

(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

By

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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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with survivors on one side and fatal cases on the other. The individual measurements are noted hourly or daily following trauma. The normal for cerebral venous blood (34-36 mm. Hg) is shown by a gray stripe.

Figure 1:

It can be seen that of the injured who later recovered 18 lie within normal levels, 19 in the level of tolerance, and only 3 below this

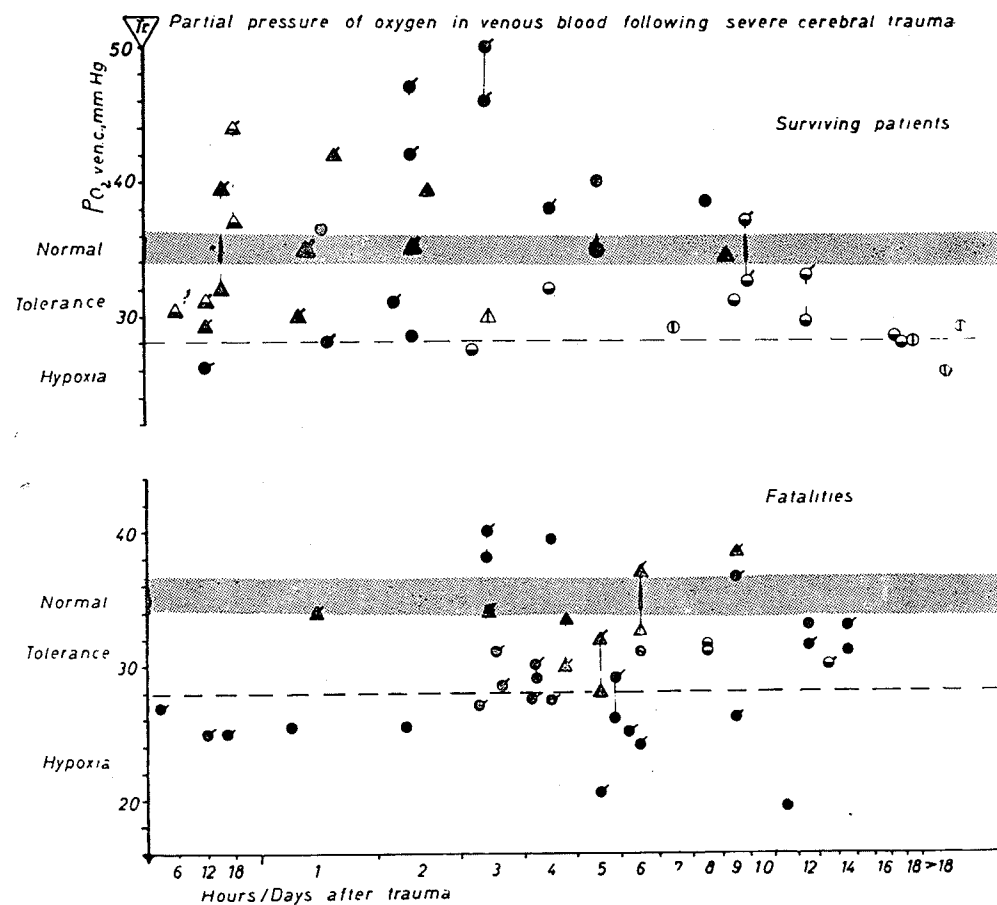


FIG. 1

level in the critical zone. The majority of the injured who were unconscious, shown by a black circle (adults) and by a black triangle (infants), inhaled an oxygen-air mixture during the investigation. In some cases it was evident that depriving them of the oxygen was followed in a few minutes by a fall in the venous oxygen tension by several millimeters.

In the fatal cases, on the other hand, it was found that only 8 were in the normal zone, 18 at the level of tolerance, while 14 clearly were in the anoxic zone. In the cases the average oxygen supply to the brain certainly had not been sufficient. When we compare the venous oxygen tension in the surviving group with that of the fatal cases, we find that the fatal cases had a lower average O_2 tension than the survivors. However, for both groups all precautions were taken to maintain an adequate O_2 supply to the brain by ensuring a free airway and additional oxygen inhalation.

The causes leading to this striking difference between the venous O_2 tension in the survival and fatal cases cannot be completely summarized. In some cases the slowing of the cerebral circulation by raised intracranial pressure plays a role. In many cases there exists a marked anemia in the acute stage with a lowering of the hemoglobin level to 10 and even 8 gm%, even when no severe external bleeding occurred.

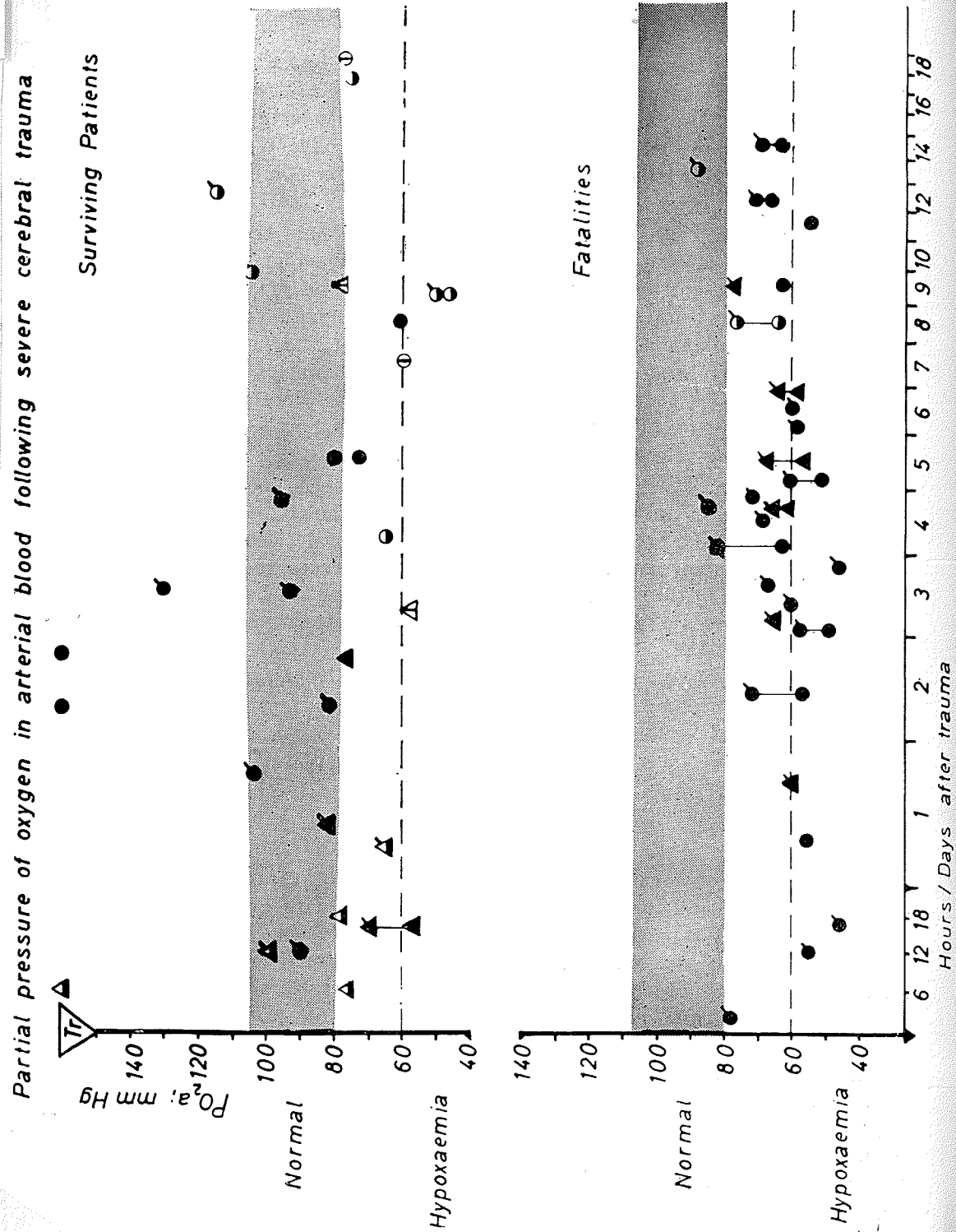
An important role is, however, played by the pulmonal alterations and for this reason we measured the arterial O_2 tension.

The normal O_2 tension in arterial blood is 80-105 mm. Hg. Below an arterial O_2 tension of 60 mm. Hg there is an increased cerebral circulation as a reaction to the anoxia.

Figure 2:

Again we demonstrate separately the values of the O_2 tension for the survivors and the fatal cases. In most cases with a favorable outcome we found a normal arterial O_2 tension. Rarely was the threshold of 60 mm. Hg passed: that was only with intentional deprivation of inhaled O_2 with obstruction of the tracheotomy tube or in cases with severe pulmonary complications.

In contrast we found 35 out of 38 fatal cases with readings below the normal, 12 of which had arterial O_2 tension values under 60 mm. Hg. Aspiration and atelectasis are the causes to be considered for these low readings obtained during the first hours following head injury. The readings obtained from the 2nd day on, as well as X-ray pictures, showed clear signs of marked pulmonary changes.



A further cause for the low cerebral venous O₂ tension seems to be the persistent hyperventilation following head injury. The persistent hyperaphia leads to respiratory alkalosis and a consequent diminution of the CO₂ tension in the blood. The carbon dioxide tension is known to be one of the most important regulators of the cerebral circulation. The injured also had their arterial carbon dioxide tension measured.

Figure 3:

Here the O₂ tension in venous blood is compared with the CO₂ tension of arterial blood. Again the readings obtained from the survivors and the fatal cases are shown separately.

In the survivors we found arterial CO₂ tensions predominantly over 30 mm. Hg. The values of the O₂ tension in the cerebral venous blood for these cases were shown in previous tables and were almost exclusively normal or at tolerance levels.

On the contrary we found that in fatal cases the arterial CO₂ tension is markedly under 30 mm. Hg, with a lower average venous O₂ tension.

The O₂ tension in all our cases of cerebral injury was reduced and these values are below the average arterial CO₂ tension for normal cases (that is 40 mm. Hg). However, the fatal cases showed markedly lower values than the surviving cases.

To summarize, we can state that in the acute state following severe craniocerebral trauma, especially in cases with a fatal outcome, an unexpectedly large number of cases demonstrated an insufficient Cerebral Venous O₂ tension - which points to an insufficient oxygen supply to the cerebral tissues.

Causes for the diminished cerebral oxygen supply include the reduced cerebral circulation, the often unexpected severe anemia and, of greatest importance, the respiratory factors.

Therefore, it is clear that we must find better methods for treatment and to avoid the pulmonary complications in addition to the generally accepted treatment by tracheotomy and bronchial toilet.

These findings also confirm the significance of the pharmacological sedation used to regulate the vegetative function and to normalize the respiration - in particular the avoidance of hyperventilation.

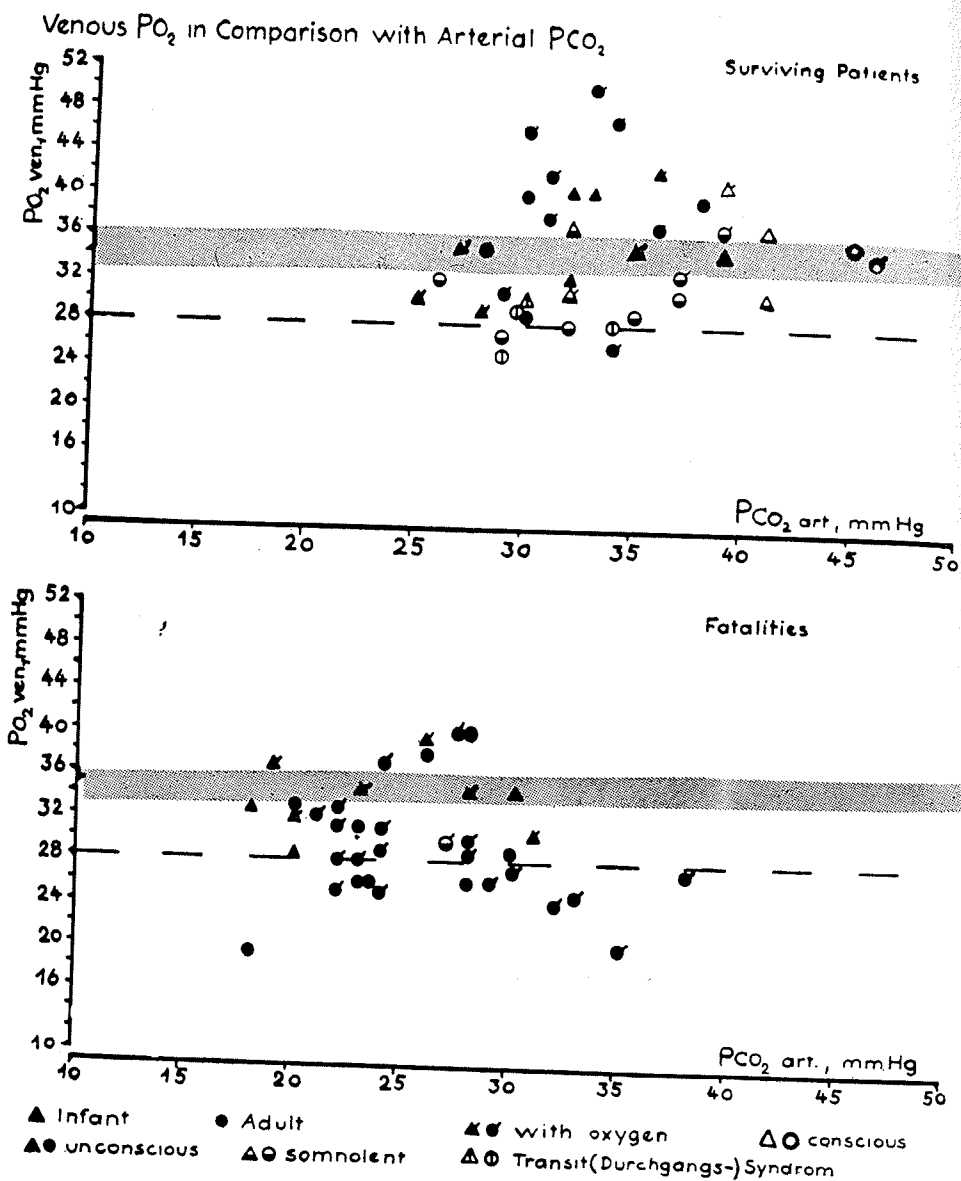


FIG 3

Résumé

La mortalité des traumatismes crâniens graves est resté très élevée pendant la dernière décade.

Une des raisons majeure de cette mortalité, est due a une diminution de la tension d'oxygène de la circulation cérébrale. Ceci a pu être mise en évidence par les mesures d'oxygène effectué sur le sang de la veine jugulaire à 85 reprises chez 20 malades.

La proportion de décés ou guérisons sont mise en parallèle avec la tension d'oxygène veineuse chez traumatisés crâniens grave.

Cette de la tension d'oxygène du sang de la circulation cérébrale est aux nombreux facteurs: H.I.C. chute de T.A. chute de Hb, à la suite d'hémorragie massive et enfin une insuffisance respiratoire ou bien au contraire une hyperventilation qui aboutit à une alcalose respiratoire modifiant la régulation de la circulation cérébrale.

A la lumière de cette étude les auteurs donnent des conseils afin de pouvoir diminuer la mortalité chez les traumatisés crâniens graves.