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Mass Lesions of the Frontal Lobes in Acute Head Injuries. A Comparison with Temporal Lesions

By

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With 5 Figures

Summary

Contusions and lacerations of the frontal lobes are very frequent; 43.4% in the whole series of traumatic brain mass lesions. Clinical, ICP, CT scan data and neuropathological findings in patients with such lesions are analysed and correlated. Moreover, the clinical features and the outcome of frontal masses undergoing surgery are also compared with similar lesions located in the temporal lobes.

Frontal lesions cannot be differentiated on purely clinical grounds and the factors governing the outcome in both locations are the same. On the whole, surgical indications nowadays seem to be rather rare; only lesions behaving truly as expanding lesions with obvious intracranial hypertension benefiting from surgery.

Brain contusion-laceration syndromes in general can no longer be considered separate entities. Neither should they be included in the miscellaneous group of "traumatic intracranial mass lesions", since the pathophysiological significance of purely extracerebral effusions is entirely different.

Traumatic contusions and lacerations and/or intracerebral haematomas, whether frontal or located elsewhere, should, instead, be considered in the context of head injuries of a different degree of gravity, as having collateral features which, on occasion, may call for surgical management.

Keywords: Head injuries; frontal contusion; temporal contusion; intracranial hypertension; computer assisted tomography; traumatic intracerebral haematoma.

Introduction

In the early phase of the neurosurgical treatment of traumatic cerebral mass lesions (contusions, lacerations and/or intracerebral haematomas), mainly foci located in the temporal poles or in the

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fronto-temporal region were taken into account [6, 17, 21, 37]. Accordingly, a "syndrome of temporal lobe contusion" was described [21], whilst, from the clinical standpoint, much less attention was paid to frontal lobe contusions. Subsequently, it was realized that frontal lesions due to different mechanisms are quite common in neurosurgical as well as in neuropathological series.

The actual incidence of such lesions, in our own and in other authors' experiences [2], seems to have increased steadily since the CT scan has become the screening procedure of choice in neurotraumatology. As a matter of fact, in the pre-CT scan era, frontal contusions with no relevant midline shift and particularly bilaterally symmetric lesions with no shift at all were often overlooked on account of their misleading angiographic appearance. By contrast, temporal lesions never involved any diagnostic problem. It is to be stressed, as well, that the CT scan can reveal a significant number of deeply located haemorrhagic contusions (corpus callosum, basal ganglia, brain stem), as already pointed out long ago by Rowbotham [31] from post-mortem material.

However, even in our first series [27] published as far back as 1964, in which only operated patients were dealt with, frontal and fronto-temporal lesions accounted for 27% of the whole group. Casella *et al.* [3], on the basis of 150 personal observations, encountered purely frontal lacerations in 18% and fronto-temporal foci in 22%. In the most recent angiographic series [16] the number of frontal lesions has increased to 37% plus an additional 5% of fronto-temporal haematomas. In the very large clinical material recently reported by Vigouroux and Guillermain [40]. 40% of the patients had frontal or fronto-temporal lesions (29% frontal and 11% fronto-temporal).

At the Regional General Hospital of Ancona so far we have operated on 108 patients with traumatic brain mass lesions, 34 of whom had frontal contusions, lacerations and/or intracerebral haematomas. On occasions intracerebral masses were associated with skull lesions and/or to extracerebral effusions.

The high rate of occurrence of frontal lesions has further increased to 43.4% since the CT scan was introduced to neurotraumatology. In Bousigue's [2] CT scan material the frontal lobe seems even more frequently involved than the temporal: 71 frontal contusions versus 55 temporal contusions. As to the number of patients, frontal contusions were encountered in 39%, temporal contusions in 38%, whilst double or multiple contusions were present in 37%. By and large, these data are supported by the neuropathological findings reported by Ch. Espagno [9, 10].

On these grounds, we think that the problems related to traumatic

frontal lobe lesions deserve a more thorough analysis. The main question that arises is whether they really have separate clinical features, prognosis and therapy or whether they are, instead, only an aspect of traumatic brain damage devoid of any specificity. In order to try to answer to this question we have reviewed our clinical and neuropathological material.

Patient Population and Methods

Criteria for Surgical Intervention

With regard to the criteria for surgical intervention in patients with traumatic intracerebral lesions, we can distinguish three phases:

First phase: Only severely ill patients were submitted to an angiographic examination and whenever a well-defined space-occupying lesion was disclosed, immediate surgery was carried out with rare exceptions.

Second phase: Most of the patients who were already deeply comatose in the first 12 hours after injury were deemed unsuitable for surgery. Moreover, patients with less severe impairment of consciousness and stagnant symptomatology were often treated conservatively.

Third phase: This commenced when the CT scan became the routine screening procedure in unselected patients. The overall number of lesions disclosed in this way has definitely increased. Nevertheless, a number of lesions have been almost "silent".

Long-term ICP monitoring as criterion for making a therapeutic decision was often used in the past few years.

On the whole, while on the one hand traumatic brain expanding lesions are nowadays more frequently detected, on the other, the surgical indications appear to be more and more limited. At least in our series, the majority of patients were operated during the pre-CT scan epoch.

Summary of Patients

Our clinical material consists of three groups of patients:

1. The patients with frontal lesions undergoing surgery over the past years (34 cases).

2. The overall series of cases with traumatic cerebral mass lesions diagnosed by CT scanning in the past three years (83 cases). In this group all the mass lesions in the brain are included whatever their location. Altogether 36 patients had frontal lesions.

3. Clinical, ICP and CT scan data were correlated in patients with purely parenchymal frontal lesions (21 cases). In all the patients long-term ICP recording was implemented by Lundberg's intraventricular technique. The highest sustained mean ICP values measured during active therapy (osmotic, assisted ventilation when necessary, etc.) were noted.

For comparison, the data concerning the epidemiology, clinical features and the outcome of temporal lesions observed and operated on in the same periods are summarized as well.

Finally, he have also examined the post-mortem findings in 17 patients diagnosed as having relevant frontal lesions on the basis of the CT appearance.

In classifying the patients clinically, we have used the Glasgow Coma Scale [30]. So, we distinguish:

A) deeply comatose patients (3 to 5 points on G.C.S.),

B) patients with intermediate disturbances of consciousness (6 to 10 points),

C) patients with minor impairment of consciousness (more than 10 points).

Group 1: Patients submitted to surgery.

The *patients with frontal lesions* are reported in Table 1.

Of the 7 patients who were already deeply comatose in the first 24 hours only 2 showed a clear-cut period of lucidity.

Table 1. *Overall Results of Surgery. I. Frontal Lesions*

Coma rating	Associated surg. lesions	Early surgery 24 h	24–72 h surgery	Delayed surgery	Alive	Dead	Total
3 to 5 points	—	+			—	7	7
3 to 5 points	—		+		—	3	3
6 to 10 points	—		+		1	3	4
6 to 10 points	—			+	3	2 *	5
10 points	—			+	2		2
3 to 5 points	epidural haem.	+			2	1	3
3 to 5 points	acute subdural haem.	+			2	3	5
10 points	comp. depressed fractures			+	4	1	5
Total					14	20	34

* Death from extracerebral complications.

Early surgery was ineffective for the entire group: no obvious clinical benefit ensued and all patients died.

Three additional deeply comatose patients were operated upon at a later stage (24 to 72 hours after injury). None benefitted from surgery.

Four patients with intermediate disturbances of consciousness were operated on within 36–48 hours of injury. One recovered very slowly and the remainder died (one from uncontrollable intracranial hypertension and 2 from late extracerebral complications. The latter patients were never responsive).

Five patients with intermediate disturbances of consciousness underwent delayed surgery (5 to 12 days after injury): three re-

covered uneventfully and 2 died from late extracerebral complications.

Two patients with minor impairment of consciousness were operated on 5 and 8 days after injury respectively. Both made a satisfactory recovery.

In 8 deeply comatose patients the parenchymal lesions were associated with extracerebral effusions (epidural haematoma in 3 and

Table 2. *Overall Results of Surgery. II. Temporal Lesions*

Coma rating	Associated surg. lesions	Early surgery 24 h	24–72 h surgery	Delayed surgery	Alive	Dead	Total
3 to 5 points	—	+			—	13	13
3 to 5 points	—		+		2	12	14
3 to 5 points	—			+	2	4	6
6 to 10 points	—	+			1 *	1	2
6 to 10 points	—		+		3	2 **	5
6 to 10 points	—			+	3	2 **	5
10 points	—	+			1	—	1
10 points	—		+		11 ***	—	11 *
3 to 5 points	epidural haem.	+			1	—	1
3 to 5 points	acute subd. haem.	+			3	6	9
3 to 5 points	acute subd. haem.		+		2	—	2
10 points	comp. depressed fractures	+			1	—	1
Total					30	40	70

* Severely disabled.

** Death from extracerebral complications.

*** In two patients only a thin subdural haematoma was removed through a small craniectomy.

an acute subdural haematoma in 5). All patients were operated on within 24 hours of injury. Two of the 3 patients with epidural haematoma and 2 of the 5 patients with subdural haematomas survived.

Finally, 5 patients with open compound depressed fractures and large frontal lacerations but with minor disturbances of consciousness were operated on 4 to 6 days after injury. Four patients recovered and 1 died from septic complications.

During the same period, 70 patients with *temporal lesions* were operated on. The breakdown is as follows (Table 2).

Table 3. *Patients with ICP Monitoring. I. Frontal Lesions*

No.	Sex	Age	Lesion	Coma rating	ICP	Therapy	Post-op. ICP	Outcome
1	M	17	F BL	A	60	op. 2nd day	35	died 10th day
2	M	17	F BL	A	70	medical	—	died 5th day
3	F	23	F BL	B	40	op. 2nd day	40	recovered
4	M	31	bil. F BL	B	25	medical	—	died (sepsis)
5	M	62	F BL	B	10	medical	—	recovered
6	M	52	F H	B	25	op. 5th day	15	died (bronchopn.)
7	M	45	F H	B	45	op. 2nd day	50	died 10th day
8	M	16	FT BL	B	40	barb. COMA	—	recovered
9	M	56	F BL	B	45	VD 4th day	15	recovered
10	M	67	F H	B	45	VD + op. 10th day	10	died (pulm. emb.)
11	M	73	F BL	B	60	VD 2nd day	—	died (pulm. emb.)
12	M	21	F BL	B	70	op. 3rd day	30	died (bronchopn.)
13	M	33	F BL	B	40	medical	—	recovered
14	M	27	bil. F BL	B	20	medical	—	died 5th day
15	M	53	F BL	B	40	VD + op. 10th day	15	recovered
16	M	33	F BL	C	40	medical	—	recovered
17	M	40	F BL	C	50	VD 5th day	15	recovered
18	M	58	bil. F BL	C	10	medical	—	recovered
19	M	65	F BL	C	30	VD 4th day	15	recovered
20	M	48	F BL	C	65	VD 10th day	20	died 27 th day (septic renal failure)
21	M	57	F BL	C	15	medical	—	recovered

Abbreviations: A = 3 to 5 points on G.C.S., B = 6 to 10 points on G.C.S., C = 10 points on G.C.S., F = frontal, T = temporal, BL = brain lacerations, H = intracerebral haematoma, VD = ventricular drainage.

If *we compare the results obtained in both series* we see that they correspond closely. In actual fact, all the patients with purely parenchymal lesions who were in deep coma and were operated on within 24 hours of injury died. Only patients with extracerebral effusions, whether subdural or epidural, managed to survive under the same conditions.

The survival rate was likewise very low in deeply comatose patients operated on later, namely between 24 hours and 72 hours after injury.

Table 4. *Patients with ICP Monitoring. II. Temporal Lesions*

No.	Sex	Age	Lesion	Coma rating	ICP	Therapy	Post-op. ICP	Outcome
1	M	17	T BL	A	60	op. 2nd day	30	died 10th day
2	M	42	T BL	A	60	op. 1st day	25	died 7th day
3	M	20	T BL	A	60	op. 1st day	30	died 5th day
4	M	32	T BL	A	70	medical	—	died 3rd day
5	M	68	T BL	A	50	op. 3rd day	45	died 20th day (bronchopn.)
6	M	77	T BL	A	55	op. 4th day	—	died after operation
7	M	17	T BL	B	40	op. 6th day	35	recovered
8	F	16	T BL	B	40	op. 2nd day	30	recovered
9	M	20	bil. T BL	B	20	medical	—	recovered
10	M	59	bil. T BL	B	15	medical	—	died 20th day (pulm. emb.)
11	M	67	bil. T BL	B	45	VD 2nd day	—	died 6th day (pulm. emb.)
12	M	56	T BL	B	15	medical	—	recovered
13	M	73	T BL	B	20	medical	—	died 7th day (bronchopn.)
14	M	48	T H	C	45	op. 12th day	20	recovered
15	M	47	T H	C	45	op. 3rd day	15	recovered
16	M	64	T H	C	60	op. 5th day	15	recovered
17	M	64	T BL	C	45	op. 7th day (decompr.)	10	recovered
18	M	40	T H	C	40	op. 9th day	35	recovered
19	F	17	T BL	C	20	medical	—	recovered
20	F	75	T BL	C	45	medical	—	recovered
21	F	55	T BL	C	55	VD 4th day	15	recovered
22	M	52	T BL	C	15	medical	—	died 10th day (hepatic failure)
23	M	58	T H	C	40	EVD + op. 8th day	18	recovered

Abbreviations: A = 3 to 5 points on G.C.S., B = 6 to 10 points on G.C.S., C = 10 points on G.C.S., T = temporal, BL = brain laceration, H = intracranial haematoma, VD = ventricular drainage.

In patients with intermediate disturbances of consciousness the postoperative mortality rate was still considerable, mostly from extracerebral complications which took place at a later stage in patients who had not definitely improved after operation.

Finally, in patients with a minor impairment of consciousness (more than 10 points on G.C.S.) there was no mortality. All these patients but one underwent surgery more than 3 days after injury.

Group 2: Overall series of patients diagnosed by CT scanning. This series provides the most reliable review of the present status of the whole problem in epidemiological as well as in clinical terms.

In the past three years 83 patients were diagnosed by CT scanning as having traumatic intracerebral masses. In the whole group 36 patients (43.4%) showed frontal or fronto-temporal lesions. Four patients exhibited coup-contrecoup lesions in the temporal and in contralateral frontal lobe.

Only 10 patients with frontal masses were operated on and in 4 of them early salvage procedures were attempted with no benefit.

Altogether, in one third (15 patients out of 47) of the cases with temporal masses surgical procedures were performed.

Group 3: Patients with purely parenchymal lesions in whom clinical, ICP and CT scan data were correlated (Tables 3 and 4).

The patients in whom additional information was required to make a therapeutic decision were more thoroughly studied. No patients with an extracerebral effusion are included in this group. Moribund patients are excluded as well. ICP monitoring was never performed in cases with a definite tendency towards deterioration or improvement.

Altogether 7 patients with frontal lesions were operated on and 7 were submitted to continuous EVD.

13 patients with temporal lesions underwent surgery and 4 were treated by continuous EDV.

On the whole, the data recorded in the patients with temporal lesions (Table 4) are broadly in line with those concerning the frontal masses listed in Table 3.

As regards ICP behaviour specifically, it is noteworthy that on occasions high ICP values were well tolerated. On the other hand, some patients with huge lesions, either in the frontal or in the temporal lobes (Table 3 cases 5, 18, and 21; Table 4 cases 12 and 19), exhibited a normal or slightly raised ICP and mild symptomatology. It to be stressed, as well, that in the latter patients the CT scan examination disclosed relevant midline shifts (up to 2.5 cm) which lasted for some weeks.

Comment

On the whole, in terms of outcome related either to coma scoring or the operative timing, our experience broadly coincides with Vigouroux and Guillermain's [40]. These authors have recently reported

one the largest series (833 cases) published in literature. However, in Vigouroux and Guillermain's patients, the overall mortality after surgery was somewhat lower in the patients with frontal lesions than those with temporal lesions, 41.5 and 47% respectively.

Neuropathological Findings

Post-mortem data collected from 17 patients are available. Fourteen patients died without speaking. One patient with a huge frontal clot exhibited a clear-cut 3-hour period of lucidity. An additional patient who also had a large epidural frontal effusion had a short period of lucidity followed by a state of deep coma with bilateral pupil unresponsiveness (3 points on G.C.S.). Finally, one patient, after several days of coma, became alert and responsive but eventually died from sepsis and renal failure.

Altogether, 6 patients with the deepest degrees of coma (3 to 4 points on G.C.S.) succumbed to uncontrollable fulminating intracranial hypertension within 72 hours of injury 10 patients died at a later stage, mostly from extracerebral complications; at any rate none of them at any time scored more than 8 to 9 points in the G.C.S.

The neuropathological findings in our cases with parenchymal frontal lesions can be listed under the following headings:

a) Morphology and location of the frontal lesion.
b) Skull lesions.
c) Extracerebral effusions associated with brain lesions.
d) Other lesions due to primary impact located elsewhere.
e) Secondary lesions caused by intracranial hypertension.
f) Microscopic lesions.

a) We have observed purely baso-frontal lesions in 8 patients, convexity haemorrhagic lesions in 4 (Fig. 1), separate lesions located both in the basal portion and in the convexity of the frontal lobe in 2 cases, polar contusion in 2, baso-frontal and temporo-polar contusion in 1. In 11 patients out of 17 the frontal lesions were bilateral and almost symmetrical. The size of the lesions was relatively small (2×2.5 cm) in some patients and extremely large (up to 7×9 cm) in others. In all cases the lesions were surrounded by perifocal brain oedema. In 3 cases the head of the nucleus caudatus was involved (Figs. 2 a and b). In 3 cases the haemorrhagic lesions reached the genu and the first portion of the corpus callosum and in one case the putamen, the anterior thalamus and the hypothalamus were disrupted as well (Figs. 2 a and b). In a patient who was conscious a small haemorrhagic lesion associated with a large

ischaemic area was encountered. The CT scan had disclosed a large hypodense area occupying almost the whole of the frontal lobe with a 1 cm midline shift.

b) Frontal fractures were encountered in 6 cases. In one of them the floor of the frontal fossa was dislocated by a compound fracture with multiple fragments penetrating the frontal lobe. In one case there was a long temporal fracture involving the floor of both middle fossae. In one case there was a long occipital fracture.

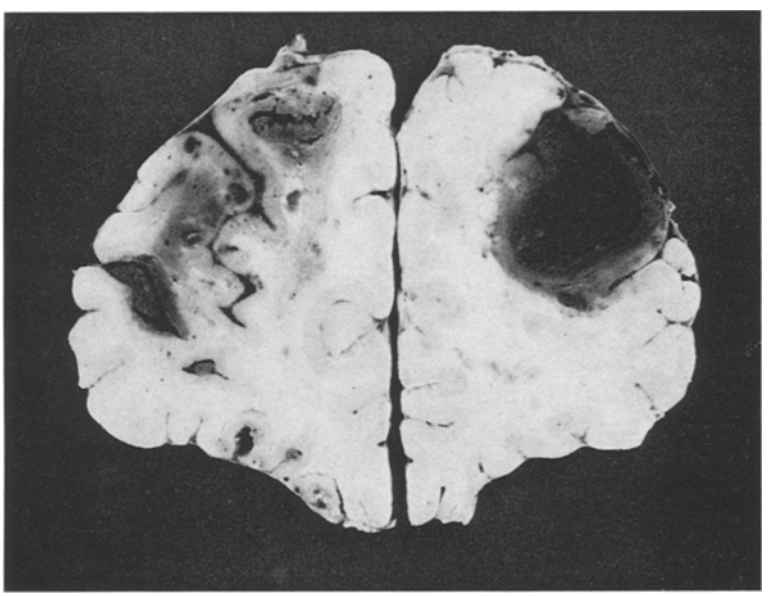

Fig. 1. Bilateral haemorrhagic contusion in the convexity of the frontal lobes (case 3)

c) In 3 cases epidural clots were removed at operation and in 6 additional cases subdural effusions were evacuated. In one patient thin bilateral subdural haematoma (0.7 cm thick) were encountered.

d) Additional large haemorrhagic contusions located elsewhere were present in 8 cases. In 6 patients the temporal lobe was involved to a greater or a lesser extent: in one case symmetrical foci in the internal capsule were present as well. In the last case a large lesion, partly haemorrhagic and partly ischaemic, was observed in the capsulo-pallidal region. In one case there were symmetrical haemorrhages in the nuclei caudati. In 2 the associated lesions were located in the parietal lobes and in 2 additional cases haemorrhagic contusions were found in the cerebellar hemispheres. In addition, the corpus

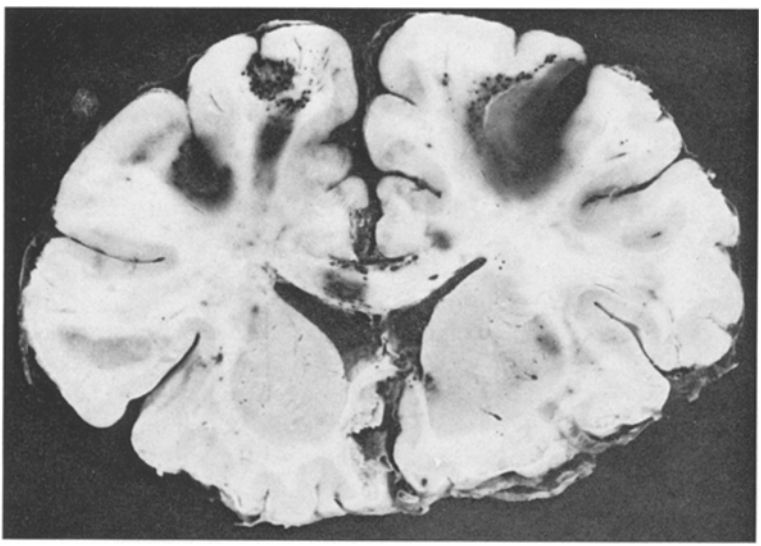

Fig. 2 a. Bilateral subcortical haemorrhagic contusions, multiple haemorrhagic lesions in the corpus callosum, in the left portion of the fornix and in the left nucleus caudatus

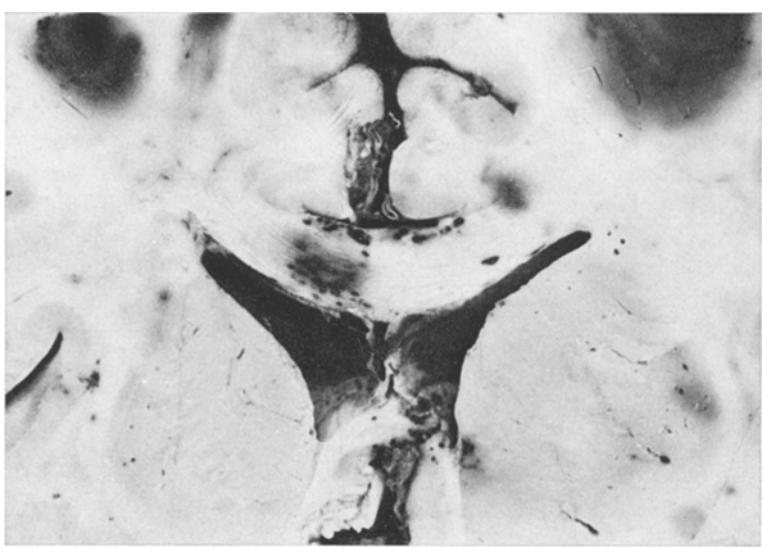

Fig. 2 b. At higher magnification the lesions in the fornix are clearly visible

callosum was damaged by contusional haemorrhages in 4 cases (Figs. 2 a and b). In one patient multiple haemorrhagic foci and areas of softening were located in the hypothalamus (mamillary body), in the crus and in the centrum semi-ovalis of both hemispheres. In one case a large area of softening involved one capsula interna. Multiple small cortical necroses were observed in nearly all cases.

e) Inasmuch as some degree of intracranial hypertension developed in all patients secondary lesions were constant. In all the 6 patients who succumbed to fulminating intracranial hypertension severe bilateral hyppocampal herniation with noticeable cortical necrosis were encountered. Moreover, in 3 of these cases the brain-stem was distorted and large haemorrhages in the median raphe were disclosed. Finally, in one case a bilateral haemorrhagic necrosis of both cerebellar tonsils was present as well. Bilateral uncal herniation was likewise found in 3 additional patients who had exhibited less severe degrees of intracranial hypertension and died later from extracerebral complications. Extensive cortical ischaemic lesions were found in 6 patients. Smaller degrees of uncal and hyppocampal herniation were noticed in all the remaining patients who died at a later stage.

Microscopic Examination

We shall confine ourselves to describing a few selected observations concerning patients in whom the CT scan had depicted well-limited and apparently isolated haemorrhagic frontal contusions. None of these patients succumbed to fulminating intracranial hypertension and all died later of extracerebral complications. One had a three-hour period of lucidity before becoming deeply comatose. He was operated on early and the frontal clot removed. The remainder exhibited only intermediate disturbances of consciousness but were never responsive. In both cases intracranial hypertension was managed by EVD and in one of them the frontal clot was subsequently evacuated.

Case 1

M. M., a 68-year-old man, was kicked by a cow in the fronto-parietal region. After a three-hour period of lucidity he became rapidly comatose (4 points on G.C.S.). In the results of the CT scan a large left haemorrhagic frontal laceration with 2 cm midline shift was disclosed. Surgery was immediately performed and a large frontal clot was removed and the surrounding contused and swollen brain resected. Only a slight clinical improvement ensued and the patient remained scarcely reactive (5 to 6 points on G.C.S.), dying 10 days after injury from bronchopneumonia.

At post-mortem a very large haemorrhagic left frontal lesion was found, impinging on the nucleus caudatus and the hypothalamus, with blood penetration of the ventricle (Figs. 3 a and b). No gross lesions of the brain-stem were encountered.

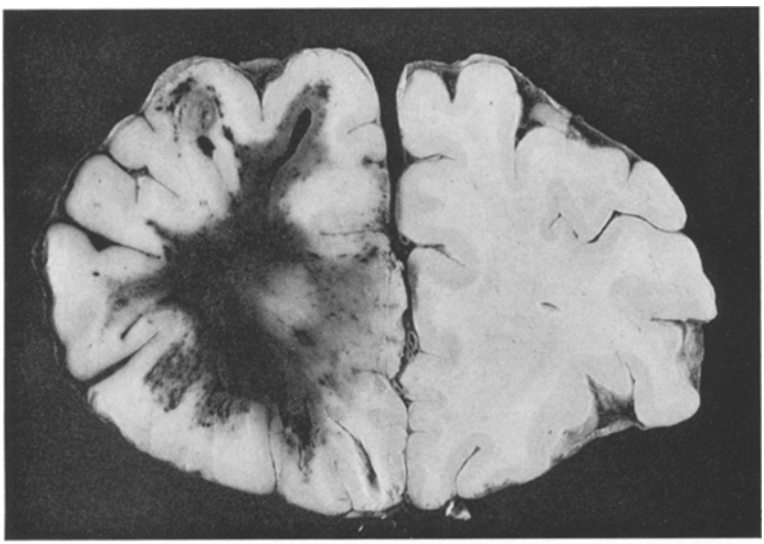

Fig. 3 a. Large haemorrhagic contusion (case 1)

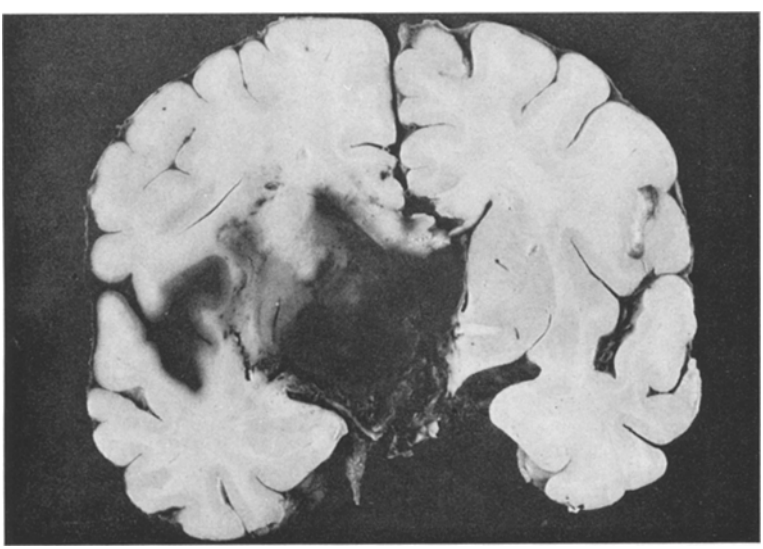

Fig. 3 b. The haemorrhagic lesion also involves the hypothalamus and the nucleus caudatus

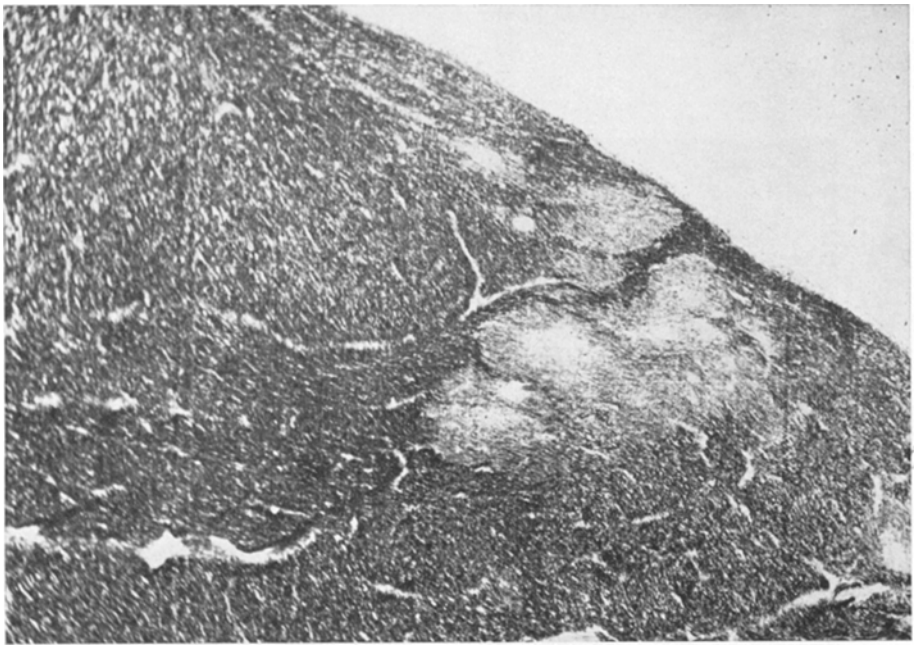

Fig. 3 c. Ischaemic lesions in the corpus callosum (Luxol fast blue 2.5×)

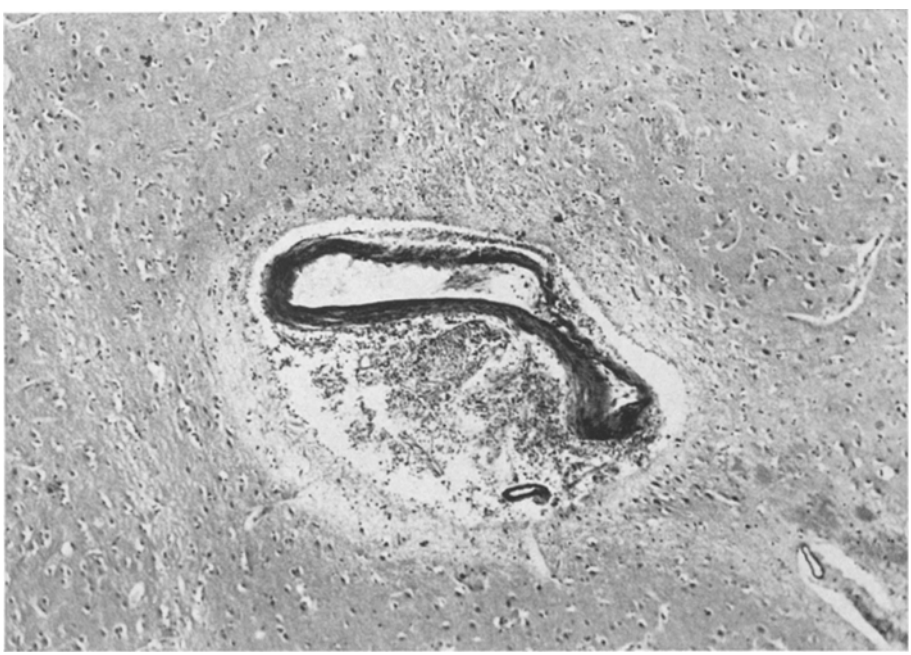

Fig. 3 d. Subcortical white matter: marked enlargement of the perivascular space with a cuff of erythrocytes and macrophages (Haematoxylin-Eosin 16×)

Microscopic examination: Scattered ischaemic microinfarts in the corpus callosum (Fig. 3 c) and widespread foci of cortical necrosis were seen. Marked enlargment of the perivascular spaces with cuffs containing erithrocytes and macrophages (Fig. 3 d) were evident together with microglial clusters and retraction balls in the corpus callosum. Microfocal necrosis was present in the medial lemniscus (Fig. 3 e).

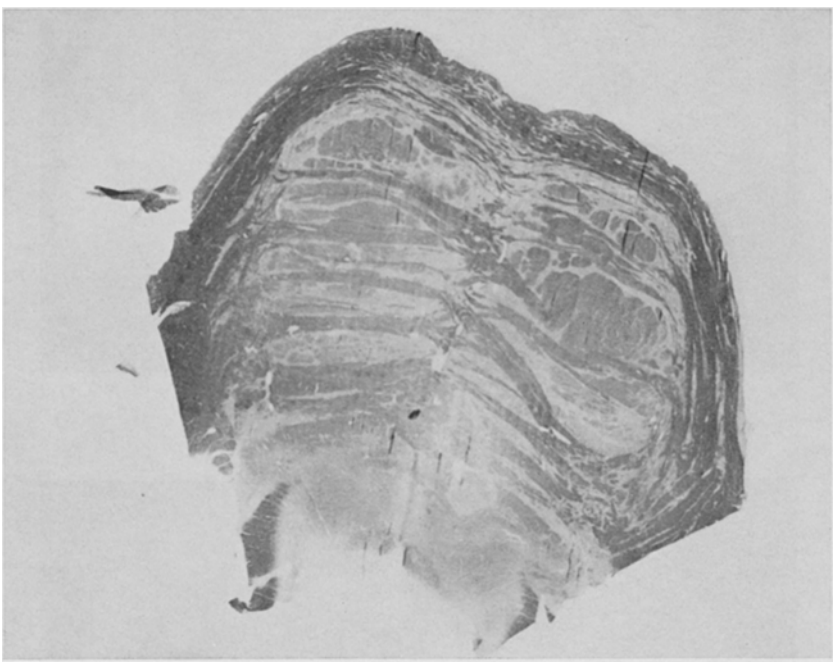

Fig. 3 e. Microfocal necrosis in the medial lemniscus (Luxol fast blue)

Case 2 (Table 3, case 10)

P. G., a 63-year-old man, was struck by a car. After injury he remained confused but the state of consciousness deteriorated in the hours following the accident. On admission, 8 hours after injury, he showed intermediate disturbances of consciousness (7 to 8 points on G.C.S.). The CT disclosed a large right frontobasal haemorrhagic contusion with a 2 cm midline shift. ICP was measured at once and since it was very high (50 mm Hg), continuous EVD was instituted and maintained for 8 days with an exit pressure of 20–25 mm Hg. The clinical condition remained unchanged. On discontinuing drainage, the ICP was unmodified and therefore the frontal clot was removed 10 days after injury. Intracranial hypertension slowly subsided but the patient did not show any definite improvement, remaining akinetic. He died 22 days after injury of a massive pulmonary embolism.

At post-mortem the findings in the brain can be summarized as follows: There was a large haemorrhagic contusion of the right frontal lobe (Fig. 4 a), a necrosis

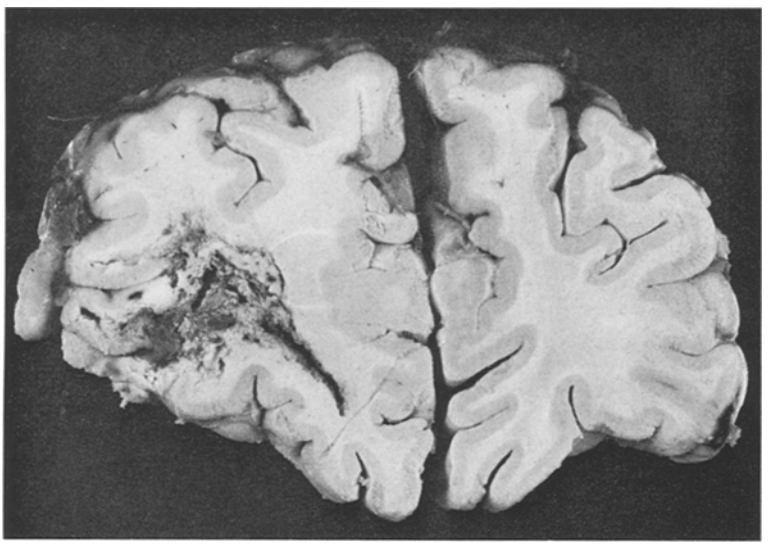

Fig. 4 a. Frontobasal haemorrhagic contusion (clot removed surgically 12 days previously) (case 2)

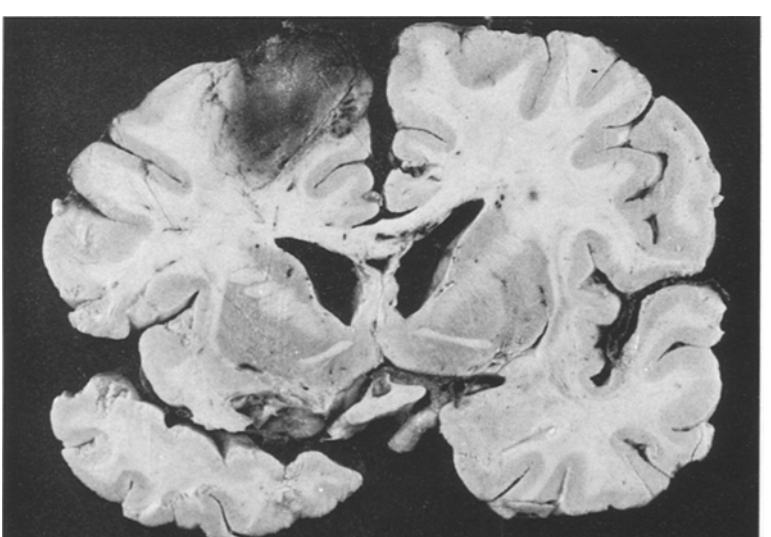

Fig. 4 b. Haemorrhagic contusion in the right frontal convexity (not revealed by the CT scan). Haemorrhagic lesions in the corpus callosum

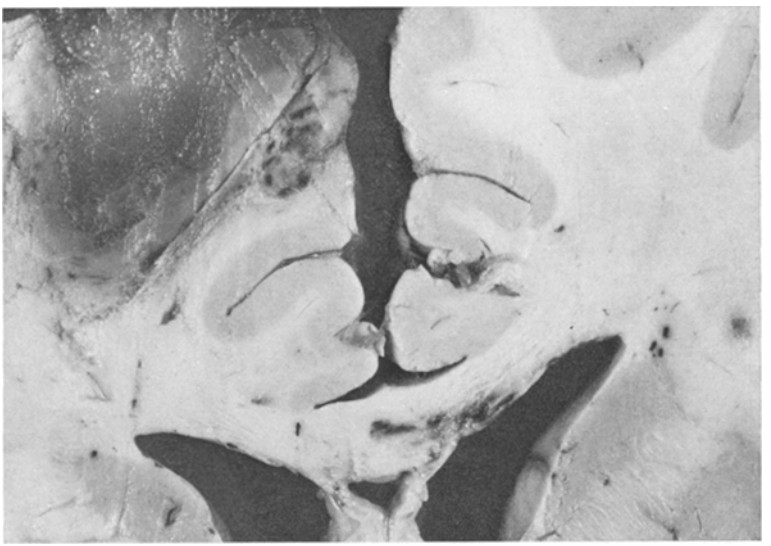

Fig. 4 c. The callosal haemorrhages at higher magnification

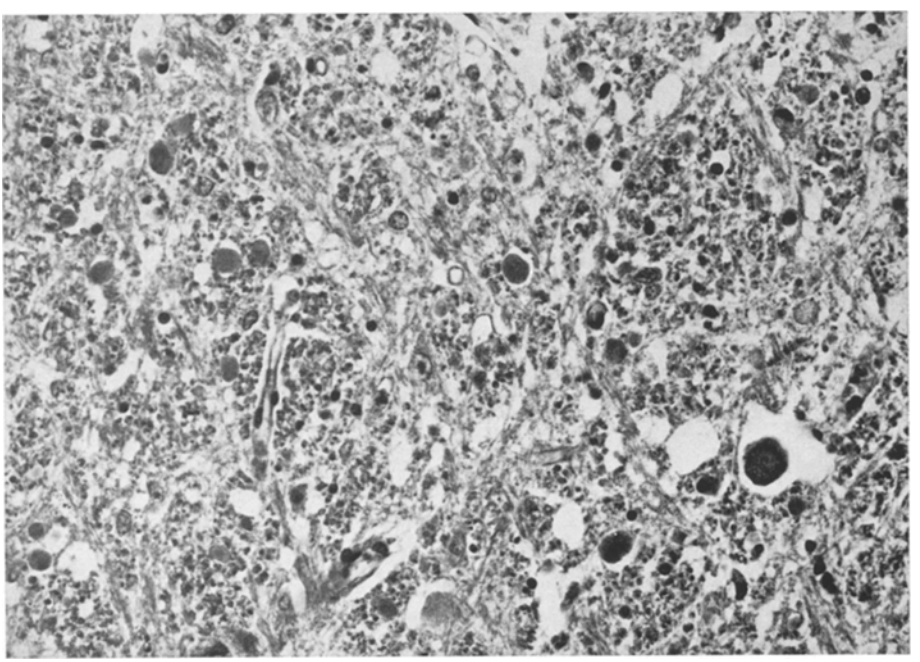

Fig. 4 d. Retraction balls in the mamillary body (Haematoxylin-Eosin 40×)

of the left upper frontal gyrus (Figs. 4 b and c), haemorrhagic foci in the corpus callosum (Fig. 4 c) and ischaemic softening in the right internal capsula area. No gross lesions in the brain-stem were noticed.

Microscopic Examination: There was an ischaemic infarction in the right median nucleus of the thalamus and an ischaemic lesion of the upper hypothalamus. Widespread cortical necrosis was present in the right hyppocampal gyrus and in the uncus. Microglial stars and retraction balls were seen both in the left mamillary

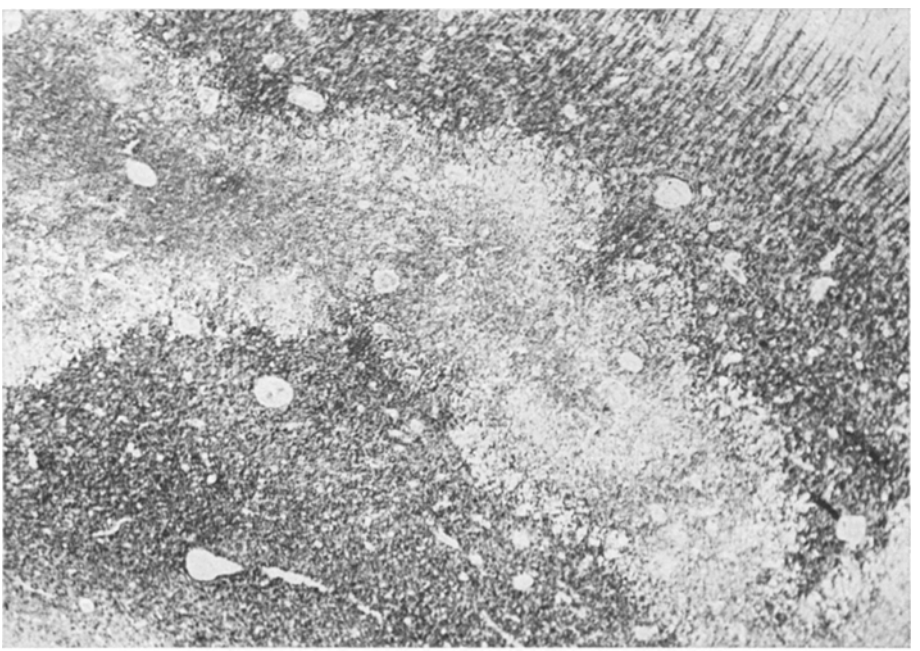

Fig. 5. Ischaemic lesion in the radiations of the corpus callosum (Luxol fast blue 2.5×)

body (Fig. 4 d) and throughout the pons and midbrain tegmentum. Extensive cortical and subcortical necroses were present in the frontal lobe and diffuse cortical ischaemic lesions in all sections of both hemispheres.

Case 3 (Table 3, case 11)

A. P., a 73-year-old man, was admitted with intermediate disturbances of consciousness (7 points on G.C.S.) 2 days after a road accident. A CT scan revealed a thin bilateral subdural effusion and a symmetric haemorrhagic contusion in both frontal lobes. The ICP was very high (60 mm Hg) and therefore EVD was immediately instituted. The ICP was maintained between 20 and 25 mm Hg. The clinical condition was not modified and the patient died 12 days after injury from bronchopneumonia and pulmonary embolism.

At post-mortem:

Gross Examination: A thin bilateral subdural hematoma (0.7 cm thick) was seen. There were bilateral haemorrhagic frontal lacerations and on the right

side the lateral portion of the corpus callosum was directly involved (Fig. 1). Bilateral hyppocampal herniation was present but no gross lesions of the brainstem.

Microscopic Examination: Multiple cortical necroses were evident in both frontal lobes with white matter ischaemic lesions throughout the frontal lobes. There was both softening of the radiations of the corpus callosum (Fig. 5) and of the left centrum semiovale. Widespread foci of cortical necrosis were seen in the left temporal lobe and ischaemic necrosis of the white matter of the temporal lobe. Cortical microinfarction was present in the left uncus and a paramedian ischaemic necrosis in the lower pons with astrocytic gliosis, microglial stars and retraction balls.

Comment

The observations we have reported clearly indicate the extension of the microscopic lesions that accompany apparently well-limited frontal lacerations. It is very likely, as many authors have repeatedly stressed, that such lesions often play a crucial role in determining neurological disorders and, in particular, impairment of consciousness. The question as to whether these alteration are due to the primary impact or to vascular disorders related or unrelated to intracranial hypertension nearly always remains unsolved. In any case, the focal mass lesions depicted by the CT scan are often only the most impressive aspect of a much more complicated neuropathological situation.

Discussion

Multiple mechanisms (direct impact, different dynamic forces such as acceleration-deceleration, rotation etc.) are responsible for brain contusion-laceration. A great deal of experimental research [7, 8, 11—15, 23—25, 30, 38] has been carried out to elucidate these biomechanical effects on the brain.

As Espagno [9] sums up, the biomechanical phenomena caused by craniocerebral injuries can be grossly classified into two main groups:

a) Static shocks, which are relatively rare in practice. They only cause local phenomena of bone distortion and, consequently, direct brain lesions. Only if the whole skull is deformed may diffuse brain lesions ensue.

b) Dynamic shocks with head displacement, which are far more frequent than the former and give rise to local as well as diffuse lesions.

In our neuropathological material no cases of lesions from direct impact were included. Only two patients were lucid and then died; the remainder were never responsive during the whole entire course

of their illness. In all cases at post-mortem multiple lesions, presumably due to the primary impact, were encountered in addition to secondary lesions from intracranial hypertension. In none of our patients who underwent necropsy was a purely frontal lesion observed. In our observations, therefore, the disturbances of consciousness and neurological deterioration may be ascribed both to widespread lesions and to intracranial hypertension brought about by the frontal masses.

In about two thirds of the patients the frontal lesions were bilateral. It is likewise noteworthy that frontal convexity lesions seem to be more frequent than previously realized.

Our neuropathological findings are broadly similar to the data reported in literature. Ch. Espagno [9, 10] observed bilateral contusions in 64.15% of his cases and lesions of the corpus callosum in 38.5%. Chodkievicz [5] found bilateral symmetric contusions in 47% of 52 patients operated on for supposedly unilateral brain contusions on angiographical grounds.

From CT scans, Lanksch *et al.* [20] and Bousigue [2] lay emphasis on the common incidence of large multiple lesions located in all areas in the brain. Moreover, several associated lesions are not detectable in the CT scan. Adams *et al.* [1] point out that the outcome depends to a great extent on diffuse white matter lesions caused by shearing forces developed at the very moment of the primary impact [4, 22, 26, 33–35]. Snoek *et al.* [32] have recently described widespread lesions even in patients with no intracranial haematomas. We have also observed such lesions in all patients who died at a later stage.

For these reasons, in many cases the CT scan alone may not account for the poor outcome. Furthermore, in the specific case of frontal contusions and lacerations, the CT scan may not be entirely reliable in outlining the posterior limits of the lesions and particularly their relation to the basal ganglia, corpus callosum and hypothalamus. As a matter of fact, at post-mortem we have seen that apparently lobar lesions sometimes definitely impinge on the anterior third of the corpus callosum, the nucleus caudatus, the septal region and hypothalamus and so forth. Anyway, Lanksch *et al.* [20] properly emphasize that the CT scan is only a part of the diagnostic effort and cannot replace critical evaluation of neurological symptoms and signs. They maintain that the demonstration of a large contusional haemorrhage does not constitute adequate grounds for surgical intervention.

From the pathophysiological standpoint, the role played by the focal parenchymal lesions themselves, whether frontal or located elsewhere, in giving rise to acute intracranial hypertension with pressure

gradients, brain shift and brain-stem distortion, is fundamentally different from that of purely extracerebral effusions.

In actual fact, the latter behave as truly expanding lesions akin to inflatable balloons used in the experimental animal to produce a rapidly decompensating intracranial hypertension with all its allied phenomena. By contrast, in the very early phase after head injury the role of parenchymal contusion-lacerations is not so obvious. In the first hours they can seldom account for the acutely rising intracranial hypertension with deep coma and signs of brain-stem dysfunction. In the vast majority of cases the focal lesion is nothing but the most impressive feature of diffuse brain damage with widespread vasomotor disturbances due to severe impairment of autoregulation. In clinical practice, when operating on very acute cases, we notice that after removing the necrotic tissue the surrounding brain remains swollen or even keeps on swelling further. The cerebral tissue is hyperaemic with red veins and it makes haemostasis extremely difficult and time-consuming. Even the most extensive decompression very seldom manages to control intracranial hypertension. Moreover, even though the ICP may on occasion be definitely reduced by surgery the outcome nevertheless, always remains very poor.

In a large percentage of cases extracerebral effusions are associated to brain contusions [40]. Even in such cases, in which the surgical decision is dictated by the presence of extracerebral effusions [40], the ultimate outcome depends to a greater extent on the entity of the associated brain damage. We have observed that in deeply comatose patients, the outcome was definitely poorer in cases with purely parenchymal lesions: none of our patients survived after operation carried out during the early stage following injury. Conversely, some comatose patients with extracerebral effusions associated with large parenchymal contusions made a satisfactory recovery after early surgery. In the latter cases, therefore, the state of coma can presumably be attributed mainly to extracerebral effusions. Moreover, very thin extracerebral effusions may cause a rapid clinical deterioration in the patient in whom large cerebral foci have been well tolerated for some days. The surgical evacuation of an incompressible extracerebral mass is followed by dramatic clinical improvement and a fall in the previously raised ICP to normal levels. Much emphasis must be laid, therefore, on the severe effects that minimal extracerebral masses may produce when the breakpoint of the pressure/volume curve is definitely displaced to the left and, consequently, brain elastance is elevated. Cerebral contusion-lacerations and/or haematomas may behave as purely or almost purely expanding lesions at a later stage, because of the size of the lesions, perifocal oedema and circulatory

disorders. Lanksch *et al.* [20] have observed by performing sequential CT scans, that an increase in the midline shift accompanying the deterioration in the neurological status results from the expansion of brain oedema and not from the extension of the haemorrhage itself. On account of increasing intracranial hypertension, these patients may deteriorate rapidly or develop subacute syndroms making surgery necessary. Nevertheless, the role that disturbances in CSF dynamics may play in maintaining long-standing intracranial hypertension should not be disregarded [28, 29].

In assessing the significance of traumatic intracerebral mass lesions in raising the ICP, two main factors are to be taken into account. Firstly, the ICP values have a totally different meaning depending on whether a given patient develops mild or severe impairment of consciousness. Secondly, the time elapsed since injury is of crucial importance as well. Consequently, in patients exhibiting not too severe disturbances of consciousness immediately after injury and developing intracranial hypertension with clinical deterioration some days later, the focal intracerebral mass is likely to behave as a true space-occupying lesion. Conversely, the earlier the syndrome of intracerebral hypertension and the more severe the impairment of consciousness, the more relevant is the role of the associated lesions and vascular disorders. In this case the focal mass, therefore, appears to be only a collateral feature. Finally, it must be said that some patients exhibiting disorders of consciousness and other symptoms and signs usually attributed to intracranial hypertension purely on clinical grounds, have in fact a normal or slightly raised ICP, despite extensive and on occasions multiple brain lacerations. We should, accordingly, stress once more the practical value of ICP monitoring for making a therapeutic decision in doubtful cases [29].

As regards the frontal lesions in particular, they cannot be clinically differentiated from contusions of the temporal lobes. The overall outcome of frontal contusions, whether operated on or conservatively treated, seems to be quite similar to that of temporal lesions. The factors governing the outcome are actually the same in both locations. Nevertheless, contusions and laterations due to direct impact are far more frequent in the frontal lobes. Such lesions, despite very marked bone and brain disruption, often have a good outcome. Our policy with these injuries is rather conservative. We operate in cases of severe involvement of the paranasal sinuses and relevant aesthetic damage, or persistent CSF leakage. Otherwise, the prognosis of frontal injuries due to dynamic shocks is not fundamentally different from that of lobar contusions located elsewhere. It depends to a much greater extent on the associated lesions and/or functional

disorders than on the locations of the contusions themselves. Therefore, in predicting the outcome, clinical conditions and, above all, the state of consciousness, is more reliable than the neuroradiological appearance [29].

In conclusion, we feel that the so-called "brain contusion syndromes" irrespective of whether frontal, temporal or elsewhere, can no longer be considered separate anatomo-clinical entities. Neither should they be included in the very miscellaneous group of "traumatic intracranial mass lesions" since the extracerebral effusions have a totally different pathophysiological significance.

Traumatic intracerebral masses (contusions, lacerations and/or intracerebral haematomas) should, instead, be considered, in the context of cranio-cerebral injuries of a different degree of gravity, as having collateral features which, on occasions, may behave as true space-occupying lesions and call for surgical treatment.

On the whole, the surgical indications in traumatic cerebral mass lesions in general appear to be rather limited nowadays [41]. Surgery is very seldom justified during the very early stage after injury, since in too severely ill patients it does not prevent devastating complications which are not influenced by decompression. Vigouroux and Guillermain [40] no longer perform operations during the very acute stage. Conversely, in many patients intracranial hypertension does not develop or can safely be controlled by conservative therapy and the operation is avoided or carried out at a later stage when the patient is in a much better condition. Surgery, by contrast, is indicated in cases in which brain masses behave as evolutive expanding lesions, giving rise to intracranial hypertension with a rapid clinical deterioration, or to long-standing hypertensive syndromes not influenced by medical therapy or ventricular drainage. On these grounds, it can easily be explained why the best results of surgical intervention are achieved at a later stage, 5 to 8 days after injury [39, 40]. At that time disturbances of autoregulation and brain oedema have subsided to a great extent and the patient with localized lesions can be operated on successfully. The choice of the treatment in cases with intermediate disturbances of consciousness, showing no definite signs of improvement or deterioration, still remains extremely difficult. We feel that whenever the ICP does not exceed 20 mm Hg with a stable tracing and narrow pulsation, and, therefore, brain elastance is presumably not elevated, little benefit, if any, is to be expected from surgery. Widespread lesions are likely to be responsible for impaired consciousness rather than a focal mass. Conversely, if the ICP is frankly raised, usually we first try ventricular drainage with an exit pressure slightly in excess of the normal ICP [28, 29]. In this

manner it is possible to keep the intracranial hypertension under control throughout the acute stage. If the ICP is not normalized and no obvious clinical benefit is obtained, we perform delayed surgery 4 to 6 days after injury. Small craniotomies, often under local anaesthesia, are quite sufficient to manage the cerebral lesions with minimal risk.

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