

SURVIVORS OF INTRACRANIAL HEMORRHAGE (ICH)

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Intracranial Hemorrhage is one of the most common causes of death in newborn and in premature babies. With improving Neonatal Intensive Care more and more infants are able to survive ICH, but the quality of survival does not seem to be encouraging (2, 3). On the other hand, we found that about 40 % of our patients surviving clinically diagnosed ICH never showed any abnormality.

Material:

From 1972 to 1976 43 newborn and premature infants who survived ICH were treated in our Newborn Intensive Care Unit. 27 infants, i.e. $63 \pm 13 \%$ (90 % - confidence range) were premature with a gestational age of 36 weeks p.m. or less, the remainder of 16 had a gestational age of 37 to 40 weeks p.m.. Onset of symptoms was seen in 18 babies ($42 \pm 14 \%$) during the second day of life, significantly more often than during all other days (1st to 30s), see Fig. 1.

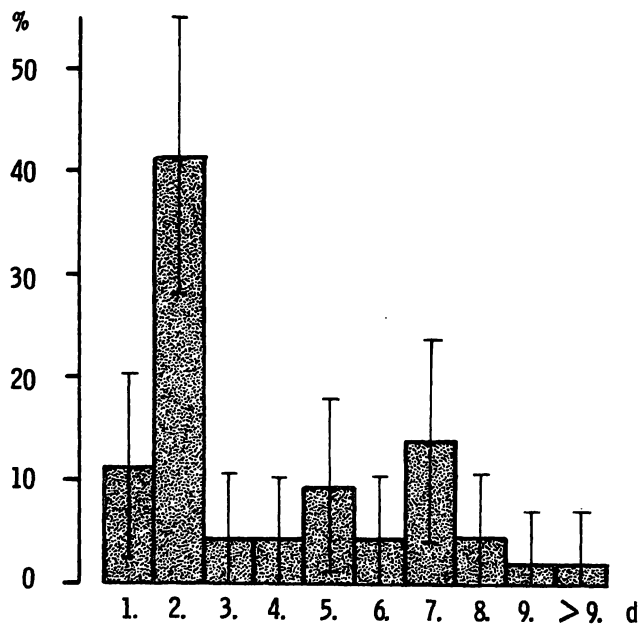


Fig. 1:
Onset of symptoms of ICH. d = day.
Length of the bars indicates 90 % - confidence range, also in the following figures.

33 out of 43 infants had blood stained cerebrospinal fluid (CSF), i.e. $77 \pm 12 \%$. If bloody CSF was found by lumbar puncture, a cisternal tap was added to exclude iatrogenic bleeding. 27 infants ($63 \pm 13 \%$) showed a sudden drop of hemoglobin - concentration (Hb) in the capillary blood of at least 3 g / dl within one day, 31 babies ($72 \pm 12 \%$) had convulsions, 27 ($63 \pm 13 \%$) suffered from apneic spells, and 17 ($40 \pm 13 \%$) developed marked hyperexcitability, see Fig. 2. If CSF was not found to be blood stained, a sudden drop of Hb together with at least two of the neurological symptoms mentioned above were challenged before making the diagnosis of ICH. Follow up examinations were done by staff members during our consultation hours for high risk infants. Only a few babies

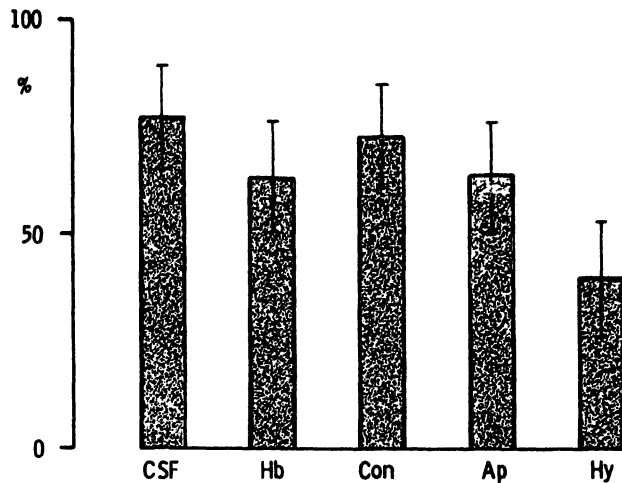


Fig. 2:
Frequency of symptoms
of ICH.
CSF = blood stained ce-
rebrospinal fluid.
Hb = sudden drop of he-
moglobin - concentra-
tion in capillary blood.
Con = convulsions.
Ap = apneic spells.
Hy = marked hyperexci-
tability.

were followed by their own pediatricians. Final judgment about the outcome of the infant was made when the infant was at least one year old. All judgments were based on the biological age, i.e. the age of the infant was reduced to the time after a gestation of 40 weeks p.m..

Results:

Out of our 43 survivors of ICH 6 infants ($14 \pm 10\%$) showed severe neurologic defects, including marked cerebral palsy, impaired mental development, or microcephalus. Amazingly, no case of hydrocephalus was found.

6 other infants were detected to suffer from mild neuromotor handicap. In 14 infants ($33 \pm 13\%$) we saw transitory neurologic defects, such as prolonged persistence of primitive reflexes, slight cerebral paresis of one limb, or retardation of developmental milestones. All these troubles disappeared within some months under physiotherapy, sometimes also without any treatment.

17 survivors ($40 \pm 13\%$) never showed any abnormality (Fig. 3). These results do not show any correlation with the severity of symptoms during the neonatal period.

The pattern of neurologic damage was the following one (Fig. 4): Quadriplegia was found in 7 ($16 \pm 10\%$) of the survivors, hemiparesis in 5 ($12 \pm 9\%$). Most of the abnormal infants, namely 14 ($33 \pm 13\%$) showed mild neuromotor impairments without any characteristic pattern.

Discussion:

Our follow up study of the 43 survivors of ICH from our New-born Intensive Care Unit from 1972 to 1976 shows that the quality of survival is not at all as catastrophic as we had assumed from the previous available literature. On the other hand the rate of severely handicapped infants is higher than the average found in prematurely born infants (review of the recent literature s. 1) and even higher than in survivors of neonatal meningitis (5, 6: 8 to 9%).

Our study is limited in two respects:

1. Clinical diagnosis of ICH does not allow any interpretation about localisation and size of bleeding. No differentiation is possible between subarachnoid and intraventricular hemorrhage.

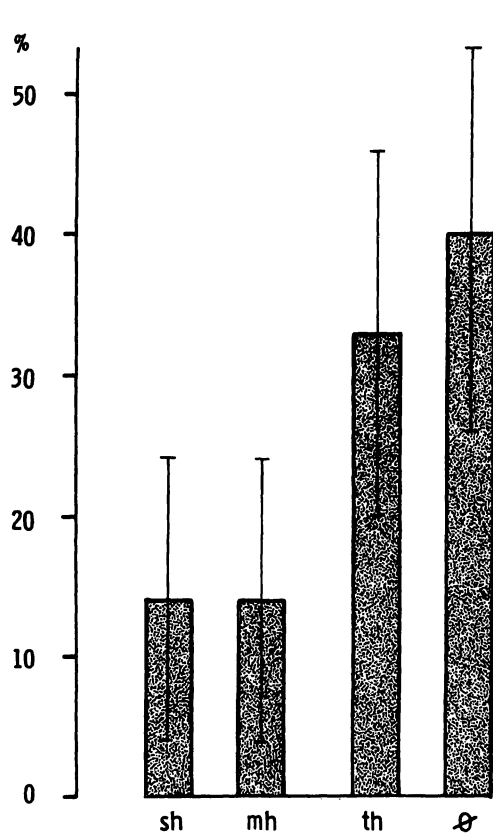


Fig. 3:
Frequency of sequelae after ICH. sh = severe handicap. mh = mild handicap. th = transitory handicap. ø = no sequelae.

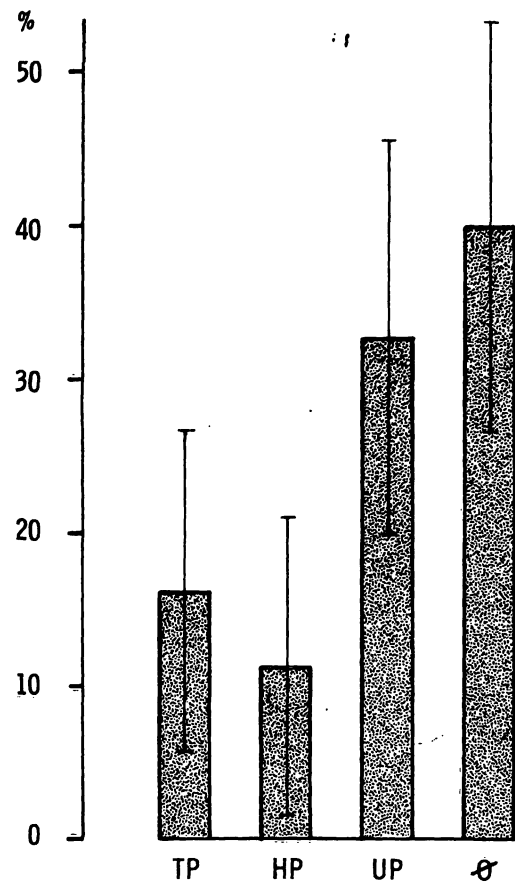


Fig. 4:
Frequency of different patterns of neurologic sequelae of ICH. TP = quadriplegia. HP = hemiparesis. UP = uncharacteristic cerebral palsy ("formes frustes"). ø = no handicap.

2. Long term follow up examinations were not made. Information about localisation and extension of ICH may only be gained by means of computerized tomography of the cranium (CT). It seems that routine use of CT will detect by far more cases of ICH than clinical diagnosis alone: Recently a cooperative study was undertaken by the American Society for Pediatric Research: 633 premature infants with birth weights below 1,500 grams were examined. 280 (44 %) had signs of ICH in the CT-scan. 80 infants out of 121 (66 %) survived ICH (4). Therefore one might conclude that only more severe forms of ICH can be diagnosed clinically. On the other hand it may be of particular interest to get information about the prognosis of these latter forms which can be diagnosed everywhere. Although we do not have any knowledge about the long term prognosis of our survivors, e.g. concerning behaviour disturbances or intellectual development, we may state that severe

neurologic sequelae are not to be expected so often that cessation of life saving measures seems to be justified after clinical diagnosis of ICH alone.

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