Short reports

subacute ventriculitis in some cases, and multiple pulmonary thromboses leading to hypertension and eventually to heart failure (Clark, 1969). In addition, 40% of such shunts require revision operations (Clark, 1969). Most of the deaths in treated cases are due to shunt complications (Guthkelch, 1967). Compressive cranial wrapping can stop hydrocephalus within 6 months if applied from an early stage, without the risks mentioned above (Epstein et al., 1973).

Animal experiments suggest that in neonatal hydrocephalus the skull expands because the sutures are lax and unable to resist the slightly raised intracranial pressure (Hochwald et al., 1972a; Epstein et al., 1973). If the bones of the cranial vault are supported by an external pressure the ventricular CSF pressure rises slightly to the point at which it may open a stenosed aqueduct or increase transventricular absorption of CSF (Epstein et al., 1973). Such absorption occurs by passage of CSF through the ventricular wall, whence it is absorbed within 600 μm of the ventricular surface (Lux et al., 1970) into the blood vessels of the brain parenchyma which drain to the straight sinuses (Sahar, Hochwald, and Ransohoff, 1970). It has been suggested that the fluid crosses the cerebral cortex to reach the subarachnoid space in babies (Davson, 1972), but this does not occur in the experimental animal (Sahar et al., 1970). In addition, the increased pressure may reduce production of CSF by the choroid plexuses (Hochwald et al., 1972b).

In the treatment of neonatal hydrocephalus the intraventricular pressure at the initiation of head compression is in the region of 500 mm of water. After 2–3 hours it falls to ½ of this value, and on removing the head compression it falls to half its pretreatment level (Epstein, Wald, and Hochwald, 1974). The pressure does not cause cortical atrophy (Epstein et al., 1973). When treatment is begun at the age of 1–3 weeks, the pathways of absorption have opened up and the sutures have fused sufficiently after 6 months for the skull to resist excessive expansion. Treatment can then be stopped and further head growth resumes at the normal rate.

Originally an elastic bandage was used to apply the pressure (Epstein et al., 1973). 2 cases have been reported in which apnoeic attacks and incessant crying may have been caused by over-tight winding of the bandage round the head (Meyer, Price, and Reubel, 1973). In the second of Meyer's cases this view is supported by the fact that the head circumference diminished by 1·25 cm in only 5 days. A pneumatic helmet has been produced so that accurately controlled and measured pressures can be applied (Epstein et al., 1974) and the staff of the Medical Physics Department at our hospital are developing a similar device.

The use of an elastic net cap has the advantages of simplicity, ease of application, and reproducibility of pressure from one application to the next. Also, the tension can be readily varied to maintain head growth at the norm by varying the number of layers.

Summary

A simple method of applying compressive cranial wrapping for neonatal hydrocephalus is described and its beneficial use in one patient is described.

REFERENCES


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Neonatal gastric hyperacidity

Further analysis of oxytocin effect

Previous reports about gastrin levels in the neonate have shown that babies born spontaneously have higher plasma gastrin levels in the umbilical
vein than do babies born after oxytocin induction (Rogers et al., 1974). The suggestion was made that a maternal component of the umbilical venous gastrin was responsible for the higher gastrin levels in the spontaneous group, and that this component may be responsible for neonatal gastric hyperacidity. It was further suggested that this hypothetical gastrin transfer was reduced by oxytocin. This present study was designed to confirm or refute this hypothesis.

Materials and methods

Umbilical arterial and venous blood was obtained from 12 babies born spontaneously and from 11 babies delivered after oxytocin-induced labour. Samples, collected by direct vessel puncture, were centrifuged in lithium heparin tubes upon withdrawal. The separated plasma was stored at -20°C and gastrin assay was performed by radioimmunoassay. The antisera used was raised to synthetic human gastrin I (2-17) conjugated to ovalbumin using glutaraldehyde. Synthetic human gastrin I was labelled with 125 Iodine (Amersham) and standard gastrin was obtained from the Medical Research Council. Separation of antibody-bound from free hormone was achieved using Dextran-coated charcoal. Cross-reaction with cholecystokinin-pancreozymin is 1/10 000 on a molar basis (Ardill, 1973).

Gastric secretion was sampled from 12 babies after spontaneous labour and from 7 babies after oxytocin-induced labours. Specimens were obtained at birth and 4 hours after delivery before the baby was fed. An orogastric tube was passed at birth and gastric emptying was as complete as possible. The aspirate was stored at -20°C until the pH was measured using wide-range litmus paper. The viscosity of many of the specimens made them unsuitable for analysis by pH meter.

All babies were spontaneous deliveries, not asphyxiated at birth, and weighed <2·5 kg. Results were analysed by the Student's 't' test.

Results

Gastric acid secretion. The mean pH at birth and at 4 hours was 6·21±0·406 SD and 2·78±0·324 SD after oxytocin labour and 6·00±0·465 SD and 4·25±0·673 SD after spontaneous labour. The fall in pH after both types of labour was significant (P<0·005, oxytocin; P<0·05, spontaneous).

The less profound fall in gastric pH after spontaneous labour was attributed to incomplete emptying at initial aspiration of the stomach contents in several of the spontaneous group. The difference in pH between the groups at birth or at 4 hours was not significant.

Gastrin secretion, arterial, and venous levels. Venous and arterial gastrin levels were higher after spontaneous labour than after oxytocin induced labour. A venous arterial gastrin difference was noted after spontaneous and induced labour. This was more marked in the former case though the difference was not significant.

The difference in venous and arterial gastrin levels is shown in the Fig. A significant rise from arterial to venous levels was seen after induced labour (P<0·05), but the rise was not significant after spontaneous labour.

Discussion

The observed fall of gastric pH in the first hours of life confirms previous reports (Miller, 1941; Avery, Randolph, and Weaver, 1966). If oxytocin interferes with the transfer of a maternal component of cord gastrin then an increase in neonatal gastric acidity might be expected after spontaneous labour, but no difference in gastric pH values was found. Our results do show that both arterial and venous gastrin levels are higher after spontaneous labour, which confirms our previous observation on the influence of oxytocin infusion on cord gastrin levels (Rogers et al., 1974).

As shown in the Fig., venous gastrin levels were not greater than arterial levels in all instances. However, a venous arterial difference was present which seems to indicate that venous cord blood has a gastrin contribution from either maternal or placental sources. Though there was no signifi-
cance in this difference, the mean value was greater after spontaneous labour. This suggests that oxytocin interferes with gastrin transfer. On the other hand paired differences of arterial and venous gastrin levels show a significant rise ($P < 0.05$) after oxytocin induction, but after spontaneous labour this rise was not significant ($P < 0.1$). In the latter group venous-arterial gastrin changes tend to be more definite, whether in a positive or negative direction. If oxytocin did interfere with gastrin transfer then one would expect paired venous-arterial gastrin differences to be more significant after spontaneous labour, but we were unable to show this.

Our results neither exclude the hypothesis of maternal/placental gastrin transfer nor conclusively support the contention that oxytocin significantly reduces such a transfer.

**Summary**

Plasma gastrin levels were measured from umbilical, arterial, and venous blood at birth from babies after spontaneous labour and after oxytocin induction. No significant difference in levels was found between the groups. Gastric pH at birth and at 4 hours was measured in a further group of 'spontaneous' and 'induced' babies. A fall in gastric pH was apparent in both groups, but gastric acidity was not altered by oxytocin infusion. Our results neither exclude the hypothesis of gastrin transfer to the fetus during labour nor support the contention that oxytocin reduces such a transfer.

**References**


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