonstrated from our study. Literature reports of mortality due to premature aneurysm rupture during surgery range from 3% to as much as 70% [2,9,10,14].

In regard to the location of the aneurysm and the incidence of premature rupture, the high rate of intraoperative bleeding in anterior communicating aneurysms reflects the difficult topographical situation of these aneurysms as well as the surgical difficulties they cause, which, according to the experience of many authors, are especially pronounced under the circumstances of early surgery [3,8,11-13].

Considering the moment of aneurysm rupture during the surgical procedure, it is obvious that intraoperative rupture during the craniotomy period almost invariably leads to a fatal outcome, whereas rupture during the dissection or clipping period can usually be managed satisfactorily by the experienced neurosurgeon, with good postoperative survival in many of the patients.

The routine use of induced hypotension has come under some criticism in recent years. Some authors have denied the right to render the whole brain oligemic or even ischemic in order to reduce blood flow in the one vessel bearing the aneurysm. In contrast temporary clipping has been increasingly advocated, and some authors have reported long clipping times without or with only minimal neurological deficits [1,6]. These reports are substantiated by our data, in which temporary clipping is shown to have a greater margin of safety than induced hypotension, with consequent better results. Should the absolute necessity of using hypotension arise, a decrease in the SBP of more than 40% from the resting blood pressure of the patient has to be avoided.

Whether the application of so-called cerebroprotective substances can make hypotension or temporary clipping safer by prolonging the time of ischemic tolerance needs to be further elucidated.

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Temporary Vessel Occlusion by Microvascular Clips

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Introduction

Microvascular procedures for cerebral aneurysms or revascularization of small arteries require the application of microvascular clips. In 1911, CUSHING [1] became the first to describe the application of vascular clips for the occlusion of bleeding cerebral vessels. Increasingly authors are recommending the application of some temporary clips before preparation and occlusion of an aneurysm without regard to possible vessel wall lesions being caused by the clips themselves. To ensure the successful performance of the finest surgical techniques these clips are expected to bring about the temporary occlusion of a cerebral vessel combined with minimal vessel wall lesion. We therefore set out to study the effects of different microvascular clips with a diameter of 1-2 mm on the vessel wall and measured blood flow in the temporarily clipped arteries.

Method

Forty carotid arteries of male Wistar rats were divided into four groups (A-D) and occluded for 60 min by different microvascular clips. These segments of the artery were fixed by intracardial perfusion of the animals with glutaraldehyde and examined by light (LM) and scanning electron microscopy (SEM).

Group A was a control group without temporary occlusion of the carotid artery.
In **group B** the carotid artery was occluded by an alpha-type clip (Biemer FD 560) with an occlusion force of 35 g.
In **group C** carotid artery occlusion was done by a pivot-type clip (Heifetz) with an occlusion force of 115 g and 5 x 1.5 mm length.
Group D was occluded by an alpha-type clip (modif. Mehdorn) with an occlusion force of 40 g and a length of 4 x 1 mm.

Additionally these clips and a temporary Sugita clip with an occlusion force of 65 g were used to clip the abdominal aorta of rats for 30 min. Changes of blood flow volume in this artery were determined by a Doppler vessel clamp with an accuracy of ±12.8% for 30 min after reopening the clip [6].

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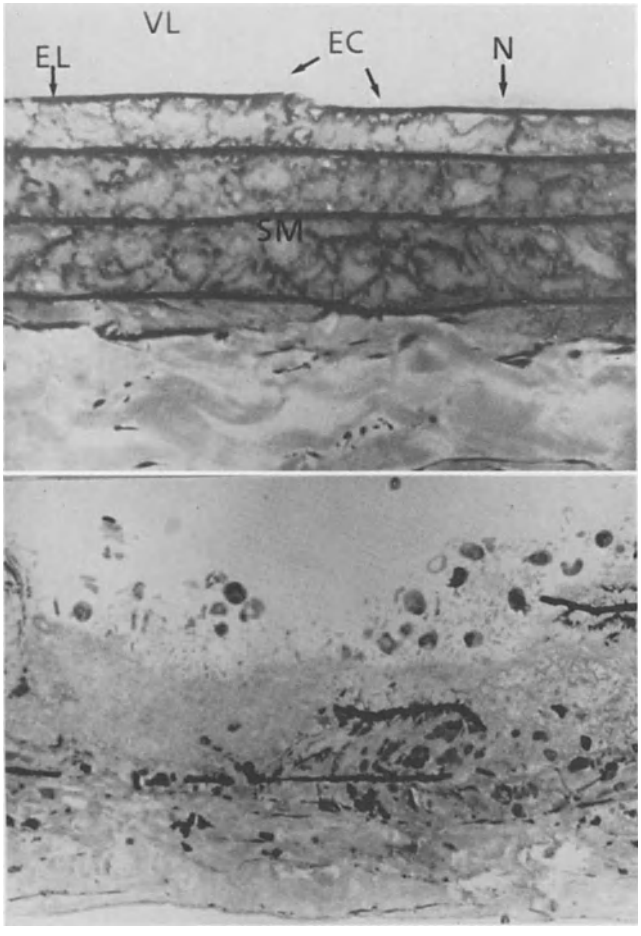

Fig. 1. Light micrograph (above) of the normal common carotid artery (group A) shows a smooth endothelial cell lining (EC), endothelial nuclei (N), internal elastic lamina (EL), medial smooth muscle cell (SM), and vessel lumen (VL). X 1000. The arterial segment below was compressed by a clip and reveals crater formation with destruction of endothelium, internal elastic membrane, and medial layers of smooth muscle cell up to connective tissue of the adventitia

Results

In control group A SEM examination of the vessel wall demonstrated a regular tissue structure with protrusions of nuclear endothelial cells with digs and folds in the longitudinal direction of the vessel. LM showed a tissue wall with a lamina of spindle-like endothelial cells near the lumen, with protruding nuclei lying close to the membrana elastica, followed by circular layers of smooth muscle cells (Fig. 1).

As tissue lesions did not differ qualitatively between the groups in the region of the applied clips, the results for groups B-D may be summarized as follows:

SEM showed a flattened endothelial layer with loss of physiological nuclear protrusion. Lesion of the endothelial lamina, followed by denuding of subendothelial connective tissue, was accompanied by leukocyte adhesions and platelet aggregation (Fig. 2).

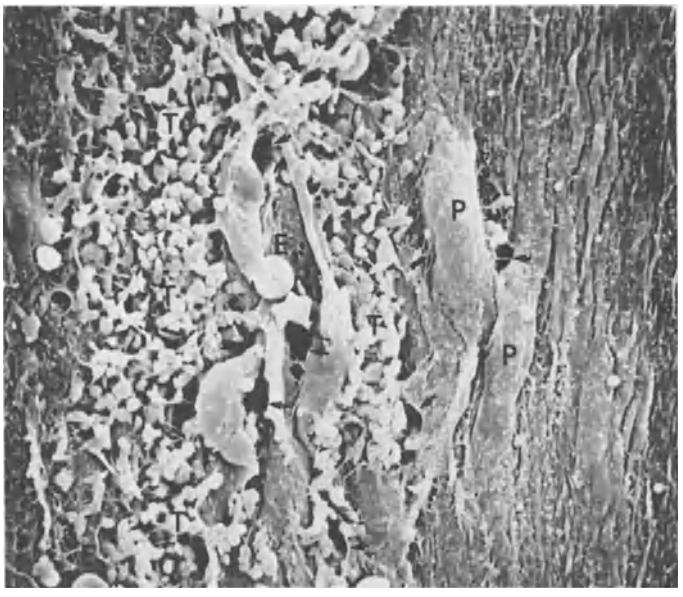

Fig. 2. Scanning electron micrograph of an arterial segment compressed by a Biemer clip (group B). Platelets (P) are adherent to the endothelium. Slight crater formation (arrows) seen (E). X 1200

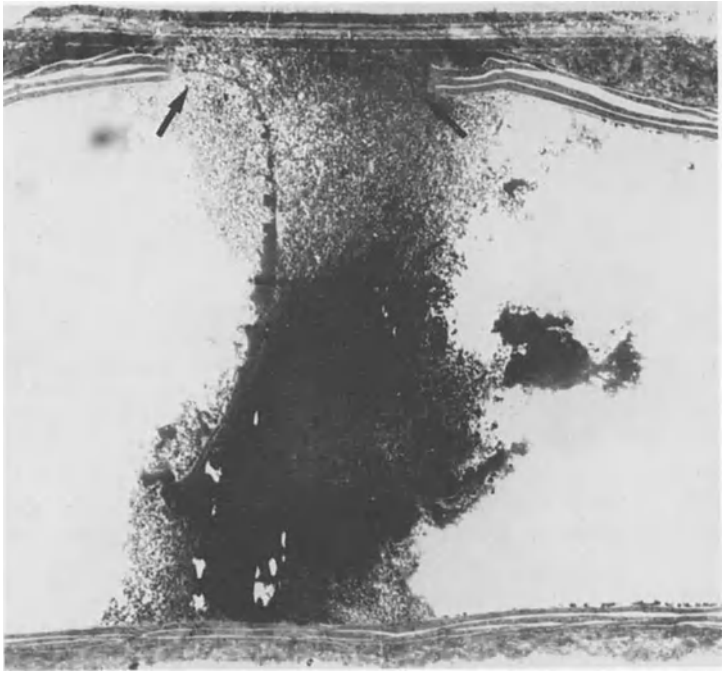

Fig. 3. Light micrograph of an arterial segment compressed by a Heifetz clip (group C). The vessel lumen is occluded by a thrombus. Parts of the vascular wall are destroyed (arrows). X 16

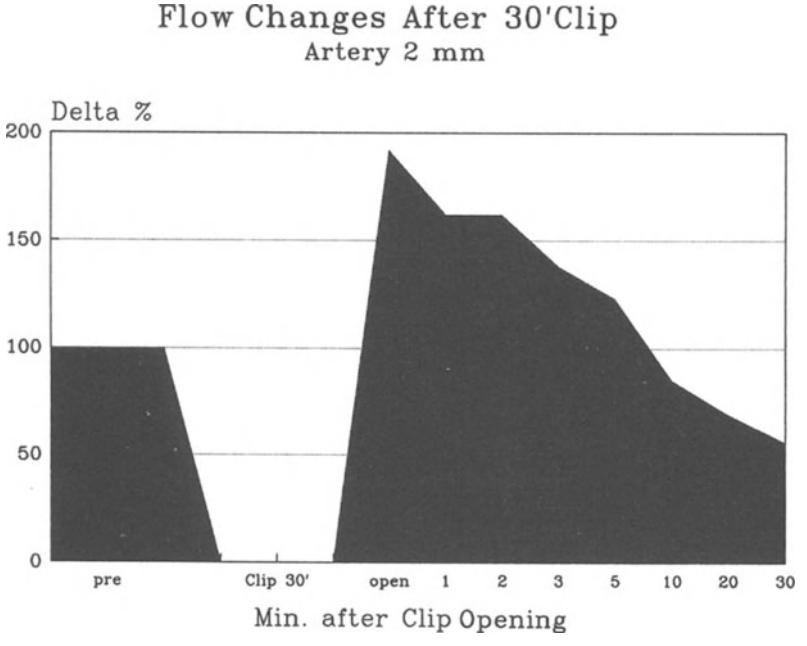

Fig. 4. Mean changes of blood flow in arteries with a diameter of 2 mm after closure with microvascular clips for 30 min

LM showed diapedesis of endothelial lamina by neutrophil granulocytes. The cytoplasm of smooth muscle cells was vacuolated, and the nuclei were pyknotic in some cases. In severe lesions there was disruption of the endothelial lamina, with crater formation and lesion of the media cells (Fig. 1). In one case of group B these vessels wall lesions led to a secondary thrombotic occlusion of the examined vessel (Fig. 3).

The quantitative examination showed segments of arteries closed by alpha-type clips with an occlusion force of 35 or 40 g to display lesions of the endothelial surface, slight alterations of the media, some pyknotic nuclei, or vacuolated cytoplasm. The pivot-type clip, with an occlusion force of 115 g, caused destruction of the intima and media cells, and severe crater formation with thrombosis of the vessel.

A follow-up examination of blood flow in the temporarily occluded artery showed a characteristic behavior (Fig. 4): Immediately after opening the clip, blood flow showed values far above steady state. Values then decreased steadily until in the 10th min they sank below the steady state value and remained virtually unchanged at a low level from the 30th min. These changes were not significantly different between the groups. The mean value in relation to steady state showed a 92% increase after clip opening; after 10 min it was 15% below the steady state value and after 30 min, 46% below.

Discussion and Conclusion

Our studies provide evidence that even microvascular clips available today, with minimal occlusion force, cause vessel wall lesions when used temporarily for periods between 30 and 60 min.

FEIN [4] was able to reveal stenoses angiographically at the sites where Biemer clips were applied in five patients who underwent cerebral revascularization operations. Because this clip produces 35 g of force and the serrated blades increase the focal distribution of the stress load, the Biemer clip may damage the vessel in a similar manner to that demonstrated in our investigations.

Furthermore, nearly all clips utilized have a lever system so that Archimedes' law applies: the force exerted by the lever is inversely proportional to the distance from the fulcrum. Therefore the force exerted by the blade near the fulcrum is greater than the force exerted at the tips. These types of clip produce the least force in the closed resting position. So a thick-walled vessel that prohibits a clip from closing fully receives more force than does a thin-walled vessel that can be compressed to a greater extent.

DUJOVNY et al. [3] showed in a computer simulation model that the calculated shear forces were very high at the points where the vessel folds over itself. This indicates that the greatest amount of stress is on the inner layer of a vessel closest to the fulcrum and that this region is most susceptible to "corner mirror lesion."

In accordance with SZILAGYI et al. [5], we found in our blood flow measurements a shortlasting hyperperfusion in the first 5 min after opening the clip, followed by a drastically reduced flow in the early postinjury period. Dodson et al. contended that the autoregulatory function of the vessel might itself be altered by such a clip [2].

These changes cannot be marked off from peripheral tissue alterations produced by transient ischemia. Irrespective of this question, the reduced flow may increase the risk of thrombotic vessel occlusion, so that additional rheological measurements should be discussed.

We were bound to conclude that no clip currently available allows small arteries to escape temporary occlusion unscathed. This endothelial trauma, which could readily compromise even the finest surgical techniques, can be reduced by using clips with minimal occlusive forces, and with smooth, long, and broad blades, which should be gently applied [7] as far from the fulcrum as possible.

An electronic microclip would be desirable that can alter its force and blade configuration as its sensors control the vessel wall.

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Aneurysmal Location and Operative Timing

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The timing of aneurysm surgery is often thought to be beyond discussion, but a retrospective study of 415 aneurysms operated on between 1970 and 1986 in Hannover revealed results which made it clear that the timing of surgery will remain a major topic of controversy. Management involving early operation [7] was not endorsed in all instances, and this result led to a reassessment of the problems of timing aneurysm surgery [2].

In the Federal Republic of Germany there are about 7000 subarachnoid hemorrhages (based on US data), or 11-13 per 100 000 inhabitants, per year [3,4,8]. Of these patients, 2500 will die immediately from the hemorrhage without possible medical intervention. A further 2000 will die because of rebleedings, pre- and/or postoperative cerebral vasospasm, and operative and medical problems. Only 2500 patients become functional survivors with no or minimal deficits. These statistics were so impressive that the approach of delaying surgery was changed to operating on all ruptured aneurysms within 72 h if possible. The rationale behind this change was as follows:

1. Clipping of the ruptured aneurysm will definitely prevent recurrent hemorrhage.
2. Evacuation of subarachnoid blood clots during early surgery may reduce the amount of spasmogenic substances which are thought to be released during posthemorrhagic clot lysis, consequently reducing the occurrence and the sequelae of vasospasm [1,3,5,6,9,10].

This study was designed to answer the following questions:

1. Did the operation in the acute posthemorrhagic stage (within 72 h) improve the results?
2. Are there differences in the results according to the location of the aneurysms?

Patients, Methods, and Results

Between January 1979 and December 1986 526 patients were admitted for subarachnoid hemorrhage (SAH), as confirmed by CT and/or lumbar puncture. In only 415 patients was an aneurysm found to be the cause of SAH.

All the patients underwent surgery using standard microsurgical techniques with clipping of the aneurysm. No wrapping or coating tech-

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Table 1. Age and sex distribution of the patients (n = 415)

Age	Male	Female
10-19	7	5
20-29	22	22
30-39	32	54
40-49	51	52
50-59	35	57
60-60	17	50
70-79	0	11
	164	251

niques were used. Surgery was performed under neuroleptic anesthesia. Intraoperative hypotension was not used regularly but was occasionally found necessary during the final dissection of the aneurysm.

The age and sex distribution of the 251 female and 164 male patients is presented in Table 1. Of the 415 patients, 181 (45.2%) were operated on within 72 h and 234 (54.8%) were operated on after the 3rd day. Postoperative angiographic control was performed 3-8 weeks after surgery. All the surviving patients were reexamined after a minimum interval of 6 months following surgery according to the guidelines of the Cooperative Aneurysm Study using the criteria of the Glasgow Outcome Scale.

In both the early and the delayed surgery groups the anterior cerebral artery (ACA) represented the most common location of the aneurysm (35.5% and 32.5% of cases, respectively), followed by the internal carotid artery (ICA) (33.1% and 27.4%) and the middle cerebral artery (MCA) (22.7% and 23.1%) (Tables 2, 3). Only two (1.1%) aneurysms of the posterior circulation were operated on within 72 h, whereas these aneurysms accounted for 7.7% of the delayed group. Fourteen pa-

Table 2. Location of aneurysm and outcome (Glasgow Outcome Scale) in patients in whom surgery was delayed

	All groups		I + II		III		IV + V	
Aneurysmal location	n	%	n	%	n	%	n	%
A. carotis interna	64	27.4	47	73.4	2	3.1	15	23.7
A. cerebri media	54	23.1	40	74.0	3	5.6	11	20.4
A. cerebri anterior	76	32.5	59	77.6	3	3.9	14	18.4
A. vertebr. basilaris	18	7.7	13	72.2	0		5	27.8
Multiple aneurysms	22	9.4	17	77.3	1	4.5	4	18.2
	234	100.0	176	75.2	9	3.8	49	20.9

Table 3. Location of aneurysm and outcome (Glasgow Outcome Scale) in patients undergoing early surgery

	All groups		I + II		III		IV + V	
Aneurysmal location	n	%	n	%	n	%	n	%
A. carotis interna	60	33.1	44	73.3	9	15.0	7	11.7
A. cerebri media	41	22.7	29	70.7	4	9.8	8	19.5
A. cerebri anterior	64	35.5	40	62.5	7	10.9	17	26.6
A. vertebr. basilaris	2	1.1	1	50.0	1	50.0	0	
Multiple aneurysms	14	7.7	7	50.0	0		7	50.0
	181	100.0	121	66.9	21	11.6	39	21.5

tients (7.7%) with multiple aneurysms were operated on early, while in 22 patients (9.4%) surgery was delayed.

The results showed that early surgery reduced rebleedings to less than 50%, from 23.2% in the delayed group to 11% in the early group (Tables 4, 5).

Internal carotid artery aneurysms rebled in 27% of the delayed cases; the corresponding figures for MCA aneurysms and ACA aneurysms were 24.1% and 19.7% respectively. Regarding the early operations, ICA aneurysms rebled in 13.3% of cases, MCA aneurysms in 14.6%, and ACA aneurysms in only 6.3%. The results regarding the location of the aneurysms demonstrated a comparably good outcome of ICA and MCA aneurysms (73.3% and 70.7%) in the early group and in the delayed group (73.4% and 74.0%). There was a striking difference in good outcome of the ACA aneurysms between the early group (62.5%) and the delayed group (77.6%).

To complete the results it has to be pointed out that even grade IV and V patients (according to Hunt and Hess) were operated on, especially if there was an additional space-occupying intracerebral hematoma. The results demonstrated an insignificantly better progno-

Table 4. Recurrent hemorrhage (delayed operation)

Aneurysmal location	n	Recurrent hemorrhage		Frequency of recurrent hemorrhage regarding aneurysmal location
A. carotis interna	64	17	7.3%	26.6%
A. cerebri media	54	13	5.6%	24.1%
A. cerebri anterior	76	15	6.4%	19.7%
A. vertebr. basilaris	18	3	1.3%	16.7%
Multiple aneurysms	22	6	2.6%	27.3%
	234	54	23.2%	

Table 5. Recurrent hemorrhage (early operation)

Aneurysmal location	n	Recurrent hemorrhage		Frequency of recurrent hemorrhage regarding aneurysmal location
A. carotis interna	60	8	4.4%	13.3%
A. cerebri media	41	6	3.3%	14.6%
A. cerebri anterior	64	4	2.2%	6.3%
A. vertebr. basilaris	2	0		
Multiple aneurysms	14	2	1.1%	14.3%
	181	20	11.0%	

sis in the early group than in the delayed group. This supports the performance of surgery even in these poor-risk patients.

This study confirms that the preoperative neurological status of patients suffering from SAH to a large extent determines the postoperative outcome (cf. Tables 6, 7).

Discussion

Analyzing our data and in answer to our introductory questions, we were able to demonstrate that the rebleeding rate was reduced by early operation to less than 50% of that following delayed operation. To demonstrate a reduction of the occurrence of vasospastic complications is much more difficult, but based on occasionally repeated angiograms, clinical diagnosis, and CT criteria, it can be asserted that the occurrence of cerebral vasospasm in about 30% of the late surgery group was reduced to about 15% in the early surgery group.

Returning to our questions we have to conclude:

1. That a definitive answer concerning the superiority of early or delayed surgery for the treatment of ruptured intracranial aneurysms cannot be given. Early surgery was not as convincing as we had initially thought, whereas delayed surgery led to better results than expected.

Table 6. Preoperative neurological status and outcome (Glasgow Outcome Scale) in patients in whom surgery was delayed

	All groups		I + II		III		IV + V	
Hunt and Hess grade	n	%	n	%	n	%	n	%
I + II + III	195	83.3	157	89.7	7	3.6	31	15.9
IV + V	39	16.7	19	48.7	2	5.1	18	46.2
	234	100.0	176	75.2	9	3.8	49	20.9

Table 7. Preoperative neurological status and outcome (Glasgow Outcome Scale) in patients undergoing early surgery

	All groups		I + II		III		IV + V	
Hunt and Hess grade	n	%	n	%	n	%	n	%
I + II + III	143	79.0	105	73.4	13	9.1	25	17.5
IV + V	38	21.0	16	42.1	8	21.1	14	36.8
	181	100.0	121	66.9	21	11.6	39	21.5

2. The aforementioned conclusion is based on consideration of all intracranial aneurysms, but if only the results regarding ICA and MCA (i.e., not ACA) aneurysms are examined, the early operated patients are seen to fare distinctly better.

As regards surgical management, this means: Ruptured aneurysms should undergo early surgery if the location of the aneurysm is at the ICA and MCA. In cases of ACA aneurysms only grade I and II patients should be operated on early, and then by the most experienced surgeon. These aneurysms tend to rebleed to only a minor degree and their outcome was best after delayed surgery in the present series [11,12].

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Ventral Transvertebral Intradural Approach in Cervical and Thoracic Lesions

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Intradurally and ventrally situated extramedullary or intramedullary lesions can be operated on from a dorsal, dorsolateral, ventrolateral, or ventral approach. Disadvantages of the dorsal approach consist in the danger of damaging the spinal cord. The direct way to the ventral spinal cord is the ventral approach [2,3,7,14,15].

Because of the need for thoracotomy in the lower region and sternotomy in the upper region of the thoracic spine, in combination with vertebrectomy, the ventral approach is doubtless the more elaborate and should therefore be restricted to specific indications.

We report on three cases of ventrally and intradurally situated lesions which we removed via the familiar ventral approach in combination with vertebrectomy.

Case Reports

Case No. 1

This 51-year-old man with neurofibromatosis had suffered for 1 year from increasing tetraparesis accompanied by multiple paralyses, especially in the region of the diaphragm, the recurrent nerve, and the shoulder girdle musculature on the left side. A large tumor was palpable in the lateral neck triangle on the left.

Neuroradiological examinations: The plain X-rays of the cervical spine showed a pronounced malposition with development of kyphosis at the level of C3. MRI demonstrated six extraspinally situated tumors and a further intraspinal premedullary tumor at the level of C3-C5 (Fig. 1).

Operation: On 1 October 1985, the tumors were operated on from a neck incision on the left side. The intradural intraspinal portion was imaged by partial vertebrectomy of C3, C4, and C5 with opening of the dura and removed completely. Altogether, two intradurally localized tumors and eight extraspinally situated tumors were found.

Postoperative course: Postoperatively, the tetraparesis and paralyses in the region of the shoulder girdle and the left arm improved to such an extent that the patient could once more fully practice his profession as a bookkeeper. Investigation of the sensory evoked potentials of the tibial nerve and the median nerve revealed a marked

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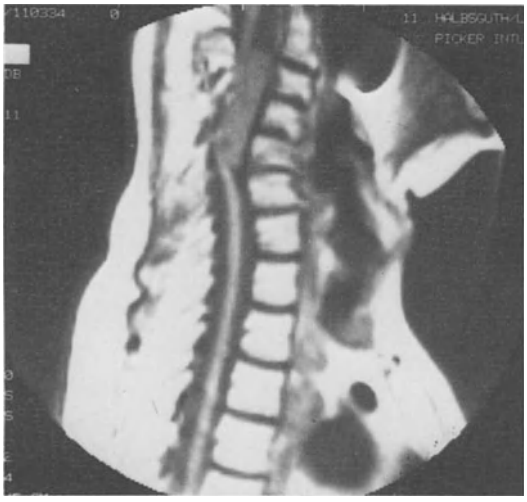

Fig. 1. MRI: intradurally located tumor at the level of C3-C5. Pronounced malposition of the cervical spine

improvement in the cortical stimulus response after 7 weeks and 17 months. The histological investigation revealed neurinomas.

Case No. 2

This 71-year-old woman was operated on elsewhere in 1981 for an intradural spinal meningioma at the level of C6-T2 by laminectomy. The readmission took place because of increasing paraparesis of the legs.

Neuroradiological examinations: MRI showed a ventrally situated tumor recurrence at the level of C7-T2 (Fig. 2). Owing to the planned approach, aortography was performed; it did not reveal any anomalies.

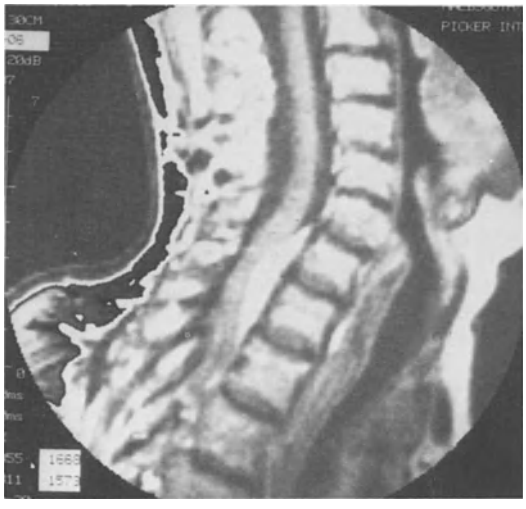

Fig. 2. MRI: meningioma situated ventral to the spinal cord at the level of C7 and extending to T2

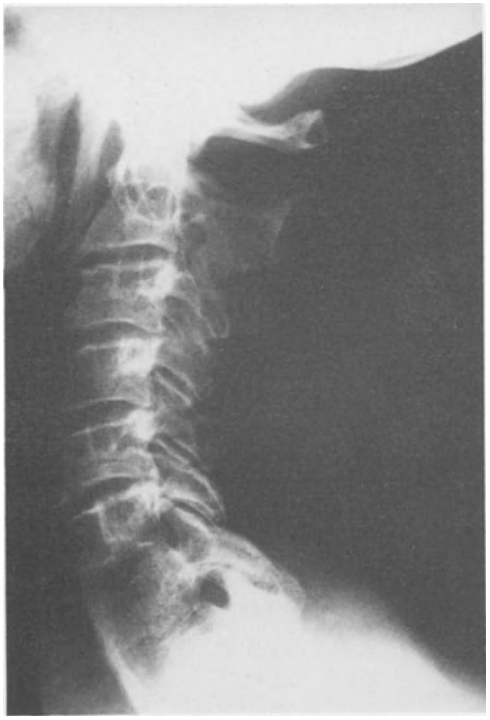

Fig. 3. Plain X-ray of the lateral cervical spine (same patient as in Fig. 2): postoperative X-ray after vertebrectomy and stabilization with a bone graft from the iliac crest

Operation: Because of the prior dorsal operation and the ventrally localized tumor recurrence, we decided to carry out cervical sternotomy, which was performed on 6 July 1987. Here, the dura was imaged from ventral and opened with resection of the 7th cervical and the 1st thoracic vertebral bodies. The tumor, which originated from the ventral dura, was resected. The tumor was dissected out from spinal cord and removed completely. Afterwards, a dura patch was inserted and stabilization was performed with an autologous bone graft from the right iliac crest (Fig. 3).

Postoperative course: Postoperatively, the paraparesis improved well, so that the patient could again look after her household on her own. The tibial somatosensory evoked potentials showed this improvement by shortening of the latencies and increase of the amplitudes.

Case No. 3

This 24-year-old woman suffered several spinal hemorrhages which led to the diagnosis of spinal arteriovenous malformation at the level of T9 and T10. The malformation was partially resected from dorsal by laminectomy in 1979 elsewhere. In the subsequent period, there was renewed hemorrhage with increasing paraparesis of the legs.

Neuroradiological examinations: MRI and angiography showed a right ventral intramedullary location of the malformation (Fig. 4). The feeding arteries came above all from the left.

Operation: Owing to the presence of laminectomy and the ventral position of the lesion, we decided to make an anterolateral transpleural

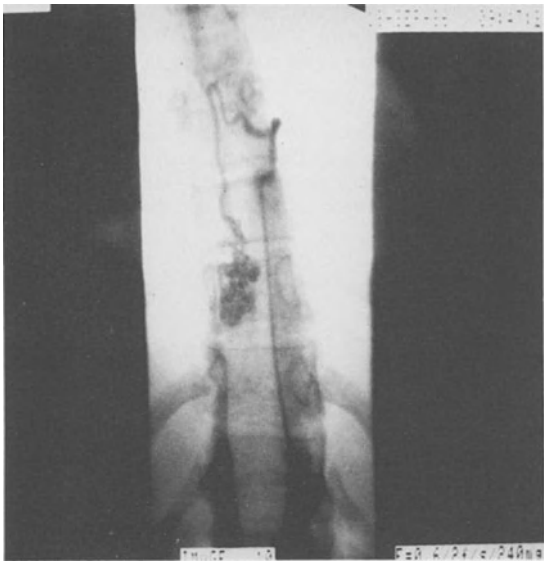

Fig. 4. Spinal angiography: representation of the arteriovenous malformation with blood supply via the radicular artery from the left (Prof. Dr. H. Hacker, Department of Neuroradiology, Johann Wolfgang Goethe-Universität, Frankfurt a.M.)

approach from the right, with resection of the thoracic vertebral bodies T9 and T10; this was done on 15 October 1986. After opening the dura, the individual feeding arteries could be isolated and coagulated, and the malformation removed. After closing the dura with a patch, stabilization was carried out with an autologous bone graft from the iliac crest.

Postoperative course: Neurologically, the paraparesis did not improve appreciably.

Discussion

According to BREIG (1979), a lesion situated ventrally to the spinal cord in the region of the vertebral column leads, in pathophysiological terms, to a traumatization of the spinal cord and to transmitted alterations [1]. The traumatization of the spinal cord arises above all in flexion movement by stretching of the nervous structures, and can lead gradually or even suddenly to appreciable neurological deficits.

For surgical treatment in particular of degenerative diseases, of tuberculous lesions, and of tumors in the region of the spine, a large variety of ventral approaches have been developed and modified. In the region of the cervical spine, the ventral approach has been developed above all in degenerative diseases [3,10,11,15]. In the thoracic region, the approach to the upper thoracic spine was first described as cervical sternotomy in one of the rare vertebral disk prolapses by CAUCHOIX, BINET and EVRARD in 1957 [2]. The ventral approaches to the lower thoracic spine were employed primarily for treatment of injuries and in spinal tuberculosis and abnormalities [4-9,12-14]. We have used these familiar approaches in order to operate on intradural lesions. For this purpose, partial or complete vertebrectomy is necessary. Afterwards, stabilization is indispensable. We prefer autologous bone grafts from the iliac crest.

The advantages of the ventral approach in the lower region of the thoracic spine are that the thoracotomy can be extended to the ventral or lateral part of the spine and the surgeon obtains a sufficiently wide angle of vision.

On the basis of our experience, we can observe that the familiar ventral approaches to the spine can be extended to intradural without difficulties. In lesions with a ventral extramedullary or intramedullary location, only this ventral transvertebral intradural approach can be successful in extirpation.

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Lateral Approach for Resection of Anterior Craniospinal Tumors

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Introduction

The dorsolateral approach for surgical resection of ventrally located craniospinal tumors [14] was elaborated to a lateral approach in the further course in relation to the upper spinal canal and the ventral surface of the brain stem of the medullary plate. In the following, the operative technique as well as the results of surgery are described in 22 patients with extradural and intradural craniospinal and spinocranial tumors with an exclusively ventral location. Patients with a craniospinal or spinocranial tumor with a lateral or dorsal location have not been included in this study.

Surgical Technique

In the sitting position, an angular incision is extended far to lateral to about 4 cm above the mastoid process and the midline up to about C6 to C7 on the right or the left, in accordance with the predominant extension of the tumor, and the head is turned to the side of the predominant extension of the tumor (Fig. 1). The skin flap dissected angularly from the musculature corresponding to the cutaneous incision is fixed far to lateral in order to avoid obstructions to vision. The neck musculature is severed transversely at the level of the nuchal plane with a remnant at the occiput remaining for the lateral suture. It is dissected free of spinous processes and transverse arches from C1 to C3 on the side of the angular incision in the midline, mobilized, and also fixed far to lateral to avoid obstruction to vision. The dorsal atlas arch up to the lateral mass or up to the vertebral arterial sulcus and the dorsal arch of the second vertebral body up to the site of axis insertion are exposed and freed of soft tissue. In the dissection of the vertebral artery and the root C2 which now follows, the numerous thick-caliber veins must be carefully dissected free around the artery and the root, coagulated, and covered at an early stage with cotton wool pads to avoid air embolism. In anterior spinocranial tumors, resection of the lateral part of the atlas arch up to the lateral mass is not necessary as a rule. Lateral partial or total resection of the C2 dorsal arch extending up to the site of insertion of the axis body is sufficient. In tumors within and above the foramen magnum, on the other hand, the lateral part of the atlas arch and the bone ridges projecting from the lateral mass and below the sulcus of the vertebral artery or the vertebral artery up to the foramen of the transverse process of the atlas are removed and the vertebral artery is mobilized in the entire horizontal section (Fig. 1). In this way, a displacement of the vertebral artery is possible to cranioventral. This position of the ver-

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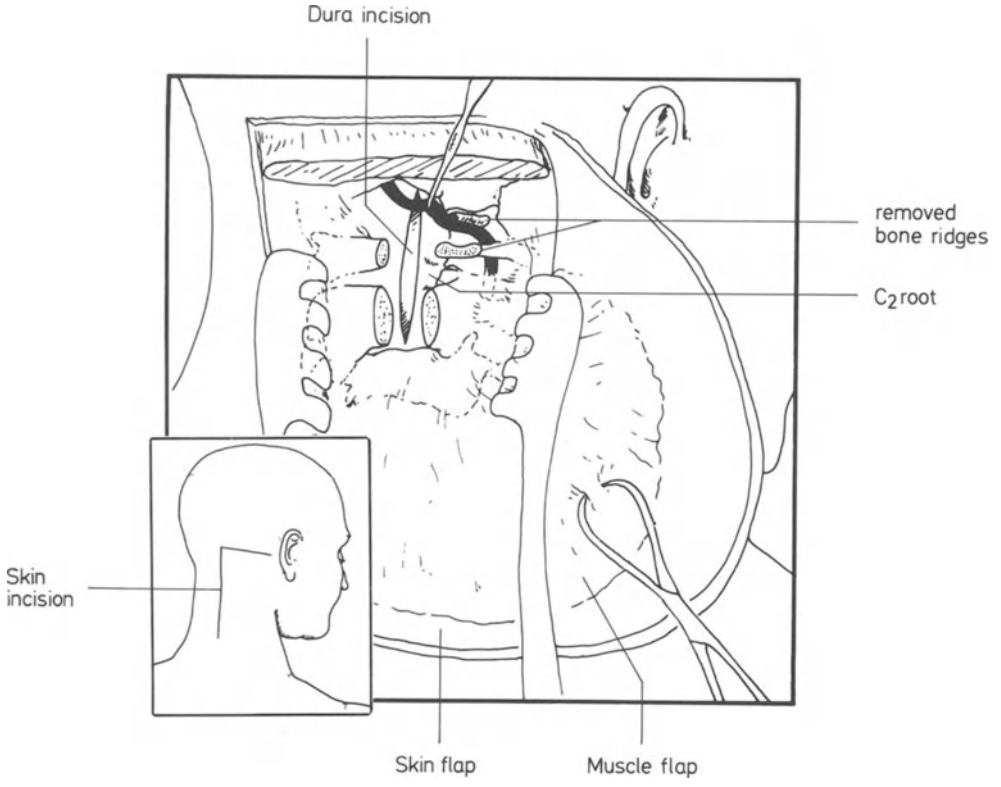

Fig. 1. Position of the head and neck in accordance with predominant extension of a tumor to the right side. Approach to the craniocervical junction with exposure of the vertebral artery on the right side

tebral artery can be retained during the entire operation by the assistant with a bent dissector or with a self-retractor. By mobilization of the artery, the approach is facilitated above the C2 root and ventral to the point of entry of the vertebral artery into the intradural space. In extradural tumors, it is entirely feasible to remove a tumor via an approach between the vertebral artery and the C2 root or below the C2 root. In intradural tumors, the dura is opened behind the C2 root and extended longitudinally ventral to the site of entry of the vertebral artery in the cranial and caudal direction. Since the vertebral artery initially runs upwards and then to the midline after its entry into the dura, there is no danger of its being injured. The large opening in the dura created in the longitudinal direction is enlarged by traction and fixation of dura holding sutures over a wide area to anterior and posterior. For a better intradural access in tumors extending to cranial, the root C2 is displaced to caudal after opening the arachnoidea; the denticulate ligament and the uppermost denticulate tip are severed cranial and dorsal to the vertebral artery, and the intraspinal fibers of the accessory nerve are also mobilized and displaced to dorsal. In this operation as well as in the further course, damage to the laterally situated ganglion on the spinal part of the cranial nerve XI should be avoided as far as possible. The exposure of anterior tumors in the lower clivus region as well as in the greater foramen is made possible by this technique. The medullary plate is now situated dor-

sally in the field of vision of the surgeon, and thus outside the danger zone. Intradural ventrally located craniospinal meningiomas emanating from the clivus are as a rule demonstrated in aged patients. In order to avoid any mechanical damage to the vulnerable medulla, the tumor should be approached ventrally (if possible with a Cusa instrument), primarily at the site of attachment, and reduced in size. Only afterwards is it possible to luxate the dorsal part stepwise to ventral and to remove it without exerting pressure on the medullary plate. In intradural tumors, dural suture is carried out continuously. The musculature, fascia, and skin are closed layer by layer.

Case Report

A 76-year-old woman patient with increasing tetraparesis finally became unable to walk and bedridden about 2 months before admission to hospital. On admission to the hospital, the legs could be moved to a minimal extent, but could not be raised from the bed. The left arm could just be raised from the bed, but only minimal finger movements were possible on the right side. CT and nuclear magnetic resonance tomography indicated an anterior craniospinal meningioma (Fig. 2) which was more extensive on the right side. A small space not occupied by the tumor was still present only on the left side. Two days before the operation, the paresis on the right side intensified to paralysis. Central respiratory disorders occurred from time to time. The operation took place using the previously described technique in a sitting position by an angular incision on the right side with turning of the head to the right side. After exposure of the lateral parts of the C1 and C2 arches, the projecting bony parts of the lateral mass above and below the vertebral artery as well as the most lateral part of the dorsal atlas arch were resected. The vertebral artery was mobilized over its entire horizontal distance up to the point of entry into the dura. The dura was opened directly behind the C2 root and the opening extended longitudinally in the cranial direction. The opening of the dura was also extended cranially as far as possible after displacement of the vertebral artery in the craniventral direction. After opening of the arachnoidea and severance of the denticulate ligament, the meningioma was shown up well (Fig. 3). The meningioma was initially approached with the Cusa instrument at the site of attachment and reduced in size. Only afterwards were the parts which had extended more dorsally in the direction of the

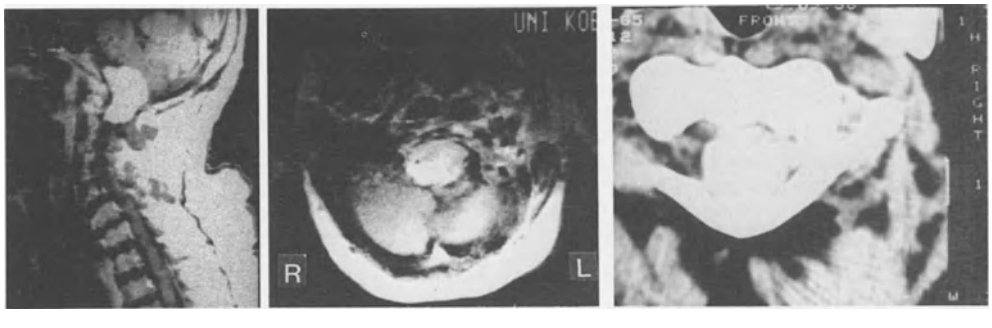

Fig. 2. MRI (left, middle panel), CT (right panel): Huge meningioma of the foramen magnum. A small space not occupied by the tumor is still present only on the left side

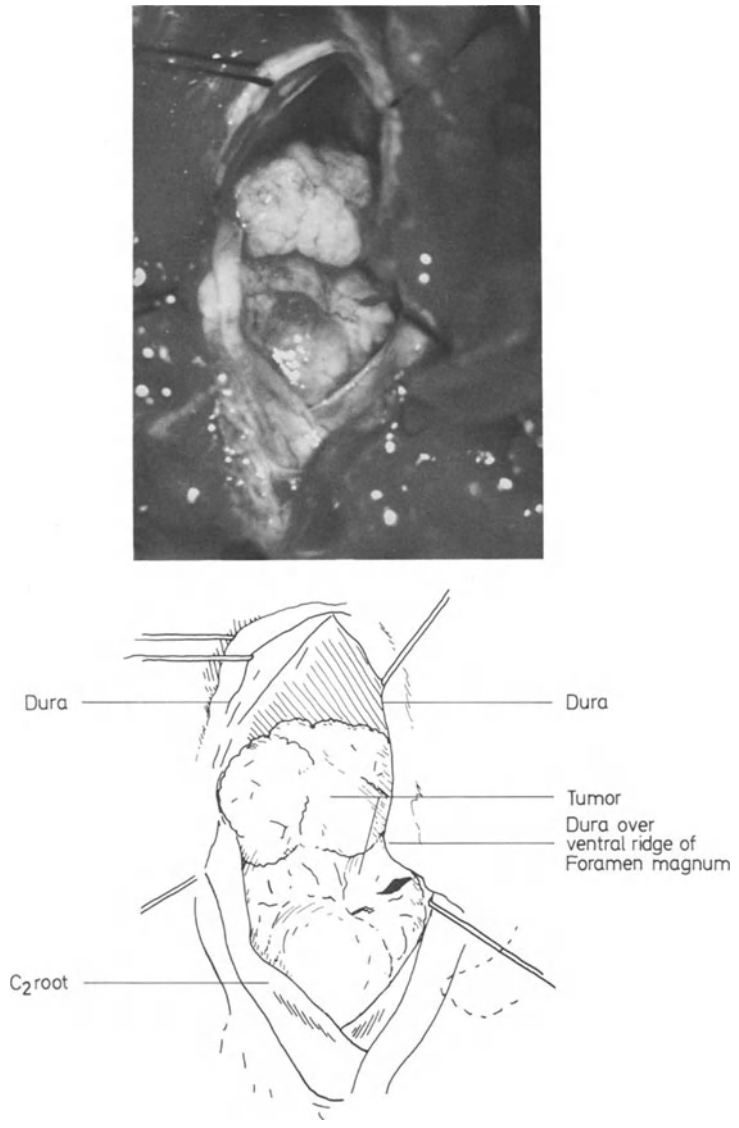

Fig. 3. Same case as Fig. 2. After the opening of the dural and arachnoidea and severance of the denticulate ligament, the meningioma is clearly visible

medullary plate luxated to ventral and finally resected completely. Only when the dorsal dural layer was retracted further to dorsal after removing the tumor could the surgeon see the ventral surface of the medullary plate (Fig. 4).

The patient recovered rapidly after the operation. She was already able to walk 6 weeks after the operation. A dextrolateral arm paresis which was still present also showed a good tendency to regress in the further course. Despite the good clinical improvement of the neurolo-

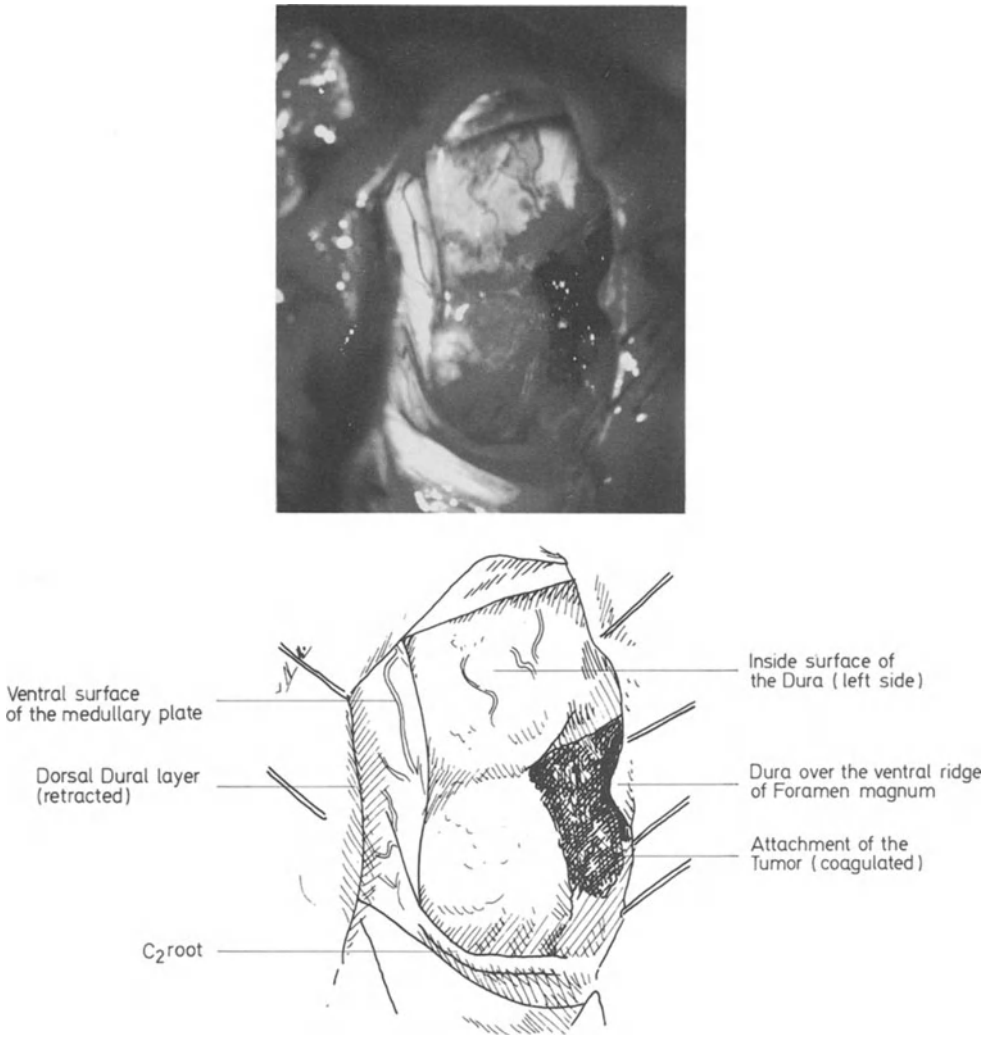

Fig. 4. Same case as Figs. 2 and 3. After removal of the tumor the ventral surface of the medullary plate can be seen if the dorsal dural layer is retracted further dorsally

gical symptoms, the anterior tumor cavity could be discerned in MRI even 6 weeks after operation, as in other cases with anterior craniospinal meningiomas. The medullary plate had not yet unfolded completely (Fig. 5).

Patients and Results of Surgery

A total of 22 patients with anterior craniospinal and anterior spinocranial tumors were operated on with this approach. Table 1 shows the type diagnosis, the craniospinal or the spinocranial extent, and the location (extradural, intradural). The catamneses (6 months after the operation) are listed in Table 2. In all patients, an almost

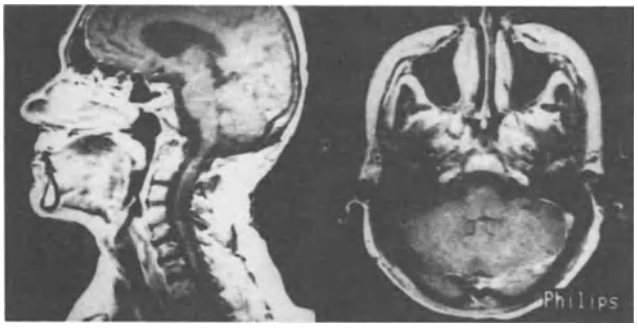

Fig. 5. Same case as Figs. 2-4. MRI 6 weeks after surgery. The medullary plate had not yet unfolded completely

complete regression of the neurological deficits was demonstrated after this period. Only a 35-year-old woman patient with an anterior craniospinal meningioma in which large amounts of air were aspirated repeatedly from the pulmonary artery by the anesthetist during the operation in the sitting position remained unconscious after the operation. She showed a mydriasis on the right side and extensor convulsions on the left side, and died on the third postoperative day. The autopsy showed a massive swelling of the brain which was more pronounced on the right. A massive arterial air embolism of the cranial vessels with corresponding typical alterations in the brain tissue was demonstrated to be the cause. This arterial air embolism was the result of an unknown and clinically irrelevant open oval foramen at the heart. The open oval foramen was not discovered in routine preoperative internist investigations.

Discussion

Since the description, definition, and analysis of craniospinal tumors [2,5,7-9,15,16] the paradoxical symptoms and the difficulties in diagnosing these tumors have been emphasized [6,30, containing further references]. Myelography in the region of the foramen magnum [17,18] and computer tomography have not led to any appreciable

Table 1. Type, location, and extent of 22 tumors operated on via a lateral approach in the craniospinal or spinocranial region

		Extension		
Tumors	No.	Foramen magnum		C1-C2
Intradural				
Meningioma	15	9		5
			1	
Neurinoma	2	1		1
Epidermoid	1		1	
Extradural				
Chordoma	1			1
Hemangiopericytoma	1	1		
Osteoid osteoma	1			1
Plasmacytoma	1			1

Table 2. Neurological findings in 22 cases of anterior craniospinal or anterior spinocranial tumors

Tumors		Neurological signs before surgery		Neurological signs 6 months after surgery			
		R	R+M	R	R+M	None	Death
Meningioma	15	2	13	4	1	9	1
Neurinoma	2		2	2			
Epidermoid	1		RM + increased intracranial pressure			1	
Intradural	18	2	16	6	1	10	1
Chordoma	1		1			1	
Hemangiopericytoma	1	Pain				1	
Osteoid osteoma	1	Pain				1	
Plasmacytoma	1		1			1	
Extradural	4	2	2			4	

R, radicular; M, medullary

improvement in the diagnosis of these tumors. Only MRI has provided a major advance in their diagnosis [21,24], so that they are diagnosed more frequently today. This is illustrated by the fact that more ventrally located craniospinal tumors have been diagnosed and operated on in the last 4 years at the Neurosurgery Division, University of Cologne than in almost 30 previous years. If the literature of the last 10 years is considered with regard to the approach, then reports on a dorsal suboccipital approach predominate [11,12,19,30]. A more precise differentiation of the location (anterior, lateral, posterior) mostly took place intraoperatively in accordance with the diagnostic possibilities available. Like the reports from other authors, the reports from the Mayo Clinic, which concern the largest number of cases (120 benign intradural tumors of the foramen magnum), refer to the difficulties in surgery in the ventral location, which was as a rule the cause of a lethal outcome or inoperability of the tumors [19,30]. Owing to the disappointing results of surgery of ventrally located tumors in the craniospinal region carried out via the dorsal suboccipital approach, the transoral approach recommended for extradural tumors by MULLAN et al. [22] has recently also been applied in occasional cases with intradural lesions [1,3,4,20,28]. Extradural tumors with a cervical extension can also be approached via the transoral route [23]. On the other hand, apart from the necessary resection and retrospective replacement of the axis, intradural tumors with extension into the cervical region are difficult to approach. The main problem is the frequently occurring postoperative CSF fistulae [3,13,20,29]. The transcervical, transclival approach recommended for clivus tumors by STEVENSON et al. [26], which has been used more rarely, is not suitable for intradural tumors. SUGIJA-

MA et al. [27] mentioned a technique with an approach from the side in the lateral position without describing it in detail.

The dorsolateral paracondylar approach recommended by SEEGER [25] for aneurysms of the vertebrobasilar region was also used for craniocervical tumors by GILSBACH et al. [10]. The lateral approach to the upper spinal canal and lower clivus region described above, which has up to now been applied in 22 patients with ventrally located craniospinal and spinocranial tumors, is suitable for the resection of anterior craniospinal and spinocranial tumors and carries a low risk. Apart from slight microsurgical variations for dissection and mobilization of the vertebral artery, this approach is well known to the neurosurgeon with experience of the dorsosuboccipital approach for removing tumors from different locations. Owing to the lateral approach, the medullary plate is outside the field of operation and is thus not in danger of mechanical damage. This approach can be applied both for extradural and for intradural tumors. Tumors with extensions above the caudal half of the clivus or in front of the pons cannot be reached via this approach. The transoral approach also appears to be more suitable in extradural tumors which extend far to cranial (clivus chordoma).

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Brain Death

Diagnosis of Brain Death

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Except for single contributions in 1977 and 1985, so far our society has discussed brain death only at the annual convention in Göttingen in 1968, organized by Karl August BUSHE 20 years ago (BUSHE 1970). Today we consider brain death the irreversible cessation of all integrating brain functions, while the cardiorespiratory functions are maintained with artificial ventilation (FROWEIN 1986; FROWEIN et al. 1987).

Diagnosis of brain death is based on certain prerequisites and the clinical findings of coma, brain stem areflexia, and apnea. As proof of the irreversibility of the loss of brain functions, additional investigations are required: angiography demonstrating cerebral circulatory arrest, EEG showing electrocortical silence, abolition of brain stem auditory evoked potentials, or confirmatory clinical investigation after an adequate waiting period, the length of which depends on the kind of underlying brain lesion and the age of the patient. Specific guidelines for these investigations were developed internationally in a first and second generation of brain death criteria (FROWEIN et al. 1987).

From our contribution in Göttingen and numerous publications, in particular by WALKER [1981, 1985, 1987], it became apparent that the consecutive order of symptoms during the development of brain death may be variable (FROWEIN and POHL 1970).

The terms "cortical," "neocortical," and "brain stem death" will not be considered here. At the Neurological Congress in 1984, organized by GÄNSHIRT, the variable clinical courses were discussed: e.g. electrocortical silence (ECS) preceding apnea by 10 h after a traumatic supratentorial lesion (FROWEIN et al. 1985) and apnea preceding ECS in infratentorial lesions like cerebellar hemorrhage, infratentorial operation, or basilar artery thrombosis (FROWEIN et al. 1985; HACKE et al. 1985; FERBERT et al. 1985).

In 7 out of 97 primary supratentorial brain lesions we found ECS preceding apnea by 1, 3, 7, 8, 12, 13, and 27 h. These courses are not controversial as the diagnosis of brain death is based on the complete clinical findings of coma, brain stem areflexia, and apnea all together.

These observations are confirmed by transcranial Doppler sonography revealing a shuttle flow in the basal vessels of the brain in patients in coma grade IV as a sign of cerebral circulatory arrest with preserved respiration. As early as 1970 a preserved basilar artery flow was documented in similar cases by vertebral angiography;

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such documentation is now easier with Doppler sonography through the foramen magnum.

In acute primary infratentorial lesions, however, apnea may precede ECS or the abolition of the intracerebral parts of the auditory evoked potentials. Therefore, the Bundesärztekammer (1986) concluded that in primary infratentorial lesions the additional documentation of ECS is mandatory for the diagnosis of brain death. In 5 out of 13 patients with primary infratentorial brain lesions leading to brain death, persisting EEG activity was found 5, 7, 8, 14, and 18 h after documentation of apnea. In the literature, such observations have been reported in six patients with infratentorial hemorrhages or tumors and in six patients with basilar artery thrombosis (FROWEIN 1986; FERBERT et al. 1986; JANZEN et al. 1985; HAUPT 1987). It should be noted that in these cases the documentation of EEG was not continuous and the onset of ECS may have been earlier.

Therefore it is our main concern today to gather more experience on the synchronous and asynchronous loss of single brain functions during the development of brain death.

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Differentiated Diagnostic Measurements in Determining Brain Death in Clinical Practice

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Introduction

In recent years the possibilities of transplantation of different organ systems have been enlarged. The term "multiorgan donor" has been created. Because of the vulnerability of the organs to be explanted, quick and reliable determination of the moment of brain death has become necessary.

Three diagnostic procedures (neurological examination, cerebral angiography, and electroencephalography) have been established [1-5]. Today evoked potential measurements are also recommended and the use of transcranial Doppler sonography is progressing.

Patients and Methods

In a retrospective study, 111 patients (52 females and 59 males) were analyzed. They were treated because of different cerebral diseases in the neurosurgical ICU. The average age of the patients was 51.9 years; the youngest was aged 15, the oldest 84. The average duration of stay in the ICU was 9.7 days. The underlying diseases were severe brain injuries in 48 patients, spontaneous intracerebral hemorrhage in 18, subarachnoid hemorrhage in 21, and malignant brain tumors in 24.

The criteria of brain death employed corresponded to the guidelines of the German Bundesärztekammer and of the German EEG society [1,2].

Results

Of the 111 patients only 30 fulfilled the stringent criteria employed to define multiorgan donors (a stay in the ICU up to 2 days and age up to 45) (see Table 1). Explantation was done in ten cases. In all these cases, the determination of brain death was established by a clinical examination followed by angiography. In none of the organ donors was brain death established by electroencephalography.

In 77 patients a diagnosis of brain death was not necessary and the clinical follow-up examination was finished by evaluating the reliable criteria of death. In these cases organ explantation was not possible.

Electrophysiological examinations were done as follow-up studies until the stage of brain death.

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Table 1. Diseases of multiorgan donors of the total group

	SAH	ICH	SBT	Tumor	Total
Age < 45 years ICU < 2 days	5	3	21	1	30
Explantation	4	1	5	0	10

SAH, subarachnoid hemorrhage; ICH, intracerebral hematoma; SBT, skull-brain trauma; ICU, intensive care unit

Discussion

The retrospective analysis of patients who died in the neurosurgical ICU showed that only one-third fulfilled the stringent guidelines for multiorgan donors. Angiography was done mainly in patients with severe skull-brain trauma and multiple organ lesions because of the unstable circulatory situation. In these cases quick and reliable diagnosis of brain death was only possible by means of angiography. Those patients with a longer stay in the ICU had electrophysiological follow-ups and therefore brain death could be established by clinical examination and EEG or evoked potential measurements. These were also done in patients with subarachnoid hemorrhage and subsequent deterioration.

In nearly half of the patients, brain death diagnosis by means of medical apparatus was not necessary because the patients' history contraindicated explantation. Most of these patients had a history of heart or circulatory diseases.

Summary

Retrospective analysis of 111 patients who died in the neurosurgical ICU showed that brain death diagnosis employing apparatus was not necessary in nearly half the patients because explantation of their organs was not possible because of adverse diseases in the history. Only a third of the patients dying in the ICU fulfilled the stringent criteria for multiorgan donors. Most of them were patients with severe skull-brain injuries and threatening circulatory collapse, and brain death could be established most quickly by angiography. Electrophysiological investigations and clinical examination proved of value in the follow-up of long-stay patients. These results, obtained retrospectively, will certainly be important in future management.

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Neurosurgical Diagnosis of Brain Death in the Peripheral Hospital Preceding Multiorgan Donation

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In recent decades rapid development of intensive care medicine has made dissociated brain death/coma dépassé [3] with artificially maintained cardiorespiratory function a diagnostic problem. After a long period of debate, brain death is today defined as the full clinical picture of brain stem death [1,4,5] with apnea, coma, and loss of brain stem reflexes. With this diagnosis established, organ donation can be considered in appropriate cases. The success of organ donation, however, is critically dependent on rapid and close cooperation between several medical specialties.

For 2 years at the University of Düsseldorf, outpatient diagnosis of brain death has been practised by the Department of Neurosurgery and the Section of Nephrology of the Department of Internal Medicine upon the suggestion of the "Kuratorium Heimdialyse und Organspende." The set-up and diagnostic procedures of the medical-neurosurgical investigation team are based on the Recommendations of the Scientific Commission of the Federal Medical Council (FMC) "Criteria of Brain Death" in its 1986 revision [5].

The institution of a "transplantation duty rota" ensures rapid deployment of a neurosurgeon and an internist in the peripheral hospital. The neurosurgeon on call can be contacted by a Euro-bleeper. The Kuratorium Heimdialyse also provided a transportable EEG recorder.

In the peripheral hospital the team takes a detailed history and excludes reversible causes of the clinical picture of brain death such as hypothermia and metabolic or drug-induced coma. Furthermore primary and secondary brain damage is noted. Thus excluding false-positive cases, clinical diagnosis is established to demonstrate apnea, coma, and brain stem areflexia. The test for apnea can be performed in any ICU by allowing $paCO_2$ to rise to 60 mmHg, disconnecting the patient from the ventilator under diffusion of oxygen (6 l O_2/min) through an NG tube introduced into the trachea. Following a 30-min recording of an isoelectric EEG, a first brain death protocol is signed [5]. The internist investigates the feasibility of multiorgan donation and institutes organ-centred maximal therapy. Blood samples for HLA typing are taken in the peripheral hospital. To avoid unnecessary effort, relatives' consent has to be obtained by the colleagues treating the patient before the team is called in. In patients without relatives or cases with medicolegal uncertainties the coroner on duty should be informed.

We have not included patients who clearly did not fulfill the brain stem death criteria. Frequently brain stem reflexes were still preserved; history and diagnosis remained obscure in one case and

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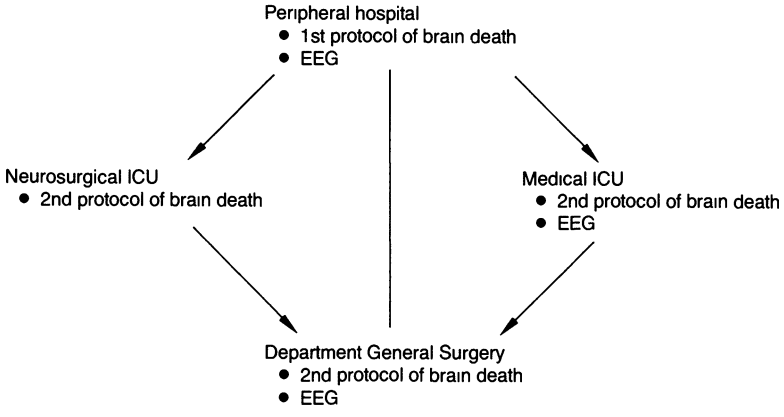

Fig. 1. Course of events in outpatient diagnosis of brain death

another patient was still being treated with muscle relaxants at the time of investigation.

With the diagnosis of brain death established, the organ donor is transferred from the peripheral hospital either straight to the Department of General Surgery or temporarily to the neurosurgical or medical ICU of the University of Düsseldorf (depending on the availability of IC beds). In the Department of Neurosurgery evoked potential studies can be performed. However, according to the FMC recommendations their use is limited in the context of organ donation and they do not lead to a further reduction of observation time.

After completion of diagnosis with a second brain death protocol in the ICU by the neurosurgeon, explantation of donor organs is performed in the Department of General Surgery under continued organ-specific maximal therapy (Fig. 1).

First experiences have shown that by interdisciplinary cooperation with colleagues in the peripheral hospitals, misconceptions were avoided in the outpatient diagnosis of brain death which previously had repeatedly thwarted organ donation in cases of attempted suicide, head injury, and resuscitation.

The obvious advantages of this procedure of diagnosis of brain death by a mobile investigation team are shortening of observation time and recruitment of potential organ donors.

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Experience with Determination of Brain Death and Organ Donation

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In a retrospective analysis, we report on our experience with determination of brain death on the basis of the patients at the neurosurgical intensive care unit of the University Hospital in Frankfurt in the period from 1980 to 1987. The following three criteria are of particular practical significance: the epidemiology of the mortalities, the practical significance of the diagnosis of brain death with the EEG, and the choice of organ donors.

Patients and Methods

In the period from 1980 to 1987, 3850 adult patients received intensive care and 543 (14.1%) died.

The epidemiological data are based on information from the Federal Office of Statistics in Wiesbaden [9]. The mortality rates in West Germany are broken down in accordance with the 9th Revision of the ICD [8]. The numbers 851, 852, and 800.3 are subsumed as head injuries. The numbers 530, 431, and 432 are listed under spontaneous hemorrhages. Numbers 191, 225, 253, and 239.1 are considered under brain tumors.

Brain death is established in accordance with the guidelines of the Bundesärztekammer from 1982 and 1986 [3,4]. In certain cases, EEG examinations are carried out as a supplementary measure for determination of brain death. The technique of EEG recording follows the guidelines of the German EEG Society [7].

The choice of organ donors is carried out in collaboration with the Department of Nephrology of the Johann Wolfgang Goethe University, Frankfurt.

Results

Epidemiology

Between 1980 and 1987, 3850 patients were treated in the neurosurgical intensive care unit. On average, 481 patients were treated per year (range: 378-582). Etiologically, head injuries (44.2%) predominate among the 543 who died, followed by spontaneous intracranial hemorrhages (29.2%) and brain tumors (22.8%). Roughly one-fifth of the patients died within 24 h after admission (Table 1).

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Table 1. Causes of diseases in the deceased persons (numbers in parentheses indicate those who died within 24 h after admission)

Year	No.	Etiology			
		Head injuries	Spontaneous intracranial hemorrhages	Brain tumors	Other[a]
1980	64 (17)	32 (11)	19 (4)	12 (1)	1 (1)
1981	68 (15)	34 (11)	23 (3)	11 (1)	
1982	59 (6)	24 (4)	10	22 (2)	3
1983	60 (7)	28 (5)	11 (2)	19	2
1984	80 (17)	28 (9)	30 (6)	19 (1)	3 (1)
1985	78 (14)	34 (7)	27 (4)	14 (2)	3 (1)
1986	71 (7)	31 (4)	21 (3)	14	5
1987	63 (17)	29 (10)	17 (3)	13 (3)	4 (1)
	543 (100)	240	158	124	21

[a] Spinal lesions 11 times, brain abscess twice, brain infarction four times, hydrocephalus four times.

The statistics on causes of death in West Germany show a decrease in the absolute and relative number of deaths due to head injuries in the years 1980-1986. The annual mortality due to spontaneous brain hemorrhages has displayed a slight decrease since 1985. No appreciable change can be discerned for brain tumors (Tables 2-4). Comparison of those among our patients who died with the overall deaths in West Germany shows that in relative terms the number of patients

Table 2. Deaths from head injuries in West Germany (Federal Office of Statistics, Wiesbaden) (ICD/9 nos. 851, 852, 853, 854, 800.3, 801.3) in relation to the number of deaths on the neurosurgical intensive care ward

Year	Deaths from head injuries				
	West Germany			Neurosurgical intensive care unit	
	No.	Percentage of total deaths	Deaths per 100 000	No.	Percentage of deaths in West Germany
1980	7448	1.04	12.10	32	0.43
1981	7370	1.02	11.95	34	0.46
1982	7245	1.01	11.75	24	0.33
1983	7451	1.04	12.13	28	0.38
1984	6824	0.98	11.15	28	0.41
1985	5878	0.83	9.63	31	0.53
1986	5715	0.81	9.36	29	0.51

Table 3. Deaths from spontaneous intracranial hemorrhages in West Germany (ICD/9 nos. 430, 431, 432) in relation to the number of deaths on the neurosurgical intensive care ward

Deaths from spontaneous cerebral hemorrhages					
	West Germany			Neurosurgical intensive care unit	
Year	No. (SAH)	Percentage of total deaths	Deaths per 100 000	No.	Percentage of deaths in West Germany
1980	9436 (1529)	1.11	15.33	19	0.20
1981	9579 (1475)	1.12	15.53	23	0.24
1982	9457 (1365)	1.13	15.34	10	0.11
1983	9507 (1425)	1.12	15.48	11	0.12
1984	9254 (1316)	1.14	15.13	30	0.32
1985	8772 (1310)	1.06	14.37	27	0.31
1986	8345 (1332)	1.00	13.67	21	0.25

SAH, subarachnoid hemorrhage

Table 4. Deaths from brain tumors in West Germany (ICD/9 nos. 191, 253, 239.6) in relation to the number of deaths on the neurosurgical intensive care ward

Deaths from brain tumors					
	West Germany			Neurosurgical intensive care unit	
Year	No.	Percentage of total deaths	Deaths per 100 000	No.	Percentage of deaths in West Germany
1980	3864	0.54	6.28	12	0.31
1981	3891	0.54	6.31	11	0.28
1982	3942	0.55	6.40	22	0.56
1983	4113	0.57	6.70	19	0.46
1984	4115	0.59	6.73	19	0.46
1985	4161	0.59	6.82	14	0.34
1986	4031	0.57	6.60	14	0.35

with head injuries who die on the neurosurgical intensive care ward has increased since 1985. For 1987, there are as yet no figures from the Federal Office of Statistics. A similar tendency is to be observed in respect of spontaneous brain hemorrhages. Comparison of the mortality rates for brain tumors does not show any demonstrable alteration.

Determination of the Time of Brain Death with the EEG

In the years 1980-1987, one or several EEG recordings (a total of 1060) were performed in 364 of those who died; an isoelectric EEG was performed 92 times. The EEG is recorded as a supplementary measure for determination of brain death after the clinical investigation in order to end the therapy, including the artificial ventilation, or if an organ explantation is planned. A repetition of the EEG recording with demonstration of electrocerebral silence was necessary in 19 of the above-mentioned cases, since the EEG was disturbed by artifacts.

EEG follow-up studies are performed in the prestages of brain death in all infratentorial processes. A dislocation between a still well-pronounced electrical basal activity within the alpha or beta range and loss of brain stem functions or the extinction of the early acoustic evoked potentials is often shown in these cases.

Organ Donation

The choice of possible organ donors is made in collaboration with the Department of Nephrology and the Transplantation Working Group at the University Hospital. Establishment of brain death and the written consent of the nearest relatives form the basis for an organ donation. In about one-quarter of the planned organ explantations, the relatives refused to give their consent. Besides the fundamental rejection of an organ explantation, restrictions with regard to the organ explantation are made by relatives, e.g., agreement to kidney explantation but not to multiorgan donation. Retrospective objections to the procedure were made only once with examination of the protocols and findings.

Exclusion criteria for kidney donation are primary disease of the kidney, malignancies outside the brain, high-risk groups, known arterial hypertension, infectious diseases, and religious and cultural reasons. Between 1980 and 1987, 184 explantations were performed, and of these 41 were in neurosurgical cases.

Discussion

Epidemiology

Deaths from head injuries in West Germany decreased markedly in the period 1980-1987. A similar trend is also to be observed in respect of spontaneous cerebral hemorrhages. Compared to this, the relative proportion of those who have died on the neurosurgical intensive care ward has increased. A possible explanation for this is that the more severe cases are being increasingly admitted to the neurosurgical unit. The number of deaths from brain tumors has remained the same. Here, it is to be considered that the data of the Federal Office of Statistics are based on the entries of the cause of death on the death certificate [2] and not, for example, on the result of autopsy.

EEG

As a supplementary investigation, the EEG has retained its practical value in the determination of brain death. If electrocortical silence is shown for at least 30 min during a continuous registration, then brain death can be observed in adults without a further observation period [1,3,4]. The extinction of the acoustically evoked brain stem potentials may replace an EEG recording in primary supratentorial brain damage. EEG follow-up examinations are absolutely necessary in primary infratentorial processes [5,6,10,11]. Employing the criteria of the German EEG Society [7], the appraisal is unequivocal.

Organ Donation

The criteria for the selection of organ donors are relatively uniform in the different study groups [6]. In recent years, and especially since 1985, the indication for organ explantation has been appreciably extended. The age for the donor is no longer a contraindication: the biological age of the organ and the age of the recipient are regarded as crucial. Kidney malformations are no longer an absolute objection. In horseshoe kidneys, a separation of the organs can be performed in many cases. In infectious diseases, especially Australia antigen-positive hepatitis, transplantation is occasionally possible when the modus of infection in the donor is known and the recipient is also positive. The basis for the decision to plan an organ donation is precise recording of investigative findings. Here, the protocol of the Bundesärztekammer has proved effective [3]. Retrospective objections to the procedure must be reckoned with, but such objections are rare.

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On Problems in the Determination of Brain Death

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Diagnosis of brain death is only possible in cases of complete and permanent loss of function of the brain. If brain death is diagnosed, all further therapeutic procedures are purposeless.

There is no doubt that brain death must be established clinically and that the irreversibility of this state must be documented for a suitable time. It is necessary to know exactly the development of a disease in order to reach the diagnosis and to exclude transient restrictions of cerebral function. The purpose of other diagnostic procedures is to aid the clinical diagnosis and to shorten the time until definitive determination of brain death.

In our neurosurgical clinic in Berlin-Buch in the last 10 years we have had to decide on the question of brain death in 57 patients (Table 1). In all cases the determination and documentation of brain death was performed according to the principles proposed in 1973 by the Society of Neurology and Psychiatrics and the Society of Anesthesiology of the German Democratic Republic. The decision on brain death must be reached by a team consisting of at least two specialists, one neurologist and one anesthetist. In our clinic this team is completed by the specialist for intensive care and the director of the clinic.

In all our patients the course is exactly documented on a supervisory protocol. If all clinical signs of brain death are manifest - i.e. (a) unconsciousness without any reaction to pain and auditory stimuli, (b) absence of spontaneous respiration, and (c) total cerebral areflexia - the protocol must be compiled by the neurologist. The findings have to be controlled three times within 12 h (Table 2).

Table 1. Diagnoses in 57 patients with documented brain death

Supratentorial brain tumors	15
Infratentorial brain tumors	6
Brain injuries	18
Aneurysms and angiomas	11
Intracerebral hematomas (nontraumatic)	4
Occlusion of basilar artery	1
Inflammatory diseases	2

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Table 2. Protocol for documentation of irreversible loss of cerebral function

1.	Name:				
	Date of birth:				
	Date of study:				
1.1.	Diagnosis:				
	Primary damage of brain:	yes - no			
	supratentorial:	yes - no			
	infratentorial:	yes - no			
	Secondary damage of brain:	yes - no			
	Onset and progression of the illness:				
1.2.	Exclusion of intoxications, neuromuscular blockade, primary hypothermia, hypovolemic shock, endocrine or metabolic coma	yes - no			
2.	Clinical identification of determinant symptoms of brain function loss				
	Examination:				
	date				
	time				
			1.	2.	3.
2.1.	Coma - deep unconsciousness with muscular atonia, absence of spontaneous movements, and lack of responsiveness to external stimuli				
2.2.	Absence of spontaneous respiration				
	(since date: time:				
	Apnea test				
2.3.	Cerebral areflexia				
2.3.1.	Reflex of accommodation				
2.3.2.	Pupils dilated				
2.3.3.	Oculocephalic reflex				
2.3.4.	Vestibulo-ocular reflex				
2.3.5.	Corneal reflex				
2.3.6.	Trigeminal pain reaction				
2.3.7.	Reflex of trachea and pharynx				
2.4.	Additional findings (for example spinal reflexes)				
2.5.	Actual clinical findings:				
	Blood pressure				
	Pulse				
	Temperature				
3.	Additional diagnostic procedures:				
3.1.	EEG isoelectric				
	Date/time	yes - no	signature		
	Date/time	yes - no	signature		
3.2.	Cerebral angiography				
	Circulation arrest	yes - no			
	Date/time		signature		
4.	The loss of brain function has lasted for ...hours. In summary of all findings documented in this protocol, the brain death of the patient is diagnosed				
	date:	time:			
Neurologist	Anesthetist	Treating physician			

Three hours after the first documentation of brain death we perform an EEG examination. In 50 of our 57 patients we saw an isoelectric EEG. In all our cases neurological and EEG examination were performed by the clinic neurologist. The EEG examinations were performed for at least 30 min under special technical conditions. In seven of our 57 patients we found residual EEG activity in spite of documentation of all signs of clinical death. In those patients EEG examination was repeated, and in all of them we saw an isoelectric EEG within 24 h. We conclude that residual activity in the EG in cases of loss of function of brain and brain stem is always the forerunner to complete loss of cerebral function and is never effective for the whole organism. The determination of residual EEG activity excludes the diagnosis of brain death at this time; the diagnosis of brain death cannot be made before documentation of an isoelectric EEG.

Only in four of our patients did we perform cerebral angiography additionally. Twice we saw some different vertebrobasilar vessels far within the posterior fossa. In these cases EEG activity was no longer seen and the neurological signs of brain death had been registered for 24 h.

Summary and Conclusions

This report deals with our experiences in the diagnosis of brain death in 57 patients. After manifestation of all symptoms of brain death a protocol must be filled out. All clinical examinations have to be repeated three times within 12 h.

Three hours after the first manifestation of all symptoms of brain death an EEG examination must be performed for at least 30 min under special conditions. If there is any residual activity, EEG examination must be repeated within 24 h.

The diagnosis "brain death" is documented by a protocol, signed by at least three specialists.

Latency of Recovery and Electrical Silence of Auditory Evoked Potentials and the Electrocorticogram After Peracute Complete Brain Ischemia of 2-30 Minutes' Duration

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Introduction

The time from the end of complete cerebral ischemia to the reappearance of first potentials of the electrocortigram has been determined in several investigations [e.g. 1,5,7,8,9]. This time interval is called the latency of recovery. There are only a few data about the latency of recovery of auditory evoked potentials after complete brain ischemia, although evoked potentials have great clinical importance for diagnosis and prognosis. The purpose of this paper is to determine the latencies of recovery of the early and middle latency auditory evoked potentials after peracute complete global ischemia lasting 2-30 min. Moreover, the latency of recovery of the electrocorticogram is determined in order to compare the latencies of recovery of auditory evoked potentials with those of the electrocorticogram. Finally, the duration of electrical silence of the auditory evoked potentials is compared with the duration of electrical silence of the electrocorticogram.

Materials and Methods

Complete peracute global ischemia lasting 2 min (n = 6), 5 min (n = 6), 10 min (n = 6), 20 min (n = 6), or 30 min (n = 6) was induced in dog brains perfused with an extracorporeal system. Details of the methodological procedure have already been described [1,5]. The ischemia began at brain temperatures of 37°C. During ischemia of 2 min, 5 min, 10 min, 20 min, and 30 min duration the temperature decreased by 0.2°, 0.2°, 0.3°, 1.1°, and 1.9°C respectively and reached 37°C again 2-20 min after the end of ischemia. The methodological procedure for the registration of the early and middle latency auditory evoked potentials and the electrocorticogram has been described in another paper [4]. Auditory evoked potentials were elicited using 0.2-ms alternating clicks of 70-90 dB above hearing level presented binaurally at a rate of 0.92 Hz.

The latency of recovery of the auditory evoked potentials is the interval between the end of complete ischemia and the first potentials which can be discriminated from the background noise. The duration of electrical silence of the auditory evoked potentials is the interval between the complete extinction of the last evoked potentials which can be discriminated from the background noise during complete ischemia and the reappearance of the first evoked potentials which can be discriminated from the background noise after the end of complete ischemia.

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Electrocorticographic recordings were carried out with a gain of 25 μV/cm. The latency of recovery of the electrocorticogram is the interval between the end of complete ischemia and the reappearance of the first potentials with a minimum amplitude of 3 μV. The duration of electrical silence of the electrocorticogram is the interval between the complete extinction of the last potentials with an amplitude of 3 μV during complete ischemia and the reappearance of the first potentials with a minimum amplitude of 3 μV after the end of complete ischemia.

Only one period of complete ischemia was performed on each brain.

Results

The longer the peracute complete brain ischemia continued, the later the auditory evoked potentials reappeared after peracute complete global brain ischemia. Figure 1 gives representative examples of the early auditory evoked potentials after ischemia of 2 min, 5 min, 10 min, 20 min, and 30 min duration. In Fig. 2 representative examples for the reappearance of the middle latency auditory evoked potentials after ischemia of the same durations are shown.

Figure 3 shows the latencies of recovery of the early and middle latency auditory evoked potentials and of the electrocorticogram of all experiments after ischemia. Only 5 of 30 brains showed the same latencies of recovery of the early and middle latency auditory evoked potentials. In the other cases the latencies of recovery of the early auditory evoked potentials were shorter than the latencies of recovery of the middle latency auditory evoked potentials. The latencies of recovery of the electrocorticogram were longer than those of the evoked potentials in all brains.

Figure 4 gives the mean values of the duration of electrical silence of the early and middle latency auditory evoked potentials and the electrocorticogram. The durations of electrical silence of the early

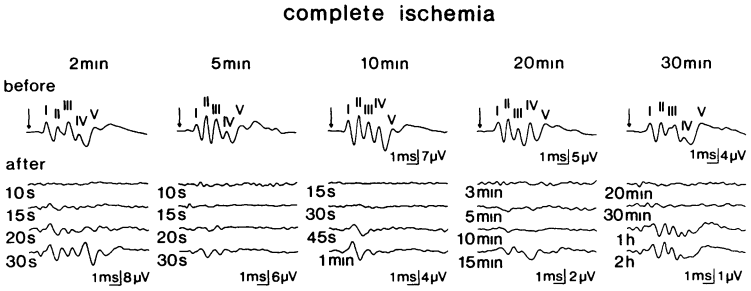

Fig. 1. Early latency auditory evoked potentials before and after complete brain ischemia of 2 min, 5 min, 10 min, 20 min, and 30 min duration. The **first tracing** of each column was recorded before complete ischemia, the following tracings after complete ischemia. The **second tracing** is a registration immediately before the reappearance of the evoked potentials. The **third tracing** is the registration of the first reappearance of evoked potentials. The **following two tracings** are later registrations. The **figures left of the columns** indicate the duration of reperfusion after the end of complete ischemia. The **arrows** indicate the click signal

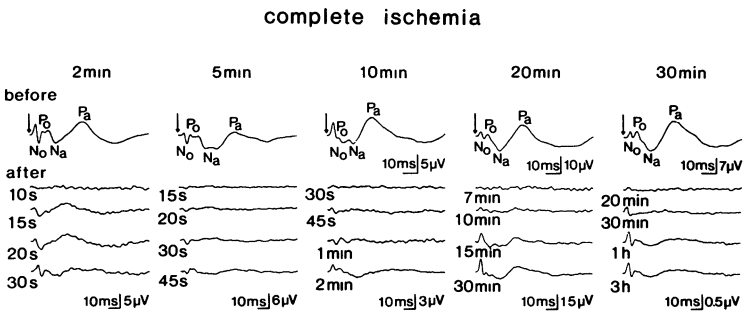

Fig. 2. Middle latency auditory evoked potentials before and after complete brain ischemia of 2 min, 5 min, 10 min, 20 min, and 30 min duration. Further explanations as in Fig. 1

and middle latency auditory evoked potentials were the same in only 3 of 30 brains. In the other 27 brains the durations of electrical silence of the early auditory evoked potentials were shorter than those of the middle latency auditory evoked potentials. In all brains the durations of electrical silence of the electrocorticogram were longer than those of the early and middle latency auditory evoked potentials.

Discussion

Data presented in this paper concerning the latency of recovery of electrocorticogram correspond to those reported in other papers [1,5,7,8,9].

Our experiments revealed that the latencies of recovery of auditory evoked potentials are shorter than the latencies of recovery of the

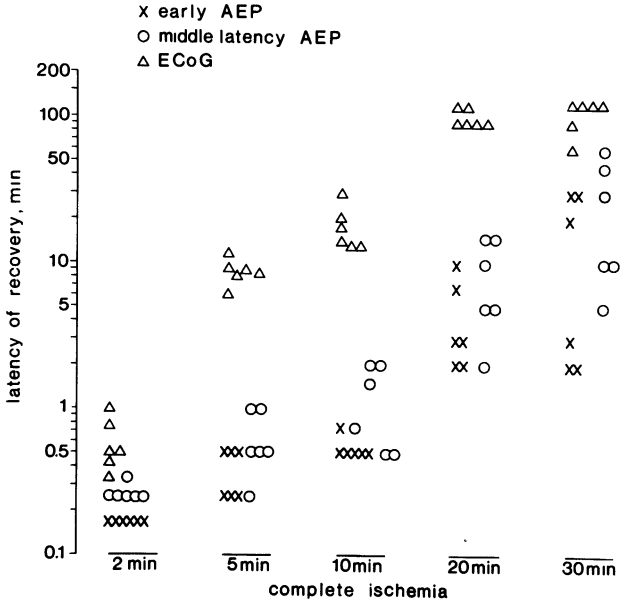

Fig. 3. Latencies of recovery of early and middle latency auditory evoked potentials (AEP) and electrocorticogram (ECoG) after complete brain ischemia of 2 min, 5 min, 10 min, 20 min, and 30 min duration. Single data of each set of six experiments

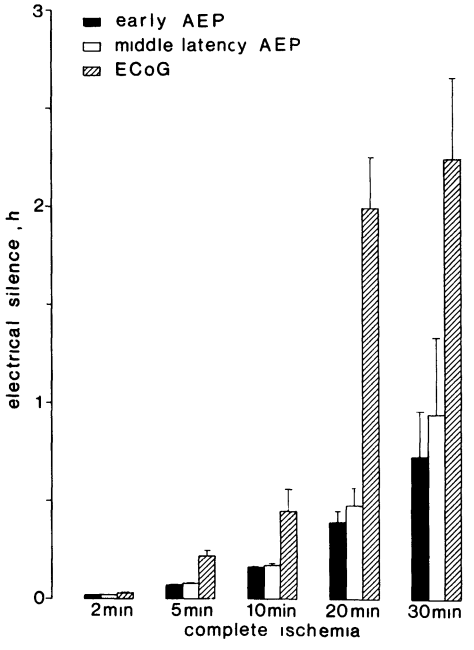

Fig. 4. Duration of electrical silence of early and middle latency auditory evoked potentials (AEP) and electrocorticogram (ECoG) after complete brain ischemia of 2 min, 5 min, 10 min, 20 min, and 30 min duration. Mean values and standard deviations

electrocorticogram. This is in agreement with the fact that the survival time (i.e., the time from the onset of peracute complete brain ischemia until the complete extinction of the potentials) is longer for the auditory evoked potentials than for the electrocorticogram [4].

The durations of electrical silence (Fig. 4) demonstrate the different resistance of the auditory evoked potentials and of the electrocorticogram to complete cerebral ischemia. In a study by SOHMER et al. with various degrees of lowered cerebral perfusion pressure the electroencephalogram also became isoelectric before the early auditory evoked potentials. After complete ischemia for 30 min SOHMER et al. found no more recovery, whereas in our experiments the latencies of recovery ranged between 2 and 30 min. These differencies between the data of SOHMER et al. and our results may be due to different conditions of recovery.

This paper only reports on the beginning of recovery of evoked potentials. A report on the further recovery of auditory evoked potentials will appear in a later publication. Single observations of our data have been reported previously [2,3].

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Methodological and Technical Problems in the Confirmation of Brain Death by Evoked Potentials

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Introduction

A number of clinical criteria have been established and internationally accepted as sufficient to document total loss of brain function which can be equated to the death of the individual [1,8].

The comparison of various national brain death codes shows that there is agreement on the clinical signs of brain death: coma, absence of all cranial reflexes, and apnea. Variations, however, exist in the number of clinical examinations and the number of physicians required to make the clinical diagnosis of brain death. Also, various technical methods are accepted to corroborate the clinical diagnosis. By the standards put forth by the West German Medical Association in 1982 [8], the clinical signs of brain death must be present for 12 h and confirmed by two physicians if the diagnosis is to be made by clinical examination alone. To shorten this procedure, three technical procedures can be implemented alternatively to confirm the clinical diagnosis in supratentorial disease after the first examination: EEG, angiography, or somatosensory and brain stem auditory evoked potentials [3,9]. In infratentorial disease, however, the development of the brain death syndrome can follow different patterns. Therefore, evoked potentials can only be implemented in conjunction with EEG recordings [2].

The methodological and technical problems of the use of evoked potentials in the diagnosis of brain death are to be discussed further.

Numerous publications [e.g., 4,7] have shown that abolished acoustic evoked brain stem potentials (BAERs) are associated with brain death. Also, there is widespread agreement that bilaterally abolished cortical median-evoked somatosensory potentials (SEPs) can be a sign of loss of cortical function and in cases of supratentorial disease invariably indicate poor prognosis. Bilateral SEP loss alone, however, is not sufficient to document brain death [6]. The confirmation of the clinical signs of brain death as defined above by evoked potentials is dependent on the subsequent loss of all intracerebral components of BAERs and SEPs.

Results

We examined 67 patients in a state of clinically confirmed apneic brain stem areflexia as defined previously [8,9]. Sixty-two patients suffered from primary supratentorial disease (brain infarction and hemorrhage, tumors, subarachnoid hemmorrhage, and anoxia) and five

patients had suffered infratentorial vascular accidents. After clinical diagnosis of brain death, all 67 showed abolished intracerebral components of BAERs; 29 patients demonstrated neither intracerebral nor extracerebral BAER waves. In 53 examined patients, all cortical SEPs were abolished and in 15 cases, additionally registered spinal SEPs were preserved. Forty-three patients were examined only in the state of brain death so that prior patency of the acoustic pathways of the brain stem could not be proven.

Typical Patterns

A 35-year-old patient suffered a left hemispherical embolus. On the 5th day of treatment, the BAERs were only mildly altered on the left and otherwise within normal limits. The SEPs were abolished bilaterally as a sign of poor prognosis. On the 9th day the clinical signs of brain death were present. At this time BAERs and cortical SEPs were bilaterally abolished whereas the spinal SEP C_2 was still preserved (Fig. 1). At the same time, an isoelectric EEG was registered (Fig. 2). This course demonstrates the typical rostrocaudal development of the brain death syndrome.

Technical Problems

Technical problems in the use of evoked potentials for the confirmation of brain death usually arise from electrical interference

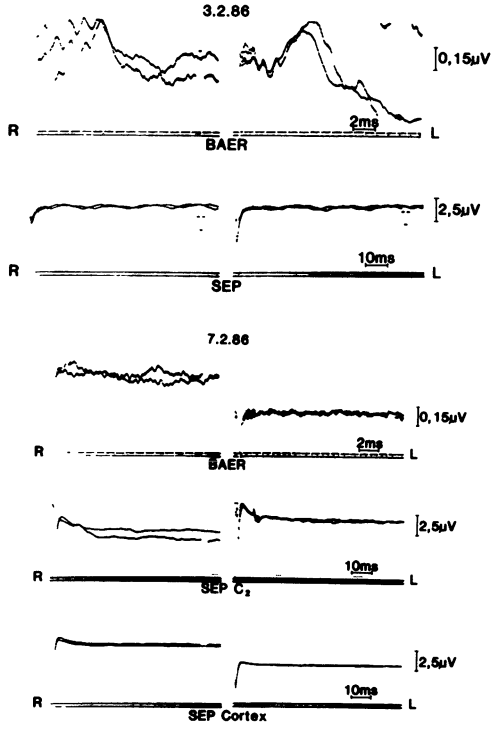

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Fig. 1. Cerebral embolus. Preserved BAERs and abolished cortical SEPs on day 4. Loss of BAERs and cortical SEPs with preserved spinal SEP C_2 on day 9

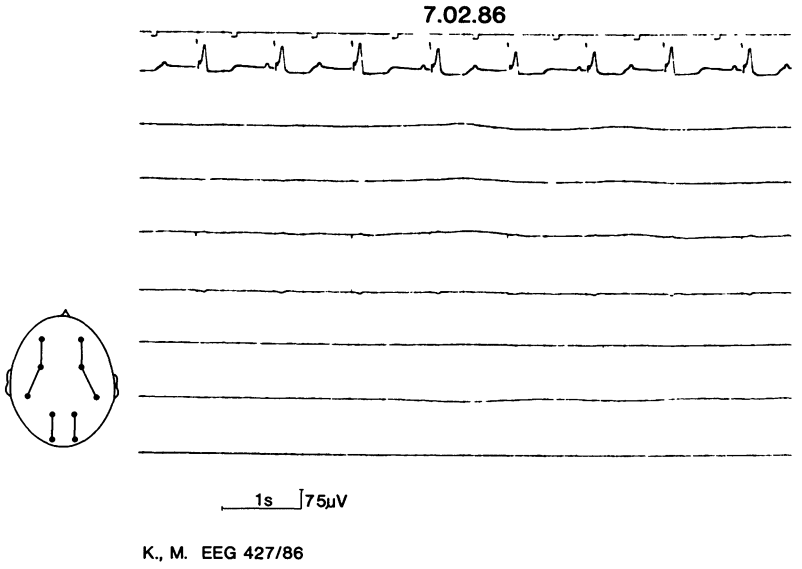

Fig. 2. Same patient as in Fig. 1, isoelectric EEG on day 9

emitted by the electrical apparatus on intensive care units: respirators, monitors, and infusion equipment. Whenever possible, all nonessential equipment should be switched off and the registration performed far away from the respirator. Even then, many tracings will be contaminated by electrical interference. In cases of doubt, three or more averaging cycles can help to clarify the findings. In some cases, cranial muscular activity can also distort the tracings; this interference can easily be avoided by the administration of muscle relaxants. This finding does not contradict the diagnosis of brain death [5].

Methodological Problems

The principle of confirmation of brain death by evoked potentials lies in the demonstration of subsequent loss of all intracerebral components of BAERs and SEPs. In many cases, especially in massive intracerebral and subarachnoid hemorrhage with rapid development of tentorial herniation, evoked potentials can be absent at first examination. In this situation, patency of the acoustic pathways of the brain stem prior to brain death cannot be proven. This was the case in 43 of our examinations. Evoked potential testing, therefore, must be performed as soon as possible in critically ill patients. Exact knowledge of primary etiology and localization of disease is also most important. In supratentorial disease where the brain death syndrome develops in a rostrocaudal direction, evoked potentials are reliable to document complete cessation of brain function. In infratentorial disease, such as infratentorial hemorrhage but also in severe meningoencephalitis with predominant affection of the brain stem, BAERs and SEPs can be abolished in the presence of persisting EEG activity for a prolonged period. In these cases, evoked potential findings can only be interpreted together with EEG findings [2]. A patient suffering from brain stem infarction demonstrated bilaterally abolished BAERs at first examination with preserved SEPs. One day later, both BAERs and SEPs were absent (Fig. 3); however, on the

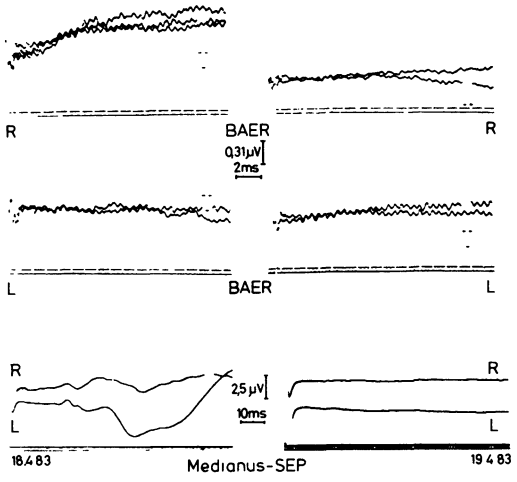

Fig. 3. Brain stem infarction. Abolished BAERs with preserved SEPs at first examination; abolished BAERs and SEPs on the following day

following day, unquestionable cortical EEG activity was registered (Fig. 4). The patient died on the next day. This course shows the caudal to rostral development of the brain death syndrome in infratentorial lesions and proves that in spite of loss of evoked potential responses EEG activity can persist in some cases.

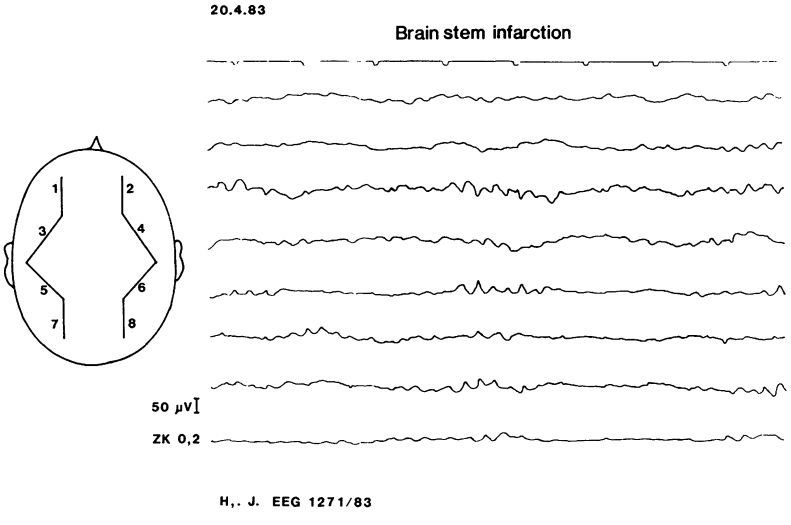

Fig. 4. Same patient as in Fig. 3: preserved EEG activity 1 day after loss of BAERs and SEPs

Summary

Evoked cerebral potentials have been demonstrated to be a viable noninvasive reliable method to confirm the clinical signs of brain death. Technical problems are usually surmountable; however, a number of methodological reservations must be kept in mind. The examinations must demonstrate successive loss of BAER components; therefore, initial testing must be done as early as possible. The etiology and primary localization of disease must be known. In uncertain cases and especially in primary infratentorial disease, evoked potentials can only be used to confirm the clinical signs of brain death in conjunction with EEG examinations.

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Nasopharyngeal Recording of Subcortical Somatosensory Evoked Potentials in Brain Death

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Introduction

If the recording of somatosensory evoked potentials (SEPs) is used as an ancillary method in the diagnosis of brain death, the distinction between cerebral and extracerebral SEP components is essential: the former must be absent in (whole) brain death, whereas the latter may be preserved. Usually, extinct cortical potentials with facultatively recordable neck potentials are considered a typical finding in brain death [7,8,11]. However, the neck potential N13 (after median nerve stimulation), under clinical conditions usually recorded from neck-to-scalp electrode montages, is contributed to by two components: N13a (horizontal dipole in the lower cervical spinal cord) and N13b [longitudinal dipole in the lower brain stem, corresponding to the third farfield potential (P14) in scalp-noncephalic reference recordings] [6,10]. These two subcomponents can be separately recorded: N13a from lower posterior against anterior neck [5] and N13b from nasopharynx against a midfrontal reference [13]. The application of this recording technique in brain-dead patients is described here for the first time.

Patients and Methods

Nine brain dead patients (ages 19-67 years; seven males, two females) were studied. Eight had supratentorial lesions (hemorrhage, tumor, brain edema), and one suffered from large bilateral acoustic neuromas.

The brain death syndrome had lasted between 6 and 72 h at the time of evoked potential recording. In all patients, the median nerve was stimulated at the wrist, in some cases on both sides. Scalp and neck recordings were made from Teflon-coated needle electrodes; the nasopharyngeal lead consisted of an isolated silver wire with a (nonisolated) ball-shaped tip.

Details of stimulus and recording parameters are listed in Table 1.

Results

Two typical SEP constellations found in brain death are shown in Figs. 1 and 2: N13b (Pgz-Fz) was regularly absent, while N13a (Cv7-Jugulum) was preserved in most cases. As a rule, the scalp far-field potential P14 was lacking (Fig. 1), but in two cases it could

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Table 1. Stimulus and recording parameters

Stimulus site	N. medianus (wrist)
Stimulus intensity	Just above motor threshold
Stimulus duration	0.2 ms
Stimulus repetition rate	3/s
Electrode montages	C'3-Fz, C'4-Fz, Fz-NC, Pgz-Fz, Cv7-Jugulum, Cv7-Fz, Cv2-Fz
Electrode impedance	<2000 Ohm
Filter setting	20-2000 Hz
Analysis time	20 ms, 50 ms
No. of samples averaged	500-1000

Pgz, median nasopharynx; Cv2/Cv7, skin over spinous process of 2nd/7th cervical vertebrae; NC, noncephalic reference (hand contralateral to stimulation side); Fz, C'3, and C'4, electrode positions at the scalp according to the 10-20 system

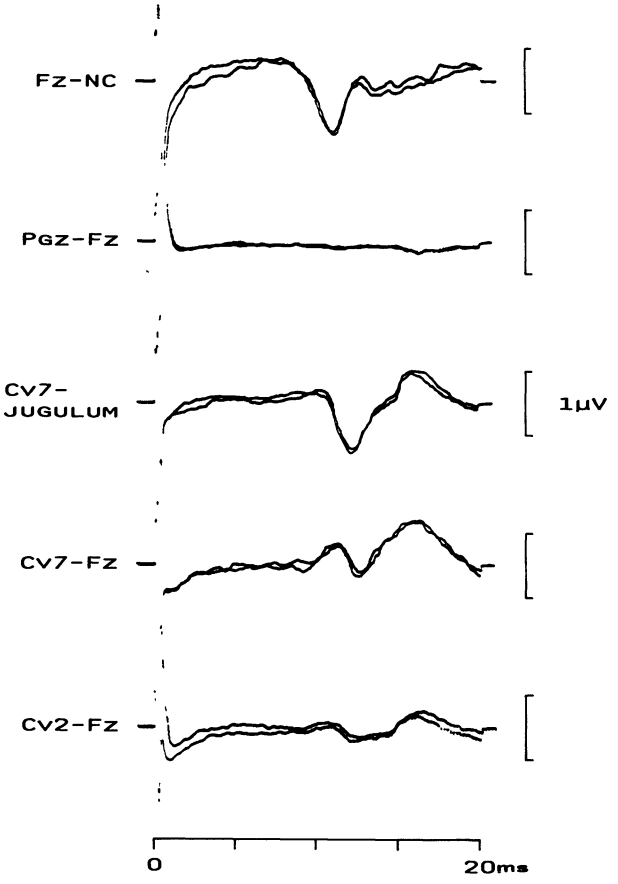

Fig. 1. Subcortical SEPs several hours after brain death had been established in a 59-year-old patient suffering from recurrent subarachnoid hemorrhage. N13b in Pgz-Fz and P14 in Fz-NC absent; N13a in neck recordings preserved. For explanation of electrode positions, see Table 1

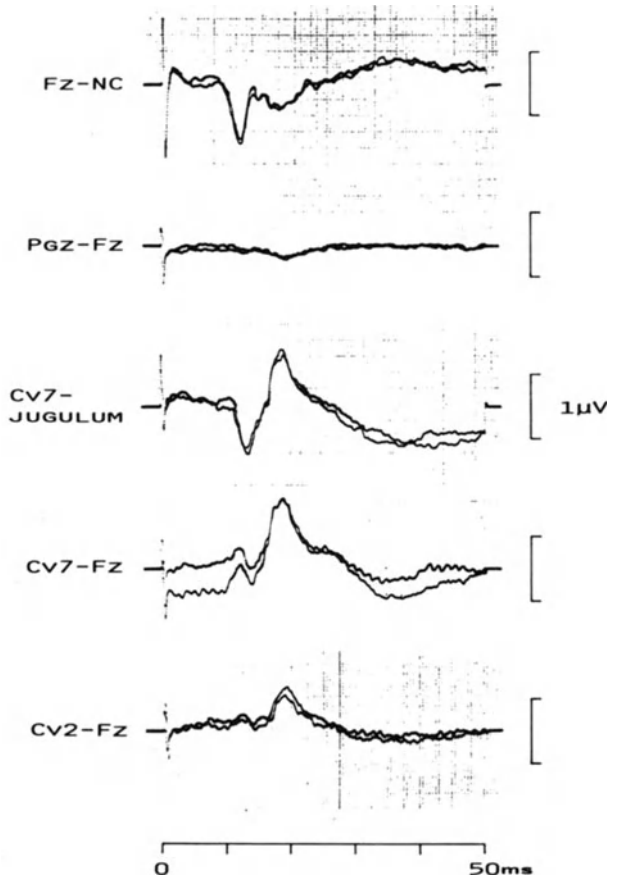

Fig. 2. Subcortical SEPs 20 h after brain death had been established in a 53-year-old patient suffering from left frontal intracerebral bleeding. No N13b in Pgz-Fz, but a low amplitude P14 in Fz-NC. N13a preserved. For explanation of electrode positions, see Table 1

be reproduced, having a very low amplitude (Fig. 2). The findings in detail are as follows:

C'3-Fz, C'4-Fz: Cortical potentials were absent in all patients (not shown in Figs. 1 and 2).
Fz-NC: P9 was preserved in every patient; P11 was distorted or not identifiable; P14 was lacking in seven cases and reproducible, but of very low amplitude, in two cases.
Pgz-Fz: N13b was lacking in all patients (including those showing a low amplitude P14 farfield potential in Fz-NC); in some cases, a very flat positive wave with the same latency as P14 was seen.
Cv7-Jugulum: N13a was reproducible in six patients, the dorsal root potential ["N10," cf. [5]] in all cases.
Cv7-Fz: N13 was preserved in those six patients showing N13a in Cv7-Jugulum.
Cv2-Fz: The same as in Cv7-Fz, but with markedly lower amplitude.

In contrast to these results, an example of subcortical SEPs in deep coma is shown in Fig. 3. This patient sustained a severe brain stem injury (without brain death) following head trauma and had no cortical potentials. Here, the N13b component is clearly visible in Pgz-Fz (and, as third farfield potential, in Fz-NC).

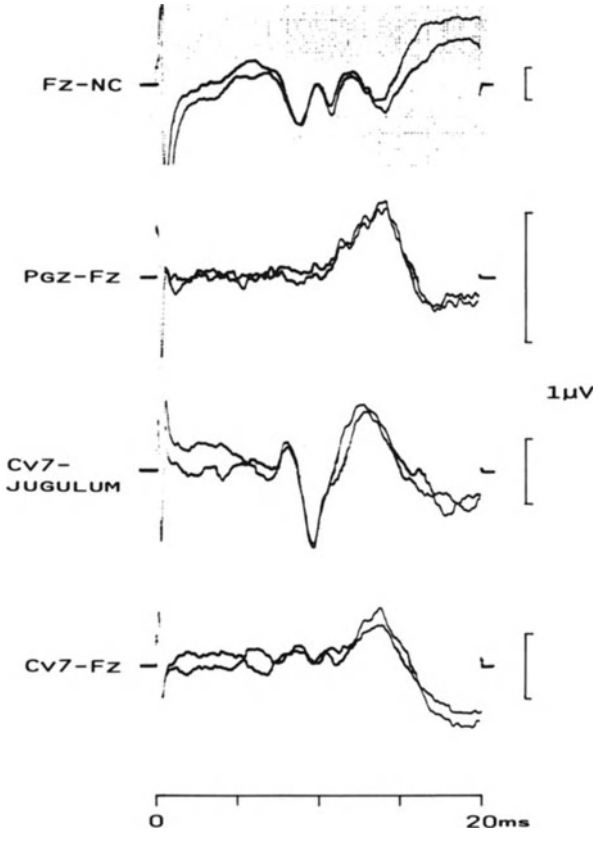

Fig. 3. Subcortical SEPs in a comatose (not brain dead) 39-year-old patient with signs of severe brain stem injury due to head trauma. N13b in Pgz-Fz (and P14 in Fz-NC) clearly preserved. For explanation of electrode positions, see Table 1

Discussion

As could be expected from previous work by other authors [1-4,7-9,11,12] cortical potentials were absent in every brain-dead patient, whereas spinal (N13a) and peripheral (P9, "N10") components were preserved in most cases. The interpretation of these findings is clear: they are the electrophysiological correlate of a dead brain and a still functioning spinal cord and peripheral nervous system.

Less obvious is the significance of the component N13b (or P14, respectively), generally attributed to lemniscus medialis, or nucleus cuneatus [6]. It is the most important finding of this study that, in recordings from Pgz-Fz, N13b was lacking in every brain-dead patient (Figs. 1, 2), whereas this wave was regularly recordable in living patients even with severe brain stem injury (Fig. 3) [14].

In noncephalic referenced scalp recordings (Fz-NC), this component appears as P14 (or P13/14). In brain (stem)-dead patients, this component should be lacking (and it is in most cases, cf. Fig. 1), but in two of our own patients, P14 was reproducible with a very low amplitude (Fig. 2). This phenomenon has also been described by other authors [1,3,4,11]; it seems to be inconsistent with whole brain death (including brain stem).

We suggest that in some cases of brain death syndrome there may be residual electrical activity in the caudal medulla oblongata (below the tip of the nasopharyngeal lead), resulting in a very small P14 farfield potential without N13b in Pgz-Fz. This hypothesis is supported by SEP recordings in the progression from coma to brain death; moreover it could explain why in some brain-dead patients a very flat positive wave in Pgz-Fz with the same latency as P14 in Fz-NC can be recorded [14].

With subcortical SEP recording only from neck electrodes (in neck-to-scalp montages), it would have been difficult to clearly distinguish the brain stem N13b from the spinal N13a component, as is demonstrated by the preservation of an N13 wave in brain death even in higher cervical regions.

Conclusion

Nasopharyngeal SEP recording facilitates the differentiation of spinal and brain stem subcomponents of the neck potential, which is crucial in electrophysiological brain death studies. It therefore provides a useful ancillary test in the assessment of this clinical syndrome and seems highly sensitive to brain death, yielding no false-negative results in all hitherto investigated patients.

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Is the Loss of Evoked Potentials and Brain Stem Reflexes as Investigated Electrophysiologically Proof of Brain Death?

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Introduction

Since 1984, besides the electroencephalogram (EEG), evoked potentials and electrically as well as mechanically triggered brain stem reflexes have also been registered at the Neurosurgery Division, University of Giessen. Since 1984, nine patients have been observed in whom there was clinical evidence of cerebral death (dilated or moderately dilated fixed pupils, clinical brain stem areflexia, respiratory arrest, flaccid muscular tonus, abolished muscle monosynaptic reflexes) and loss of the evoked potentials and the brain stem reflexes investigated electrophysiologically, but in whom the EEG still showed electroencephalographic activity.

Material and Methods

Nine adult patients (26-68 years old, mean 40.55 years) are reported on in whom early acoustic (BAEP), somatosensory (SEP: N_{14}/N_{20}) and visual (VEP: P_{100}) evoked potentials and among the brain stem reflexes the electrically evoked blink reflex as well as the mechanically evoked glabella reflex and masseter reflex were elicited. Before triggering the blink reflex, the muscular response from the orbicularis oculi muscle in stimulation of the facial nerve was registered. The technique for registration of evoked potentials and brain stem reflexes has been described in detail elsewhere [4]. The underlying causes of damage were craniocerebral traumata in five cases, spontaneous primary infratentorial diseases in three cases (one cerebellar mass hemorrhage and two basilar thromboses), and secondary brain damage in the form of global cerebral hypoxia after circulatory arrest of cardiac origin and resuscitation in one case.

Results

All patients displayed clinical signs of brain death at the time of the investigation. The registration of the BAEPs revealed a zero line or a still preserved wave I, absence of cortical SEPs, and a zero line in registration of VEPs (Fig. 2). In stimulation of the facial nerve, a muscular response could be recorded in all patients. This was slightly reduced in amplitude in only two cases. All early and late reflex responses of the blink reflex and the glabella reflex were absent. The masseter reflex was also absent on both sides (Fig. 3). On the other hand, the EEG recording still displayed spontaneous activity which was altered in the form of an alpha EEG (case 1) as

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the most severe form of general alteration or in terms of burst suppression EEG (Fig. 4) with development to the zero-line EEG.

Case 1. A 58-year-old female patient suffered a basilar arterial occlusion with development of a complete bulbar brain syndrome. In this condition, all evoked potentials and the brain stem reflexes investigated electrophysiologically were negative, whereas electroencephalography showed an alpha EEG.

Case 2. A 27-year-old female patient suffered an open craniocerebral trauma with diabetes insipidus and development of a bulbar brain syndrome from the 4th day. In the registration 2 days after the trauma, the VEP was still approximately normal, and the masseter reflex which could be evoked on one side was pathological; all further potentials and reflexes were absent. The control registration 2 days later revealed a loss of all reflexes and potentials, whereas the EEG recorded afterwards revealed distinct electroencephalographic activity.

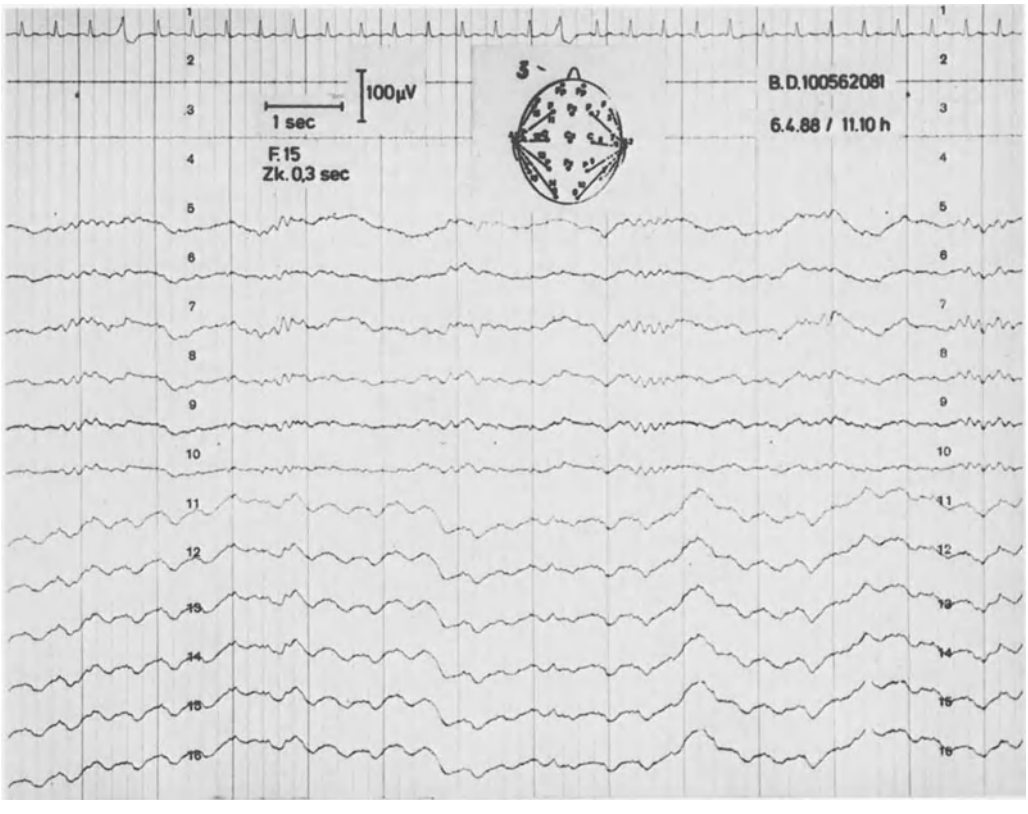

Fig. 1. A 27-year-old male patient with severe brain contusion. In the EEG, burst suppression pattern with 5- to 7-s bursts roughly every 2-3 s

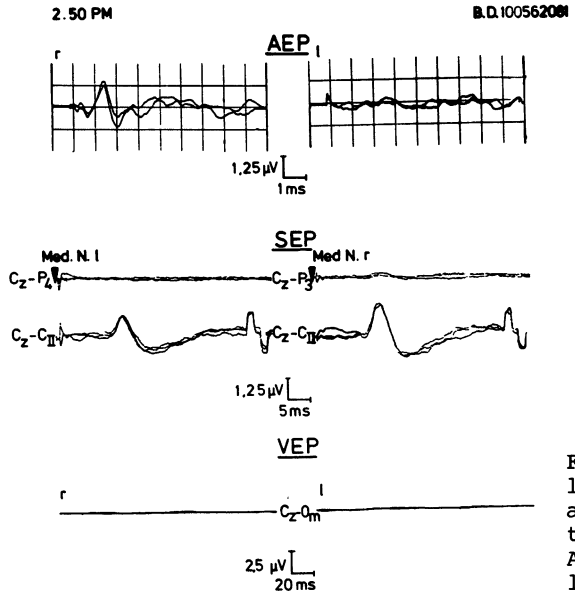

Fig. 2. The same patient with loss of BAEPs on the left side and loss of BAEP waves rostral to wave I on the right side. Absent cortical SEPs. Zero-line VEP

Case 3. A 27-year-old male patient with severe brain contusion which was more pronounced on the left showed 5- to 7-s bursts in the EEG approximately every 2-3 s (Fig. 1). The evoked potentials registered 3 1/2 h later merely showed a peripherally generated wave I on the right side. In the registration of the SEPs, a neck SEP which could be evoked over C_2 was shown, whereas the VEP displayed a zero line (Fig. 2). All oligosynaptic and polysynaptic reflex responses were absent (Fig. 3). The EEG which was afterwards recorded once more over the right hemisphere revealed a declining burst suppression pattern with development to the zero-line EEG (Fig. 4).

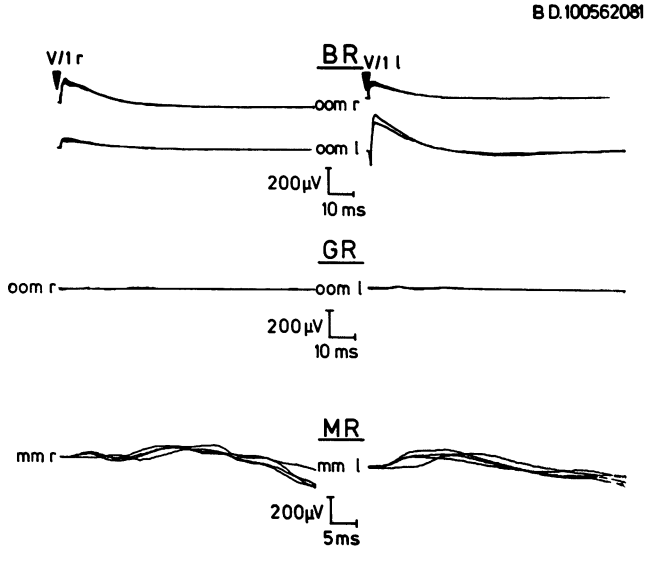

Fig. 3. The same patient. Loss of the electrically evoked blink reflex and the mechanically evoked glabella and masseter reflexes

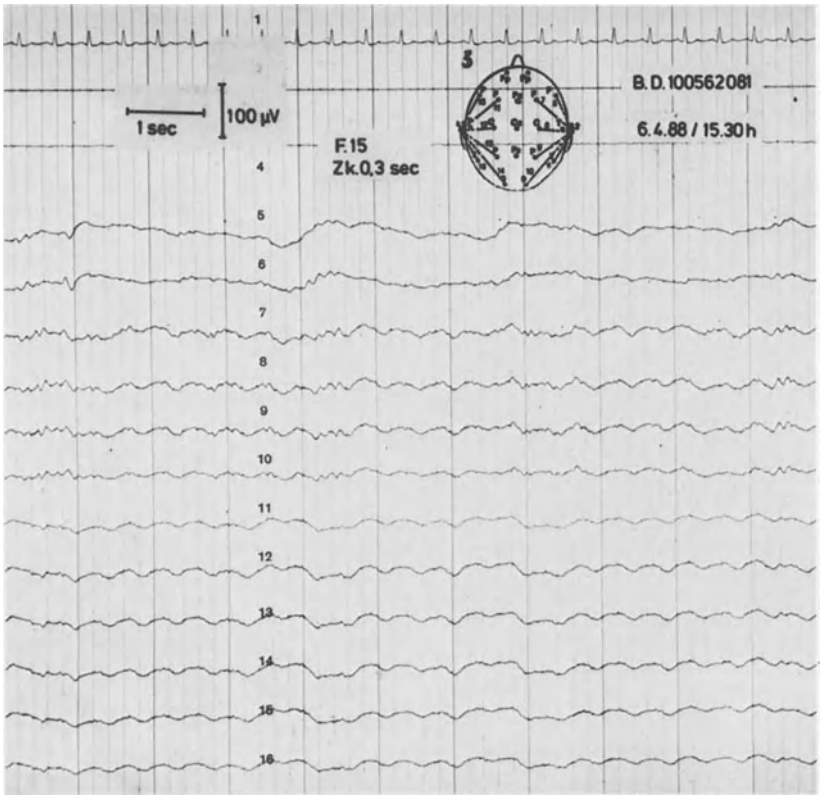

Fig. 4. The same patient after registration of the evoked potentials and brain stem reflexes. The EEG shows a declining burst suppression activity over the right hemisphere with development to the zero-line EEG

Discussion

Within the space of 4 years, we saw, in nine patients with clinical signs of brain death, an electrophysiological pattern which documented a loss of the cortical and brain stem functions investigated on the basis of acoustic, somatosensory, and visually evoked potentials as well as the electrically evoked blink and mechanically evoked glabella and masseter reflexes. On the other hand, the spontaneous EEG of these patients still revealed electroencephalographic activity. This constellation of findings caused us to question the value of evoked potentials and electrophysiologically investigated brain stem reflexes in the diagnosis of brain death: the loss of potentials and reflexes may document a loss of function in certain nuclear areas and of specific sensory and reflex tracts in the central nervous system. However, it does not prove cerebral death, if this is defined as a condition of electroencephalographic zero-line activity. Our findings indicate a dissociation between induced bioelectrical processes on the one hand and spontaneous electroencephalographic activity on the other hand. It is not difficult to explain this dissociation in primary infratentorial lesions. The absence of a visually evoked potential might also be interpreted on the basis of the conditions in the circulation, at least for patients

with basilar artery thrombosis. It is more difficult to explain the dissociation of the electrophysiological findings described in primary supratentorial lesions. For this purpose, it appears necessary to resort to the familiar neurophysiological investigations according to which diencephalic structures as pacemakers or oscillators of the EEG influence large cortical areas [1-3].

It is conceivable that impulses still emanate from the neuronally nonspecific thalamic system which is not precisely defined histologically [5], whereas the specific thalamocortical systems are already defunct. Here, further clinical and experimental studies are necessary in order to be able to interpret the findings.

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Multimodality Evoked Potentials in the Diagnosis of Brain Death

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When evoked potentials are used to determine brain death, primary supratentorial lesions must be distinguished from infratentorial lesions, as the order of the loss of different brain functions varies.

According to the guidelines for the determination of brain death by the Bundesärztekammer (BÄK) [6], documentation of the stepwise abolition of brain stem auditory evoked potentials (BAEP) in serial investigations in primary supratentorial lesions may be used as proof of the irreversibility of the loss of all brain functions after the clinical signs coma, cranial nerve areflexia, and apnea have been established [3-6]. As a minimum, wave III has to be preserved at a first investigation, as waves I and II are considered to be generated peripheral to the brain stem.

In 56 patients with coma grades I-III, we found only one patient who presented with the absence of auditory evoked potentials at the initial investigation. When he regained consciousness, it turned out that he had been deaf.

Patients and Methods

In ten patients serial recordings of somatosensory evoked potentials (SEP), visual evoked potentials (VEP), BAEP, and the 40-Hz AEP [1,2] during the development of brain death were possible. Of these patients, six had primary supratentorial lesions and four had primary infratentorial lesions.

Findings

For each lesion one illustrative case will be presented:

Supratentorial brain lesion: A 42-year-old female (1619/86) had a subarachnoid supratentorial hemorrhage. CT clearly demonstrated blood in supratentorial subarachnoid spaces. She was admitted with severe clouding of consciousness; SEP, VEP, and BAEP were reproducible. She lapsed into coma; 7 h later cranial nerve areflexia and apnea were recorded. Another 6 h later the abolition of SEP, VEP, and BAEP was documented (Fig. 1).

Infratentorial brain lesion: A 71-year-old female (1653/86) lapsed into coma from a posterior fossa hematoma she developed on anticoagulant therapy. While there was no reproducible SEP and BAEP, the VEP

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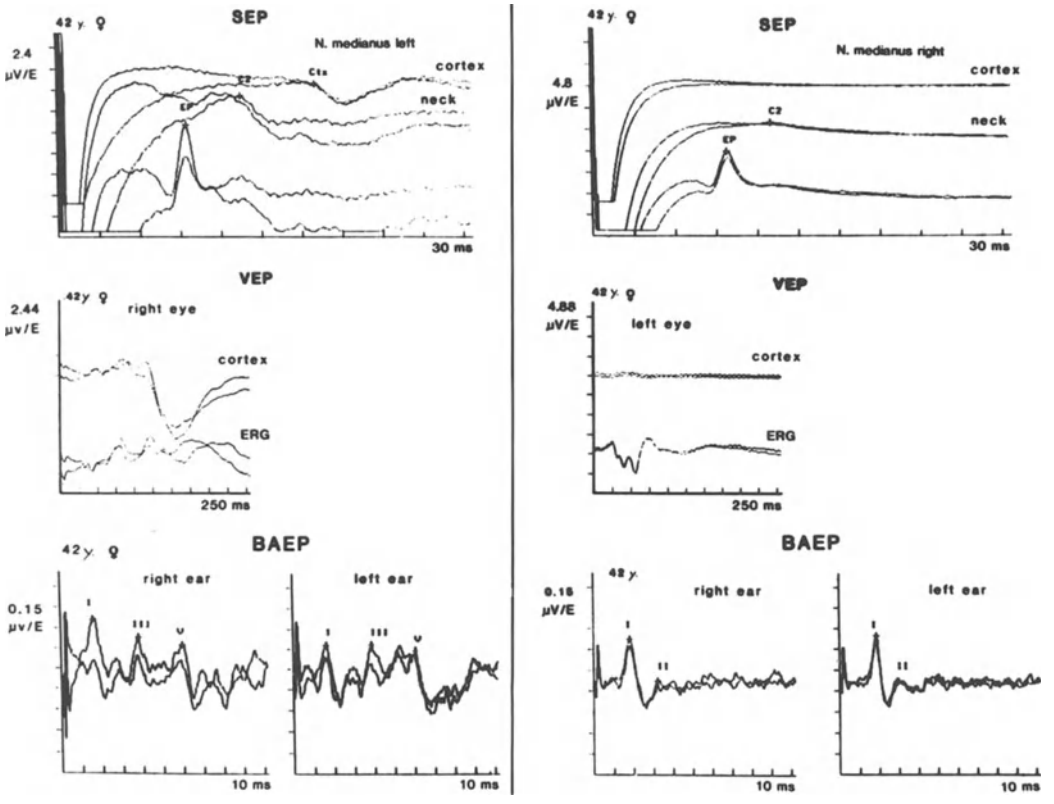


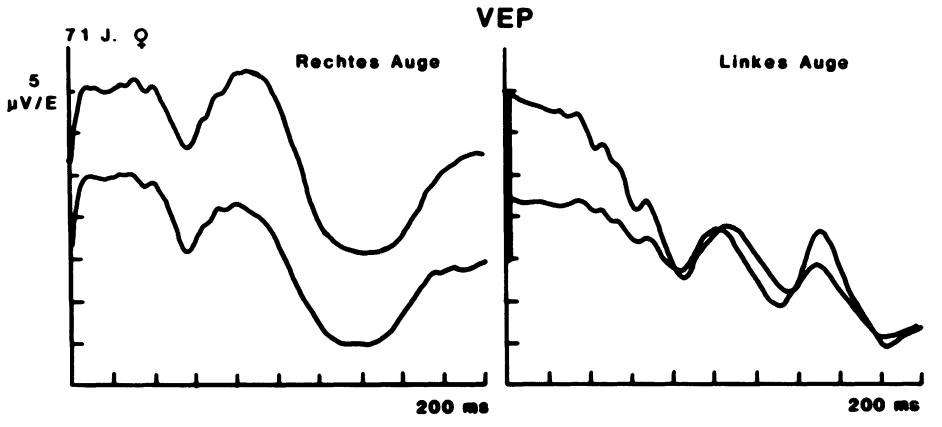
Fig. 1. Abolition of SEP, VEP, and BAEP in subarachnoid hemorrhage. (Preserved potentials on the left, abolished potentials on the right)

proved to be reproducible. Twelve hours after onset of coma, cranial nerve areflexia and apnea were found; EEG still revealed α-activity. Another 5 h later, the VEP was no longer reproducible (Fig. 2).

As defined by the BÄK [6], the irreversible loss of all brain functions is required for the determination of brain death. In infratentorial brain lesions, however, cortical function may outlast the loss of brain stem function. Thus, in infratentorial lesions, an EEG demonstrating electrocortical silence is always mandatory.

The 40-Hz AEP was found to coincide with the BAEP except for two cases. In the 42-year-old presented above, the second BAEP was abolished except for waves I and II. At the same time, the 40-Hz AEP was partly reproducible (Fig. 3).

In a 39-year-old female (47/87) with intracerebral hematoma and coma grade IV, a preserved wave I of the BAEP was found on one side. An abnormal but still reproducible 40-Hz AEP was found on the same side (Fig. 4).



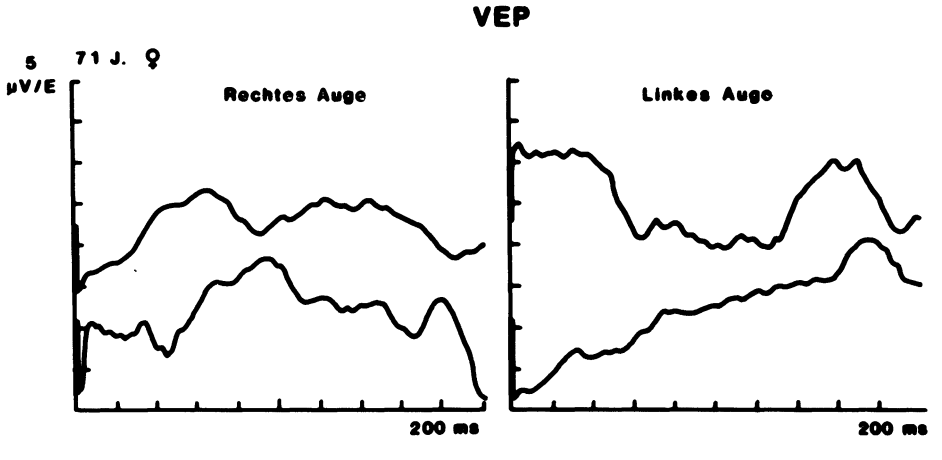


Fig. 2. Abolition of VEP in infratentorial hemorrhage (Rechtes Auge = right eye; Linkes Auge = left eye; J. = year)

Conclusions

Explanations for a partly preserved 40-Hz AEP along with abolition of wave III of the BAEP can only be speculative. More clinical experience is needed.

From our experience so far, abolition of the BAEP in serial investigations in supratentorial brain lesions after the documentation of

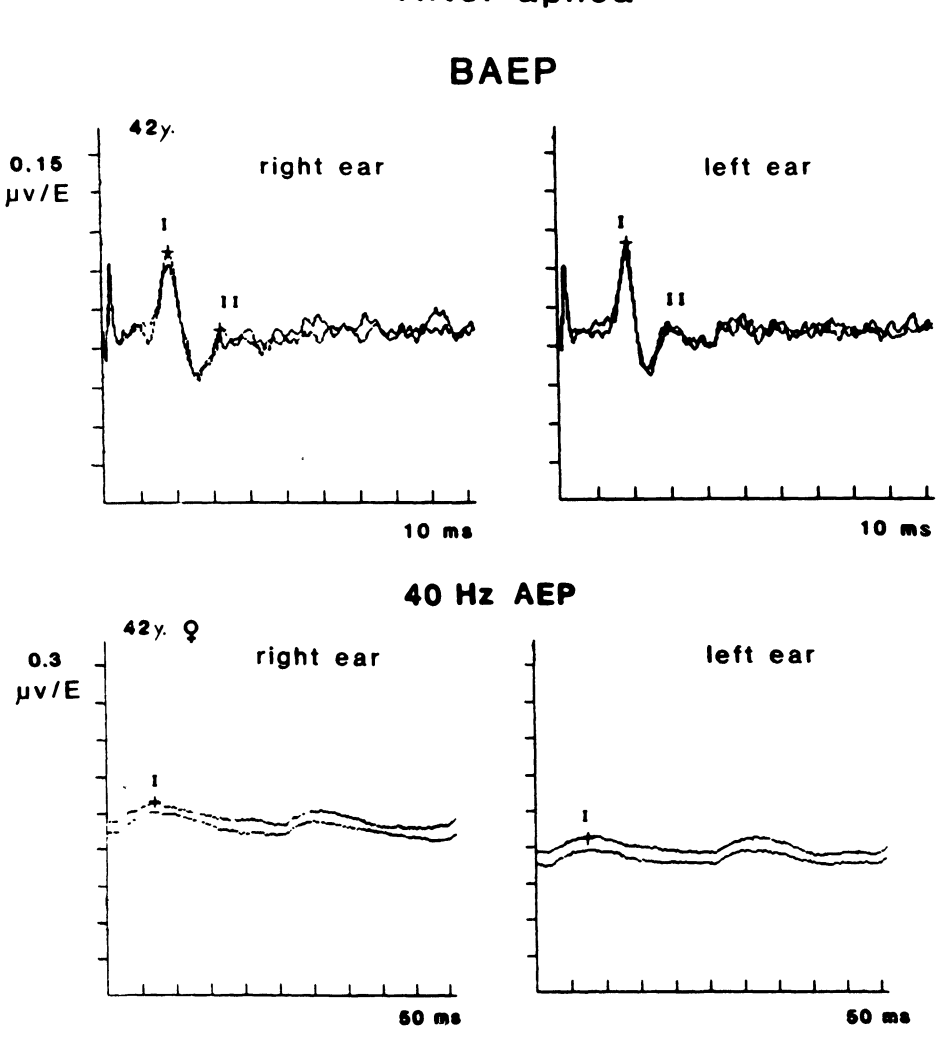


Fig. 3. Bilaterally preserved reproducibility of 40-Hz AEP with preserved wave II of BAEP

coma, cranial nerve areflexia, and apnea is sufficient to demonstrate the irreversible loss of brain functions.

Summary

Serial recordings of multimodality evoked potentials in ten patients demonstrated the stepwise abolition of all responses. The order of the loss of cortical and brain stem function, however, depends on the location of the primary brain lesion. Preserved cortical function in spite of coma, cranial nerve areflexia, and apnea after primary infratentorial lesions stresses the need for EEG recording of electrocortical silence. In two cases reproducible potentials of the

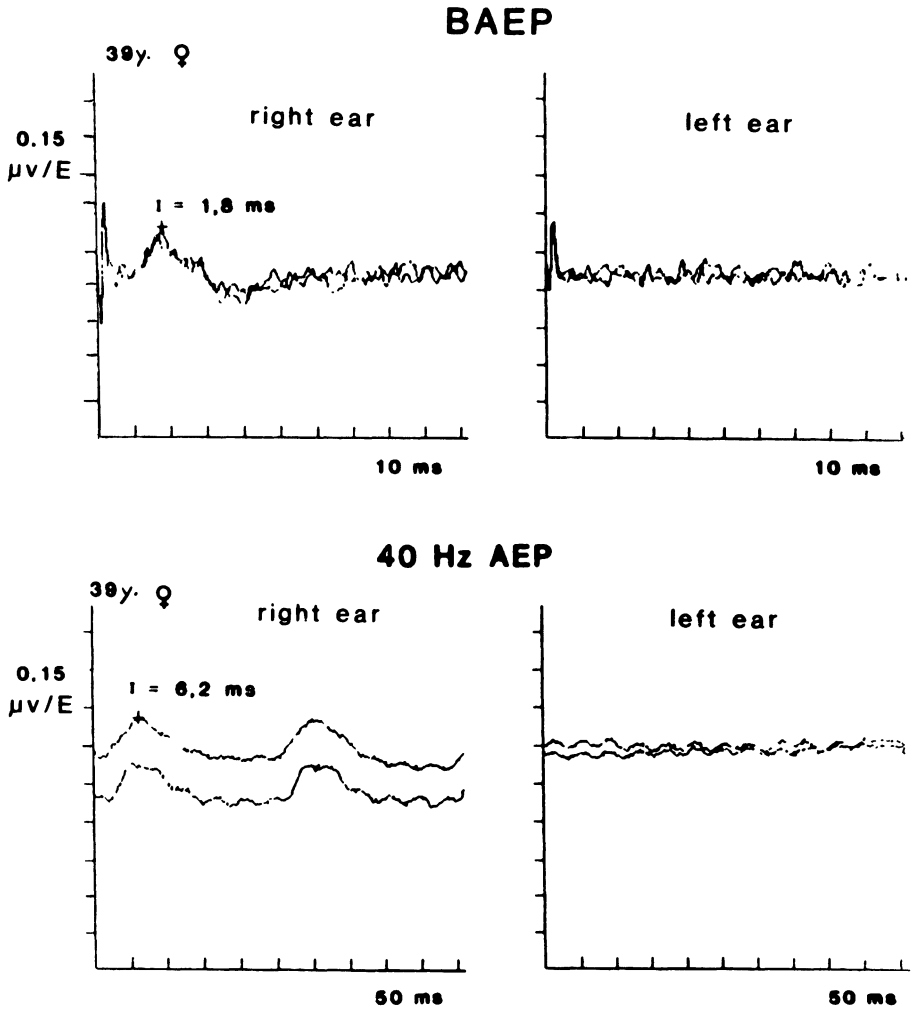


Fig. 4. Unilaterally preserved reproducibility of 40-Hz AEP with preserved wave I of BAEP on the same side

40-Hz AEP were found although the BAEP was preserved up to wave II only. It is concluded that more data on the 40-Hz AEP in brain death is desirable.

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The Value of Motor Potentials Following Transcortical Stimulation in Diagnosing Brain Death - First Results

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Introduction

Electrical stimulation of the motor cortex has until now been restricted by the necessity of opening the skull to apply the electrodes. The intact scalp and skull offers considerable resistance to the conventional methods of electrical stimulation. In 1980 MERTON and MORTON [6] introduced a technique to stimulate the motor cortex without any special preparation. The principle of this method is to apply a single very brief high voltage stimulus [7]. By means of this technique electrophysiological assessment of the corticospinal pyramidal tract is now possible, which was not the case using conventional methods of evoked potentials.

Patients and Methods

Twenty-eight comatose patients in the intensive care unit were studied by motor evoked potentials (MEPs) following cortical and spinal stimulation, by brain stem auditory evoked responses (BAERs), and by median nerve somatosensory evoked potentials (M-SSEPs). The clinical features of these patients are listed in Table 1. The classification of coma was based on WFNS coma scaling [1]. The diagnosis of brain death was made using the criteria of brain death published by the Federal Chamber of Physicians in 1982 [2]. Transcranial cortical stimulation was performed with a commercially available Digitimer D 180 Cortical Stimulator. This stimulation unit delivers a high voltage shock (max. 750 V) of short duration (time constant 50 μs). To stimulate the brain, the anode was placed over the hand area of the motor cortex, corresponding to positions F3/C3 and F4/C4 of the international 10-20 system. The cathode was applied 6 cm in front of

Table 1. Clinical features of patients

No.	Sex	Age	Diagnosis	
28	m 12	Median 45 yrs.	Head injury	12
	f 16	Range 8-67 yrs.	ICH	7
			SAH	4
			Others	5

ICH, intracerebral hematoma; SAH, subarachnoid hemorrhage

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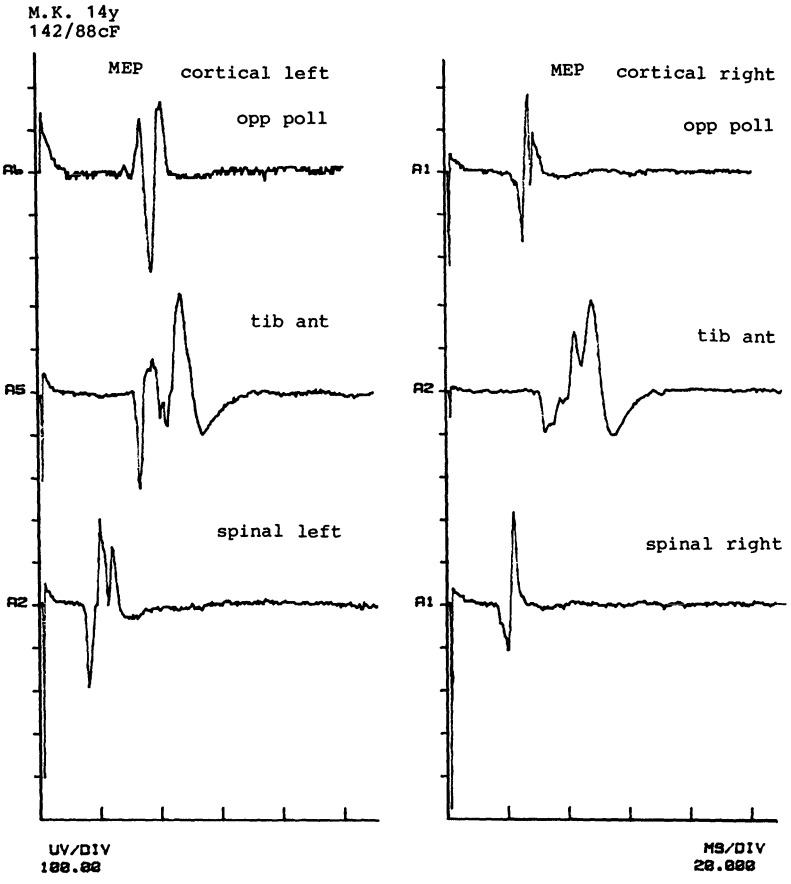

Fig. 1. Motor evoked potentials recorded from thenar muscles (opp poll) and anterior tibial muscles (tib ant) following cortical and spinal stimulation

the anode in the same axial plane. For spinal stimulation the anode was placed in the midline over the C6 spinal process and the cathode over the D2 spinal process. Recordings were made using silver needle electrodes in the contralateral thenar muscles. Signals were registered on the Compact Four (Nicolet) and stored on floppy discs. Filters were set at 30 Hz and 3 kHz and the time base chosen was 100 or 200 ms. Parameters of evoked potential recording are described elsewhere [8].

Results

A summary of the results is given in Tables 2 and 3. For spinal stimulation, stimulus intensity was between 30% and 70% of maximal stimulus intensity, and for cortical stimulation, 60%-100% of maximal stimulus intensity. Latency to onset of EMG response in thenar muscles was about 21 ms for cortical stimulation and 14 ms for spinal stimulation (Table 2).

Table 2. Latency to onset of EMG response in thenar muscles to spinal and cortical stimulation

	Latency				L-R difference	
	$\bar{x}$		S		$\bar{x}$	S
	L	R	L	R		
Cortical-thenar	21.6	21.2	1.6	1.7	0.9	0.8
Spinal-thenar	14.3	14.4	1.6	1.9	0.8	0.6
Central conduction time	7.5	7.0	1.2	1.6	0.7	0.8

Thirty-three recordings were made in 28 patients. In all cases a muscle action potential was registered following spinal stimulation. No muscle action potential was evoked in 19 of 33 recordings following cortical stimulation. In one case MEP was missed on one side. Seven of 19 recordings with absent cortical MEPs were made in patients classified as coma grades I-III. All other recordings with absent cortical MEPs were in patients classified as coma grade IV or belonging to the group of "brain dead" who showed deep coma, absent brain stem reflexes, and apnea. The three patients classified as coma grade IV showed absent cortical MEPs. In two of these patients BAERs were preserved on both sides and M-SSEP was absent bilaterally. In the third patient only M-SSEP was found to be intact on both sides, while BAERs were missing bilaterally.

All patients with deep coma, absent brain stem reflexes and apnea (classified as brain dead) showed absence of cortical MEPs and BAERs as well as disappearance of the primary cortical responses N20/P25 of M-SSEP. MEPs following spinal stimulation were preserved in all cases.

Discussion

Motor evoked potentials can also be recorded in patients in a comatose state. In awake patients cortical stimulation of higher intensity can produce some discomfort. All patients studied here received some sedative drugs in respect of mechanical ventilation. No vegetative reactions, e.g., increase in heart rate or arterial blood pressure, were observed by continuous monitoring following spinal or cortical stimulation. No seizures or other concomitant reactions were noticed during our procedure.

Sedative drugs given continuously in medium dosage (alfentanil 1.5 mg/h and midazolam 9 mg/h) apparently do not influence recording of spinal and cortical MEPs. Whether there is some interference with high dosage sedatives or barbiturates is beyond the scope of our experience. Two patients received high dosage barbiturates because of raised intracranial pressure. Both showed normal muscle action potentials following spinal stimulation but absent cortical MEPs. We are

Table 3. Results of brain stem auditory evoked responses (BAERs), median nerve somatosensory evoked potentials (M-SSEPs), and motor evoked potentials (MEPs)

Patient groups, WFNS coma scale	No. of patients = recordings	BAERs Left/right + +	BAERs + 0 +	BAERs 0 0	M-SSEPs Left/right + +	M-SSEPs + 0 +	M-SSEPs 0 0	MEPs Left/right + +	MEPs + 0 +	MEPs 0 0
Coma I	6 = 7	◆◆◆	◆	◆◆◆	◆◆◆ ◆◆	◆◆		◆◆◆ ◆◆◆		◆
Coma II	8 = 10	◆◆◆ ◆◆◆ ◆		◆◆	◆◆◆ ◆◆◆ ◆	◆◆◆		◆◆◆ ◆◆	◆	◆◆◆ ◆
Coma III	4 = 4	◆◆	◆	◆	◆◆◆		◆	◆◆		◆◆
Coma IV	3 = 3	◆◆		◆	◆		◆◆			◆◆◆
"Brain dead"	7 = 9			◆◆◆ ◆◆◆ ◆◆◆			◆◆◆ ◆◆◆ ◆◆◆			◆◆◆ ◆◆◆ ◆◆◆

+, correct evoked potential; 0, evoked potential lost

not sure whether the absence of cortical MEPs is to be attributed to the barbiturate effect or to cortical or brain stem lesions.

The latencies following cortical and spinal stimulation do not differ significantly from those of awake patients described in the literature [4,5]. In coma grades II and III half of the patients showed absent cortical MEPs. BAERs and M-SSEPs seem to be more stable in the deep coma state. This finding is confirmed by the recordings of patients in coma grade IV.

Loss of cortical and brain stem function (patient group "brain dead") is in all cases linked with absent cortical MEPs, as well as loss of BAERs and primary cortical responses of M-SSEP.

The advantages of cortical MEPs are simple handling, few problems with artifacts, and time sparing in relation to the evoked potential technique. A single stimulus evokes a muscle action potential, and averaging methods are unnecessary. In relation to BAERs and M-SSEPs, cortical MEPs seem to be less stable in the deep coma state. In the course of brain stem dysfunction due to transtentorial herniation, cortical MEPs seem to be affected earlier than BAERs and M-SSEPs. The influence of drugs (sedatives in high dosage, barbiturates) has not yet been clearly defined. More experience is necessary to evaluate the role of cortical MEP recording in diagnosing brain death. Further possibilities may emerge in the future using transcranial magnetic stimulation.

Summary

In 28 patients of the neurosurgical intensive care unit motor evoked potentials (MEPs) were studied in different coma stages and in brain death. The first results showed that MEPs seem to be less stable than BAERs and SSEPs. All patients with deep coma, loss of brain stem reflexes, and apnea showed absent cortical MEPs, BAERs, and SSEPs.

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Motor-Evoked Potentials and Transcranial Doppler Sonography During Development of Cerebral Circulatory Arrest

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Introduction

Personal experience with monitoring of motor-evoked potentials (MEPs) [4] in patients suffering increased intracranial pressure has shown the high stability of motor responses, which are sometimes recordable even up to clinical brain death. Since with transcranial Doppler sonography (TCD) a noninvasive assessment of intracranial hemodynamics is available [1], it was the aim of our study to correlate MEP and TCD findings and to define the degree of cerebral blood flow necessary for elicitability of motor responses. Therefore, we compared the results of both tests in 46 patients suffering increased intracranial pressure and report on our findings.

Patients and Methods

Forty-six patients (24 males, 22 females) from 17 to 89 years (average age 41 years) were studied. All were comatose, intubated, and artificially ventilated and finally died from increased intracranial pressure caused by a severe closed head injury (n = 34), intracranial hemorrhage (n = 8), or subarachnoid hemorrhage (n = 4). The observation time ranged from 1 to 21 days.

Motor evoked potentials were elicited by transcranial electrical stimulation of the motor hand area. Stimulus strength was increased until distinct electromyographic (EMG) responses from the contralateral thenar muscles were recordable or the absence of any response could be documented despite a maximum stimulus strength of 750 V. In each case the lower cervical region at C6-7 was also stimulated to make sure of an intact peripheral pathway. TCD examinations were performed transtemporally on the internal carotid and the proximal middle cerebral arteries on both sides.

The MEP findings were divided into three categories. Type I means well-preserved responses following transcranial stimulation. In type II amplitudes are noticeably diminished to below 200 μV, and in type III they are absent on both sides despite a maximum stimulus strength of 750 V, while they are still preserved following lower cervical stimulation (Fig. 1).

The TCD findings were also divided into three categories. Type I means reduced diastolic flow velocities. In type II diastolic flow is absent on both sides, and in type III only oscillating flow or systolic peaks are present or the signals are completely abolished (Fig. 2).

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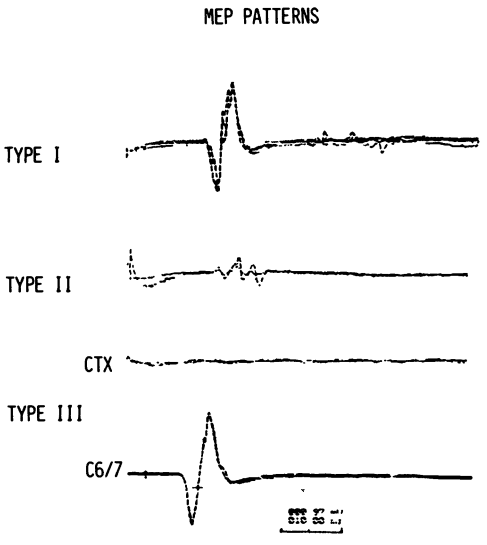

Fig. 1. MEP patterns. Recordings of three patients are shown. Type I: bilaterally preserved EMG responses. Type II: bilaterally noticeably diminished MEPs. Type III: bilateral loss of EMG responses following transcranial (CTX) stimulation; bilaterally preserved responses following lower cervical (C6/7) stimulation

Results

Table 1 shows the combination of MEP and TCD results. In 46 patients 85 recordings were performed. Twenty-one patients (75.0%) with reduced diastolic flow velocities (TCD type I) showed distinct EMG responses (MEP type I), while amplitudes were diminished in five (17.9%) and completely absent in two (7.1%) cases. With absent diastolic flow (TCD type II), MEP amplitudes were noticeably reduced (MEP type II) in 17 cases (53.1%), but distinct in six (18.9%) and absent in nine (28.1%).

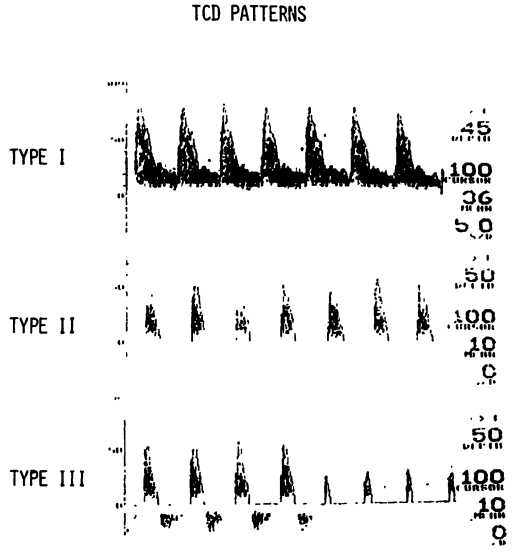

Fig. 2. TCD patterns. Recordings of three patients are shown. Type I: reduced diastolic flow velocities. Type II: absent diastolic flow. Type III: oscillating flow, systolic peaks or absent signals

Table 1. Combination of MEP and TCD Results

TCD / MEP	I		II		III		Total	
	No.	%	No.	%	No.	%	No.	%
I	21	75.0	6	18.9	1	4.0	28	32.9
II	5	17.9	17	53.1	-	-	22	25.9
III	2	7.1	9	28.1	24	96.0	35	41.2
Total	28	100.0	32	100.0	25	100.0	85	100.0

EMG responses were absent in all but one patient with oscillating flow, systolic peaks, or absent TCD signals. Figure 3 shows a typical example of MEP and TCD findings during development of intracranial circulatory arrest.

Discussion

Our results show a close relationship between MEPs and TCD findings during development of intracranial circulatory arrest. Usually distinct EMG responses could be recorded in cases with reduced diastolic flow velocities. Typically, MEP amplitudes decreased with absent diastolic flow and were abolished in all but one case with oscillating flow, systolic peaks, or absent TCD signals. Thus, for elicitability of MEPs only a minimum cerebral blood flow seems to be necessary. This might also be an explanation for other experience we had which did not reveal any prognostic significance of preserved MEPs in comatose patients.

The patient with preserved MEPs despite oscillating flow seems to be unusual. This concerned a 27-year-old man who was comatose due to a subarachnoid and intracerebral hemorrhage. MEPs and TCD were examined 4 days after clipping of a middle cerebral artery aneurysm when the patient showed clinical signs of brain death. It is possible that in this case due to skull defects (although the bone flap had been reimplanted) foci of high charge density occurred [2,3] which allowed the impulse to overcome damaged supratentorial structures, thus exciting deeper structures such as the brain stem.

To conclude, excitability of MEPs elicited by transcranial electrical stimulation is given even when there is a minimum cerebral blood flow. In our opinion, these findings are only valid in supratentorial and secondary brain stem lesions. In primary brain stem lesions, other results would have to be expected, such as reduced or abolished motor responses, while supratentorial TCD findings would not be noticeably influenced.

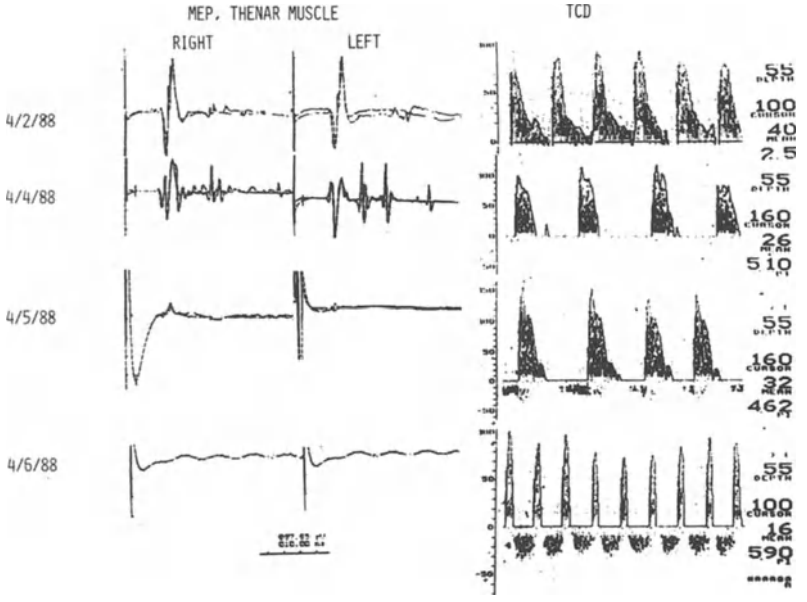

Fig. 3. MEPs and TCD in a single patient with a severe closed head injury. Recordings on 4/2/88 show MEP and TCD types I. With increased intracranial pressure (4/4/88) diastolic flow is absent (TCD type II), coinciding with MEP type II. With beginning of oscillating flow (4/5/88) there is only a minimum EMG response which is completely absent (MEP type III) with appearance of clear oscillating flow (TCD type III) on 4/6/88

Summary

In a total of 46 patients suffering under increased intracranial pressure, motor-evoked potentials (MEPs) and transcranial Doppler sonography (TCD) were examined to consecutively monitor functional and hemodynamic changes during development of cerebral circulatory arrest. It was the aim of our study to correlate MEP and TCD findings and to define the degree of cerebral circulation necessary for elicitability of motor responses. With regard to three MEP and three TCD types, we found that usually distinct motor responses could be obtained with reduced diastolic flow velocities (type I). Typically, MEP amplitudes decreased noticeably with absent diastolic flow (type II) and were abolished in all but one case with the appearance of oscillating flow (type III). To conclude, under transcranial electrical stimulation, descending pathways may still be excitable with minimal cerebral blood flow when recovery of the neurological condition is no longer possible.

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Neuropathology of Brain Death in Relation to Continuously Measured Intracranial Pressure

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Introduction

The brain death syndrome results from increase of the intracranial pressure (ICP), which causes an irreversible arrest of the cerebral perfusion. However, ICP values continuously obtained with the hitherto existing methods in relation to the blood pressure (BP) partially seem to contradict this pathogenesis. Thus, RICHARD et al. [1] observed in 21 patients three patterns of the terminal ICP/BP relation, each of which occurred in one-third of cases:

Type I : ICP > systolic BP
Type II : ICP > diastolic BP
Type III : ICP < diastolic BP

Especially in the cases of type III measurements, the explanation of the intracranial circulation arrest is difficult. The question arises as to whether the neuropathology can make a contribution to the explanation. Can the circulation stop in the type III cases be confirmed by postmortem examination, and are peculiarities observable hinting at various effects of a lower ICP?

Material and Results

From our neuropathological file of more than 300 brain death cases we reexamined all the cases of exclusively supratentorial space-occupying lesions with continuously measured ICP in the period of transition to brain death and with sufficient neuropathological documentation. "Sufficient" means the presence of histological sections from the boundary of the intra- and extracranial circulation, at least from the intracanalicular part of the nervi optici and from the hypophysis (sometimes supplemented by the upper cervical cord), because the changes in other parts of the cerebral total infarction are unspecific [2].

We present 16 cases (Table 1). They are nearly equally distributed among the three ICP/BP types. Because morphological changes at the border zones are first demonstrable 8 h after the onset of brain death, as we have shown previously [2], the number of evaluable cases is reduced to 14. Cases of type I and II are combined because of their small numbers. Within the so formed two groups the use of intraventricular or epidural measurement was nearly equal.

Sure morphological signs of intracranial circulation arrest are **demarcations** with myelin palor in the canalicular part of the nervus

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Table 1. Neuropathologically examined cases with brain death syndrome (BD) and continuous measurement of ICP. V, ventricular fluid pressure; E, epidural pressure

I	4 , with BD period of > 8 h	2	7	2V, 4E, 1V+E
II	5	5		
III	7	7		3V, 4E
	14	14		

opticus and in the upper cervical cord at the boundary of the intracranial blood supply. Another such demarcation arises within the anterior lobe of hypophysis by necrosis. In both types of ICP/BP relation these demarcations were demonstrable in all cases (Table 2). Another change occurring exclusively in cases of brain death is shearing of cerebellum at the edge of the foramen magnum with **displacement of cerebellar particles** into the spinal subarachnoid space. This was also shown in two of the seven cases in each group (Table 2). Since the frequency of this phenomenon increases with time, we show in the table the periods of brain death, which also correspond in both groups.

Other morphological changes occur independent of the development of the brain death syndrome and are signs of the terminal herniation. **Pontine hemorrhages** result from shearing of dorsal parts against basal fixed parts by axial mass shifting. In their frequencies in the two ICP/BP groups we might expect a reflection of the different development of ICP, but as shown in Table 3, the difference was only small and not significant. Sometimes a **cortical hyperemia** is conspicuous not only on the side of the lesion but also contralaterally. This results from final cerebral vasoparalysis with blood engorgement. This was found in both groups nearly equally as well (Table 3).

Conclusion

In all cases of type III with subdiastolic ICP values the clinical diagnosis of brain death could be confirmed by neuropathological proof of typical demarcations at the border zone of the intracranial circulation. Other findings were also similar to those in group I/II with relatively higher ICP values. From the neuropathological point of view we can accept the same pathogenetic mechanisms in both groups, which result in the arrest of the intracranial circulation.

Table 2. Changes proving the intracranial circulation arrest. BD, brain death

BD period		Demarcation	Spinal cerebellum
I/II	13-30 h	7/7	2/7
III	10-27 h	7/7	2/7

Table 3. Changes associated with terminal herniation

	Pontine hemorrhage	Cortical hyperemia
I/II	4/7	4/7
III	2/7	3/7

It follows that the ICP values obtained with present neurosurgical practice do not yield in all cases measurements which are usable for the calculation of the cerebral perfusion pressure. Therefore, at present the ICP values cannot be accepted as an additional sure diagnostic criterion of brain death.

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The Use of Transcranial Doppler Ultrasound Monitoring in the Determination of Brain Death

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The exact diagnosis of cerebral death has two objectives. In the first place there are considerations about whether or not to continue treatment; secondly, and of increasing importance in recent years, the question of removal of organs for transplantation is raised.

Current methods of establishing a diagnosis are:

1. **Clinical and neurological examination,** indicating complete loss of cerebral function: absent pupillary responses, no reaction to painful stimuli, absence of reflexes, including brain stem reflexes, lack of response to caloric stimulation, and absence of spontaneous breathing even during hypercapnia (apnea test).

2. **Electroencephalography,** demonstrating an isoelectric pattern. As a bedside method of investigation this is an elaborate procedure and therefore time consuming. In patients under barbiturate therapy the results are not fully reliable. There is often some form of electrical interference, especially when recordings with increased sensitivity are made in an intensive care unit. Recently recordings of acoustic evoked potentials have also been used for this purpose.

3. Investigations of cerebral circulation:

a) **Four-vessel angiography** [4,8]. This investigation calls for patient transportation and is also time consuming. The administration of contrast agents to patients who are potential organ donors is a disadvantage.
b) **Sequential isotope scanning** [7]. The applicability of this method of investigation depends on the availability of an isotope, so that it cannot be performed everywhere. It would also require a mobile gamma-camera.
c) **Extracranial ultrasound Doppler sonography** [3,9]. This investigation is not completely reliable. Patients have been described in whom a brief reversal of flow was detected, with later recovery of circulation. Duplex scanning, however, has been shown to be a reliable method. It allows precise investigation and differentiation of circulation in internal and external carotid arteries.
d) **Transcranial ultrasound Doppler sonography** (TCD) [1,2,5]. This method of investigation allows measurement of direction of flow, pattern of flow, and flow velocity in intracranial vessels. The technique has been developed by Aaslid and co-workers and is available in most neurosurgical units, usually as a routine method of monitoring vasospasm in patients with subarachnoid hemorrhage. The method has been routinely applied in our clinic since 1985. A disadvantage of this procedure is the possible interference of the signal caused by

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other electrical appliances present. In a small number of patients it is impossible to detect a transtemporal signal.

In 1984 AASLID, and later RINGELSTEIN, MOSKOPP, and RIES, demonstrated the typical flow pattern in circulatory arrest caused by brain death [1,2,6]. The "to-and-fro" movement described by Yoneda et al. in extracranial Doppler sonography [9] was also detected by TCD.

It is questionable whether TCD is reliable in establishing cerebral death. In the literature there is evidence that in experienced hands the presence of an oscillating flow correlates with cerebral death and an isoelectric EEG in all cases [6]. Brain death may, however, be present without a fluctuation in the flow pattern [6]. This indicates that only the presence of such a flow pattern ascertains the diagnosis of cerebral death.

In patients with a severe neurological condition or suspected brain death TCD should be performed as soon as possible. CT may demonstrate the presence of a midline shift and thus facilitate the investigation. A complete investigation with TCD takes only a few minutes. In the case of an improvement of the clinical situation further investigations are not necessary. In the event of a deterioration it can be easily repeated. Progressive reduction of diastolic flow velocity and clinical deterioration necessitate repeated TCD investigations. On demonstration of an oscillating pattern preparations for removal of donor organs may be made. In such cases we believe that recording of an EEG is unnecessary. A proposal for the diagnostic management is summarized in Fig. 1.

The usefulness of the method was studied in 17 consecutive adult patients. In eight cases clinically established brain death was the result of a subarachnoid hemorrhage or intracerebral hematoma, in eight cases there was a severe head trauma, and in one patient there was deterioration following the removal of a large meningioma. In three patients, all with clinical signs of brain death, it was not possible to record a signal. TCD could not be performed earlier in these patients, so the absence of the signal allowed no conclusions on TCD alone. In all patients a recent CT scan was available. In our

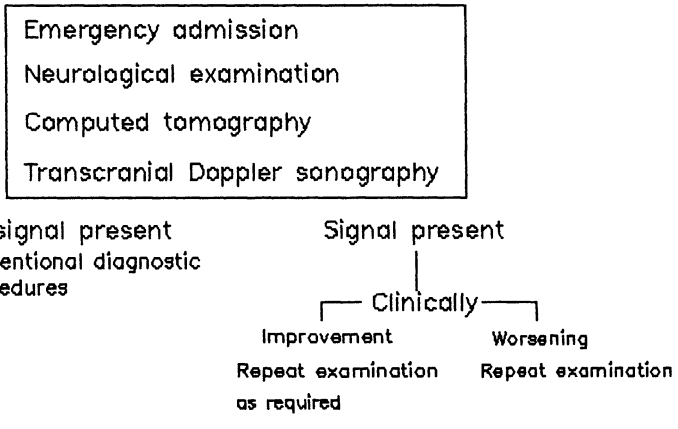

Fig. 1. Flowchart of management scheme in patient with suspect cerebral death

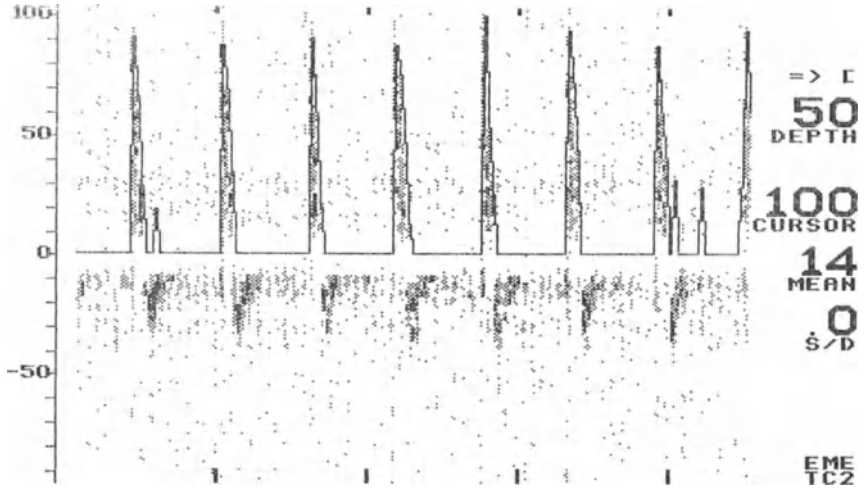

Fig. 2. Typical "to-and-fro" movement signal in clinically brain dead patient

experiences the findings on CT are not essential for the accurate recording of TCD. The experiences of the investigator seems to be far more important.

In the 14 patients in whom it was possible to demonstrate a transtemporal Doppler signal, we found two types of signal:

- an oscillating signal as mentioned above (Fig. 2)
- a signal showing only a systolic peak without inversion of the diastolic signal

In all cases the patients were cerebrally dead following clinical criteria (apnea test, caloric stimulation, no brain stem reflexes, no reaction to painful stimuli). In ten patients an EEG was recorded within 1 1/2 h, showing a flat curve in each case.

In summary our study has led to the following conclusions:

1. Our results, and those reported in the literature, show that TCD is a reliable method in the investigation of cerebral death.
2. The diagnosis of cerebral death may only be established in the presence of an oscillating flow or the absence of continuous diastolic flow providing the signal is distinct.
3. In our experience the transorbital signal at a depth of 60-70 mm is very variable and difficult to validate. An oscillating signal has only been detected in two patients, so transorbital TCD seems to be unreliable.
4. Measurements of flow in the basilar artery in ventilated patients are very elaborate. As they do not contribute any useful information they are superfluous.
5. In the case of legal circumstances requiring EEG recording, TCD can aid in predicting the moment the EEG will yield an isoelectric pattern. Thus a single recording will suffice.
6. TCD is not influenced by drugs such as barbiturates.
7. TCD in experienced hands is an instant method of investigation that can be repeated at any time and as often as necessary.

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Comparative Evaluation of Angiography and Transcranial Doppler Sonography in the Determination of Intracranial Circulatory Arrest

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Introduction

Although brain death is diagnosed on the basis of well known clinical neurological signs of permanent loss of cerebral functions [6], angiography is often used to additionally determine the intracranial circulatory arrest (ICCA), particularly when an organ explantation is discussed. However, this invasive method may be harmful to the brain so that it should not be used to determine ICCA in a patient who does not completely fulfil the clinical criteria of brain death, e.g., because of the suspicion of intoxication. Furthermore, it requires that an angiographic suite is available permanently, it is expensive, and it may be time consuming. The need for a simple bedside procedure with at least the same reliability to diagnose ICCA is evident.

Transcranial Doppler sonography (TCD) has been used for a variety of clinical conditions [1,4,5,8] and because of its approach to the basal arteries it has been acclaimed to be more accurate for the study of cerebral circulatory pattern than is conventional extracranial Doppler sonography. In order to elucidate the value of TCD to determine ICCA, a prospective study was undertaken using TCD in addition to clinical judgment of comatose patients in the intensive care unit. Additionally, a retrospective analysis of angiograms of patients who had undergone cerebral angiography over the last 4 years to determine ICCA was performed to determine the "flow pattern" in this particular group of patients.

Material and Methods

Between April 1984 and 1988, 341 patients died in our department or were examined by staff members with the assumed diagnosis of brain death. Forty of them underwent cerebral angiography in order to determine ICCA; 31 of them could be reevaluated retrospectively. Since TCD had been introduced into our department and become a routine method, it was used in 27 comatose patients for a total of 43 examinations to determine ICCA.

Angiography was performed with intraarterial contrast injection into the aortic arch in combination with digital subtraction imaging in the vast majority of patients. Only in one patient studied early in the series was contrast medium injected selectively into the extracranial arteries, in combination with conventional angiography. In ten patients, contrast was injected into the vena cava superior through the antecubital vein.

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Transcranial Doppler sonographic studies were performed using the TC 2-64B from EME (Überlingen, FRG), which is a pulsed Doppler system with frequencies of 2, 4, and 8 MHz. This machine allows for evaluation of both the major extra- and intracranial arteries. A complete study was attempt in every case, including the carotid and vertebral arteries in the neck, the middle and anterior cerebral arteries (MCA and ACA respectively) through the temporal window, the ophthalmic arteries (OA) through the orbita, and the basilar artery (BA) via the foramen magnum.

In seven patients, TCD studies prompted cerebral angiography within such a short time interval that the results of the two studies could be compared.

Results

Angiography

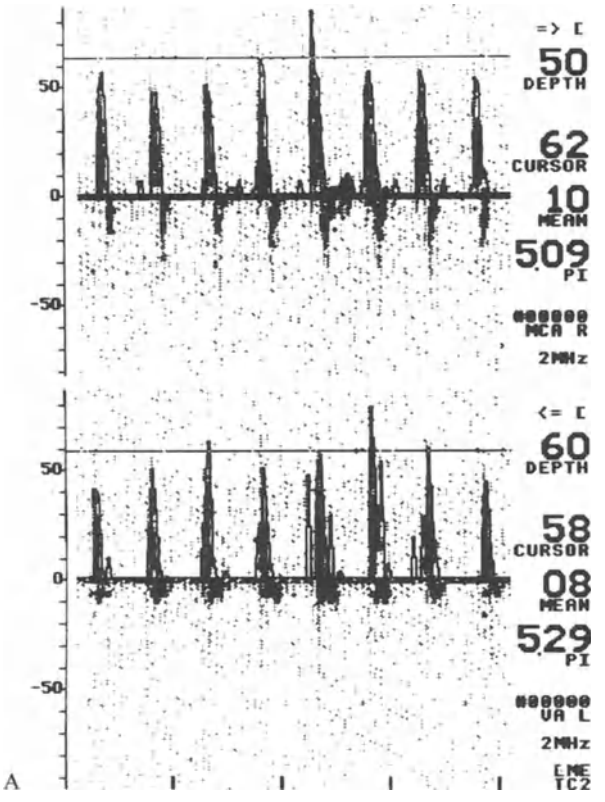
Angiography showed contrast medium in all carotid bifurcations, and extension of the contrast column up to the skull base in two-thirds. The carotid siphon was contrasted in one-fifth, and the MCAs were shown in 1/31. In the posterior circulation, the vertebral arteries were stained in three-quarters, the BA was shown in 19%, and the posterior cerebral arteries were shown in 16%. The flow in the stained intracranial arteries was so slow that ICCA could be assumed.

Transcranial Doppler Sonography

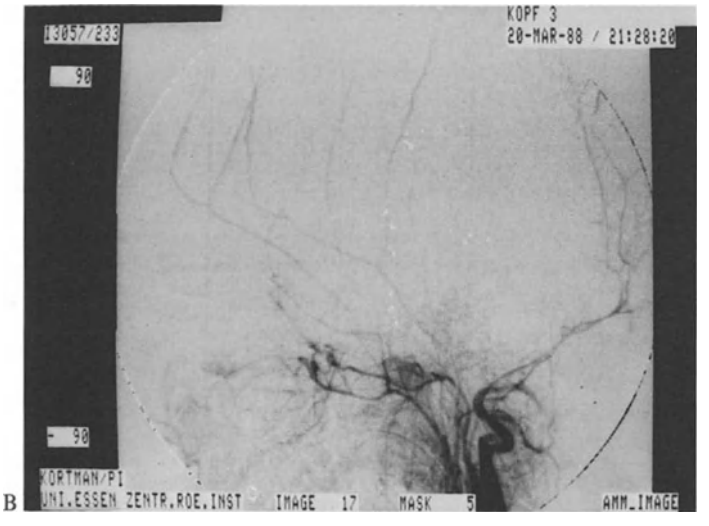
In normal patients, TCD demonstrates a flow pattern in the intracranial arteries which is characterized by a systolic peak and a slow return of velocity to the diastolic valley. The ratio between peak systolic and lowest diastolic velocities can be calculated to give either the ratio itself, the "pulsatility index" (PI) as defined by GOSLING [3] as the difference between systolic and diastolic flow velocity divided by mean velocity or the "Pourcelot index" [7], which both represent the vascular resistance. Because better ways to characterize the flow curves are currently lacking, these figures are used to characterize the flow pattern as intracranial as opposed to extracranial. The TC 2-64B's built-in computer analyzes the flow curves and computes and displays automatically the PI.

In the comatose patients reported in this study, the flow pattern changed in such a fashion that a higher and sharper systolic peak could be observed, followed by a rapid flow reduction at the end of systole, and in the diastole sometimes another sharp hill developed. In some patients, the diastolic flow was reversed, resulting in an undulating flow pattern. In late stages prior to central loss of circulatory regulation and cardiac arrest, either a flow pattern as shown in Fig. 1 was observed (sharp systolic peaks are followed in all insonated arteries by zero-flow in diastole or a minor negative flow), or the MCA/ACA complex could not be insonated at all; this was the case in one-half of the patients. If the MCA/ACA complex could not be demonstrated at all through the temporal window, the transorbital route used to insonate the carotid siphon and the ACA showed the flow pattern just described. Similar flow was demonstrated in the BA, although in a few patients at this stage the BA still had an undulating flow pattern.

Under the assumption that, in addition to a systolic flow, a diastolic flow is essential to maintain an intracranial circulation, the



A



B

Fig. 1 A, B. One-year-old child clinically diagnosed as brain dead following blunt head injury. **A** TCD shows sharp systolic peaks followed by narrow negative diastolic flow. **B** Angiography confirms the ICCA

absence of diastolic flow, that is the pure presence of sharp systolic peaks, was used as the criterion for ICCA. Forty-three TCD observations were performed in 27 patients; in 21 observations, clinical diagnosis of brain death was followed by ICD findings which corresponded to ICCA. In three cases with a clinical diagnosis of brain death, the TCD findings were suggestive of ICCA but did not comple-

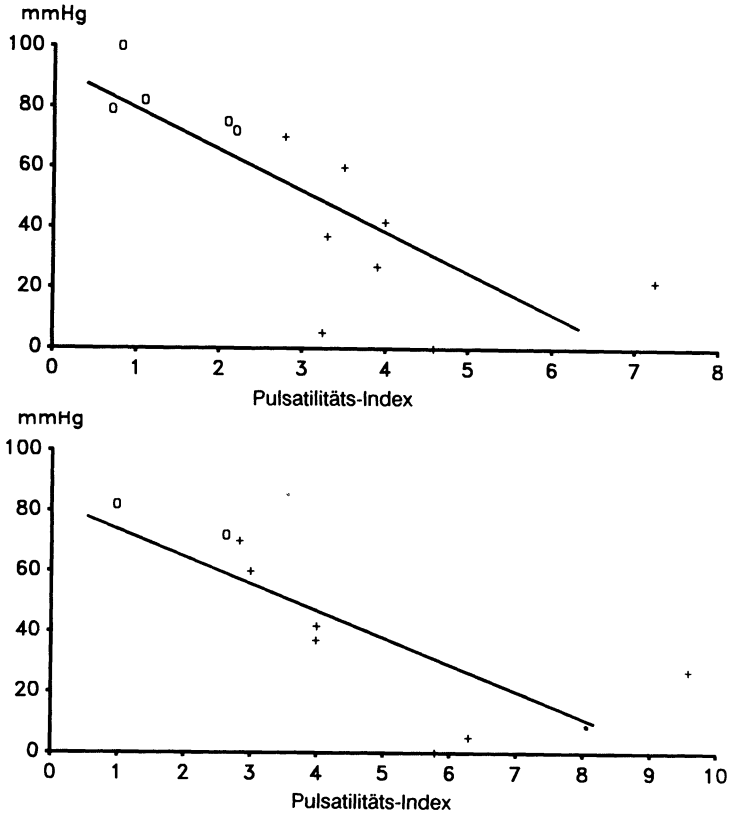


Fig. 2 a, b. The relationship between perfusion pressure (mmHg) and pulsatility index (a) for supratentorial arteries and (b) for infratentorial arteries. 0 represents a comatose patient while + represents a clinically brain-dead patient (Pulsatilitäts Index = pulsatility index)

tely fulfil the above-described criteria. In 19 cases, TCD showed flow patterns suggestive of some intracranial circulation.

In order to test further the reliability of TCD in the diagnosis of ICCA, the curves of all patients were evaluated by using the PI. The curves obtained in brain-dead patients were opposed to those obtained in comatose patients who did not fulfil criteria of brain death. The mean PI derived from flow measurements in supratentorial arteries in 16 brain-dead patients was 4.211 ± 2.26, while in 12 comatose patients it was 1.587 ± 0.85. This difference is highly significant ($P < 0.005$). Also, in seven patients clinically diagnosed as brain dead, it was possible to compare TCD findings with angiography performed shortly after TCD evaluation. In six patients, TCD showed the typical flow pattern demonstrated in Fig. 1 A, and angiography proved the absence of intracranial circulation (Fig. 1 B). In one patient, TCD was suggestive of some residual flow, but angiography showed ICCA.

Additional information obtained from the TCD studies could become helpful in the management of comatose patients: Because we observed a high PI in patients in the state of brain death or shortly prior to this state, we arranged the data in a graphic presentation against the perfusion pressure, which was computed according to the formula of BUR-

TON [2], taking into account the intracranial pressure. Figure 2 clearly shows a trend for high PI to correspond to low perfusion pressure. The comatose patients are represented on the left part of the abscissa, and the border between the PI for the living and the dead is between 2 and 3. All patients with PI values within this range determined in all intracranial arteries died within the next 24 h.

Discussion

In view of the results reported here, TCD seems to be most helpful and accurate in the noninvasive diagnosis of ICCA. With careful evaluation of intracranial arteries, it was possible to diagnose ICCA using TCD, and TCD was correct in all cases. However, the experience is limited and has to be confirmed in a large-scale study before it should be generalized to add legally useful information to our present handling of deeply comatose patients. Some issues should be discussed.

1. TCD study is a noninvasive study that can be easily performed and repeated in a bedside manner. However, because technical mistakes can be made by the examiner, it requires an experienced examiner to give reliable results. In order to exclude the possibility of mistakenly diagnosing the noninsonability of an intracranial artery and deducing an ICCA, the same observer should perform repeat studies on a patient with as many arteries insonated as possible. This will certainly not always be possible, e.g., when a patient is referred for organ explantation from elsewhere already in the state of brain death and ICCA; in such instances particular attention should be given to the possibility of technical errors. Insonation of OA down to the carotid siphon and of the BA should usually be helpful to rule out misdiagnosis.
2. As with all other examinations of ICCA, blood pressure should be within the normal range. It is possible to demonstrate, by means of TCD, the effect of stepwise cardiac arrest on the intracranial circulation, but this is only the very final stage which is of no major clinical interest.
3. Using the formula mentioned above, TCD should become able to estimate noninvasively the intracranial pressure as it affects the flow pattern in the intracranial arteries.

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Hemodynamics of Cerebral Circulatory Arrest: Correlation Between Perfusion Pressure and Blood Flow Velocity

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Introduction

Since 1986, transcranial Doppler ultrasonography (TCD) has been used in our department to assess cerebral blood flow velocities (FV) in patients developing intracranial circulatory arrest due to intracranial hypertension. This method offered new insights into cerebral hemodynamics under conditions of raised intracranial pressure (ICP) as it allows repeated bedside recordings which are noninvasive and easy to interpret. Application of TCD and normal values have been reported in the literature [1,3,4,6,7]. The following questions arise: How do TCD spectra, which represent arterial FV in basal cerebral arteries, change with rising ICP? What is the influence of systemic arterial pressure (SAP)? Can perfusion be improved by dehydration or induced systemic hypertension?

Clinical Material and Method

Thirty patients were studied who suffered from and finally died of intracranial hypertension with developing intracranial circulatory arrest due to severe head trauma in the majority of cases. ICP was measured using unilateral Gaeltec probes (Gaeltec Ltd. Dunvegan, Isle of Skye, Scotland) inserted over the frontal convexity, usually on the right side. SAP was registered continuously through radial artery catheters. Patients showing no TCD signal upon the initial investigation were excluded. Partial arterial CO_2 values measured 30-35 mmHg during the TCD evaluation. SAP and ICP were documented continuously on computer-generated printouts, so that the interrelation between FV in basal cerebral arteries (TCD) and cerebral perfusion pressure (CPP = SAP - ICP) could be studied.

Results (Figs. 1-4)

Correlation Between TCD and CPP

Normally, the pulsatility amplitude of FV in basal cerebral arteries is about 0.5, i.e., end-diastolic FV is 50% of the systolic peak value. A consistent relationship was found between CPP and the shape of the FV spectrum outline in severe intracranial hypertension. With rising ICP, the diastolic FV decreases while systolic peak FV remains unchanged. This results in a pronounced systolic-diastolic pulsatility (high resistance flow). Diastolic FV becomes zero when diastolic ICP reaches diastolic SAP. A further decline of CPP leads to the

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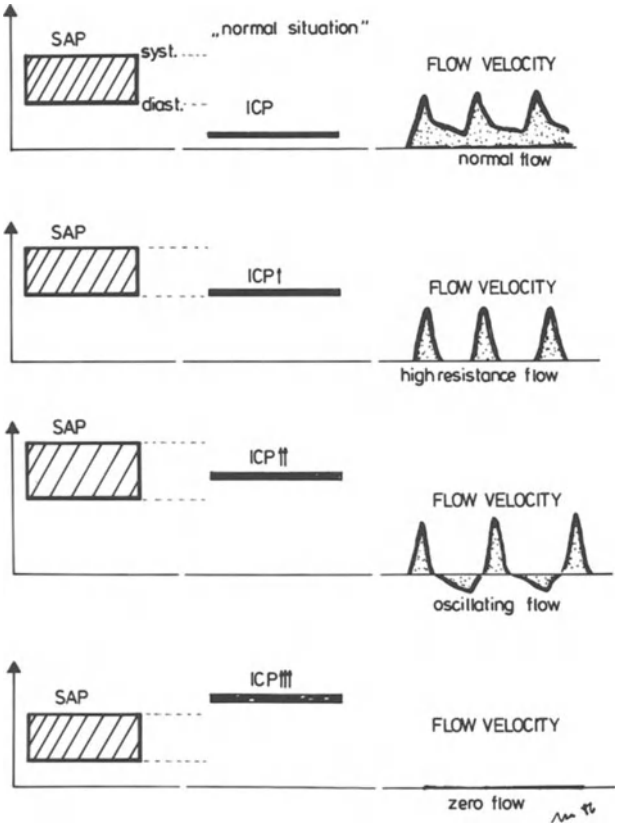

Fig. 1. Relationship between SAP, ICP, and FV as measured in the middle cerebral artery by TCD. In all displayed TCD spectra, upward deflections indicate blood flow toward the Doppler probe, whereas downward deflections appear with flow directions away from the probe

occurrence of diastolic backflow (biphasic flow). When the time averaged mean forward flow equals the backflow, the pattern is called oscillating. Oscillating TCD profiles have been shown to correctly predict angiographic nonfilling of the anterior intracranial circulation [5]. Following the oscillating pattern, a profile with characteristic small systolic spikes develops (Fig. 2), which then leads over to zero flow. These patterns also correspond to angiographic circulatory arrest [5].

In cases of circumscribed mass lesions causing intracranial hypertension, the existence of different pressure compartments within the skull can be demonstrated by TCD, e.g., showing circulatory arrest on one side with less severe disturbance on the other.

Effect of Therapy for Intracranial Hypertension

Dehydration (mannitol) proved effective provided the TCD spectra had not yet become oscillating. Beneficial effects can be demonstrated by an increase or recurrence of diastolic forward FV upon TCD which corresponded to ICP improvement. In cerebral circulatory arrest, however, dehydration and induced systemic hypertension were ineffective as they did not change time averaged inflow to the brain.

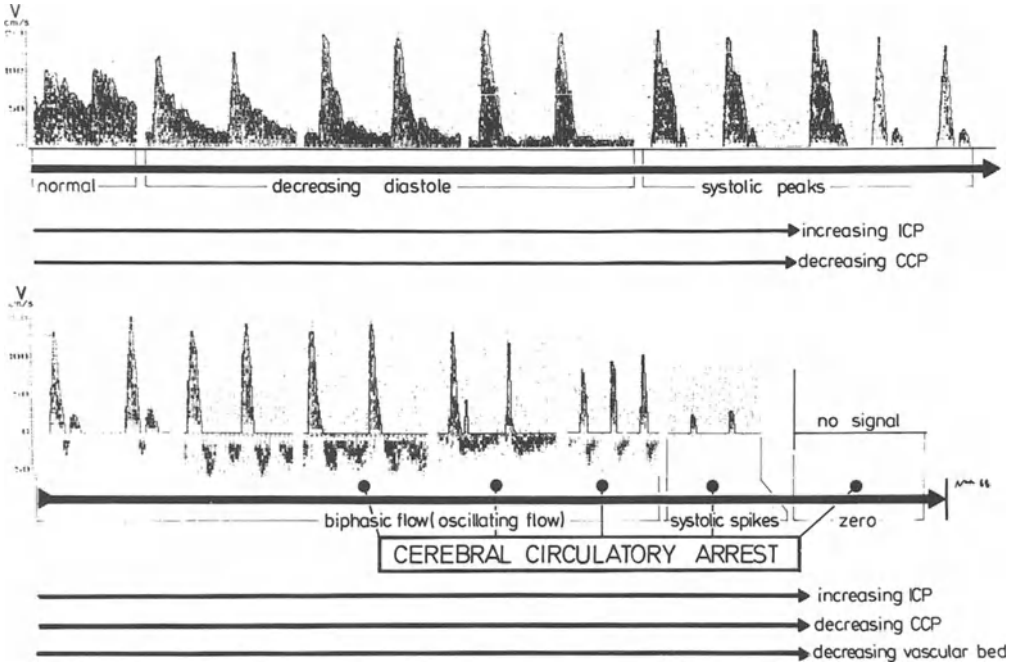

Fig. 2. Blood flow velocities as measured in the middle cerebral artery under rising ICP up until cerebral circulatory arrest as proven by angiography. Three different TCD patterns correlate with angiographic nonfilling: oscillating flow, small systolic spike flow, and zero flow (provided that there has been a signal before)

Discussion

As supposed by AASLID et al. [2], TCD can be used to assess the severity of intracranial hypertension.

Blood inflow to the skull cavity is determined by CPP and cerebrovascular resistance (CVR). As severe intracranial hypertension decreases CPP and increases CVR, blood inflow becomes reduced. The instantaneous CPP varies over the cardiac cycle, being lowest at the end of diastole. Diastolic CPP will therefore be the first to become zero in progressing intracranial hypertension. This point corresponds to the occurrence of diastolic zero FV upon TCD. With further ICP increases (or SAP decreases), diastolic CPP becomes negative, being accompanied by a diastolic backflow on TCD. When time-averaged systolic forward FV equals time-averaged diastolic reverse FV (= net zero FV, the oscillating TCD pattern), circulation in the peripheral vascular tree of the evaluated vessel has come to a standstill, as was demonstrated by intracranial angiographical nonfilling [5]. Despite extracranial stop of dye flow in these cases, the basal cerebral arteries remain patent since oscillating TCD patterns are obtainable. The site of distal outflow obstruction within this "blind duct," which must be caused by vascular collapse, remains unclear.

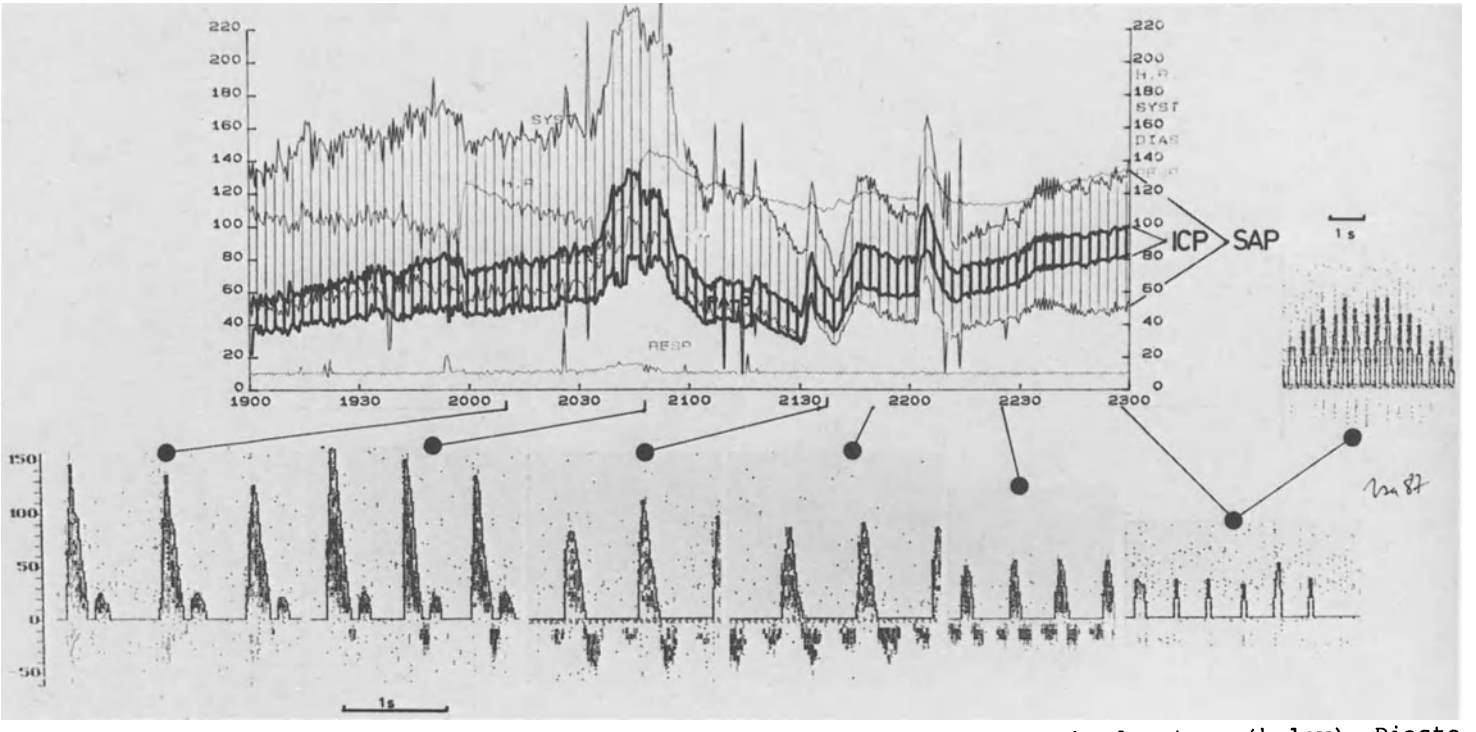

Fig. 3. Correlation between SAP, ICP, and FV as measured in the middle cerebral artery (below). Diastolic backflow (downward deflections) occurs when diastolic ICP surpasses diastolic SAP. Following oscillating flow patterns, small systolic spike flow occurs, showing respirator-dependent amplitude fluctuations

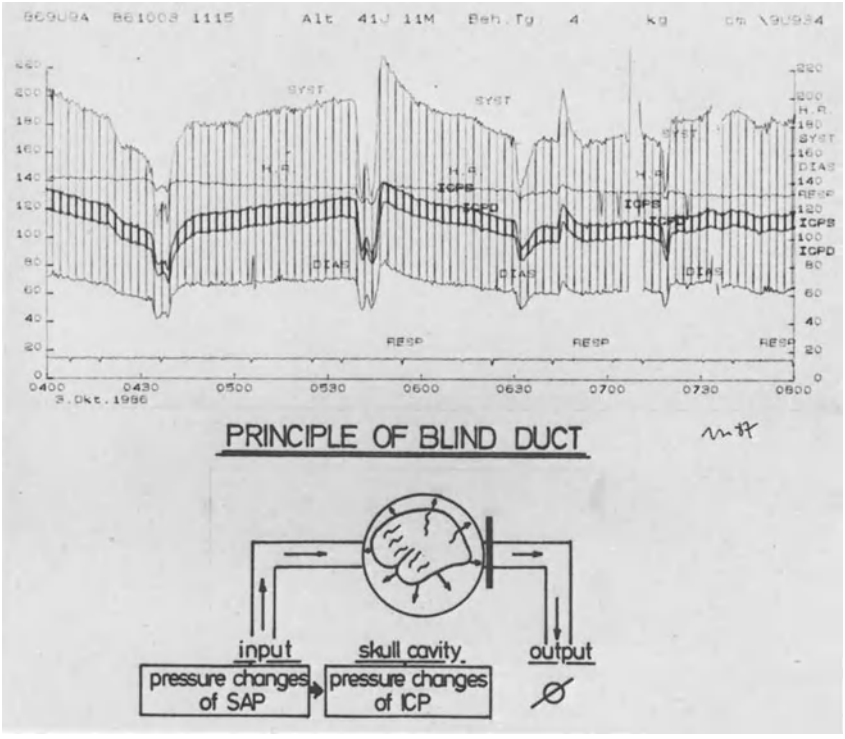

Fig. 4. Failure of systemic arterial hypertension to reestablish cerebral perfusion in a patient with cerebral circulatory arrest following head trauma and severe brain swelling. The cerebral vascular tree has become a "blind duct" with distal outflow obstruction of unknown location

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Comparison of Transcranial Doppler Sonography and Cerebral Angiography for the Diagnosis of Cerebral Circulatory Arrest

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Introduction

Cervical Doppler examinations on clinically brain dead patients have been performed since the mid-1970s. BÜDINGEN et al. [4] described the typical oscillating "compliance flow" patterns in the carotid and vertebral arteries which were explained by back-and-forth movements of the blood column in the presence of an intracranial circulatory stop. Today, transcranial Doppler sonography allows the insonation of basal cerebral arteries [1,2] which are directly affected by rising intracranial pressure [6,8]. The method might therefore be of diagnostic relevance for cerebral circulatory arrest and can be compared with the widely accepted method of cerebral angiography [3].

Clinical Material and Methods

Fifty-five patients were studied who suffered from and finally died of intracranial hypertension with developing intracranial circulatory arrest due to severe head injury and intracerebral hemorrhage in the majority of cases. Angiography was performed after clinical diagnosis of brain death and after transcranial Doppler sonographic prediction of cerebral circulatory arrest. Four-vessel angiography was performed with 15 ml of contrast medium which was injected by machine into the common carotid arteries or cervical segments of vertebral arteries. The last angiographic images were taken 26 s after contrast medium injection, and 14 shots were taken for each vessel.

Transcranial Doppler sonographic examination was performed before and after angiography through the temporal basal window with the depth set at about 55-60 mm for carotid arteries. The foramen magnum window was used to insonate the basilar and vertebral arteries at a depth of 70-90 mm. If no signal was obtained, transorbital and retromastoid insonation was also performed.

Results

Correlation of Internal Carotid Artery Filling to the Transcranial Doppler Sonographic Signal

One hundred and ten internal carotid arteries (ICA) from 55 patients were investigated (Table 1).

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Table 1. Correlation between cerebral angiography and transcranial Doppler sonography in the internal carotid artery (ICA)

Angiographic findings (n = 110)		Transcranial Doppler findings		
Filling of ICA up to:	Number of cases	No signal	Systolic spikes	Oscillating flow
1. Bifurcation, petrosus bone	57	52	4	1
2. Petrosus bone	23	6	15	2
3. Siphon	12	4	8	0
4. Siphon + ophthalmic art.	8	0	1	7
5. Minor filling of intracranial vessels after 14 s	10	0	0	10

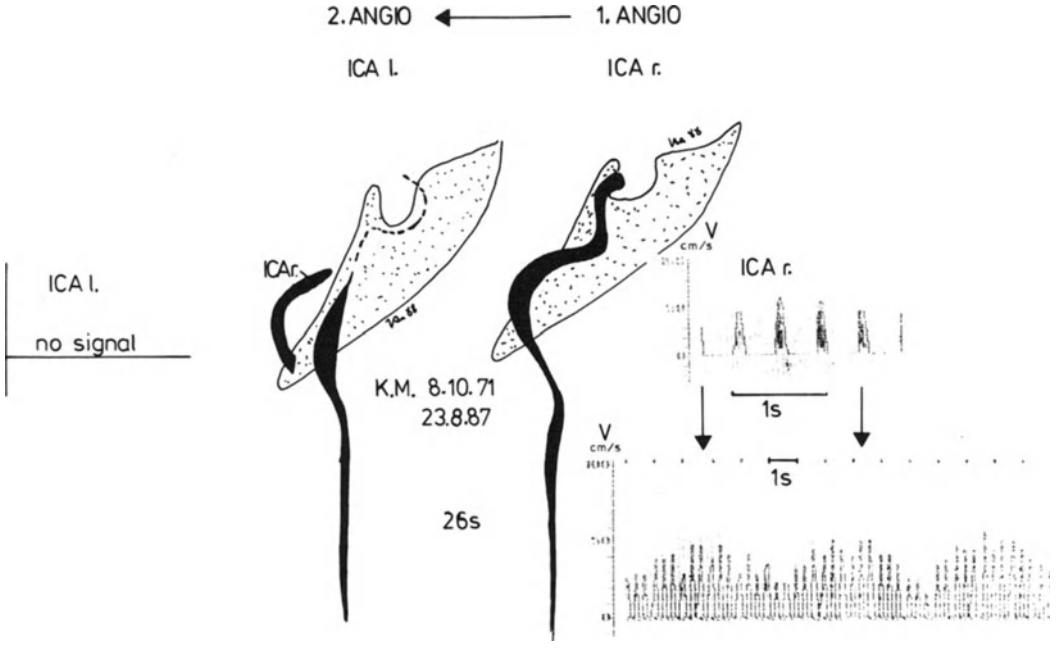

Fig. 1. Different levels of filling of ICA in cerebral circulatory arrest and transcranial Doppler sonographic findings. On the right side, the right ICA fills up to the siphon. Transcranially, systolic spikes can be recorded. These systolic spikes show a typical respiration-dependent time course when the signals are recorded at a very slow velocity. On the left side, the left ICA breaks at the level of the petrosus bone. No signal can be obtained transcranially

In 57 cases, the contrast medium in the ICA stopped at the level between the carotid bifurcation and the base of the skull. In most of these cases (52), no transcranial Doppler sonographic signal was obtained. In four cases, small systolic spikes were recorded. The systolic spike spectrum is composed of sharp and narrow peaks during systole, with a maximum flow velocity of 100 cm/s. No diastolic flow was recordable. These systolic spikes show a typical respiration-dependent wave (Fig. 1). Oscillating flow (compliance flow) was seen in only one case: the oscillating flow spectrum is defined as forward flow during systole and backflow during diastole. Cerebral circulatory arrest is reached when time-averaged mean forward flow equals the backflow.

In 23 cases, the contrast medium in the ICA stopped at the level of the petrosus bone. The majority of cases showed systolic spikes (15); no signals were obtained in six cases and in two patients, oscillating flow patterns were recorded.

In 12 cases, the contrast medium in the ICA stopped at the level of the siphon (Fig. 2). In the majority of cases (8), systolic spikes were recorded, while in four cases no signal was obtained.

In eight cases, the contrast medium in the ICA stopped at the end of the siphon with filling of the ophthalmic artery. In this angiographic situation, in most cases oscillating flow patterns were recorded (7); in one case systolic spikes were recorded.

In ten cases minor filling of thin intracranial vessels (MCA, ACA) occurred after 14 s. Cerebral veins were not seen after 26 s. Oscillating flow patterns were obtained in all of the ten cases.

Correlation of Vertebral and Basilar Artery Filling to the Transcranial Doppler Sonographic Signal

Fifty-five basilar arteries were investigated with Doppler sonography (Table 2). In some cases, retrograde flow in the contralateral vertebral artery occurred due to filling of the homolateral vertebral artery without filling of the PICA of the cerebellum.

In two cases, the contrast medium stopped in the vertebral artery at the level of the cervical vertebral column. Transcranially, no flow velocity in the vertebral artery or basilar artery was obtained.

In 42 cases, the vertebral artery flow was stopped at the level of the atlas, only filling muscular branches of the neck. Here, in most cases (41), no signal was obtained in the basilar arteries or vertebral arteries; small systolic spikes were recorded in only one case.

In five cases, angiography showed scarce filling of a thin basilar artery up to the clivus and dorsum sellae. In this condition, systolic spikes were recorded three times and oscillating flow patterns twice.

In six cases, minor filling of posterior cerebral arteries occurred after 14 s. Cerebral veins could not be seen after 26 s. Four of these cases showed oscillating flow patterns, while two showed systolic spikes.

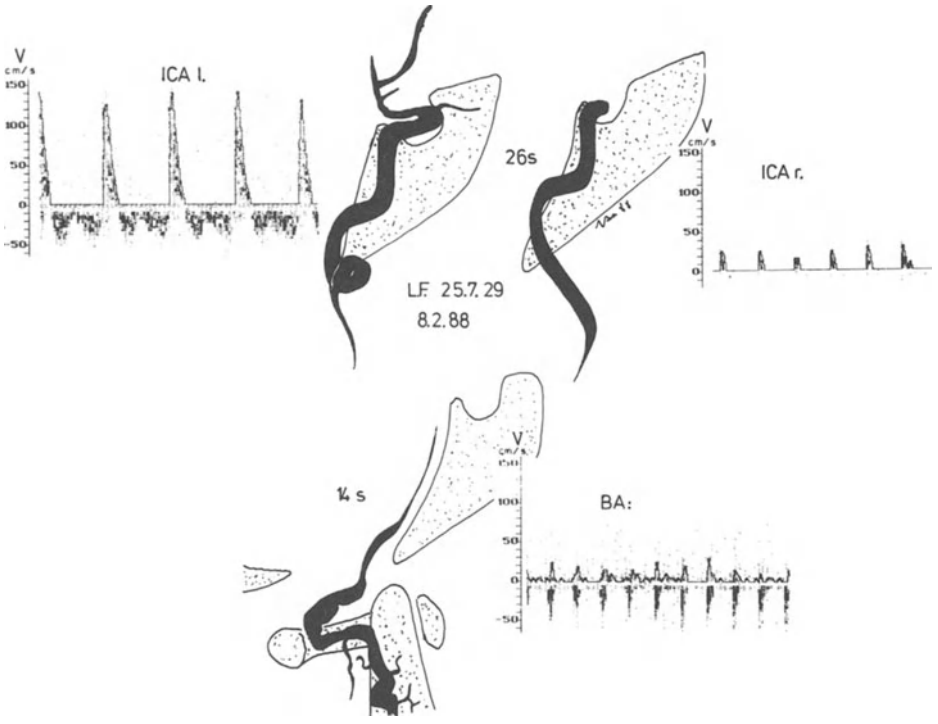

Fig. 2. Brain-dead patient with different angiographic filling of ICA. The right ICA stops at the level of the siphon with typical systolic peak flow. The left ICA shows minor intracranial filling of the proximal MCA after 26 s. Transcranial Doppler sonographic recordings show oscillating flow patterns. The basilar artery (BA) fills up to the middle part of the clivus. After 14 s, only systolic peaks can be recorded (the systolic peaks point downward because the flow velocity is away from the Doppler probe)

Table 2. Correlation between cerebral angiography and transcranial Doppler sonography in the vertebral basilar system

Angiography (n = 55)		Transcranial Doppler findings (basilar artery)		
Filling of basilar artery/ vertebral artery up to:	Number of cases	No signal	Systolic spikes	Oscillating flow
1. Cervical vertebral column, C2-C6	2	2	-	-
2. Atlas (vertebral artery)	42	41	1	-
3. Clivus-dorsum sellae (basilar artery)	5	-	3	2
4. Minor filling of posterior cerebral artery after 14 s	6	-	2	4

Discussion

The transcranial Doppler sonographic signal of flow velocities in basal cerebral arteries is influenced by cerebral perfusion pressure, cerebral vascular resistance, and the amount of open vascular beds [6]. This means that during developing cerebral circulatory arrest, the level of intracranial pressure and amount of vascular bed volume influence the Doppler flow velocity spectrum the most.

Cerebral circulatory stop and an open vascular bed can be found during subarachnoid hemorrhage [5,6]. In this condition, very pronounced oscillating flow signals can be recorded, which means that the moved blood volume is large and therefore produces a good signal [7]. Conditions with slowly increasing intracranial pressure up to cerebral circulatory arrest lead to a slowly diminishing size of the vascular bed. In this condition, the volume of oscillating blood is decreased, so that the Doppler sonographic signal becomes weaker. Therefore, the transformation of pronounced oscillating flow patterns to systolic spikes and furthermore to no obtainable signal is a sign that the vascular bed is shrinking. Accordingly, no signal can be obtained when the carotid artery filling stops at the level of the carotid bifurcation (Fig. 3).

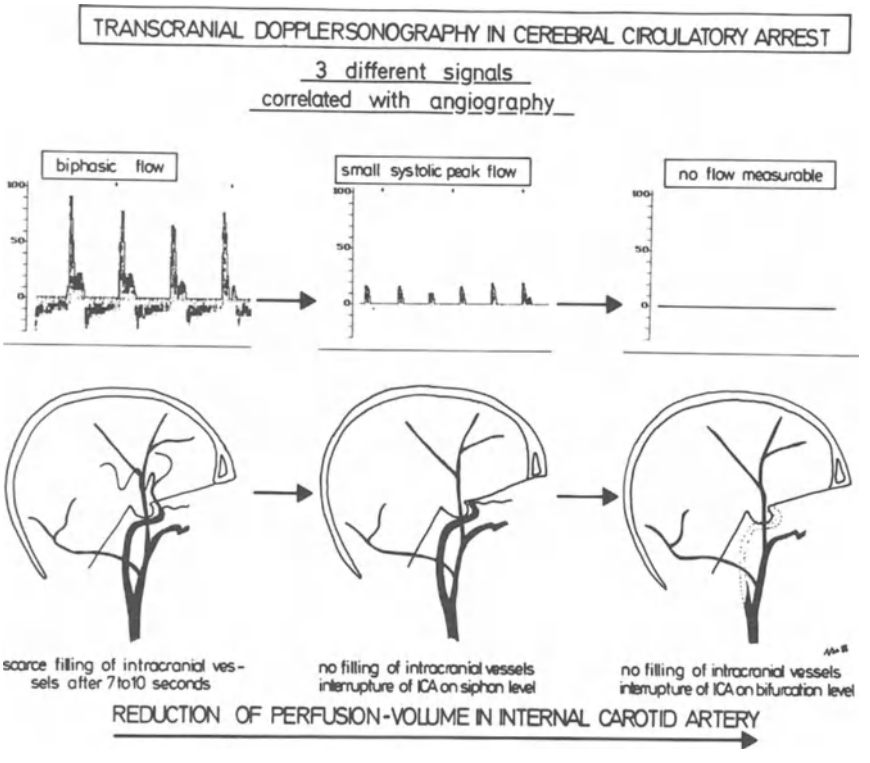

Fig. 3. Schematic drawing of angiographic and Doppler sonographic findings with reduction of intravascular bed. Doppler signals show a typical time course. Minor filling of intracranial arteries is accompanied by oscillating flow patterns. Filling up to the siphon shows systolic peaks, and contrast medium stop at the level of the carotid bifurcation shows no signal recording

Comparison with angiography shows that transcranial Doppler sonography is a very good and simple method to determine cerebral circulatory arrest. The method is accurate if certain parameters are observed:

- Time course investigation with typical Doppler spectra
- Typical Doppler signals: systolic spikes and oscillating flow patterns
- Absence of signal recording can only be accepted if it is preceded by a typical time course of Doppler spectra
- An experienced investigator

The method is less accurate if no signal is recorded, and if there is no time course investigation (normally in 4% no signal can be obtained because the skull bone is too thick) [1,2]. Air in the skull cavity leads to a total reflection of ultrasonic beams, so that no signal can be observed. Swelling and intracerebral bleeding may displace intracranial vessels, so that recording may be difficult or impossible. The method is also inaccurate if the investigator is inexperienced.

When all these factors are taken into consideration, transcranial Doppler sonography can replace angiography. Angiography should only be performed in situations which are unclear.

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Electrographic Changes in Brain-Dead Patients

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Introduction

Cardiac rhythm in brain-dead patients has been studied under artificial ventilation [3]. However, few studies have been done on cardiac arrhythmias after disconnection of the respirator, since cessation of cardiac function or electrocardiographic silence in brain-dead patients occurs spontaneously or after disconnection without any further monitoring. The aim of this study was to analyze the influence of total anoxia on cardiac function as well as on the mode and course of cardiac arrest.

Methods and Results

In 35 brain-dead patients of a neurosurgical unit, ECGs were examined before and after removal of the ventilator. In all 35 cases, criteria of brain death included coma, loss of spontaneous respiration, electrocerebral silence, and cessation of circulatory arrest in the brain documented by arteriography [4].

The causes of brain death were intracranial expansive mass lesions, severe hemorrhages or other unfavorable courses of well-established cerebral diseases, or trauma. Drug intoxication was excluded.

ECGs were recorded until circulatory arrest, and the terminal changes analyzed (Table 1). In some patients, peripheral arterial as well as central venous pressures were recorded. Blood samples were taken for evaluation of blood gas analysis, serum electrolytes, and myocardial enzymes. Cardiac output was determined. Catecholamines were not studied (Table 2).

Before disconnection of the respirator, a sinus rhythm was noted in all patients; in 12 cases there was sinus tachycardia, in 4 cases, sinus bradycardia. The time between termination of ventilation and cessation of electrocardiographic activity was 7-38 min. During this period a gradual slowing of the sinus rhythm was seen in all cases. In seven cases it was interrupted by a junctional rhythm, in ten cases by intermittent atrial fibrillation (Fig. 1). Five patients had first-degree AV block, six patients had second-degree AV block, and seven patients had third-degree AV block. Terminal atrial fibrillation occurred in three cases. In all other cases a terminal sinus bradycardia was recorded. An atrial mechanism persisted for 20 s to 3 min in all patients without any ventricular activity before the ECG became isoelectric. Ventricular tachycardia was transient and returned to a sinus rhythm in five cases; ventricular extrasystole of

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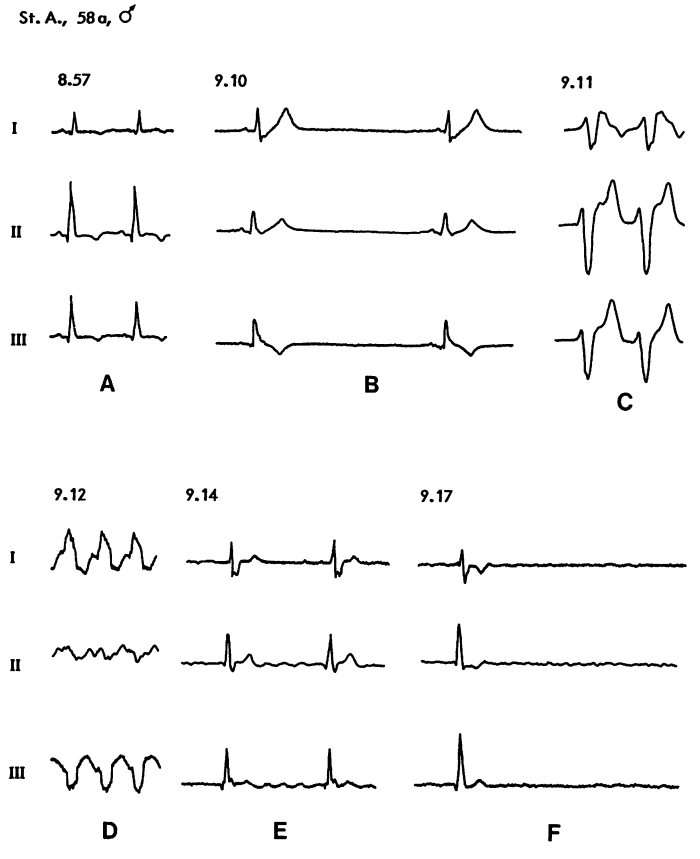
Table 1. Electrocardiographic changes in brain-dead patients (n = 35)

Preterminal rhythm	Changes of atrial activity	Changes of ventricular activity	Terminal ischemia	J-wave	Terminal rhythm	Cessation of ECG activity
Normal	AV block:	Tachycardia 5	Anterior and inferior wall	3	P-Wave only 32	Min. 7 min
Sinus rhythm 19	First degree 5	Fibrillation 6		12	Atrial fibrillation 3	Max. 38 min
Sinus tachycardia 12	Second degree 6	Premature beats 6				
Sinus bradycardia 4	Third degree 7	All changes transiently			In no case was ventricular activity observed	
	Atrial fibrillation 10					
	Junctional rhythm 7					
	Progressive slowing of the sinus rhythm 35					

Table 2. Blood gas analysis and changes of serum electrolytes after disconnection in a typical case of brain death (56-year-old patient)

	Time of sampling				
	17.29	17.38	17.43	17.47	17.50
PH	7.47	7.38	7.21	7.08	7.05
PCO_2	29.3	42.1	63.5	82.5	87.6
PO_2	63.5	17.5	7.4	4.1	4.9
BE	-1.8	-0.5	-4	-7.7	-8.7
SBC	22.7	23.6	20.9	18.2	17.5
Na	146.6	149.3	148.9	147.8	147.0
K	4.59	4.89	7.03	7.82	7.76
Ca	1.09	1.08	1.24	1.17	1.11
					(Cessation of ECG activity)



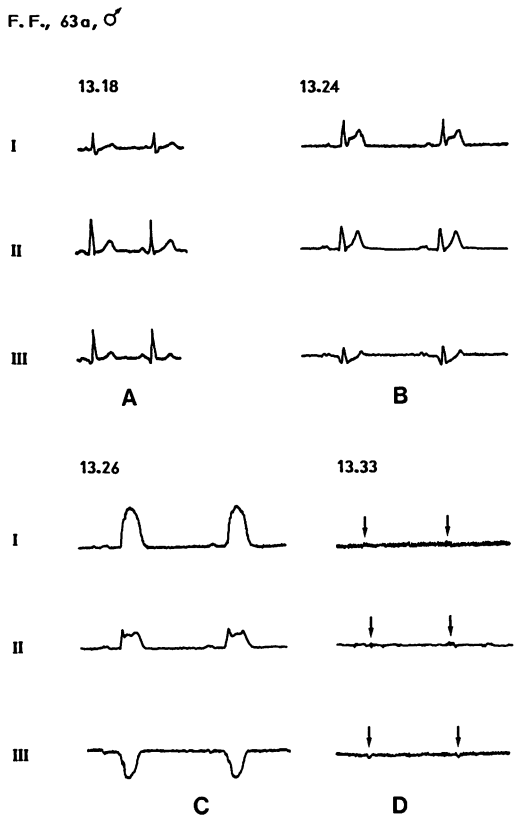


Fig. 2. A Preterminal sinus rhythm, no ischemic signs; B beginning ischemia, first-degree AV block; C complete ischemia with prominence of ST segment and T-wave; D isolated terminal atrial activity

bigeminal type occurred in three cases and atrial premature beats in three cases. A ventricular escape rhythm was never observed. In 12 cases an acute infarction of the anterior as well as the inferior wall was recorded with elevation of the ST interval until complete fusion of the R-wave and ST interval (Fig. 2). In all cases, progressive slowing of ventricular activity until asystole was present. Preterminal ventricular tachycardia was never recorded. A J-wave was observed in three cases.

Discussion and Conclusion

Electrocardiographic changes are not due to destruction of cerebral (i.e., central nervous system) structures, but result from changes in the cardiac sympathetic innervation itself [2]. Termination of ventilation with subsequent hypoxemia and anoxemia with hyperkalemia does not result in characteristic arrhythmias of the ECG or in a sudden loss of heart function. The myocardium of the ventricles seems to

Fig. 1. A Preterminal sinus rhythm, ischemic signs; B progressive slowing of the sinus rhythm, ischemia; C ventricular tachycardia; D ventricular fibrillation; E atrial fibrillation; F terminal QRS complex, atrial fibrillation

be more sensitive to hypoxemia than the atrial structures and the conduction system [1].

1. There is no correlation between electrocardiographic activity in brain-dead patients before and after disconnection of the respirator.

2. Apparently the atrial complex is less susceptible to hypoxia than the ventricular complex, since atrial activity persists up to 1 min after cessation of ventricular activity.

3. There is no correlation between preterminal and terminal ischemia: in 14 cases myocardial ischemia was present in the preterminal stage, although only seven cases showed a preterminal electrocardiographic pattern of myocardial anoxia.

4. Ventricular arrhythmias are independent of existing myocardial ischemia.

5. Disconnection from the respirator with complete anoxia in brain-dead patients does not change cardiac rhythm characteristic of sudden heart death.

The electrocardiographic studies in brain death after disconnection of the respirator give an insight into the destruction pattern of an organ in total anoxemia, but have no place in the diagnosis of brain death. However, they might be of some consequence in heart transplantation for harvesting an organ in fully oxygenized blood and perfusion of the organ itself.

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Brain Death in Fulminant Hepatic Failure

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Fulminant hepatic failure is a rare but severe illness with a rather poor prognosis. It is the result of massive hepatocellular necrosis occurring in most cases without preexisting liver disease. Viral hepatitis is the most important cause of fulminant liver failure [19,22,31]. Exogenous etiological agents include several drugs such as acetaminophen, halothane, paracetamol, valproate, and isoniazid, and toxins such as **Amanita phalloides** [17,18,21,24]. In general the survival rate of the disease averages about 20% [2,14,27,31]. Only in specialized liver units have survival rates up to 40% been reported [5,6].

Cerebral edema is one of the major complications of fulminant hepatic failure, occurring in 50%-80% of all cases [2,11,25,27,28]. At present the etiology of cerebral edema remains unclear and its importance as a cause of death in fulminant hepatic failure is a source of controversy. Well documented cases of fulminant hepatic failure with lethal cerebral edema have only rarely been published [28].

Case History

An 11-year-old girl was referred to the Children's Hospital of Cologne University with a 5-week history of nausea, abdominal pain, intermittent fever, jaundice, and progressive lethargy. Previous history and family history were uneventful. There was no history of drug exposure, infection, or intoxication.

Clinical and laboratory findings confirmed the diagnosis of fulminant hepatic failure. Serological examinations showed no evidence of any viral or bacterial infection. No hepatotoxic substances were identified. The child was disoriented and lethargic with episodes of hallucinations, consistent with the clinical signs of hepatic encephalopathy stage II [21].

A regimen of intravenous fluids, protein exclusion, cleansing enemata, and oral neomycin was started together with parenteral administration of heparin and antithrombin III. Seven combined treatments with plasmapheresis and hemodialysis were carried out between the 2nd and the 8th hospital day. Nevertheless the clinical condition of the patient deteriorated continuously.

Hepatic encephalopathy stage III-IV was evident on the 4th day, together with radiological signs of cerebral edema on CT scan examination. Clinical and neurophysiological signs of brain death

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were recognizable on the 9th day of treatment. Neurophysiological findings during the course of the disease are demonstrated in Table 1.

Postmortem microscopic examination of the liver showed a nearly complete necrosis of hepatocytes together with intranuclear inclusion bodies suggestive of a subacute type of hepatitis, probably of viral origin. The brain was markedly edematous. There was a herniation of the cerebellar tonsils. Microscopic examination of the brain showed disseminated areas of necrotic ganglial cells in both cerebral hemispheres, the cerebellum, and the brain stem. Both optic nerves showed characteristic histological signs of demarcation with pallor of myelin within the intracranial parts and normal myelin staining within the extracranial orbital parts. This demarcation can be considered as histological proof of the intracranial circulatory arrest [23].

Discussion

For the last several years the pathogenetic mechanisms of hepatic encephalopathy, i.e., the functional changes within the central nervous system, have been partly understood [7,8,13]. Currently it is thought that hepatic encephalopathy results from: (a) accumulation of toxic substances in the brain because of impaired hepatic filtering; (b) alteration of the plasma amino acid profile, resulting in accumulation of false neurotransmitters and impaired synthesis and kinetics of true neurotransmitters; and (c) increased brain tissue levels of neuroinhibitory substances such as γ-aminobutyric acid [21].

The pathophysiology of cerebral edema in fulminant hepatic failure, however, has remained unclear and hypothetical in many respects [1]. Data from animal experiments [12,33] as well as the benefical effects of osmotherapy [2,5,6,12] have led to the conclusion that cerebral edema during the early stages of fulminant hepatic failure is of the cytotoxic type. An increase in intracellular water is probably caused by the accumulation of still unidentified toxic substances in the blood which inhibit the sodium- and potassium-ATPase of the cell membranes in pericapillary astrocytes [4,5]. At this stage the blood-brain barrier is still intact. Therefore cytotoxic cerebral edema can be successfully treated with mannitol. Osmotherapy is particularly effective when started during early stages of fulminant hepatic failure and in the presence of low or only moderately raised intracranial pressure [12,20]. With progression of the disease, the vasogenic type of cerebral edema is added to the cytotoxic edema. This was demonstrated experimentally by the increased passive permeability of insulin and L-glucose into the brain tissue [4,15,32]. Osmotherapy has no beneficial effect on the vasogenic cerebral edema [33]. In several controlled clinical studies it has further been demonstrated that administration of either glucocorticoids or dexamethasone does not improve the survival rate of patients with fulminant hepatic failure and cerebral edema [29,26]. Renal failure, not infrequently associated with hepatic failure, apparently does not contribute to the development of cerebral edema [2,5].

The prognosis of fulminant hepatic failure is mainly dependent on the prevention of clinical complications during the acute phase of the disease such as bleeding, renal failure, and cerebral edema [9]. In addition to the standardized therapeutic regimen in hepatic coma, metabolic parameters can be stabilized by plasmapheresis and hemofiltration. In our own case a transient increase in the cortical activity was observed in the EEG for several hours after termination of plasmapheresis and hemofiltration, indicating that the elimination

Table 1. Clinical and neurophysiological findings in fulminant hapatic failure (N.L., ♀11 years)[a]

Hospital day	Clinical course	EEG	VECPs	BAERs
1	Hepatic encephalopathy Stage II Glasgow Coma Scale 11	Diffuse slowing triphasic waves	-	-
4	Hepatic encephalopathy stage III-IV Glasgow Coma Scale 6	Severe diffuse slowing, amplitude depression	Amplitude depression	Normal
9	Clinical signs of brain death Glasgow Coma Scale 3	No cortical activity	Absent	Left: absent Right: only wave I recognizable, later components absent

[a] Parallel to progressive clinical symptoms of hepatic encephalopathy, the EEG and the VECP findings indicate the deterioration of cerebral function on the cortical level while at BAERs are still normal (day 1, day 4). Severe abnormalities and complete absence of the BAERs on the 9th hospital day are neurophysiological signs of the herniation of the brain stem and the intracranial circulatory arrest due to excessive cerebral edema. These findings correlate with the clinical symptoms of brain death and the absence of cortical activity in the EEG and the VECPs

of toxic substances from the blood had transiently improved the metabolic situation of the CNS. The clinical symptomatology of hepatic encephalopathy, however, remained unchanged.

Diagnosis of cerebral edema in patients with fulminant hepatic failure is difficult. Papilledema is rarely noted [21] and computed axial tomography of the brain cannot be repeated at short intervals. Together with the evaluation of the neurological status, the epidural monitoring of intracranial pressure is the most important diagnostic measurement for prevention and early detection of cerebral edema. Clinical studies of patients with fulminant hepatic failure demonstrated intermittent rises of intracranial pressure in 85% of the cases. Intracranial pressure of 60 mmHg and more was associated with decerebrate posturing and/or unequal or abnormally reacting pupils [2,5,6].

Additionally, continuous EEG monitoring and the recording of visual evoked cortical potentials (VECPs) and brain stem auditory evoked responses (BAERs) provide valuable neurophysiological data on cerebral function. In contrast to the rather impressive alterations of the cortical EEG and the VECPs in patients with fulminant hepatic failure, BAERs are not influenced by metabolic coma if the intracranial circulation remains unimpaired [3]. The prolongation of central conduction time within the brain stem and the complete disappearance of BAERs are indicators for the compression of brain stem structures and the arrest of intracranial blood circulation during the terminal phase of cerebral edema [10].

At present, treatment of cerebral edema due to fulminant hepatic failure consists in osmotherapy, hyperventilation, and administration of analgesics, sedatives, and diuretics, together with controls of the serum sodium level and restriction of intravenous fluids [16]. The prognosis of the patients however, remains doubtful even in cases with early start of treatment. Further knowledge about the pathogenetic mechanisms of cerebral edema in the future may help to develop new therapeutic concepts for causal prevention and treatment, thus improving the survival rate of patients with fulminant hepatic failure.

Conclusion

Cerebral edema is a major complication of fulminant hepatic failure. Its pathophysiology is only partly understood and its significance as a cause of death has rarely been documented in the literature. We present the case history of a girl aged 11 years with fulminant hepatic failure. Brain death due to cerebral edema was documented by EEG monitoring, VECPs and BAERs, CT scan findings, and postmortem examination of the brain with histological proof of intracranial circulatory arrest. Epidural monitoring of the intracranial pressure is the most effective procedure for early diagnosis of cerebral edema, which seems to be of the cytotoxic type during earlier and of the vasogenic type during later stages. Osmotherapy has a benefical effect on cytotoxic cerebral edema in the presence of an intact blood-brain barrier. Dexamethasone and glucocorticoids do not improve the survival rate.

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Ethical and Legal Aspects of the Diagnosis of Cerebral Death in the GDR

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Dying and death have always had a particular relevance in every society. Analogous philosophical problems are also at the very center of ideological and ethical reflections nowadays, particularly when medical activities and decisions are concerned.

Questions concerning organ transplantation, presupposing the donation of organic material, have increasingly been at the forefront of public interest and have been made a subject for discussion by the mass media - unfortunately not always correctly and to the point.

The fact that death may have occurred in an individual although the heart and other organ systems are still functioning has demanded a reorientation, not only by physicians, lawyers, and theologians, but also by the general population. In the meantime the conception that the irreversible loss of all brain functions corresponds with the end of individual human existence has been generally accepted. Two important principles can therefore be stated:

1. Cerebral death corresponds with individual death.
2. The timely diagnosis of cerebral death is a human concern of comprehensive practical importance.

In order to reach these principles it was necessary to solve medical, ethical, and juridical as well as religious and moral problems.

The solution concerning the moral aspect was perhaps quite easy: at all times and with all means at our disposal we have to offer care for the benefit of the patient whose organs are of vital importance for the survival of other people after the patient's death is beyond all doubt.

From the religious point of view it was evident that nobody except the physician is able to discern and determine the exact moment of death. Pope Pius XII stated as long age as 1957 that this is solely the task of the physician and does not touch the competence of the church (Speeches of the Pope, 1958, Volume 4).

Even now improvements in the field of medicine still involve a wide sphere of juridical and ethical problems for the physician. Above all we have to point out that modern possibilities of intensive care must not be misused.

There are certainly biological bounds that have to be known and must be taken into consideration by the physician, and young colleagues must learn to recognize them. Eagerness and ignorance should not give

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rise to the exceeding of these bounds. Hippocrates said that the physician should not venture upon those who are already overpowered by sickness, and this is certainly still true today. Not only the right to live but also the right to die has to be recognized. Bacon interpreted this with his words: "as it is truly not little part of human blissfulness, that is to say to have a gentle end."

Of course the irreversible decision of the physician over cerebral death must be beyond question and therefore the highest personal sense of responsibility is necessary. To certify death is still one of the duties of the physician. Modern medicine insists on two different demands in this connection:

1. The classical symptoms of death are known. If they are evident, only the physician decides on certification.
2. The exact criteria of cerebral death are still not a general part of medical knowledge. If the mental process has obviously not yet been brought to a close, the committee must decide on certification.

Committees for certifying individual death on the basis of the irreversible loss of all brain functions are installed by the County Medical Officer of Health (M.O.H. /Bezirksarzt). Compulsory members of the committee are: one specialist in neurology and psychiatry or neurosurgery and one anesthetist. The doctor in charge can also be appointed to the committee. With regard to the personnel, the committee is variable in order to meet all required practical measures at any time. There are no legal rules prohibiting removal of organs from the just deceased, but aliens and members of the army and similar organizations are excluded.

There is no juridical requirement to speak to the relatives about the determination of death and the probable organ donation. Such decisions would go beyond the relatives' capability to judge this responsible situation. We are of the opinion that such a procedure should be excluded for ethical and humane reasons.

Consent or refusal by relatives is not possible because the very idea of ownership of a corpse is unthinkable. Ownership results as a matter of principle only from purchase, heritage, or gift, none of which is applicable in this case. The relatives are only responsible for a dignified funeral. In this connection it must be pointed out that the act of certifying cerebral death must not be taken into consideration only with regard to an organ donation.

The technical term "declaration of death" ought to be eliminated from the vocabulary of physicians, because it is a juridical statement which might be applicable to persons who are missing for a very long time. There are no ethical scruples about timely typing of seriously injured patients or other potential donors - for instance within the transition stage from midbrain syndrome to bulbar brain syndrome - because a procedure of that kind implies nothing at all. The therapeutic efforts are not at all reduced by such a situation. On the contrary: for the purpose of conditioning the donor the circulatory backup, the correction of metabolic derangements, fluid balance, electrolyte substitution, and control measures against metabolic acidosis cannot be neglected. The therapy has to be carried on to its full extent. Thus the basis for eventual survival can be created. With regard to the patient as well as to organ explantation a reduction of the therapeutic efforts cannot be justified from either the ethical or the juridical viewpoint.

According to the legal regulations the admissibility of organ donation is not dependent on the consent of the organ donor. Organ donation is based on the ethical principle of genuine mutual assistance. If the deceased has appointed by will that in the case of death organs must not be used for explantation, this has to be taken note of. Nevertheless, in cases of emergency very lengthy investigations ought to be left undone. If death occurs under suspicious circumstances the state prosecutor has to consent to the intended organ explantation.

Cerebral death can only be certified by an exact clinical examination and observation of the course. The total cessation of brain stem function corresponds with individual death. The electroencephalogram (EEG) is unsuitable for cogent evidence and has no legal documentary value.

According to the original version of the Harvard criteria, EEG is not obligatory. The poikilothermal temperature type is of essential importance. Angiography is not necessary but it is of considerable help in determining the time of cerebral death.

In conclusion:

1. Certifying cerebral death is a task of medical practice; the physicians who are entrusted with it must possess a pronounced sense of personal responsibility, requiring excellent ethical traits of character. The task of certification is in the hands of experienced and skilled specialists in the field of neurology, psychiatry, neurosurgery, and anesthesia who are in the employ of the committees appointed by the County Medical Officer of Health. Two signatures are sufficient.

2. Although certification of death is based on exact scientific facts, at first sight it does not seem to be compatible with the physician's vocational duty to cure; nevertheless, correct procedure in this regard is obligatory on a humane basis.

3. Ethical and legal fundamentals enable the physician to come to exact decisions concerning the restoration of other peoples' health in this exceptional situation, too.

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Brain Death Diagnosis in Anencephalics?

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In October 1987 organ transplantation from still breathing anencephalic donors was feigned in a television report [1] in the Federal Republic of Germany. Although the response was groundless, the report caused indignation among the public. In the region of the transplantation center in Münster the declaration of consent to organ transplantation declined. A gynecologist is on trial for murder.

Gynecologists from Europe and America have recommended use of organs from anencephalics for transplantation [2,3,5-8,11]. MARTIN in 1969 published a case of kidney transplantation from an "anencephalic monster" [8].

BELLER in 1980 proposed a hypothesis to define the legal position of anencephalics [3]. The discovery of such malformations places the gynecologist in a difficult situation: abortion after the 22nd week of pregnancy has been prohibited, but in many cases the discovery of anencephaly occurs much later. To solve this problem gynecologists have defined anencephaly as brain death. In the event of brain death no therapy is necessary. Therefore pregnancies with anencephalic fetuses may be interrupted at any time [2]. This solution, also favored by HARRISON in the United States [5], is now in practice worldwide.

When organs are intended to be taken from anencephalics for transplantation the legal situation becomes difficult, because explantation itself causes death. "Anencephaly" is a term for various malformations concerning the brain and spinal cord to different extents [10]. Therefore the time of survival ranges from early embryonic periods to several weeks after normal birth. During pregnancy the diagnosis "anencephaly" is possible by sonographic, radiological, and chemical studies. Nevertheless, brain function cannot exactly be determined before abortion, and especially potential brain stem function cannot be estimated.

The gynecological statement "anencephaly is brain death" is a form of brain death determination which comes into conflict with all rules of the Bundesärztekammer [12,13].

Neurosurgeons and neurologists must offer their comments, because they are the specialists in cerebrospinal diagnosis and therapy. In the television report one of the central questions was whether a neurosurgeon was consulted, but neurosurgeons had not been involved.

Faced with imprecisely defined conditions for this kind of brain death determination, it is not the time to discuss such problems in

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public. A horrible scenario was designed by television: the report predicted the future breeding of genetically manipulated anencephalic organ donors, carried to term by hired women. Of course, the television viewers' reactions ranged from "misuse" to "child murder."

To avoid legal complications and to safeguard transplantation medicine, the following has to be stated:

1. The statement "anencephaly is brain death" has to be refuted.

2. Brain death determination following the rules of the Bundesärztekammer is not possible in anencephalics.

3. Brain death determination fulfilling legal and medical prescriptions is impossible in anencephalics. For this reason organ explantation from anencephalic children is not practicable.

Moreover there is no requirement for explantation from anencephalic donors: In the Federal Republic of Germany about 8000 persons aged between 0 and 50 years die annually in accidents. If only half of them were to be made available for explantation, the current 20 000 patients with terminal renal insufficiency could be transplanted within a few years.

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New Research

Monitoring of Hemodynamics in Subarachnoid Hemorrhage Using Transcranial Doppler and Laser Doppler

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Introduction

The surgical treatment of ruptured cerebral aneurysms has been made safer by the use of microsurgical techniques and recent therapeutic advances. Nevertheless, postoperative vasospasm remains a major complication, producing delayed ischemic neurological deficits. Until the introduction of transcranial Doppler, there was no noninvasive method to assess the vasospasm after subarachnoid hemorrhage. Arterial narrowing causes an increase in the flow velocity and this change is inversely proportional to the diameter squared. Therefore, recording of flow velocities of the circle of Willis can be a sensitive method of monitoring vasospasm. Blood flow velocity can be measured in the circle of Willis transcranially using a 2-MHz pulsed directional velocimeter and spectral analyzer [1]. Several investigators have recently reported that transcranial Doppler may be beneficial for estimating vasospasm and cerebral ischemia [2-6]. Laser Doppler flowmetry now makes it possible continuously to measure the blood flow, blood volume, and velocity of the cerebral cortex using an implantable transducer. Hemodynamics after subarachnoid hemorrhage fluctuated due to vasospasm or increased intracranial pressure. In our department, flow velocity of the middle cerebral artery and cerebral blood flow have been measured continuously in 25 cerebral aneurysms after surgery, using a transcranial Doppler and a laser Doppler respectively. Intracranial pressure, systemic arterial pressure, pulmonary arterial pressure, and central venous pressure have been measured simultaneously. The aim of this study was to correlate the flow velocity of the middle cerebral artery with the clinical course of patients after ruptured aneurysm, and to investigate the relationship between the flow velocity and intracranial pressure. Serial changes in flow velocity, cerebral blood flow, and intracranial pressure will be discussed in relation to drug therapy, which is given to prevent the ischemic deficits after subarachnoid hemorrhage.

Materials and Methods

Flow velocity was measured using a 2-MHz transcranial Doppler ultrasound (EME and Medasonics) and cortical blood flow was measured using a laser Doppler (TSI) in 25 patients undergoing early aneurysm surgery within 3 days after subarachnoid hemorrhage. The flat probe of the transcranial Doppler was placed at the temporal region for continuous monitoring. The low profile device of the laser Doppler was placed on the cortex during surgery for the intracranial aneurysm. Cerebral blood flow, blood volume, and velocity of the cortex were estimated

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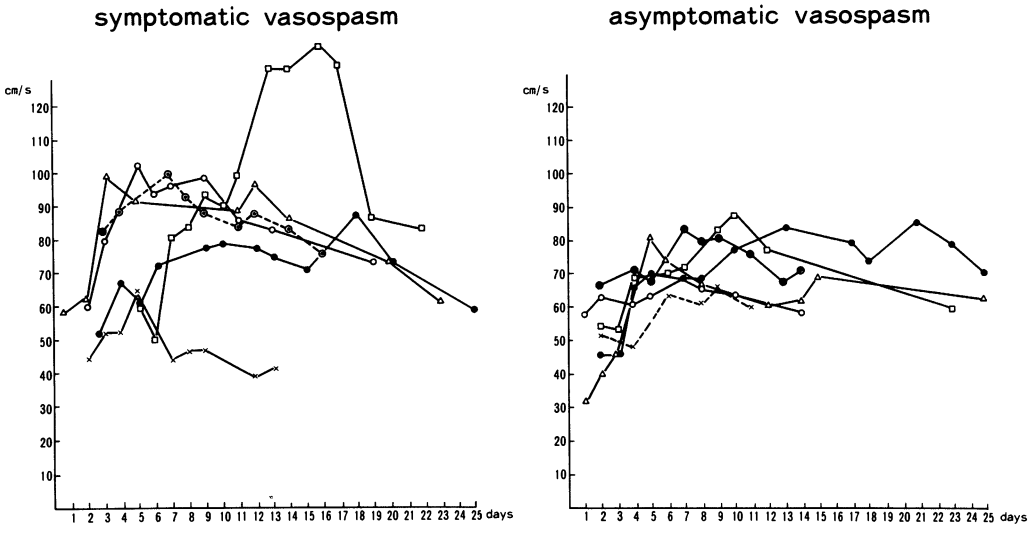

Fig. 1. Time course of mean flow velocity (cm/s) after subarachnoid hemorrhage. Increased flow velocities were seen in patients with symptomatic vasospasm as compared with asymptomatic cases

continuously. Intracranial pressure was measured by an epidural sensor (Cardio Search). A Swan-Ganz catheter was introduced supraclavicularly. Information derived directly from the Swan-Ganz catheter includes pulmonary arterial pressure, pulmonary artery wedge pressure, cardiac output, and central venous pressure. Systemic arterial pressure was also monitored simultaneously via an arterial catheter.

Results

Flow Velocity After Subarachnoid Hemorrhage

Transcranial Doppler ultrasound recording was carried out in 25 patients with subarachnoid hemorrhage during the 3 weeks after the surgery. All patients had increased flow velocity of the middle cerebral artery between days 4 and 21 after subarachnoid hemorrhage. Even in asymptomatic cases, increased flow velocity was detected by a transcranial Doppler. However, flow velocity showed a rapid increase in symptomatic cases of vasospasm in comparison with asymptomatic cases (Fig. 1). It was impossible to assess the existence of vasospasm by the actual value of flow velocity of the middle cerebral artery.

Flow Velocity Correlated with CT Grading (Fisher's Grading)

Transcranial Doppler flow velocity was compared with CT visualized blood (Fisher's CT grading). Increased flow velocity was seen in cases with Fisher's grade 3 and 4 compared with Fisher's grade 1 and 2. There was a significant correlation between diffuse deposition of subarachnoid hemorrhage and development of vasospasm evaluated by transcranial Doppler sonography (Fig. 2).

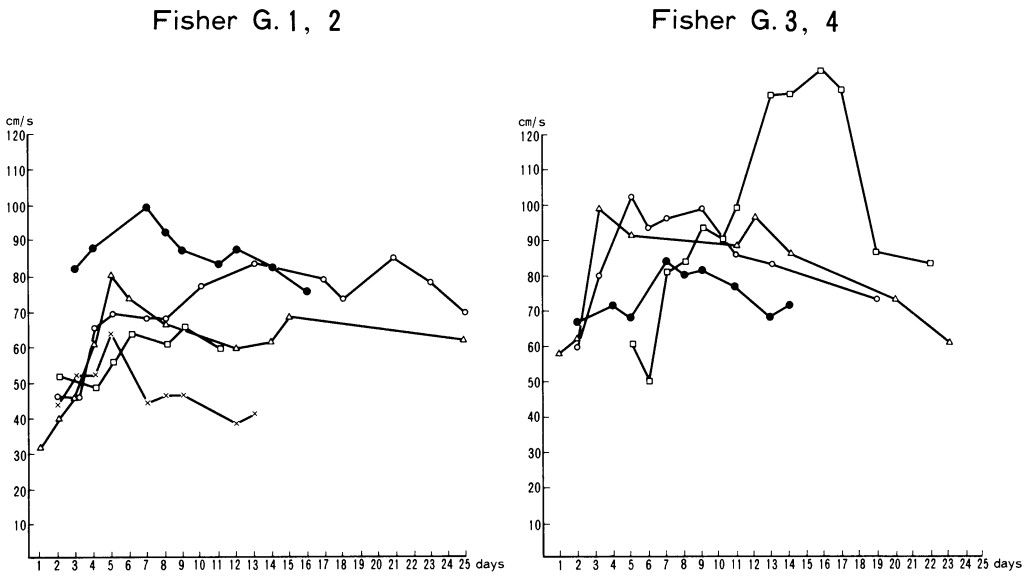

Fig. 2. Cerebral vasospasm evaluated by transcranial Doppler correlated with CT grading (Fisher). Fisher's grades 3 and 4 revealed increased flow velocities in comparison with grades 1 and 2

Flow Velocity of the Circle of Willis and Cortical Blood Flow

Flow velocity and cortical blood flow were measured simultaneously using a transcranial Doppler and a laser Doppler respectively. When the flow velocity of the middle cerebral artery increased, cortical blood flow and velocity were also increased. Figure 3 shows increased flow velocity of the middle cerebral artery and cortical blood flow after glycerol infusion. Relative changes in flow velocity detected by a transcranial Doppler are similar to those in cortical blood flow detected by laser Doppler (Fig. 3).

Effect of Glycerol, Albumin, and Calcium Channel Blocker

Intracranial pressure was reduced by glycerol infusion. On the other hand, mean flow velocity of transcranial Doppler was increased gradually, and the S/D ratio was reduced. Cerebral blood flow and velocity were shown to be increased by laser Doppler after glycerol infusion (Fig. 3). Hypervolemic hemodilution with plasma reduced cortical vascular resistance and increased cerebral blood flow. The administration of albumin increased the cortical blood flow and velocity. These data suggest that hypervolemic hemodilution with expansion of plasma volume increases cardiac output and cerebral blood flow. When a calcium channel blocker (nicardipine 1 mg) was administered intravenously, the flow velocity of the middle cerebral artery was shown to decrease gradually by a transcranial Doppler, and cortical blood flow and velocity were also decreased.

Flow Velocity and Intracranial Pressure

Intracranial hemodynamics are altered by vasospasm, and there is increased intracranial pressure after subarachnoid hemorrhage.

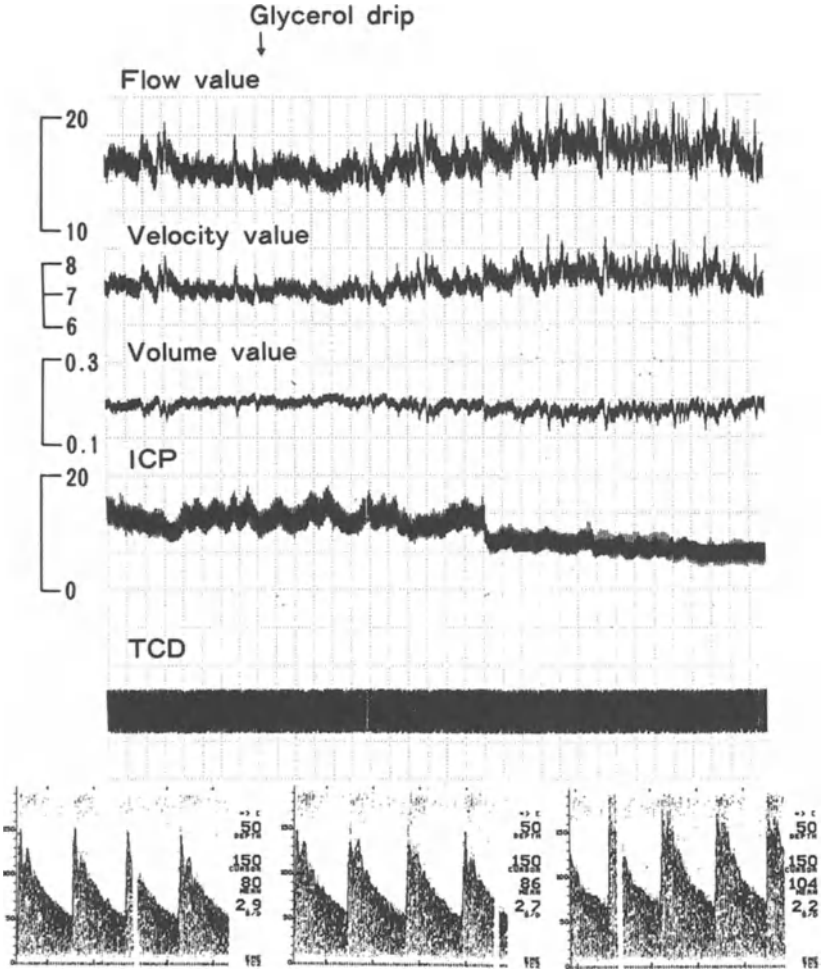

Fig. 3. Flow velocity measured by transcranial Doppler and CBF measured by laser Doppler were measured simultaneously. This figure showed increased flow velocity and CBF after glycerol infusion. Relative changes of flow velocity by transcranial Doppler were similar to CBF alteration by laser Doppler

Serial flow velocity, cerebral blood flow, and intracranial pressure were studied. Systemic arterial pressure, pulmonary arterial pressure, and central venous pressure showed relative changes simultaneously. When a pressure wave appeared, mean flow velocity was decreased and the S/D ratio was increased, which suggested increased systolic velocity and decreased diastolic velocity. However, the mean flow velocity was increased rapidly and the S/D ratio was decreased at the descending slope of the pressure wave. The same alternation of flow velocity showed reduction at the time of plateau wave. Cortical blood flow and velocity were also changeable when the pressure wave appeared after subarachnoid hemorrhage. Blood volume of the cerebral cortex was increased before the appearance of pressure wave, and blood flow and flow velocity were decreased gradually when intracranial pressure was high. The flow velocity of the middle cerebral

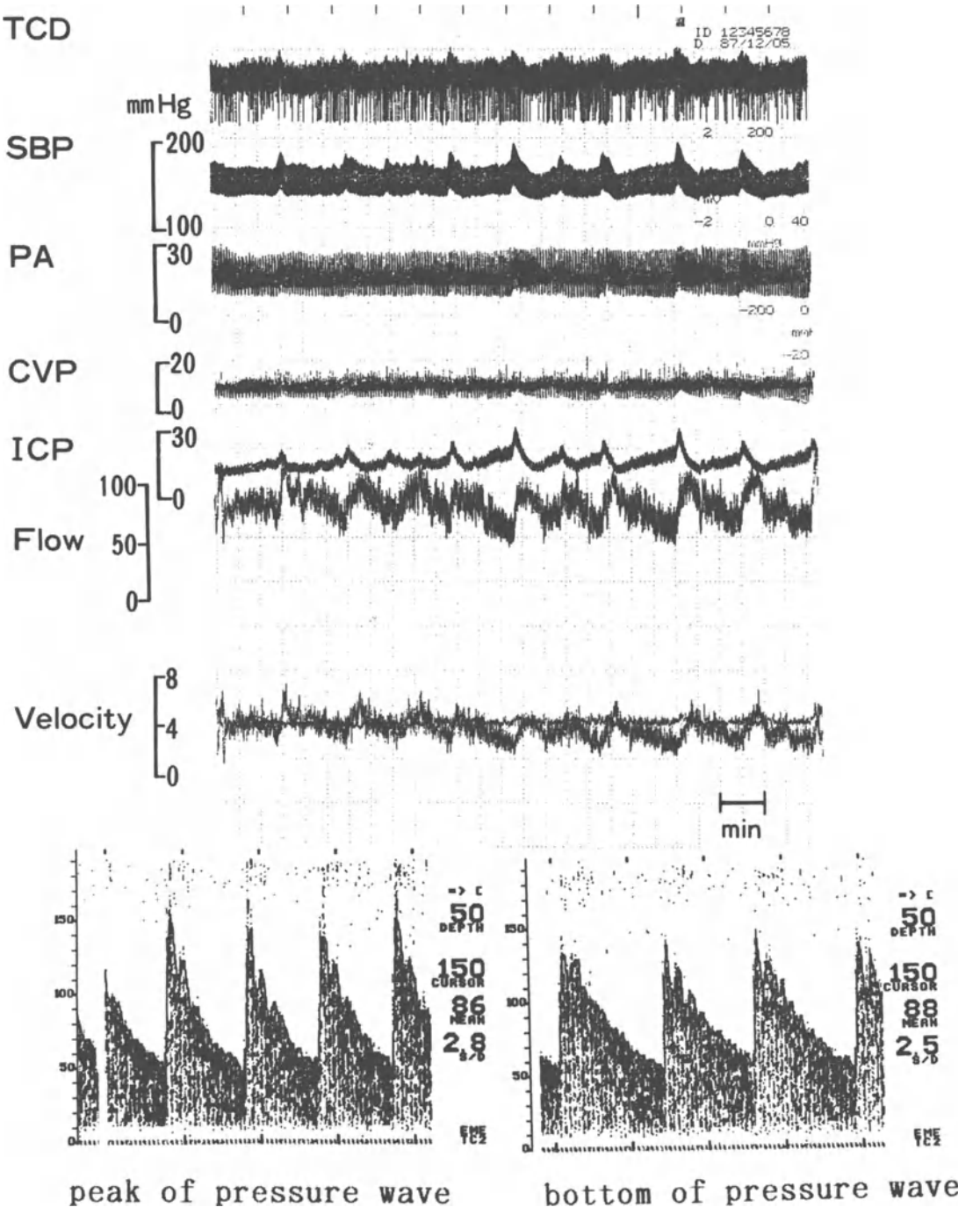

Fig. 4. Intracranial hemodynamics fluctuated due to vasospasm and increased intracranial pressure (ICP). Systemic arterial pressure, pulmonary arterial pressure, and central venous pressure fluctuated with ICP changes simultaneously. Mean flow velocity and CBF were reduced when a pressure wave appeared. Flow velocity and CBF increased at the descending slope of the pressure wave

artery, measured using a transcranial Doppler, and the cortical blood flow, measured by a laser Doppler, showed synchronous changes in response to alteration of intracranial pressure (Fig. 4).

Discussion

Transcranial Doppler ultrasound is a new method which provides real-time evaluation of flow velocity in the basal cerebral arteries. We have shown that transcranial Doppler may be useful for noninvasive continuous examination of cerebral vasospasm subsequent to subarachnoid hemorrhage. Several investigators have reported that vasospasm can be evaluated by the actual mean value of flow velocity using a transcranial Doppler (2-6). Our data suggested that it was difficult to assess the vasospasm by an absolute value of velocity. However, it was possible to estimate the existence of vasospasm by continuous monitoring. When the arteries became narrowed, the flow velocity increased rapidly in symptomatic cases; nevertheless, asymptomatic vasospasm also showed increased flow velocity. It was also impossible to evaluate the critical reduction of cerebral blood flow by the absolute value of the mean flow velocity using a transcranial Doppler. However, relative changes in flow velocity of the middle cerebral artery were similar to those in cortical blood flow and velocity measured by a laser Doppler. The flow velocity of the middle cerebral artery measured by transcranial Doppler and the cerebral blood flow measured by laser Doppler coincided relatively. Therefore, continuous monitoring is very valuable for estimating hemodynamics after subarachnoid hemorrhage.

These studies suggested that flow velocity and blood flow fluctuated instantaneously together with the changes in intracranial pressure. When the intracranial pressure became elevated, the mean flow velocity was reduced at the ascending slope of the pressure wave and the S/D ratio became elevated due to increased vascular resistance; meanwhile, the flow velocity increased at the descending slope of the pressure wave. The peak of the pressure wave coincided with the lowest flow velocity and cerebral blood flow. Cerebral blood volume increased before the appearance of the pressure wave. Transcranial Doppler and laser Doppler monitorings are very valuable in assessing hemodynamics after subarachnoid hemorrhage as well as intracranial pressure. Recent interest in intravascular expansion involves use of hemodiluting colloid infusates for the treatment of cerebral vasospasm and delayed ischemic deficits. These studies suggested that intravascular volume expansion with albumin raises cardiac output and increases cerebral perfusion. The effects of hypertonic solutions of glycerol and mannitol on flow velocity, blood flow, and intracranial pressure were studied. The present data demonstrated that glycerol or mannitol infusion decreased intracranial pressure gradually and increased flow velocity and cerebral blood flow; meanwhile cerebral blood volume was shown to be decreased by glycerol or mannitol infusion. Hypertonic solutions should be used to prevent the delayed ischemic deficits after subarachnoid hemorrhage. On the other hand, a calcium channel blocker did not increase flow velocity or cerebral blood flow. It is important to estimate the hemodynamics of vasospasm by continuous monitoring and to provide prompt treatment or prevention of vasospasm.

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Cerebral Blood Flow Measurements with ^{99m}Tc-HMPAO and ^{123}I-Amphetamine (HIPDM) in Patients with Cerebral Tumors (1)

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Introduction

New methods in the treatment of brain tumors, e.g., boron neutron capture therapy, external stereotactic irradiation by linear accelerator, and the use of chemotherapy and interferons in patients with malignant brain tumors, demand not only knowledge of the localization and histology of the lesion. Information on the pathophysiological behavior of the tumor and edematous tissue is also important for planning strategies involving these therapeutic methods. An important factor for the irradiation sensitivity of brain tumors is the oxygenation of the tissue [2,8]. Failure of radiotherapy can be explained by various factors, e.g., the presence of hypoxic or anoxic cells in the tumor area [1]. The effectiveness of chemotherapy depends on drug concentration in the tumor tissue. Both examples illustrate the importance of studies dealing with measurements of tumor perfusion.

Material and Methods

Two methods are used in measuring regional cerebral blood flow (rCBF): positron emission tomography (PET) and single photon emission computed tomography (SPECT). The high cost of the PET system prevents its widespread introduction as a screening method, in contrast to SPECT. Measuring rCBF with SPECT, lipophil substances are used which enrich in the cerebral tissue in relation to the cerebral blood flow. The first tracer is hexamethylpropyleneamine oxime, labeled with technetium 99 m (HMPAO SPECT) followed by N,N,N-trimethyl-N-(2 hydroxyl-3-methyl-5-idobenzyl)-1,3-propane-diamine, labeled with iodine 123 (HIPDM SPECT). In a study with untreated cerebral tumors we tested the value of these two cerebral blood flow tracers and tried to identify a tumor-specific rCBF pattern. The results were compared with CT and MRI. During the last 12 months we examined 36 patients with intracranial tumors: 23 females and 13 males, the aged 27-82 years. Histological diagnosis was performed in 12 cases by stereotactic biopsy, in 24 cases by surgery. There were 10 meningiomas, 22 gliomas, 2 lymphomas, and 2 metastases (Table 1).

Analytical Procedures

HMPAO SPECT was performed by a rotating gamma camera with a matrix of 64 x 64 pixels and a slice thickness of 6.25 mm (Table 2) and analyzed semiquantitatively. Our patients received 555 MBq ^{99m}Tc-HMPAO (Ceretec, Amersham Internat. plc, Amersham U.K.) in 0.9% NaCl intravenously. Ten minutes after application SPECT was carried out.

Table 1. Histological diagnosis

Meningioma	10
Low grade glioma	8
High grade glioma	14
Metastasis	2
Lymphoma	2
Total	36

HIPDM SPECT mostly was done using a rotating multidetector scanner with a slice thickness of 20 mm (Table 3) and was analyzed qualitatively. ^{123}I-HIPDM (100 MBq) was injected intravenously, followed by the examination 2 min postinjection. The cerebral uptake of HMPAO and HIPDM was then measured within the tumor area and the perifocal edema and correlated to a corresponding region of the contralateral normal hemisphere. The SPECT region of tumor and edema was determined by comparison with the corresponding CT or MRI scans. The evaluation of SPECT ought to differentiate between solid, cystic, and necrotic tumor tissue. Regions of interest (ROI) ranging from 6.25 cm^2 to 47 cm^2 were defined in tumor and perifocal edema areas and compared with the corresponding contralateral normal cerebral tissue. A cerebral index (CI) was calculated by the quotient of counts per pixel in the pathological ROI to counts per pixel in the normal contralateral ROI. A CI above 1.05 was interpreted as hyperperfusion, a CI lower than 0.95 as hypoperfusion.

Results

The blood flow measurements with HMPAO SPECT and HIPDM SPECT showed almost identical results in the meningioma group. In nine of the ten cases with HMPAO SPECT and in seven of the ten cases with HIPDM an increased perfusion rate was found in meningiomas (Table 4). Two tumors, a large meningioma in the temporoparietal region and a small fibromeningioma, were hypoperfused with HIPDM. There was only a small area of perifocal edema in both tumors. With both SPECT methods a small endothelial meningioma surrounded by edema grade 2 was hypoperfused, whereas one metastatic process showed an increased tracer uptake and the other a decreased blood flow pattern with HMPAO SPECT (Table 5). This tumor was represented in CT and MRI as a space-occupying lesion with a large area of central necrosis and a solid tumor rim surrounded by perifocal edema grade III. In contrast the

Table 2. Data acquisition and analysis - ^{99m}Tc-HMPAO

Gamma camera and computer:	Rotating gamma camera Apex 415 Elscint
Collimator:	Low energy, high resolution
Activity:	555 MBq
Rotation:	360°/60 projections
Acquisition time:	20 s/projection
Matrix:	64 x 64 pixels
Slice thickness:	6.25 mm
Spatial resolution:	7 mm FHWM
Reconstructions:	Transversal, coronal, sagittal

Table 3. Data acquisition and analysis - ^{123}I-HIPDM

Detector system:	Rotating multidetector system (Tomomatic 64)
Activity:	100 MBq
Rotation:	360°/10 s
Acquisition time:	4 min
Matrix:	64 x 64 pixels
Slice thickness:	20 mm
Spatial resolution:	17 mm FHWM
Reconstructions:	Transversal

Table 4. rCBF measurement in cerebral tumors: ^{99m}Tc-HMPAO in comparison with ^{123}I-HIPDM. Results in ten meningiomas

	HMPAO	HIPDM
Hyperperfusion	9	7
Hypoperfusion	1	3
Pathological perfusion	-	-
Total	10	10

Table 5. rCBF measurement in cerebral tumors: ^{99m}Tc-HMPAO in comparison with ^{123}I-HIPDM. Results in two metastases and two lymphomas

	HMPAO	HIPDM
Hyperperfusion	3	2
Hypoperfusion	1	1
Pathological perfusion	-	1
Total	4	4

hyperperfused metastasis showed a homogeneous contrast enhancement of the whole tumor tissue and was surrounded by a large area of edema as well. This small metastasis could not be recognized in HIPDM SPECT.

The results of rCBF measurements of the glioma group are demonstrated in Tables 6 and 7. We found almost identical results in low grade gliomas with both methods: five astrocytomas, one oligodendroglioma grade II, one subependymoma of the IIIrd ventricle, and one myxoid ependymoma grade II (Table 6). A decreased tracer uptake was seen in five of the eight cases with HMPAO SPECT and in four of the eight tumors with HIPDM SPECT, whereas two tumors with HIPDM SPECT and one with HMPAO SPECT were not detectable. Two gliomas, one astrocytoma of the thalamus, and one cystic fibroastrocytoma with contrast enhancement were hyperperfused. A small oligodendroglioma grade II could

Table 6. rCBF measurement in cerebral tumors: ^{99m}Tc-HMPAO in comparison with ^{123}I-HIPDM. Results in eight low grade gliomas

	HMPAO	HIPDM
Hyperperfusion	2	2
Hypoperfusion	5	4
Pathological perfusion	1	2
Total	8	8

not be recognized by SPECT. In the high grade glioma group (12 glioblastomas, one astrocytoma grade III, and one ependymoma grade III-IV) there was an increased HMPAO uptake in seven patients and a decreased uptake in the other seven (Table 7). With HIPDM SPECT there was an increased rCBF in the tumor area in only 4 of 14 cases. Eight tumors were hypoperfused. In two tumors we could not measure any difference between the tumor area and the normal contralateral cerebral tissue. Perifocal edema of grades II and III and, in one case, of grade I could be represented as a hypoperfused area in SPECT. Figure 1a shows the axial MRI of a 38-year-old man after contrast enhancement demonstrating a cystic thalamus tumor. The patient was operated stereotactically. Histologically we found a glioblastoma grade IV with a great pathological vascularization. The rCBF measurement with HMPAO SPECT is shown in Fig. 1b. According to the axial MRI shift in the tumor area we found a distinct increased tracer uptake represented as a white area in the thalamic region. Figure 2a shows the CT scan of a right frontal glioblastoma with contrast enhancement. The solid hyperdense tumor rim can be recognized clearly. The central tumor necrosis is seen as a distinct hypodense area. In the histological examination we found a great number of thrombosed pathological vessels. The corresponding rCBF measurement with HIPDM SPECT is demonstrated in Fig. 2b. The tumor tissue is hypoperfused, being represented as a dark region.

Discussion

Our results indicate that HMPAO and HIPDM are able to trace the rCBF in brain tumors. HIPDM has a good blood-brain extraction fraction of about 90% [5,6] and stays long enough in the tissue that it is possible to measure the cerebral perfusion with the rotating gamma camera and SPECT. In contrast to other groups [4,5], we measured an

Table 7. rCBF measurement in cerebral tumors: ^{99m}Tc-HMPAO in comparison with ^{123}I-HIPDM. Results in 14 high grade gliomas

	HMPAO	HIPDM
Hyperperfusion	7	4
Hypoperfusion	7	8
Pathological perfusion	-	2
Total	14	14

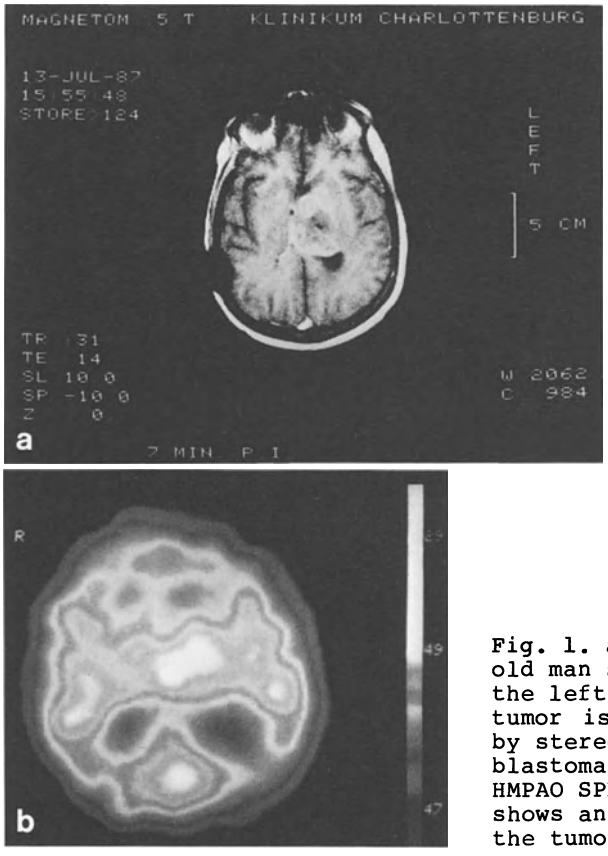
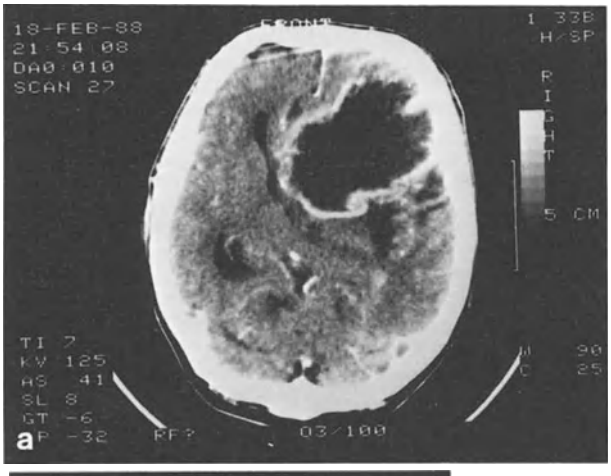


Fig. 1. a Axial MRI in a 38-year-old man suffering from headache. In the left thalamic region a cystic tumor is detectable, characterized by stereotactic biopsy as a glioblastoma. b rCBF measurement with HMPAO SPECT in the same patient shows an increased tracer uptake in the tumor area (hyperperfusion)

increased uptake of HIPDM in the tumor tissue in 15 of 36 patients. We suppose that iodine-labeled amines are able to measure rCBF in cerebral tumors. HMPAO SPECT is a method introduced for measuring rCBF in cerebrovascular disorders [3]. In our examinations we could demonstrate that HMPAO SPECT is also able to measure rCBF in tumor and edematous areas. In comparison with HIPDM SPECT there is a better imaging quality. The two methods showed a good correspondence in meningiomas, lymphomas, metastases, and the low grade glioma group. The different resolution power of the HIPDM SPECT may cause different rCBF results in the tumor areas and in the areas of perifocal edema with identical measurements. In most cases low grade gliomas were hypoperfused according to the CT findings as a hypodense lesion without contrast enhancement. The rCBF measurements of the malignant gliomas varied; 50% were hyperperfused and 50% hypoperfused. These results agree with the work of Langen et al. [7], who could not find a homogeneous blood flow pattern. In the PET study of Tyler et al. [9], 5 of 14 malignant gliomas were hyperperfused, three of them located in the thalamus. In our study one thalamic glioma also had an increased rCBF, probably resulting from the special blood supply of the basal ganglia. Comparing CT and MRI findings with the SPECT results, all tumors with increased blood flow had contrast enhancement. Small solid tumor rims were not detectable with the SPECT methods. The results of the rCBF measurements with SPECT depend on several factors such as the perfusion rate of the tumor tissue, the relation of the necrotic area to the solid tumor tissue, the relation of hypoperfused perifocal edema to the size of the tumor



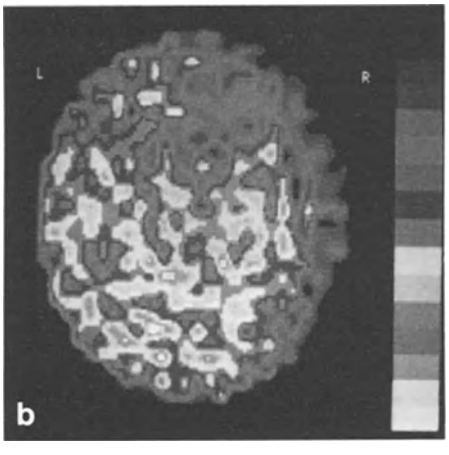


Fig. 2. a CT scan after contrast enhancement in a 49-year-old man hospitalized with general seizures. The CT scan demonstrates a right frontal glioblastoma with a large area of central necrosis and a small solid tumor rim. b The blood flow measurement of the tumor with HIPDM SPECT shows a decreased tracer uptake (hypoperfusion) in the necrotic area, visible as a dark region. The small solid tumor rim is not detectable with SPECT

(partial volume effect), and the localization of the lesion. The main advantages of the two SPECT methods, and especially of HMPAO SPECT, is the excellent imaging quality in brain tumors, and SPECT is the method of choice as regards availability and cost.

Conclusion

HMPAO and HIPDM are appropriate tracers for rCBF measurements in brain tumors. The previous results did not show a homogeneous blood flow pattern in gliomas, in contrast to the areas of perifocal edema. When PET is not available, HMPAO SPECT is the method of choice. Our results provide evidence for the importance of the SPECT methods as diagnostic tools in the planning of radio- and chemotherapy of malignant brain tumors.

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CSF Flow Visualization by Magnetic Resonance Imaging Techniques - Methods and Clinical Examples

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Introduction

It has been recognized that the circulation of cerebrospinal fluid (CSF) plays a key role in several diseases of the central nervous system. The relative inaccessibility of the subarachnoid space has impeded the pace of investigations in this area. Until now invasive techniques, such as spinal puncture with application of contrast agents or intracranial pressure measurement, have been the mainstay of studies concerning CSF flow dynamics.

Theory

Based on the different measuring components of magnetic resonance imaging (MRI), flow effects can be visualized [1]. Moving spins cause a signal alteration because of their movement within and between the different parts of the status of measurement. The formula of signal intensity takes this into consideration.

$$I = N(H)f(v)e - TE/T_2(1 - e - TR/T_1)$$

I is the pixel intensity, N(H) is the proton density, f(v) is a function of flow, e is the base of the natural logarithm, and TE and TR represent the common programmable sequence parameters, the echo delay time and the repetition time. T_1 and T_2 are tissue-dependent relaxation times. On this basis it is possible to obtain flow information from MRI, as has been well known since the early days of its use [1,2,4,7,8]. At first the signal loss (flow void) of high velocity blood flow was obvious [2]. But later signal increase caused by flow effects was also described [5]. To understand this it is necessary to recognize the known effects corresponding with flow:

1. Signal loss from:
   a) Time of flight effect (high velocity signal loss)
   b) Dephasing effect (first echo dephasing)

2. Increased signal by:
   a) Diastolic pseudogating
   b) Entry phenomenon
   c) Second echo rephasing

Signal loss from time of flight is clearly visible in arteries because the protons exposed to the first pulse are not within the plane at the readout time; this is an effect of through plane flow. Because of dephasing the signal void phenomenon depends on flow

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within the plane [4] as well; flow causes a loss of local information.

Especially two signal increasing effects depend on through plane flow. The first effect is seen in the multislice technique when the TR is a harmonic of the RR time, so that some pictures are taken during diastolic rest of blood or CSF. The entry phenomenon is evoked by unsaturated protons entering the slice causing a higher signal. These mechanisms are used for visualization of blood and CSF flow. Electrocardiographic (ECG) gating provides information about pulsatile flow alterations [2,3,5]. Other techniques without gating demonstrate complex information of pulsatile and total spin movement [6].

If the first echo dephasing effect is traceable and the flow is constant within the time of 2 x TE, rephasing can be done with a second 180° pulse of a conventional spin echo sequence. After subtraction of both pictures we only get signal from spins with laminar flow. The signal intensity is a function of the number of moving spins, i.e., of flow, not of the velocity [8].

Patients and Methods

We used a conventional spin echo multiecho which consists of 16 180° pulses and built up a subtraction image with flow information usually within the sagittal midline plane. Up to now we have examined 27 patients upon 34 occasions:

-Normal pressure hydrocephalus	9
-Hydrocephalus of various etiology	8
-Spinal examination of paraparesis	3
-Intracranial cystic lesion (no tumor)	3
-Isolated fourth ventricular syndrome	3
-Intraventricular tumor	1

Results

Figure 1 shows a normal volunteer. CSF flow of the fourth ventricle is visible, as is flow of the third ventricle with a lower signal in

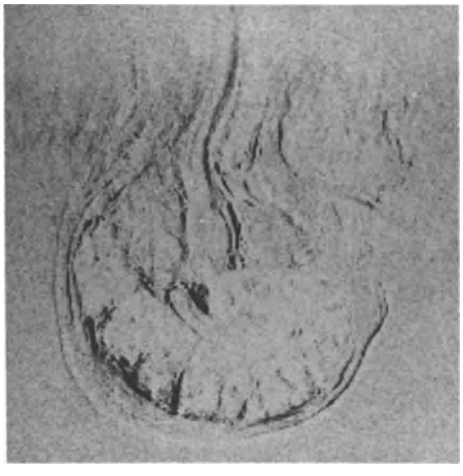

Fig. 1. Sagittal multiecho subtraction image of a normal volunteer. It visualizes the CSF flow of the third and fourth ventricles and the craniocervical junction; a strong signal is also caused by the midline veins

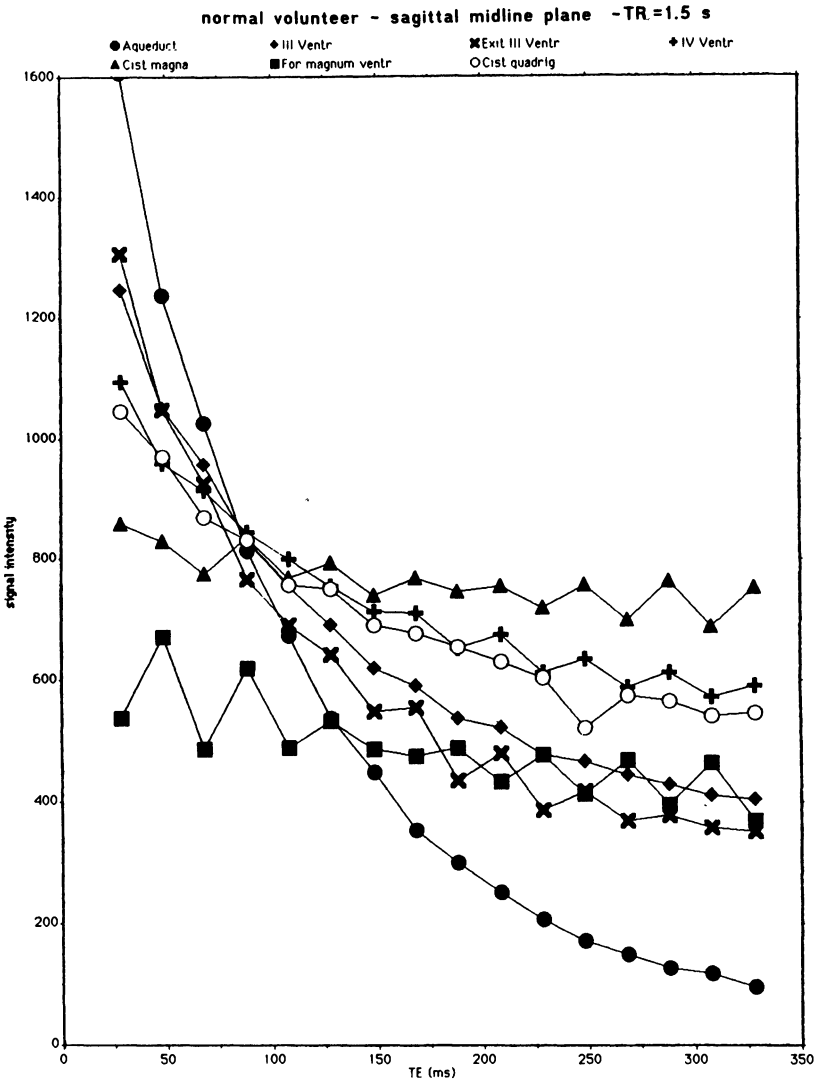

Fig. 2. The signal intensity curve of a normal volunteer shows the proton movement from special regions by down and up signal alteration. Flow is demonstrated at the foramen magnum; no flow is shown at the aqueduct

some cases. The pulsatile flow within the craniocervical junction causes a strong signal; the aqueduct was not enhanced in normal cases, but was if normal pressure hydrocephalus existed. If we added this image to a normal anatomical image of the same series, it could be shown that the flow within the basilar cistern is enhanced. This is an example of the sensitivity and local resolution of the method. It is also possible to obtain a signal intensity curve out of a region of interest. This shows whether there really is a flow effect, by down and up alteration of the signal intensity from echo to echo time (Fig. 2).

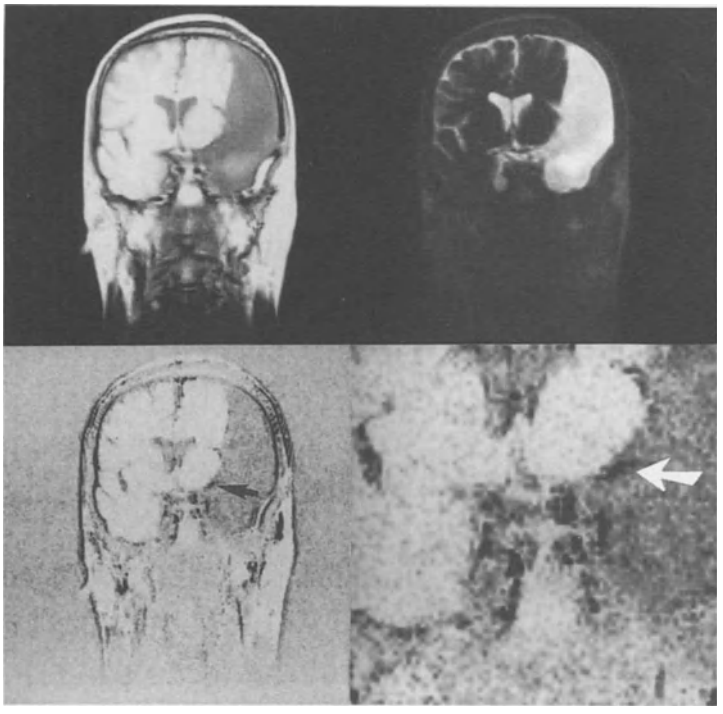

Fig. 3. The image of patient with a great temporal arachnoid cyst shows the basal CSF communication with the basal cistern system after operation by a high intensity area (arrows)

Pathological cases such as a great temporal arachnoid cyst (Fig. 3) prove that we are able to procure clinical information from this method. A strong signal at the base of the cyst shows the communication with the basal cistern system of the surgically treated arachnoid cyst.

First results from normal pressure hydrocephalus [1] demonstrate the disturbed pathological flow pattern with a strong signal of the aqueduct, and accordingly we have also seen a lowered signal at the craniocervical junction. This pattern has been normalized in our results after shunting. The method seems a new approach to the diagnosis of this progressive disturbance of CSF circulation. The spinal canal can be visualized if there is a stop of pulsatile CSF movement, as is caused by spinal blockade. It seems possible that these disturbances provoke an increased neurological deficit after lumbar puncture during myelographic examination.

Other information can be obtained from ECG gated gradient echo sequences (so-called fast imaging such as FLASH). After a first saturation time we are able to obtain information about the signal alteration within the cardiac cycle. If we use a T_2-weighted FLASH sequence, CSF movement causes a visible signal void. From alterations within the RR cycle a pulsatile inflow of CSF from the spinal canal to the extended fourth ventricle could be demonstrated and later verified (Fig. 4) by isotope cisternography. Additionally, the

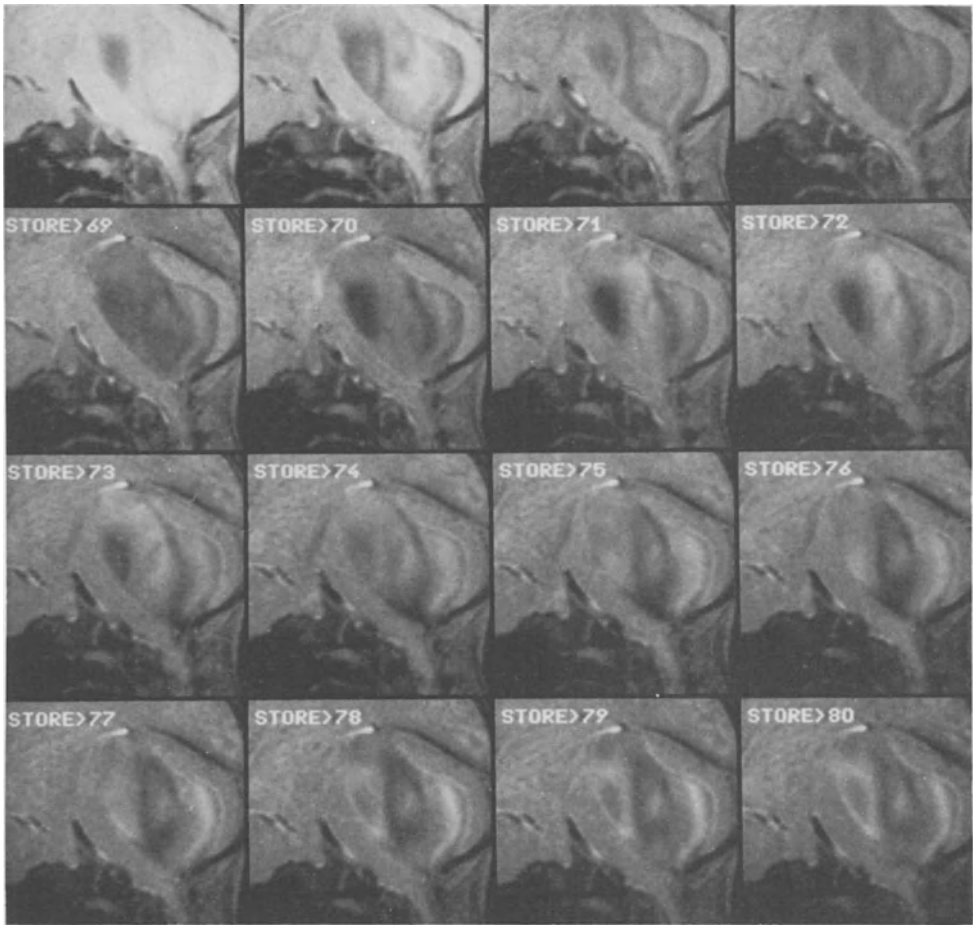

Fig. 4. Pulsatile CSF movement is demonstrated from within the so-called isolated fourth ventricle by an alternating signal void during the RR cycle. Pulsatile inflow from the spinal canal is visualized.

transmission of the pulsatile motion from the choroid plexus of the fourth ventricle is obvious within these series.

Conclusions

Our results show that the described multiecho technique is able to provide presurgical information about normal pressure hydrocephalus. The shunt function can be visualized and thus controlled after surgical treatment. Pulsatile flow visualization is able to demonstrate circulation pathways (Fig. 4).

Only a few clinical examples could be demonstrated here, but they indicate the possibilities of these MRI methods. Further experiments will be done to develop a clearly reproducible noninvasive method for CSF flow imaging without disturbance of the CSF system itself. Up to now, there is no direct way to visualize turbulences, but unlike with MR angiography this is no real problem because of lower veloci-

ties within the so-called third circulation. Faster measurement techniques such as gradient echos with phase imaging [3,5] are a promising new tool.

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Intracranial Complications After Anticoagulant Therapy

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Introduction

Anticoagulant therapy is, without doubt, one of the major achievements in medical history [1,5,7,12,15]. However, the benefit of long-term therapy has been limited to only a few conditions because of the dangers based on the risk of hemorrhage [10,11,13]. Intracranial hemorrhage, though less frequent than other bleeding manifestations, is more often fatal and more hazardous from a prognostic point of view [6,8].

It is important to develop a realistic estimate of the incidence of hemorrhage and to determine how successfully this risk can be avoided.

Results

In a series of 149 spontaneous intracranial bleedings in the year 1987 we found 21 (14%) to be due to anticoagulant therapy. Ten patients had an intracerebral hemorrhage, nine a subdural hematoma, and two a subarachnoid hemorrhage. A similar distribution was also found in the literature [14].

Intracerebral Hemorrhage (Table 1)

Of the ten patients in this report, two had a cerebellar hemorrhage. There were six males and four females. The age range was 57-78 years, and four had hypertension. It seems that anticoagulation is more dangerous in hypertensive patients. The duration of anticoagulation

Table 1. Results of intracranial hemorrhage after operative treatment

	Intracerebral hemorrhage	Subdural hematoma	Subarachnoid hemorrhage
No. of patients	10	9	2
Died	4	1	1
Neurological deficiency	1	1	-
Rehabilitated	5	7	1

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was from 3 months to 22 years. Four patients died, one had an outcome with neurological deficiency and four were rehabilitated.

Below we report one case with intracerebral hemorrhage and outcome without neurological deficiency.

Case Report. A 57-year-old man had been receiving anticoagulant therapy since cardiac surgery in 1981. After a simple injury he suffered headache. Although he had no neurological symptoms, he was admitted to hospital. On the 3rd day of hospital stay he became somnolent. Cranial computer tomography showed a bifrontal intracerebral hemorrhage (Fig. 1) and an acute midbrain syndrome emerged. Postoperatively the patient was without paralysis but had slow reactions. The outcome showed no neurological deficiency.

It is important for the outcome to recognize the situation and evacuate the hemorrhage quickly.

Subdural Hematoma (Table 1)

Of our nine patients with subdural hematoma, eight were males and one a female. The age range was 50-84 years. Three of the patients had hypertension. The duration of anticoagulation was from 1 month to 10 years. The 84-year-old man with subdural hematoma died, one patient had neurological deficiency, and seven were rehabilitated. The prognostic situation of subdural hematoma was much better than that of intracerebral hemorrhage or subdural hematoma.

Subarachnoid Hemorrhage (Table 1)

The two women with subarachnoid hemorrhage had hypertension. One patient died, one had no neurological deficiency.

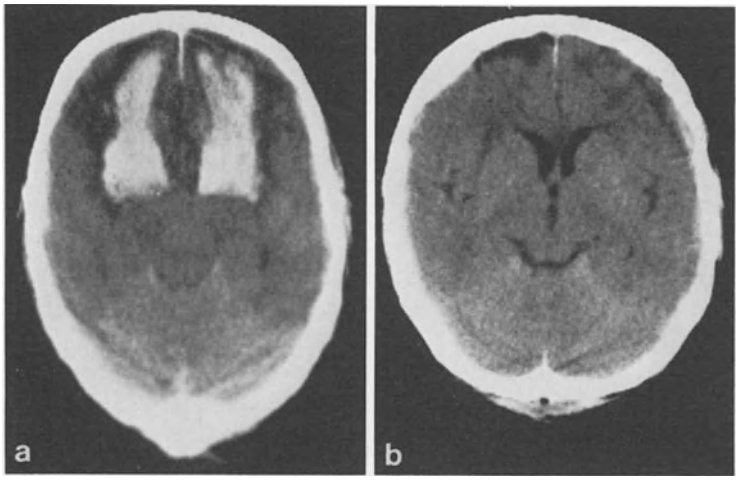

Fig. 1. a CCT: bifrontal intracerebral hemorrhage; preoperative scan in 57-year-old man. **b** Postoperative control

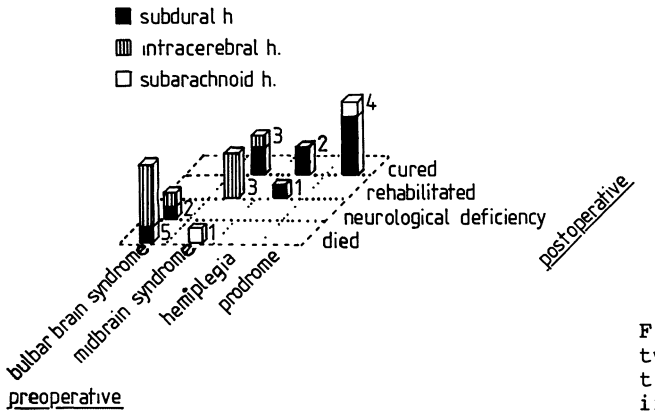

Fig. 2. Correlation between pre- and postoperative situation after intracranial hemorrhage

The correlation between the pre- and the postoperative neurological situation showed us that none of the patients with bulbar brain syndrome showed recovery. By contrast, patients with midbrain syndrome or hemiplegia had a good chance of rehabilitation (Fig. 2).

The typical clinical development with subdural hematoma often started 14 days before neurological signs occurred. The early symptoms were headache, sickness, emesis, and vertigo (Fig. 3a). The typical clinical development with intracerebral or subarachnoid hemorrhage also showed early symptoms, but the duration of this period was often short, ranging between 2 and 6 h (Fig. 3b).

The development, the localization, and hypertension were the limiting factors for the prognosis of intracranial complications of anticoagulant therapy. We found the prothrombin (Quick) index most often to be in the therapeutic range (Table 2) [4,11,12,15]. The risk of bleeding was independent of the prothrombin index.

After diagnosis the evacuation of hemorrhage was indicated. A blood transfusion or treatment with PPSB increased the prothrombin (Quick) index, so time from CCT to exploration was often less than 45 min.

Conclusions

1. Intracranial complications of anticoagulant therapy were possible in spite of drug control and a therapeutic prothrombin (Quick) index.

Table 2. Prothrombin (Quick) index

Range	No. of patients	Died
<15%	1	1
15%-30%	15	4
>30%	5	1
	21	6

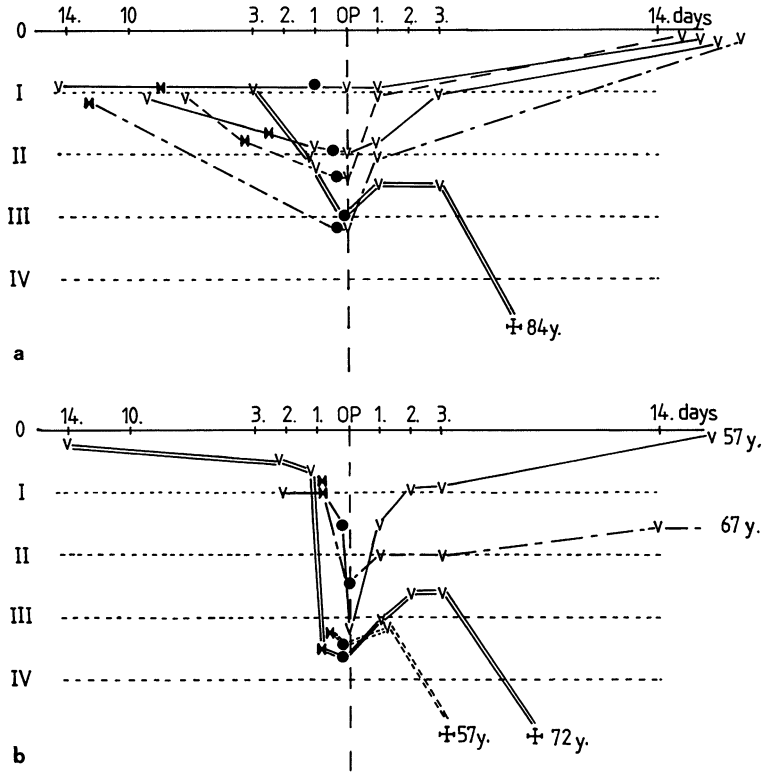

Fig. 3. a Clinical development with subdural hematoma. I, early symptoms; II, hemiplegia; III, midbrain syndrome; IV, bulbar brain syndrome. M, begin of hospital stay: ●, department of neurosurgery. b Clinical development with intracerebral hemorrhage

2. Vigorous appraisal of the benefit of long-term anticoagulant therapy had limited the indications for such treatment to only a few conditions. A risk of bleeding arose in the elderly and patients with hypertension.

3. Intracerebral hemorrhage had a poor prognosis and was the most often found complication of intracranial hemorrhage.

4. With subdural hematoma the clinical symptoms occurred slowly. Intracerebral hemorrhage had increased neurological symptoms.

5. As the compressive effects of hemorrhage may be reversed by early operative intervention, the importance of careful investigation of all patients on anticoagulant therapy who develop symptoms and signs referable to the central nervous system is evident.

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Subject Index

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Contents: Surgery of the Anterior Skull Base: Surgery of Malformations of the Anterior Skull Base. - Surgery for Trauma to the Anterior Skull Base. - Surgery for Inflammatory Complications in the Region of the Anterior Skull Base. - Surgery of Space-Occupying Lesions of the Anterior Skull Base. - Surgery of Tumors of the Orbit and Adjacent Skull Base. - Special Operative Techniques. Surgery of the Middle Skull Base: Surgery of Traumatic Lesions of the Middle Skull Base. - Surgery of Inflammatory Disorders of the Middle Skull Base. - Surgery of Space-Occupying Lesions of the Middle Skull Base. Surgery of the Posterior Skull Base: Surgery of the Internal Auditory Canal and Cerebellopontine Angle. - Surgery of Tumors of the Lateral Posterior Skull Base and Petrous Bone. - On the Problem of Paralytic Dysphagia Caused by Posterior Skull Base Tumors. Surgery of the Clivus: Introductory Remarks. - General Operative Techniques. Surgery of the Craniocervical Junction: Introductory Remarks. - Operative Technique. Surgery of the Facial Nerve and Skull Base: Introductory Remarks. - General Operative Techniques. - Special Operative Techniques.

This is the first text to consider the skull base as a whole and from an interdisciplinary point of view. It analyzes the wide spectrum of pathological entities which can affect this crossroad region, including anomalies, traumatology, tumors and infectious processes.

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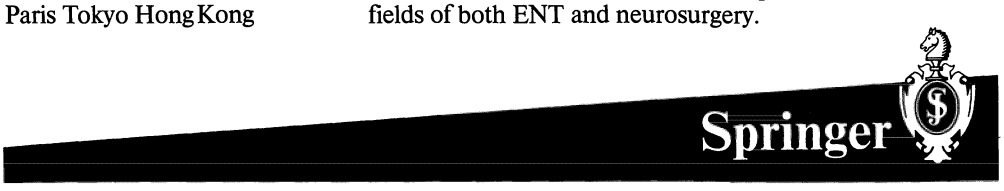

R. Unsöld, University of Düsseldorf;
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Compressive Optic Nerve Lesions at the Optic Canal

Pathogenesis - Diagnosis - Treatment

Collaborators: M. Bach, H.-R. Eggert, G. Greeven, J. DeGroot

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This comprehensive monograph opens up sensational new diagnostic and therapeutic perspectives. The topographic information is presented with excellent anatomic preparations. The wide spectrum of symptoms is taken from extensive clinical experience; they are critically analysed and compared to the ophthalmological, neurosurgical, and neuroradiological literature.
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