suffer from indigestion, acidity, clotting, and frequent vomiting? How can the nurse fail to waste away from that incessant suckling? Our peasant women are certainly more prudent about nursing their children, for they give them the breast only three or four times a day and leave them to cry as much as they please while they themselves are busy at work in the fields'.

Perhaps the most remarkable aspect of Ramazzini's writing was his emphasis on the

prevention of disease, a theme that remained generally neglected by the therapeutically orientated medical profession almost up to the present day. Nor were his clients any more responsive than they are today. In vain did he recommend personal cleanliness, the luxury of clean clothes on holidays, moderation in food, drink, and exercise, and warn of the danger when, scantily clothed, they left overheated workshops for the chilly streets.

1 Ramazzini B. *Diseases of workers*, 1713. Translated by W C Wright. Chicago: University of Chicago Press, 1940.

LETTERS TO THE EDITOR

Metabolic acidosis in premature babies

SIR,—Walter recently reviewed metabolic acidosis in newborns, however little mention was made concerning volume expansion as a possible treatment strategy.¹ In particular the role of human albumin solution (HAS) in ventilated, premature babies was not discussed.

We have studied the effect of an infusion of 15 ml/kg of 4.5% HAS, over 30 minutes, in 19 consecutive low birthweight infants receiving mechanical ventilation with a metabolic acidosis. Arterial blood was taken before and 15 minutes after volume expansion for the measurement of pH, base deficit, bicarbonate, and lactate (table). During the study ventilatory requirements remained stable.

The significant rise in pH ($p < 0.01$) and significant decrease in base deficit ($p < 0.0001$) was mirrored by a significant decrease in serum lactate ($p < 0.001$), interestingly there was no change in serum bicarbonate (paired t test).

This study shows that lactate accumulation, probably the result of hypovolaemic tissue hypoxia, is a cause of metabolic acidosis in premature babies. This acidosis is appropriately rectified by the infusion of a volume expander.

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1 Walter JH. Metabolic acidosis in newborn infants. *Arch Dis Child* 1992;67:767-70.

Traumatic neonatal intracranial bleeding and stroke

SIR,—Extending the concept of posterior cerebral artery compression associated with intracranial hypertension to other arterial distributions in the expansile neonatal skull requires reasonable proof. We believe that discrepancies in the radiological and clinical data presented by Govaert *et al* limit the strength of their final suggestion.¹

Direct demonstrations of arterial injury associated with intracranial hypertension are not presented. Several computed tomograms are misinterpreted, for example fig 1 does not show a 'basal convexity' haematoma while anatomically inaccurate interpretations are given for fig 6B. In fig 3, the haematoma is located at the level of the tentorium while the computed tomogram demonstrates a hypodensity in the territory of the proximal middle cerebral artery. This anatomical discrepancy is continued in the schematic drawing. Either the initial (case 5) or follow up (case 6) computed tomograms are not presented and consequently adequate conclusions may not be reached concerning the aetiology of the computed tomogram abnormalities. Finally, alternative clinical explanations for the computed tomogram findings such as a watershed infarct in case 1 or intraparenchymal injuries in cases 2 and 7 are ignored or discarded without sufficient cause.

Most of these infants demonstrated several neuroradiological abnormalities. Ascribing long term sequelae to one or the other may be inaccurate and legally treacherous. More importantly, the natural but inappropriate desire to correct surgically any potential problems implied by these data should be resisted until newer techniques such as magnetic resonance angiography can confirm the authors' speculations.

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1 Govaert P, Vanhaesebrouck P, de Praeter C. Traumatic neonatal bleeding and stroke. *Arch Dis Child* 1992;67:840-5.

Dr Govaert and coauthors comment:

We welcome the interest raised by our article on traumatic neonatal intracranial bleeding and stroke. The essence of our observation was the existence of hypoperfusion within the region of the middle cerebral artery ipsilateral to extracerebral haemorrhage. In the first case of our small series of seven an interval of at least two days between traumatic vacuum delivery (with subgaleal bleeding and supratentorial subdural haematoma) and subsequent hypoperfusion within the middle cerebral artery on that side suggested the possibility of vasospasm as an intermediary mechanism. This was not demonstrated with an angiographical technique and therefore we used the term hypothesis to describe that finding.

We would agree that 'basal convexity haematoma' may be a misnomer. In the absence of generally accepted defining criteria for various types of subdural bleeding we use the term to refer to supratentorial subdural haemorrhage not related to bridging veins of the superior sagittal sinus or to central tentorial injury near the great cerebral vein. Usually these collections are under the temporo-occipital lobe and along the temporal lobe convexity, as was the case in our first patient. The sections used in fig 1 do not show the 'typical' site of bleeding as visible in other sections.

In fig 3 (case 4) a basal subdural haematoma is associated with hypodensity of the mesial temporal lobe. According to all anatomy books we consulted this area is perfused by the posterior cerebral artery. This would not exclude concurrent hypoperfusion of branches of the middle cerebral artery, but we felt our computed tomogram did not show this with certainty. The hypodense areas depicted in fig 1 (case 1) cannot be a watershed arterial infarct according to the same references. It is plainly within the region perfused by the middle cerebral artery.

Some initial or follow up scans were not presented because there is a limitation to the use of scans in an article. We therefore chose to use the most representative scans. The patient in case 6 (fig 5) did not have a follow up scan, because it is our practice to perform scans only on clinical indication.

In case 2 (fig 2) there were three major reasons why we felt the haemorrhage to be

Effect of 4.5% HAS infusion in 19 low birthweight infants. Results are mean (SE)

	pH	Base deficit (mmol/L)	Serum bicarbonate (mmol/L)	Serum lactate (mmol/L)
Before infusion	7.258 (0.023)	8.98 (0.82)	17.37 (0.62)	3.56 (0.37)
After infusion	7.328 (0.025)	7.53 (0.79)	17.44 (0.71)	2.88 (0.34)