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Cranial nerve damage after paediatric head trauma: a long-term follow-up study of 741cases

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A report is given on transient and permanent (6 months) impairment of cranial nerves after paediatric head injuries (N: 741). There is a link between severity of the injury, fractures on the base of the skull, its foramina and channels, and the frequency of cranial nerve involvement. One should try to establish whether a posttraumatic dysfunction of the cranial nerves is primary or secondary in nature, i.e. due to raised intracranial pressure or haemorrhage. In children after head injuries often the cranial nerves of the oculomotor system are affected (20.2%/7.0%) — transient (permanent), followed by optic atrophy (4.88%), lesion of the trigeminal nerve (4.2%/2.2%), and the facial nerve lower motor type (4.1%/1.7%). Loss of hearing (3.3%/1.2%) and of smell (3.2%/1.2%) are less frequent in children than in adults.

There are few reports on cranial nerve (CN) damage after paediatric head injuries (HI) [19, 20, 25, 26, 28, 36, 37, 46, 53]. Most comprehensive reports are mixed series of a few children and mainly adults [1, 24, 31, 32, 35, 41, 51, 64]. CN impairment might indicate raised intracranial pressure (ICP) or expanding haematoma and is a secondary lesion then. On the other hand, all CN are close to the base of the skull or are even harboured within by bony channels or passing through various foramina. So, if there is a fracture on the base of the skull one has to look carefully for every kind of CN impairment. On the other hand, their course is beneath "silent" areas of the brain: the fronto-orbital and basal temporal lobes. If there is an additional contusion of those brain structures, the child later might present with mental retardation or regression or with perl sonality, language or memory problems; or there may be a permanent brain-organic syndrome, in particular if the damage was bilateral and/or symmetrical. Therefore it seems important to look attentively for CN impairment in every child with HI.

Patients

From January, 1970, to July, 1984, we have seen and treated 741 children who had suffered HI. They were grouped according to the duration of post-traumatic amnesia (PTA) (Table I). The ratio boys/girls was 2/1, (471/270).

As to aetiology and age, (Table II), 45.2% were involved in traffic accidents. Mortality was 4.9% in the whole material, but 8.0% in those after traffic accident. Mortality was much influenced by poly-

TABLE I

Duration of post-traumatic amnesia (PTA) in 741 children

I: no PTA, concussional symptoms present	235
II: PTA < 1 hour	168
III: PTA 24 hours	97
IV: PTA < 1 week	93
V: PTA > 1 week	111
VI: died within the first 4 weeks after trauma	37
Total:	741

NB: of the 204 patients in IV and V 183 were controlled after 6 months, some of them were followed-up more than 10 years.

trauma: 9.3% (N:289) versus 2.2% in those with only HI anaemia and hypovolaemia (N:111); severe hypoxia/cardiorespiratory arrest (N:11) occurred more often after polytrauma than after cranial monotrauma. Many of the children who were hypovolaemic or developed shock later had to be classified into groups IV—VI with the worst prognosis [14, 42, 43]. Of the 35 children who had been battered; 30 were younger than 2 years; 3 of them, died. In these physically abused children one has to accept that head injury is a recurrent event and the addition of minor and major trau'sвш even in subconcussional doses, finally might end in profound mental retardation or severe brain (and ocular) damage [17, 18, 27, 36, 37].

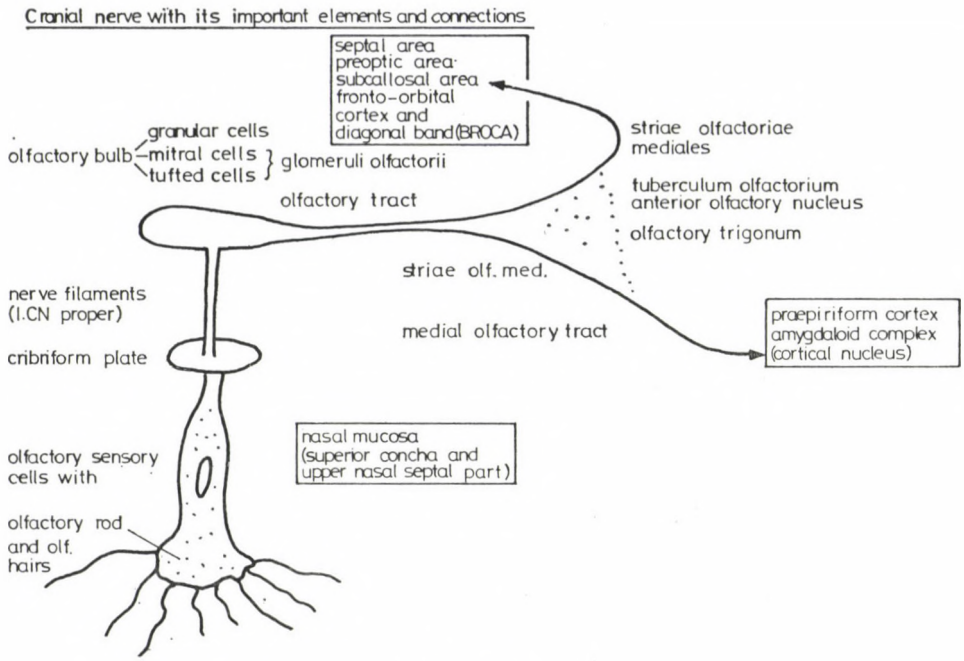
CN impairment, general considerations

Primary lesions are due to stretch, shearing, intruding bone fragments and ischaemic necrosis [1, 5, 31, 32, 44, 64]. Some damage may depend on shearing of the nerves' vessels (vasa vasorum). Basically, every accident may bring direct concussional or rotational forces into action on the human skull. Apart from its direction, the impact might be of crushing or high velocity type with acceleration-deceleration of the brain as a soft tissue content of the skull. CN passing through bony channels (II, V, VII, VIII) are more exposed to crushing forces, whereas those running through the subarachnoidal space are more exposed to shearing (rotational) forces (Graph 1) if, additionally, they are neighboured by sharp edges, wings or ligaments (I, III, IV, VI).

Secondary lesions are mainly prompted by raised ICP and intracranial haemorrhage, including intradural and interstitial bleeding of the nerve itself [5, 39, 64]. Rare causes of secondary CN damage are bacterial meningitis, adhesive arachnoiditis, aerocele, and callus formation [39, 64]. Most important is a secondary damage to the midbrain which is prompted by herniation, vasospasm and circulatory failure; there is a predilection for these in the periaqueductal grey matter and the basis pontis/mesencephali [29, 64].

Site of damage. There are four main areas (Graph 1):

1. within the brainstem in the brainstem nuclei, internuclear network (medial longitudinal bundle) and roots of exit;



GRAPH 1

2. in their intracisternal course;

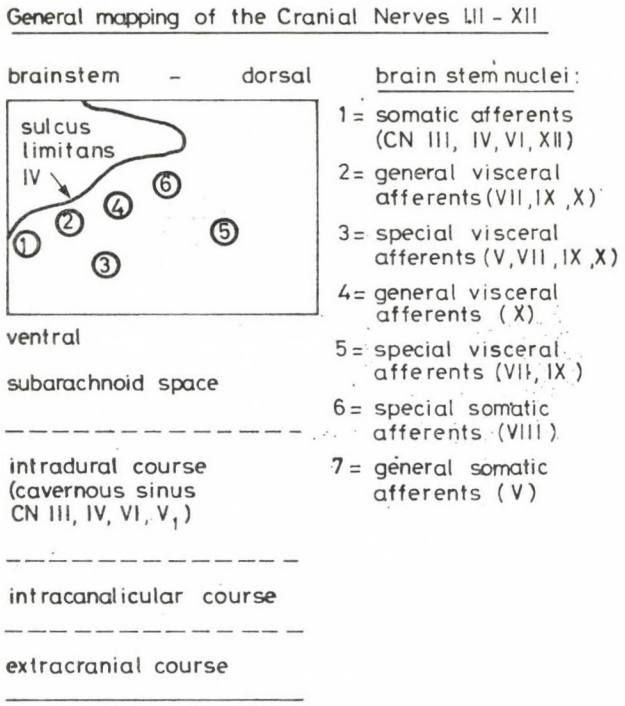
3. in their intradural (intracanalicular, intraforaminal) course;

4. in its most peripheral course the CN itself or its branches might be severed.

CN impairment, particular considerations

CN I, olfactory nerve

Disturbance of smell after HI which has an overall incidence of 3 to 20% [1, 31, 32, 35, 40, 41, 51, 60, 61, 62, 64] is age-related and not dependent on the severity of trauma. Older people (50 years and more) suffer in 50% from loss of smell after HI, even when there was not PTA at all or one of less than one hour [60, 61]. In most cases the blow was frontal or fronto-temporolateral in direction, but in 1/3 it was occipital [41, 64] and may be regarded as a contre coup effect. If recovery takes place, this occurs within 3 months in most cases [31, 32, 40, 61], but recovery has been observed even 5 years after the trauma [61]. Parosmia or olfactory scotomas arise from tearing off the olfactory filaments when they are leaving the dura (Graph 2), but it is thought to be a cortical phenomenon [1, 31, 41, 61, 62, 64]. Parosmia nearly always presents as kakosmia [40, 60, 61]. It is not always due to the violence of the accident itself but to general anoxia comparable to transient loss of smell at high altitude (>7 500 m) too [61]. Bilateral loss of taste and smell is encountered after severe HI and may be attributed to



Graph 2

damage of the ventromedial thalamic nuclei or, if there is additional nystagmus and/or IIIrd nerve palsy, of the tegmental part of the brainstem [8]. In contrast to adults, posttraumatic anosmia in children is rare, 1.4% [14, 28]. In the present series (Table III) it was transient in 2%, and permanent in 1.2%. All these children had suffered from frontobasal injuries with fractures of the anterior fossa and most of them had nasal CSF discharge and needed surgical repair of dura tears and lacerations. Loss of smell was found bilaterally only in 1 child; there was some predilection of groups IV and V with longstanding PTA, and in some very young children and some with very severe brain damage, testing of smell could not be done adequately. Parosmia or olfactory scotomas were never seen in our paediatric patients.

CN II, optic nerve and visual pathway

Optic atrophy (OA) was found in 34 children (4.8%), 33 of them were in groups IV and V (long PTA), and in 20 it was bilateral. 19 of these children were blind on one or two eyes. The worst findings were in the battered children who had had retinal haemorrhages, some of them reaching into the vitrous body, some with partial retinal detachment. All these abused children had a raised ICP, too.

TABLE II

Aetiology, length of PTA and age in 741 children with head injury

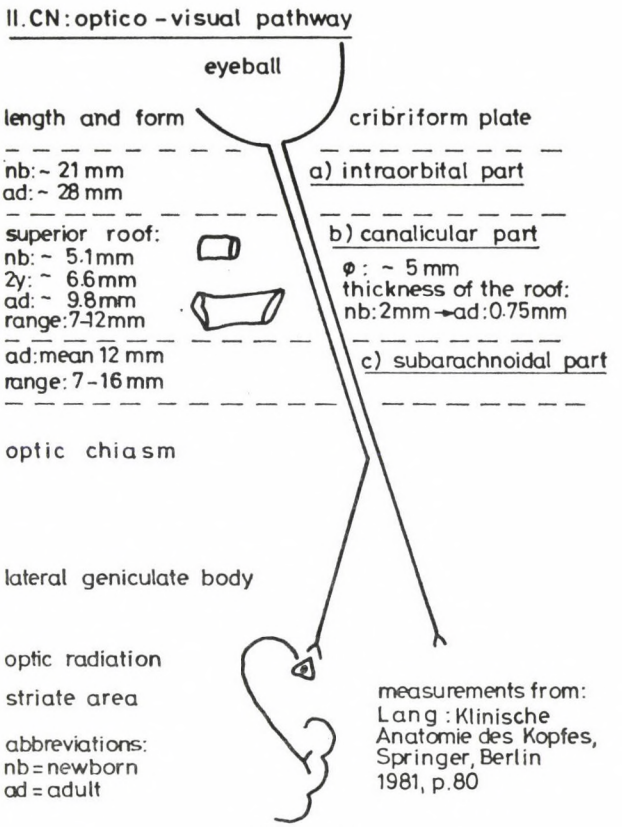
Aetiology PTA:	I	II	III	IV	V	VI	N	in per cent	1—24 mo	< 6y	> 6y
at home	122	39	12	9	2	—	184	24.8	94	58	32
at play and sport	46	49	12	6	3	4	120	16.2	1	40	79
falls (> 3m)	11	12	9	16	6	3	57	7.7	9	29	19
parental assault	11	2	4	3	12	3	35	4.7	30	4	1
other: shoot/stab	1	3	1	—	2	—	7	1.0	—	2	5
all traffic accidents	44	63	59	59	86	27	338	45.3	17	105	216
as seat passenger	4	6	10	11	14	6	51	6.9	15	15	21
as pedestrian	22	36	29	34	62	17	200	27.0	2	81	117
as (motor)cyclist	18	21	20	14	10	4	87	11.7	—	9	78
within each group:	235	168	97	93	111	37	741		151	238	352

NB: lethality figures: the whole group: 4.9% for all traffic accidents: 8.0%; for the rest: 2.5%

These findings were in accordance with data of other authors [16, 17, 27, 36, 37]. Raised ICP was the only cause for OA in 13 patients. The sites of damage to the optic nerve were besides the retina as follows (Graph 3).

During its intraorbital course, direct lesions of the II CN may occur [6, 13, 22, 24] prompted by penetrating objects. Anterior marginal tearing and ischaemic damage may lead to field defects [6, 24]. Of practical importance is a retrobulbar haematoma; clinically it should be suspected if there was an orbital trauma and unilateral amaurotic pupil. Diagnosis to-day is made by high resolution orbital computerized tomography, and after its imaging the haematoma should be drained immediately by needle aspiration. We had one such case in our series.

Lesions of the optic nerve in the optic canal: the amaurotic pupil has no direct light reaction but the consensual one is well preserved or even overshooting. The amaurotic pupil fluctuates in its width according to the light in the room. In every child with an amaurotic pupil a canal or a chiasmal lesion should be suspected and excluded [41]. We had 4 cases of tearing of the optic nerve in our patients, two of them had minor trauma [1]. One has to consider that the roof of the optic canal is thicker in young children than in adults [38]. Most lesions occur after direct frontal or frontolateral trauma; it was shown that the isochromatic lines after experimental frontal or frontolateral concussion converge to the roof of the optic canal and ipsilaterally to the suprasellar region [21]. Therefore, some authors stress the importance of canal fractures in optic nerve damage [7, 11, 13, 21] while others do not [1, 6, 22, 24, 31, 32, 59, 62]. If there is some recovery of the direct light reflex within 48 hours after the trauma, some or much restitution of vis-



GRAPH 3

ual acuity may be expected [41, 64]. As far as operative procedures are concerned, an inferior-medial approach through the middle and posterior ethmoid cells without opening of the dural sheath of the optic nerve is indicated [7, 11, 65]. Fukado is for optic nerve decompression in every case, and reviewed 353 own cases [11]. According to this author, some improvement can be achieved by this operative procedure even weeks after the trauma. All other authors indicate operative decompression only if there is progressive visual loss, short interval trauma/operation (< 48 hours) and the patient is not comatose [13, 22, 24, 62, 64]. Again, some other authors disfavour operative treatment completely [1, 31, 32]. According to Fukado [11], the symptoms of optic nerve damage in its intracanicular course are a loss of direct light reflex (100%), lesion of the lateral eyebrow (97%) and blood and/or CSF discharge from the homolateral nostril (80%). If the optic nerve is severed at the orbital apex, some other CN are involved: II, IV, VI and the first division of the Vth [13, 22, 52]. OA develops 12 days to 6 weeks after the trauma [1, 6, 20, 22, 24, 31, 32,

41, 59, 62]. The outcome is always doubtful. All of our four patients became blind permanently, as were more than 50% in larger series. In some patients light/dark discrimination or finger counting may be possible, in a few a tolerable visual acuity is restored [6, 20, 22, 24, 31, 41, 59, 62]. If there is an altitudinal or inferior quadrant field defect, this finding is in favour of an ischaemic or vascular lesion of the optic nerve [6, 22, 41, 62].

Chiasmal lesions should be suspected clinically if there is a bilateral loss of direct light reflex initially with recovery on one side during the next days/weeks, hemianopic field defects with macular sparing or splitting [1, 6, 13, 22, 31, 47] in long-term observation, and hypothalamic symptoms like transient diabetes insipidus, polyphagia with obesity [1, 13, 39, 47, 48] and, very rarely, sexual delinquency or narcoleptic attacks [48]. Further symptoms found in chiasmal lesions are fractures of the tuberculum sellae [1, 13, 41], CSF-discharge from one nostril or one of the ears [1, 13, 24, 39], see-saw nystagmus [39, 41], anosmia, or involvement of the CN III, VII and VIII. Lesions of the optic chiasm may be prompted by longitudinal shearing up to 3.5 cm [13], ischaemic necrosis and interstitial haemorrhages [5, 31]. The hypothalamic symptoms are attributed to damage of the hypothalamic-hypophyseal tract and the supraoptic/paraventricular nuclei of the hypothalamus [1, 13, 39, 41, 47, 48]. Longstanding PTA has been observed [13, 24, 31, 39, 47, 48] as it was in our 5 patients; they all belonged to group V with a PTA longer than 1 week. Posttraumatic diabetes insipidus and chiasmal lesions are not linked invariably: there are chiasma lesions without diabetes insipidus in 50% [39] and vica versa in 5/18 [48]. OA develops within months in patients with chiasmal lesions [6, 22, 31] and the disk might present with a more yellow colour [39] after peri-parasellar bleedings: yellow OA.

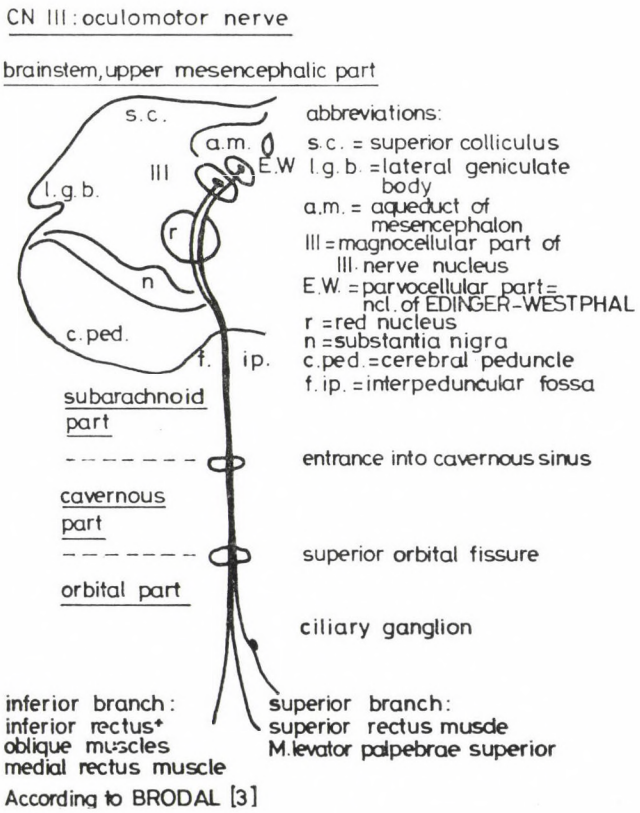
Traumatic damage to the optic tract and lateral geniculate body is exceptional; it is found mostly after penetrating injuries [24, one own case] and also after blunt trauma [5, 6]. It may be followed by OA after months and even years [13]; clinically it presents with homonymous quadrant- or hemianopsia with hemianopic loss of the light reflex and macular splitting [6, 13, 24].

Cortical blindness and damage to the optic radiation occurred in 18 (2.6%) of our patients, but it may have been missed in a few who survived in a vegetative state. Cortical blindness was observed with a short PTA as well as after long PTA at a ratio of 8/10 in our patients; the difference is in the duration of this clinical symptom, which is a few hours or days in the "commotional group" and long, weeks and even months, in the „concussion group". Most patients with posttraumatic cortical blindness have a good prognosis [6, 13, 31, 32] with the exception of penetrating occipital injuries [41]. Cortical blindness is a secondary phenomenon in

most patients, due to circulatory failure in the territory of the posterior cerebral and the anterior choroid artery, for instance during uncal herniation. Cortical blindness in children after trivial trauma with or without PTA might be related to complicated migraine in some cases [13, and some own observations].

CN III, IV and VI: oculomotor, trochlear and abducens nerves

Oculomotor nerve impairment was found most often in our series; CN 15.9% in the acute state, and 5.6% in the chronic course. This finding split up into total ophthalmoplegia in 3.9% (permanent 0.9%), and external ophthalmoplegia in 2.4% (2.5%) which means that some of the total ophthalmoplegias had resolved to partial external ones and internal ophthalmoplegia in 11.6% (2.2% permanently impaired). The III CN might be damaged in its course through the subarachnoid space (Graph 4) by mass lesions or raised ICP lying between the posterior cerebral and the superior cerebellar artery and the tentorial notch; or at its entrance into the cavernous sinus when passing the



GRAPH 4

ridge of the lesser sphenoid wing [1, 44]. When there is a raised ICP to which are due 75% of all IIIrd nerve traumatic lesion [24, 31, 32], the most vulnerable part of the nerve are its parasympathetic fibres lying superior-medially in its cisternal course [52]. If there is a III and VI CN damage without orbital fracture or sinus cavernosus fistula [1] we have to think of raised ICP in every case very carefully [20, 24, 25, 53]. In follow-up, there are some differences in the width of the pupils after uncal herniation in most cases [31, 32]. If loss of the round shape of the iris is observed, nuclear damage within the upper brainstem has most likely occurred [6, 44]. After orbital trauma, branches of the III CN may be impaired: if the medial orbital wall is broken out, there are more oculomotor nerve branches affected while after lateral wall fracture the VI, and after inferior wall fracture, the 2nd division of the V may be impaired [6, 22, 24, 31, 59, 63]. If there is enophthalmus (4 of our patients), it may be progressive at follow-up [59]. Exophthalmus, on the other hand, means a clot formation behind the globe (blood, pus), foreign body in the orbit or, if it is pulsating, encephalocele after blow-out fracture of the lesser sphenoidal wing. If there is a pulsating exophthalmus with bruit, this means a cavernous sinus a.-v. fistula, or in a child with Recklinghausen disease and no or only a faint bruit, a premorbid condition [22, 59, 63]. Recovery after the trauma can be expected in about 75% as far as all eye muscles are concerned [1, 20, 22, 24, 25, 31, 35, 59, 63, 64]; it often occurs within the first weeks and then is most likely be due to raised ICP. When fibre interruption had occurred. misdirectional phenomena are encountered, such as globe retraction on vertical gaze, horizontal gaze lid dyskinesis, adduction of the homolateral eye during upvergence, monocular vertical optokinetic nystagmus and pseudo-Graefe's sign or pseudo-Argyll Robertson pupil phenomenon [1, 31, 52, 63].

Trochlear nerve palsies most often are due to trauma [55, 56, 59]. Of our 5 cases, one was due to penetrating trauma [24], 2 to raised ICP [41] and 2 to orbital fractures [6, 31, 32, 52]. Although the IV CN has the longest course through the subarachnoid space: 75 mm, as compared to the III CN: 20 mm, and the VI CN: 15 mm [34, 38, 52], there is no preponderance of pressure damage. Apart from the 3 causes already mentioned, it might be damaged in fractures of the ala parva of the sphenoid, traumatic a.-v. aneurysms of the intracavernous part of the carotid artery, and in the superior orbital fissure syndrome [1, 31, 32, 41, 59].

Abducens nerve palsies may be uni- or bilateral. Most of these palsies are due to raised ICP and then may disappear within a few days, even when present bilaterally [own observation]. A posttraumatic abducens nerve paresis is not likely to be due to brainstem damage [63, 64]; in the young adult, preexisting disseminated sclerosis or diabetes mellitus [31, 32,

TABLE III

CN-involvement in children after head injury: a) (sub)acute = < 6 months (N: 704)
b) chronic = > 6 months (N: 683)

CN-function concerned	PTA: < 6 mo						> 6 mo					
	I—III	in %	IV+V	in%	N	in %	I—III	in %	IV—V	in %	N	in %
I:olfactory nerve	8	1.6	15	7.3	23	3.2	2	0.4	6	3.4	8	1.2
II: optic nerve: optic atrophy	1	0.2	33*	16.2	34	4.8	1	0.2	33*	16.2	34	4.8
optic nerve: amaurosis	1	0.2	18*	8.8	19	2.7	1	0.2	18*	8.8	19	2.7
cortical blindness	8	1.6	10	4.8	18	2.5	—	—	2	1.1	2	0.4
III: oculomotor nerve	28	5.6	81	39.7	109	15.9	3	0.6	35	19.1	38	5.6
IV: trochlear nerve	—	—	5	2.4	5	0.7	—	—	4	2.2	4	0.6
VI: abducens nerve	9*	1.8	19*	9.3	28*	4t0	2	0.4	5	2.7	7	1.0
III/IV/VI	37	7.4	105	51.5	142	20.2	5	1.0	44	24.0	49	7.0
all oculomotor brainstem signs	1	0.2	50	24.5	51	7.2	—	—	23	12.6	23	3.3
V: trigeminal nerve	1	0.2	29	14.2	30	4.2	1	0.2	14	7.7	15	2.2
VII: facial nerve, lower motor neurone	5	1.0	24	11.8	29	4.1	1	0.2	11	0.6	12	1.7
facial nerve, upper motor neurone	11	2.2	40	19.6	51	7.2	1	0.2	27	14.2	27	3.8
VIII: stato-acoustic nerve	13	2.6	14	6.8	27	3.8	1	0.2	7	3.8	8	1.2
IX—XII: basal CN group	2	0.4	1	0.5	3	0.4	1	0.2	1	0.5	2	0.3
patients within the groups	500		204		704		500		183		683	

* = bilaterally present: optic atrophy: 20/33; amaurosis: 6/18; abducens nerve palsy: 3/9, 3/19, 6/28 respectively

55, 56, 58], in all groups intracranial hypotension (e.g. after lumbar puncture) and phenytoin intoxication [31] should be considered. Apart from pressure on the incisura tentorii, the VIth nerve crosses under the petrosphenoidal ligament and may be sheared by up- or downward acceleration of the whole brainstem during high velocity accidents. Recovery is as good as after IIIrd nerve impairment [31, 52, 59]. After incomplete recovery, overactivity of the antagonistic muscles may take place as in IIIrd nerve muscle impairment [41] so that the corrective operation should not be delayed too long: the topic should be discussed at least 6 months after the trauma [1, 6, 22, 31, 41, 59, 63].

Oculomotor brainstem signs (Table III) most often are found in groups IV and V. Therefore they are indicators of brainstem damage in the acute phase. Among these signs we observed ocular bobbing, skew deviations, quick alternating horizontal movements ("ping-pong sign") and several forms of central nystagmus. Vertical or horizontal gaze paresis may be present with bulbus positions in dysjugate attitude in most cases of head positioning or vestibular testing as do internuclear ophthalmoplegias [1, 29, 41, 51, 63]. In contrast to these

dysjugate movements and positions, every conjugated gaze, deviation ("déviation conjugée") is in favour of hemispheric supratentorial damage on the side to which the eyes deviate, or of an epileptic seizure spreading from one of the adversive fields [19, 25, 27]. When the patient is at the end of coma, very often dysjugate horizontal eye movements are observed as well as convergence spasms [31, 32]. This period of deviant eye movement control shortens after high dose phenobarbital and neurointensive treatment for days, whereas it was observed for weeks with conventional treatment and lasts for months and even years in the apallic syndrome ("vegetative state") [own observation]. Of the oculomotor brainstem signs, 60% disappear within 6 months (Table III), convergence paresis [6, 20] and partial diplopias may present as "blurred vision" later [22, 29, 51]. These findings 6 months after trauma were found only in patients of group V. They have to be regarded as definite brainstem damage, exactly of the medial longitudinal bundle, which connects the oculomotor nuclei with the vestibular and extrapyramidal motor system [34].

CN V, trigeminal nerve

Trigeminal lesions of different kinds occurred in 4.2% in the acute stage, and in 2.2% they were still present after 6 months. In groups IV—VI, diminished or absent corneal reflex was often found (30 patients), but 6 children of group V had unilalater motor impairment, 4 of permanently. These long lasting effects on trigeminal function are in favour of midbrain damage, whereas after orbital trauma branches of the Vth may be severed: supraorbital division of the ophthalmic nerve was observed in 3 cases, or of the nasociliary part which led to neuroparalytic keratitis in 2 cases. The infraorbital branch which is affected in adults after orbital lesions more often [30, 31, 32, 41, 51] than the supraorbital nerve, was not impaired in any of our patients. Lateral crush injuries to the skull may lead to complete trigeminal impairment [1, 30, 41, 64]; they may be followed by zoster eruption [24] or posttraumatic neuralgia [1, 30, 31]. These crushing traumas are followed by complete anaesthesia of the face if the trigeminal nerve is lesioned, whereas loss of pain and temperature sense with retained feeling of touch are due to upper cervical lesions [30, 41, 64] of the descending trigeminal pathway and nuclei [34].

CN VII, facial nerve

VIIth nerve palsies of the lower motor neurone type occurred in 29 of our patients (4.1%) with 12 minor but permanent sequelae. Of these 8 were of the immediate type, in 13 paresis appeared a few days after the trauma. In 13 patients there was blood/CSF discharge from one ear, and 7 had combined lesions of the facial and acoustic nerve. VIIth nerve lesions are related to petrous bone fractures: transverse fractures extend

from the foramen magnum to the petrous bone and run perpendicularly to its longitudinal axis. These amount to 10—20% of all petrous bone fractures [1, 31, 32, 45, 49, 50 ,64] and are linked with VIIth nerve palsy in 30—50%. Many of these patients have cochlear damage, too [45]. Longitudinal fractures of the petrous bone run from the squama ossis temporalis into the mastoid and the petrous bone. Facial nerve impairment occurs in 15—20% after this type of fracture which is often accompanied by initial ear bleeding [1, 15, 18, 31, 35, 45, 46, 49, 50, 64]. The paresis may be prevented by systemic steroid administration if the X-rays show a fracture of the petrous bone and blood discharge occurs from the ear [2, 32]. Good recovery or even full restitution may be expected in about 75—90% of all cases [1, 24, 31, 35, 41, 64], but even after lesions with delayed onset some permanent sequelae may be found [54]. Peripheral facial palsies should be monitored electromyographically: 7—10 days after the injury one is able to diagnose complete or incomplete denervation [15, 18]. In children one should not perform surgical decompression unless there is still total denervation in EMG-testing after 4—6 months [15]; in adults earlier decompression is advocated by otologists [33, 41, 45, 49, 57]. After complete denervation, recovery starts in the frontal muscle, whereas if it is incomplete, the first reinnervation can be observed in the M. orbicularis oculi [50]. Axonal flow from the nuclear area starts after 8 days, and after 3 weeks it reaches the gap where the nerve is damaged [78]. Resprouting of the axons have a speed of about 1 mm/day, which means 6—9 months for the full length of the facial nerve [57]. Misdirection phenomena [45, 50] are observed: narrowing of the palpebral fissure, evident mainly in upward gaze, chin wrinkling at eye closure, or the crocodile tears phenomenon. They run in a short-circuit through the brainstem nucleus of the CN VII and are no cortical phenomena [23]. The general opinion is that infrastyloid lesions have a bad prognosis [24, 31, 32, 57; three own cases].

CN VIII, acoustic nerve

In most children, posttraumatic loss of hearing is due to haematotympanon (10 of 13 own cases with conductive impairment). Sensory hearing loss (10 of our patients) is often linked with transverse fractures of the petrous bone and VIIth nerve palsy (7 patients). Conductive deafness in children has the tendency to improve [1, 19, 46] except if there is an interruption of the ossicle chain [31]. High tone loss may be due to concussion or contusion of the cochlea, but few children are definitely deaf after trauma [14, 19, 25, 46]. The frequency of minimum hearing impairment after trauma depends on the intensity and methods of testing [31, 32] and is related to age [64].

CN VIII, vestibular nerve

We had 4 patients with impaired peripheral vestibular function, all of them recovered within months. Other

authors found 1/1.015 children with definite vestibular loss after HI [46]. According to Jennet and Teasdale [31], 27% of all patients have an abnormal caloric test until the end of the first year after HI. Of this vestibular impairment 2/3 are due to concussion of the end organ, and 1/3 to brainstem dysfunction; blood within the middle ear might reinforce postconcussional symptoms, especially dizziness and vertigo [41]. In children the "post-commotional syndrome" is encountered rarely (17 of 683 patients; 2.5%); the main complaint in this condition is abnormal fatigue and/or headache but no vertigo or dizzy feeling.

CN IX—XII, the basal group

This kind of CN impairment is more often found after missile injuries in the neck [4, 9, 19, 31, 62] than in blunt injuries with fractures passing the jugular foramen or foramen magnum [1, 10, 41, 64]. Some patients may not survive [51]. Although rare, this clinical entity called Collet syndrome [4] consists of homolateral impairment of the CN IX—XII, Horner syndrome, hearing loss and V_3 (mandibular nerve, if the fracture extends to the foramen ovale) dysfunction and nystagmus to the contralateral side. In some patients cord paralysis may lead to dyspnoea responding only to prompt tracheotomy or intubation [9, 41], while others have tachycardia, dysphagia or excessive salivation, symptoms which improve spontaneously during the following weeks [9, 10]. In one patient oesophageal achalasia was reported which responded well to psychotherapy [10]. In 2 patients after blunt injury, Wallenberg syndrome (of the lateral oblongata) was observed which was due to traumatic thrombosis of the posterior inferior cerebellar artery (PICA) [10, 12].

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