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Subdural effusion in the first six months of life

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During the period 1978–1981, among 1280 (0–12 months old) infants suffering from consequences of various pre- and perinatal cerebral lesions, positive neuroradiological changes were found in 148 cases. Of these, 56 proved to be real subdural effusions with elevated ICP and increased protein content. Periodical transfontanellar taps and drainage were effective in 49 cases. A neurosurgical operation was performed in 7 infants because of the poor effect of the above treatment. In these 7 infants the encapsulation process was nearly complete.

Early chronic subdural effusion exerts a devastating influence on the maturing brain partly by the high ICP. Early treatment often cures the process and major surgery is not needed. Early neurorehabilitation and habilitation coordinated with elimination of the effusion is the method of choice. Prudence is recommended in selecting the appropriate cases for instrumental therapy because some small effusions and fluid accumulations may disappear by the end of the first year.

The development of new diagnostic batteries for the early detection of pre- and perinatal brain lesions yielded new information on the occurrence of various morphological and physiological defects in the central nervous system. At present a complete and reliable developmental neurological investigation of the newborn or young infant consists of polygraphic studies of feeding, visual, auditive and motor behaviour, detailed analysis of all elementary motor patterns activated by various stimulus positions, EEG, and in selected cases polymyography. In addition, the investigation should include transillumination. CT, sonography, and, if necessary, transfontanellar tap, subdurography,ventriculography and various laboratory examinations. Such a complex and thorough developmental neurological investigation will reliably reveal early defects in the nervous system, and differentiate between normal variations and defects. Regular transillumination proved to be of considerable help in the early detection of abnormal brain development owing to malformations or trauma (1, 11). Computerised tomography (CT) then revealed an even higher percentage of brain abnormalities during early infancy (2, 5, 8, 10). Subdural effusion was found in a considerable number of young infants (9, 13).

Formerly, a subdural effusion was practically never detected before the second half of the first year, and it was treated by subdural taps, drainage, or neurosurgical intervention. It has been realized only recently that

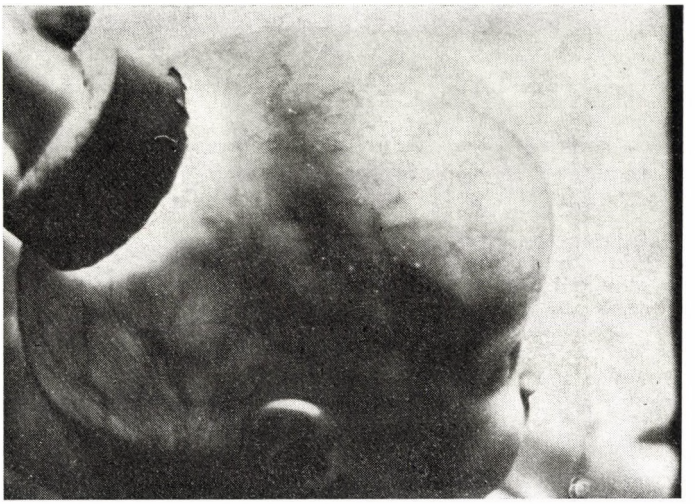


FIG. 1. Transillumination with a floodlight lamp. Large transparency reveals hydrocephalus

chronic subdural effusion may be present much earlier. The present study was focussed on this problem.

MATERIAL AND METHOD

In our department in the period 1978–1981, cerebral lesion and defective neurological function was detected in 1280 infants 0 to 12 months of age. The investigation took one week on the average. During this period a detailed and often repeated study was done. The function of cranial nerves and central control mechanisms were studied by polygraphy, sensory and activated motor functions by a thorough analysis of the stimulus-response relations, and EEG and in selected cases, polymyography were performed. All forms of objective recording of various sensory as well as motor behavioural patterns were used to obtain a sound diagnostic basis.

All infants were transilluminated using a special floodlight lamp (Fig. 1) and followed up by repeated transilluminations. If a suspicious transparency was observed, neuroradiological studies were performed. They consisted of transfontanellar subdurography, vetriculography, CT, and since the last year, transfontanellar sonography. By these means an intracranial morphological defect was revealed in 147 cases (Table I); of these in 56 infants increased intracranial pressure (ICP) and

TABLE I

Frequency of early subdural effusion

	No.	Per cent
Cerebral lesion	1280	100
Positive neuroradiology	147	11.5
Subdural effusion, 0–6 months	56	4.4

an elevated protein level in the CSF were found, with signs of a subdural effusion (Fig. 2). In all these infants, aberrant neurologic symptoms were present. They varied from serious deviations in motor and postural control, defective proportioning of muscle tone and aberrant feeding behaviour, to defective consiousness and vigility. Defective auditive and/or visual function was complicating 72% of the cases (6, 7).

CT proved to be extremely helpful in the detection of early subdural effusions but owing to the fact that laboratory analysis of the CSF was necessary for studying the nature of the subdural fluid. When the subdural space was tapped, air studies were also performed (Figs 3, 4 and 5).

ICP was recorded in selected cases in order to detect a non-manifest elevation of the ICP. An 8 channel Beckman R 411 Dynograph was used to record intracranial pressure and EEG simultaneously. Naturally all interventions were performed under sterile conditions. During the whole study which included not only 147 air studies but several hundred subdural taps, and therapeutic drainages not a single infection occurred.

Early preventive treatment was initiated in cases in which diagnostic batteries revealed subdural effusion. Therapy consisted of regular subdural taps, usually twice weekly on the affected side, or on alternating sides if the effusion was bilateral. In 7 cases a neurosurgical intervention had to be done.

Results

As shown in Table I, early subdural effusion was found in 56 infants aged 0 to 6.5 months, or 11.5% of the 1280 infants who were found to suffer from results of pre and perinatal cerebral lesions. Signs or suspicion of IC haemorrhage at or following birth was found in 26% of these cases; they all had a suspicious or clearly pathological transparency on transillumination. In 6 cases the transillumination was not positive initially but a transparency developed during the next 4 to 6 weeks. It was therefore

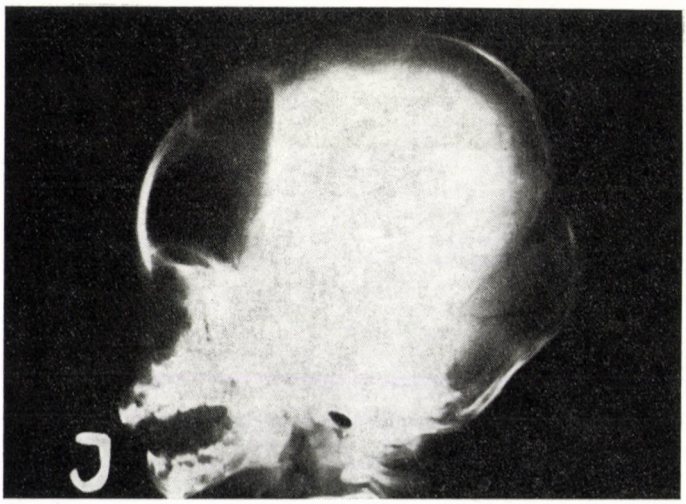


Fig. 2. Subdural effusion in a 4 months old infant. The effusion is mainly frontal

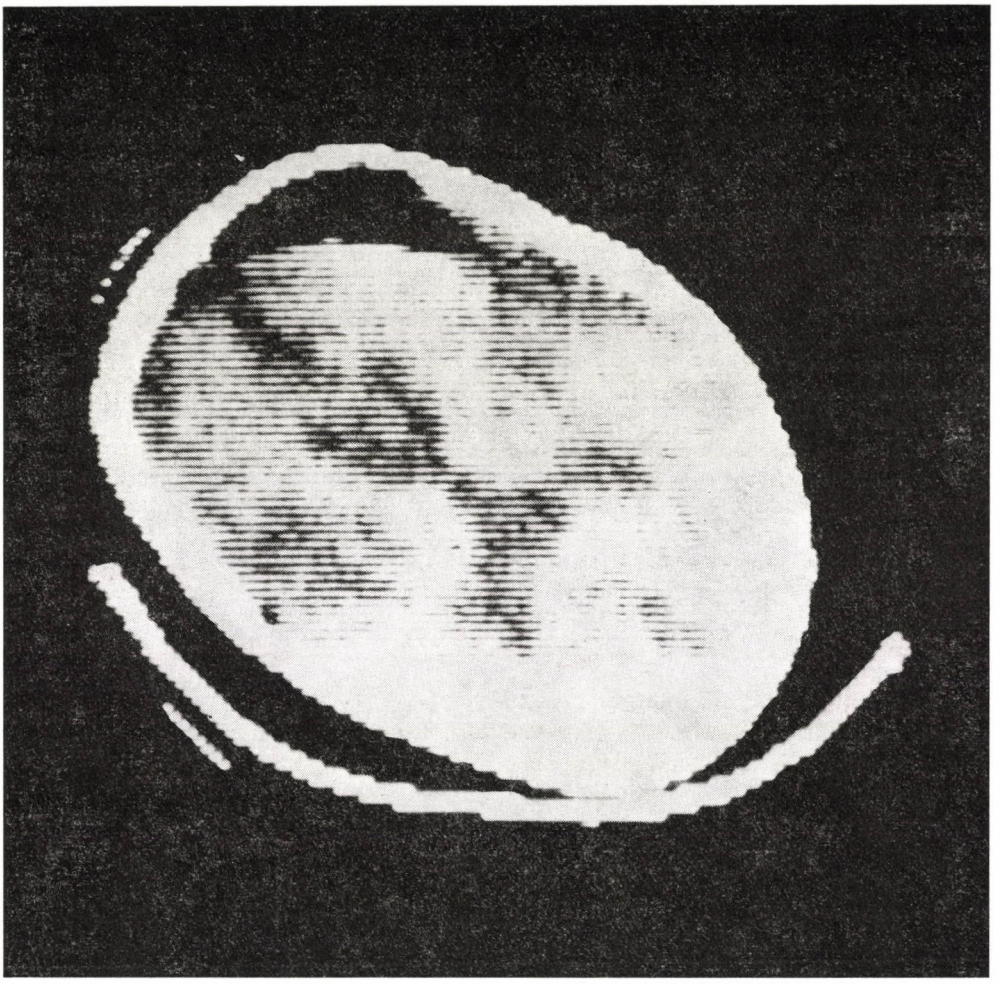


FIG. 3. CT of a subdural effusion over the frontal lobe

considered imperative to repeat regularly transillumination in infants with apparent cerebral lesion or with due suspicion of such a defect.

Repeated tapping or drainage of the subdural space eliminated the effusion in most of our cases. Neurosurgery was necessary in not more than 7 cases. During tapping and drainage the protein content of the effusion dropped from the initial very high level gradually to normal, the effusion diminished and then disappeared.

During therapy, acute and chronic changes in behaviour were recorded. In selected cases drowsiness and loss of contact with the environment were the signs of increased ICP. This was clearly observed in the slow or completely absent stimulus-response correlation, in the absence of any signs

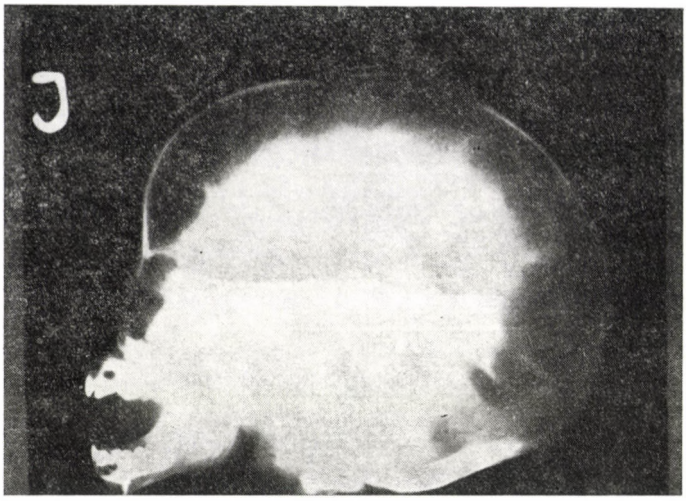


FIG. 4. Subdural effusion over the right hemisphere in a 3 months old infant. Lateral view

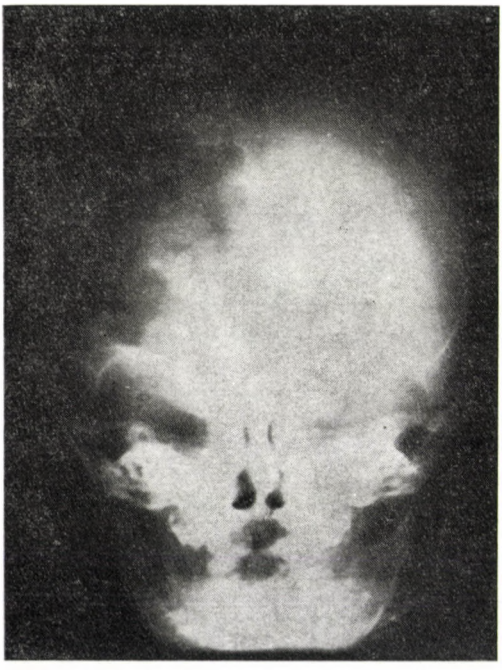


FIG. 5. Large subdural effusion over the right hemisphere of a 4 months old infant. AP view

of orientation on polygraphic monitoring or on the EEG. All signs of an active behaviour and environmental contact disappeared. In such cases, aspiration of 20–40 ml fluid from the subdural space produced dramatic changes. Not only the well-known signs of elevated ICP such as an increased fontanellar bulging, precomatose state, bradycardia, and vomiting had disappeared, but even the stimulus-response relations improved when visual or auditive stimuli were applied, and orientative behaviour, habituation and dishabituation returned to the normal level. Eventual changes in ICP during programmed exteroceptive stimulation by sound were studied in selected cases by polygraphy. It was occasionally observed in quiet, resting infants who produced definite behavioural responses to auditive stimulation that the classical orienting reaction was accompanied by a definite elevation in both ventricular and subdural pressure. Continuous monotonous stimulation inhibited the orienting reaction and simultaneously both subdural and intraventricular pressure dropped. When, however, the frequency of continuous monotonous stimulation was changed, the appearance of a second orienting reaction

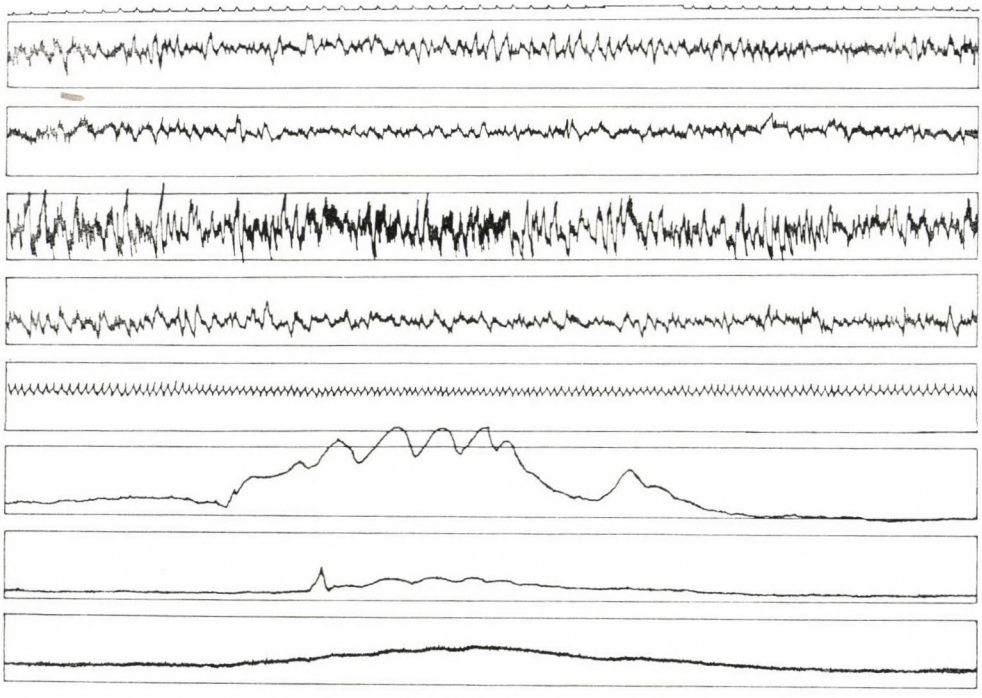


FIG. 6. Simultaneous recordings of spinal (lowest), subdural central and ventricular (upper) pressures, EEG, and ECG. Auditive stimulation was followed by a transitory pressure wave in all pressure recording channels. The 6th channel records right ventricular pressure, the 7th left subdural pressure, and the 8th spinal pressure. Channels 1–5, and paper speed and stimulus parameters as in Fig. 7*a* and *b*

(dishabituation) was again accompanied by a sudden elevation of subdural and intraventricular pressure (Figs 6 and 7*a*, *b*).

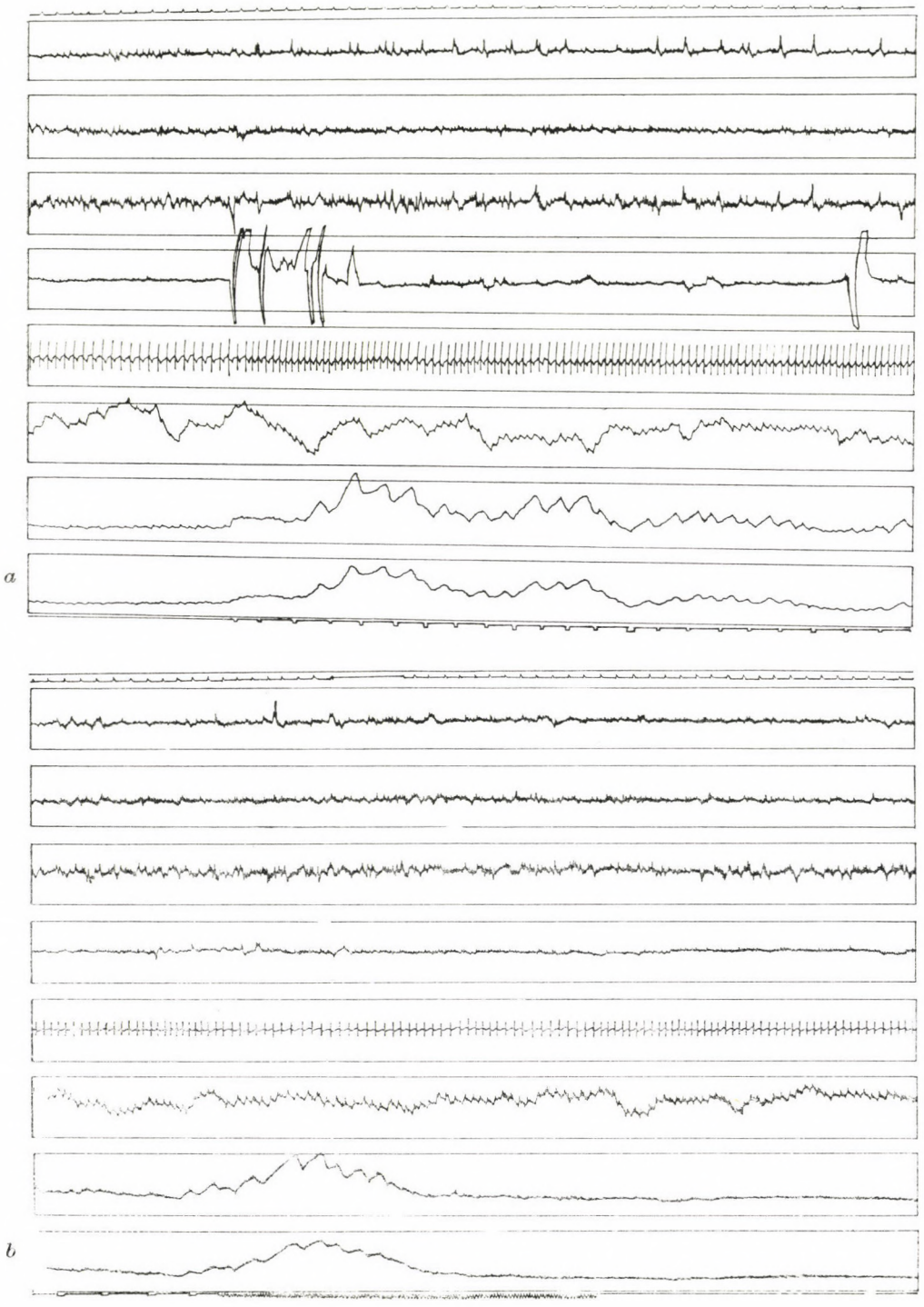
The studies have shown that not only a sudden rise of ICP can depress alertness, vigility and environmental contact of the infant but drowsy states may develop even if the rise is not constant. During prolonged ICP recording an undulating pressure was found.

CT is an important indicator of the fact that fluid has accumulated in the subdural space but not a reliable proof that the fluid represents a real effusion. In our practice the coincidence of positive neuroradiology, increased ICP, elevated protein content up to 600–2000 mg/dl in the subdural fluid seemed to verify the presence of early chronic subdural effusion. A further indicator of its presence was found when 7 of the patients had to be operated upon because of the failure of intermittent transfontanellar taps and drainage: in all these cases the two layers of the fibroblast envelope of the effusion had already begun to form even in 3 infants under the age of 3 months.

In 85% of the cases prolonged treatment by tapping and drainage was successful. The subdural effusion disappeared, the symptoms of increased ICP ceased to exist. Apart from the 7 cases in which this treatment was ineffective, in 6 cases the effusion reappeared, and a new series of tapping became necessary for making the effusion disappear.

Discussion

The occurrence of a subdural effusion with cerebral hypotrophy in the early months of life in such a high percentage means that the effusion may develop earlier than it had been thought; it may be chronic in the first months of life. The mentioned experience during operations revealed the fact that the neomembrane cover enveloping the cerebrum may develop early, though encapsulation is not complete. Thus, early detection of a real effusion may prevent the stabilization of the layers and shorten the period of brain compression. Increased ICP even if it is not constantly elevated exerts a destructive influence on the developing brain. The maturing structural differentiation represented by dendritic arborization, formation of synaptic junctions and glial proliferation are affected by the blood flow being impeded by the increased ICP. According to the classical work of Ingraham and Matson (4) the subdural haematomas discovered in early infancy are usually bilateral. In the 149 cases of Pia (12) 119 were bilateral. In our own material, too, most of the subdural effusions were bilateral and extended to the frontoprecentral region. In some cases they reached the parietal region, but we observed no effusion in the posterior fossa. Development of neomembranes and effusion formation was treated by a number of authors. Friede (3) summarized the recent information. Operative findings and our X ray studies call attention to the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fact that the hypotrophic changes in the brain can be widespread under the layer of the effusion, even in the first months of life. Earlier we have called attention to the fact that neomembrane formation can be a very early process.

According to our experience a large amount of real subdural effusion can accumulate during the first postnatal months with elevated ICP, increased protein level, and definite signs of cerebral hypotropy even in the absence of any suspicion for IC haemorrhage in the perinatal history. Such effusions sometimes accumulate when the newborn had suffered from a verified or suspected IC haemorrhage but this then became completely normal, and no symptoms suggested any danger in the following days or weeks. In these cases transillumination is sometimes negative, and transparency becomes apparent only several weeks later when neurologic symptoms develop. A complete developmental neurological examination detects these symptoms earlier than a quick routine assessment. This is one of the reasons why subdural effusions were detected in our material at an early age and why treatment could be applied with success. Another cause of the high incidence of effusions in our material was that our special department receives a large number of young infants from other neonatal centres where suspicion of a possible pre- or perinatal cerebral lesion had arisen on the basis of certain functional symptoms such as defective feeding behaviour, problems of sucking and swallowing, conspicuous postural and motor signs, disturbed muscle tone, incompetent visual or auditive behaviour, restlessness or prolonged sleeping periods, convulsions, etc. The symptomatology usually represented a mixture of these signs. In 11.5% of these cases neuroradiologic examination revealed hydrocephalus, cystic deformities, porencephaly with fluid accumulation, and real subdural effusions, with a characteristic elevation of ICP and of protein content, a sign which was never detected in other processes associated with IC fluid accumulation as for example in porencephaly.

FIG. 7*a*, *b*. Auditive stimulation was followed by distinct changes on EEG, ECG and waves in the subdural and intraspinal pressures (orientation). Monotonous stimulation produces habituation, and a change in stimulation frequency produces dishabituation with another elevation in subdural and spinal pressures

First line: time marker; paper speed: 10 mm/s

Channels 1–4: EEG (pF_1—T_3 T_3—O_1 pF_2—T_4 T_4—O_2) 30/ · 1 x · 01 10 mV/cm
Channel 5: ECG 30/1 x · 1 10 mV/cm
Channel 6: intraspinal pressure DC/30 x · 01 10 mV/cm, 1 cm = 100 mm H_2O
Channel 7: left side subdural pressure DC/30 x · 01 10 mV/cm, 1 cm = = 100 mm H_2O
Channel 8: right side subdural pressure DC/30 c · 01 10 mV/cm, 1 cm = = 100 mm H_2O
Last line: stimulus marker (stimulus was given by an EMG type PHOTO–PHONO stimulator. Repetition: 0.4 cps in Fig. 7*a* and 4 cps in Fig. 7*b*; duration: 0.2 sec; sound frequency: 1000 Hz; sound intensity: 70 dB

In cases operated upon at the end of the first year or later, the brain showed considerable changes in size, gyrification, electrical activity and function due to the effect of the effusion. There is no way to tell whether an effusion with increased ICP and elevated protein content will be eliminated by spontaneous absorption. It is highly questionable how an accumulation of 150–200 ml can be absorbed if it receives constant supply from the developing vascular layers enveloping the brain and the fact that encapsulation is almost complete at a relatively young age speaks against frequent spontaneous recovery.

Depending on the type of the functional defect rehabilitation consisted in visual and/or auditive training, application of a stereophonic hearing aid, therapeutical training and education of elementary motor patterns, electrotherapy and sensory training. All these served to reinforce the existing functional potentialities and to assist development of the CNS. It would have been paradoxical to begin a medical programme in order to rehabilitate lost functions and to prevent development of other defective functions by habilitation, and to watch meanwhile inactively the continuous compression of the brain. Maximum caution was applied in indicating tapping or drainage. They were done exclusively when clear-cut signs of early chronic subdural effusion were observed. These are a large amount of fluid, elevated ICP, elevated protein level and positive neurological signs. The fact that the first three symptoms were eliminated in 49 cases, speaks for the correctness of diagnosis. Among these 49 cases, 36 infants became completely normal and 13 became subnormal but educable; this result demonstrates the value of the prudent application of coordinated instrumental therapy and neurorehabilitation.

Various strategies have been advised for detecting and treating subdural effusions. The presence of acute and/or chronic neurological symptoms together with positive neuroradiological evidence as well as an increased protein content in the effusion and occasionally the elevated subdural pressure can be indications to adopt an active conduct. The simultaneous presence of all symptoms and evidence of a considerable amount of effusion in the subdural space justifies repeated punctures or subdural drainage, if punctures are not sufficient to diminish the amount of fluid rapidly. We fully share the opinion of those who experienced the disappearance of subdural effusion after persistent taping. As no early chronic subdural haematoma in our patients was due to previous inflammation we considered them to be of traumatic origin if evidence of difficult labour was present, or of unknown origin. Due caution is necessary to decide for a taping regime, but this seems to be a better proposal than to wait until the effusion is absorbed spontaneously. If all the above-mentioned symptoms are present, simultaneously, this waiting attitude may endanger the

developing brain. If tapping or drainage fail to solve the problem, craniotomy, neomembrane stripping, or the insertion of a valve is necessary.

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