

TRAUMATIC INTRACEREBRAL HEMATOMA

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In evaluation of t.i.c.h. (1), the following to conditions are excluded:

1. Cases with pre-existing vascular anomalies, such as aneurysms, angiomas, and bleeding tumors.
2. Cases with major cerebral laceration and t.i.c.h. of only a small area. The cases considered as t.i.c.h. only, are those in which the hematoma is a major space-occupying lesion.

Pathological anatomy:

In most cases the t.i.c.h. is to be found within the temporal or frontal lobe. Displacement of the brain with or without contrecoup and the pressure of the brain against the sphenoidal ridge or the orbital floor may play an important part. Usually the hematoma is superficially situated, and a thin layer of fresh subdural hematoma or lacerated cortex indicates the site of the intracerebral clot.

In regard to the consistency of the hematoma, one usually finds dark colored, thick blood with articles of blood coagula. In chronic cases the coagulated blood remains.

Analysis of cases:

Over a period of fifteen years, 416 cases of acute head injuries have been treated in the Pahlavi Hospital.

The following cases of intracranial hematomas were included:

intracerebral	37
intracerebellar	5
extradural	26
acute subdural	7
combined hematomas	6

79

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(1) t.i.c.h. = Traumatic intracerebral hematoma.

On the remaining 337 cases, 67 died. Autopsies performed indicated that three of these cases had a t.i.c.h. Almost 10% of those with head injuries had an intracerebral clot. This is by far a higher percentage than that reported by **Loew-Lin-Gurdjian** and other authors. The reason for this may be that the only cases admitted are those suspected to need surgical attention.

In 21 cases the hematoma was localized within the temporal lobe in 10 cases in the frontal lobe, 1 in the parietal lobe, and 1 in the occipital lobe.

In 4 cases combined hematomas were found which could be classified as follows:

t.i.c.h. in the right temporal and left frontal	———1 case
t.i.c.h. in the right temporal and subdural hematoma, left parietal	———2 cases
t.i.c.h. in the left frontal and extradural hematoma, right parietal	———1 case

Clinical symptoms:

1. **Raised intracranial pressure:** In all cases in which consciousness was not severely affected, headache was the main complaint. 17 patient had vomiting. Papilledema was found in 2 chronic cases. Progressive bradycardia sometimes is a reliable sign.

2. **State of consciousness:** Changes in the state of consciousness in various forms from a psycho-organic syndrome to a full coma is the predominant clinical picture. Any deterioration in the level of consciousness is of imperative importance. In the group of 37 cases there were not less than 28 cases in which the consciousness was disturbed in some way. In 2 of the remaining 9 cases the hematoma had a chronic course.

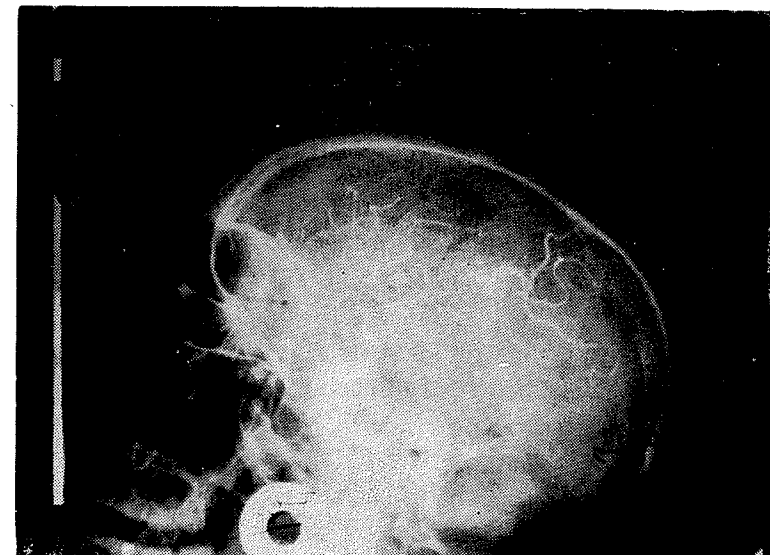
3. **Focal signs:** The paucity of focal signs is due to the involvement of the frontal or the temporal lobe.

In very acute cases, coma predominates, sometimes along with a state of decerebrate rigidity. In these cases the focal signs are not of great value. In the following, focal signs were encountered:

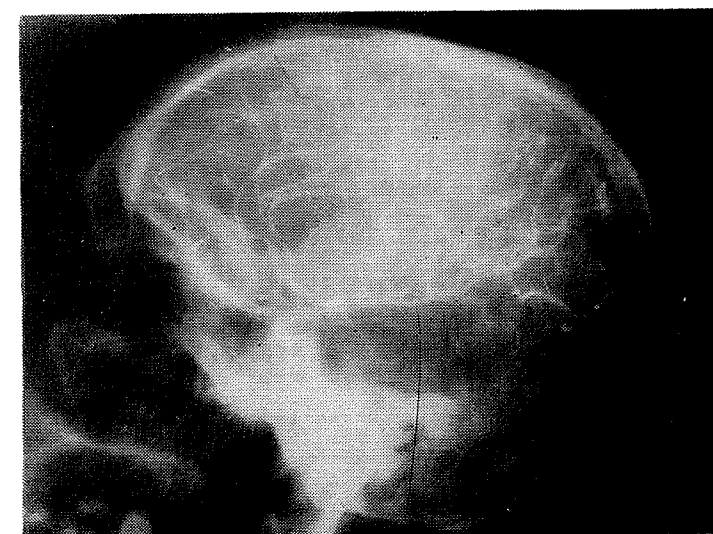
(a) **Pupils:** It was extraordinary that unilateral dilatation of the pupil occurred in only 3 cases. In one case, the hematoma was in the frontal lobe, and the contralateral pupil was dilated. In 2 cases the hematoma was in the temporal lobe.

(b) **Hemiparesis:** In 2 cases hemiplegia occurred. In both cases the hematoma was in the temporal lobe. In 12 cases of temporal-lobe hematoma hemiparesis was found. In 2 of these the paresis involved the same side as the hematoma.

(c) **Minor pyramidal deficiencies** were detected in 12 patients. 5 of them had a frontal-lobe hematoma.



Temporal lobe hematoma



Frontal lobe hematoma

(d) Disturbance of speech was found in 5 patients. In all of them the hematoma was in the left temporal lobe.

(e) 8 patients had no focal signs.

X-ray examination

19 patients had a skull fracture. Carotid angiography was the method chosen for the diagnosis of t.i.c.h. If possible, the angiography is done bilaterally using a general anesthesia. It should be emphasized that a local cerebral oedema or an extradural hematoma cannot be differentiated from a t.i.c.h. with full certainty, even with angiography.

Treatment:

Spontaneous recoveries of t.i.c.h. are reported, but this is exceptional. The hematomas are removed by small craniotomies. Actually, a large craniotomy is preferred leaving the dura open. Tracheotomy, induced euthermia, and management of electrolytes are of major importance.

Results

9 patients died following surgery. In 2 cases the hematomas were bilateral frontal. In the remaining 7 cases, 4 patients had a hematoma within the frontal lobe, and 2 patients in the temporal lobe. In almost all cases extensive cerebral damage, together with cerebral oedema, was the cause of death.

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OXYGEN SUPPLY TO THE BRAIN FOLLOWING SEVERE CEREBRAL TRAUMA

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In spite of advances in the treatment of severe craniocerebral injuries during the last ten years the mortality rate remains high, about 50%-75% (FROWEIN 1961). In order to improve this survival rate we must first determine whether or not the measures available to us ensure that an adequate oxygen supply to the brain tissues is maintained in the acute state. The oxygen tension in brain tissue is the standard measurement of the oxygen supply to the brain. It is thus possible to determine the oxygen supply to the brain by the oxygen tension measured at the end of the cerebral circulation, that is in venous blood. For this purpose we can take blood from the bulbous venae jugularis.

Along with FROWEIN and EULER we have been able to make 85 measurements of cerebral venous blood in 20 cases of recent craniocerebral injury. Of these, 13 were closed injuries, 2 open, and 5 post-traumatic intracranial hematomas.

From physiological studies, especially by SCHNEIDER and OPTIZ, HIRSCH, LUBBERS and others, it is known that the oxygen tension in arterial blood is normally 80-105 mm. mercury. On the venous side of the cerebral circulation, e.g. in the bulbous venae jugularis, the oxygen tension still amounts to 34-36 mm. mercury. Thus, if the venous oxygen tension is lower than this it would signify an abnormal intermediate oxygen tension in the brain tissue. According to SCHNEIDER and OPTIZ a venous oxygen tension of down to 28 mm. Hg is without significance, below 28 mm. Hg there is a reversible anoxic effect on the central nervous system, and with 19 mm. Hg the critical zone begins in which oxygen shortage alone is sufficient to cause irreversible damage.

Our results are demonstrated in the following slides which show the values obtained from the cases of craniocerebral injury

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