

from Schwartz-Mann and Clinical Assay (the results with these two kits showed close correlation). Steady-state plasma quinidine concentrations ranged from 4.2 to 12.7  $\mu\text{mol/l}$  (1.3 to 3.8  $\mu\text{g/ml}$ ) with an average of 7.0  $\mu\text{mol/l}$  (2.1  $\mu\text{g/ml}$ ).

All 12 patients showed a rise in plasma digoxin concentrations when quinidine was given (mean value 1.1 nmol/l (0.85 ng/ml) before quinidine, and 2.0 nmol/l (1.6 ng/ml) after quinidine). Five determinations of plasma digoxin were made in each patient after the start of quinidine treatment. One of the six patients whose plasma concentrations rose above the therapeutic range on one or more occasions developed symptoms of digitalis intoxication. In the first four patients the digoxin concentration was measured only once before adding quinidine, whereas more determinations were performed in the remaining patients to check that they had been taking the tablets as prescribed and that the digoxin concentrations represented steady-state values. The figure shows the drug concentration curves in the patient whose concentrations were measured most often.

The possibility of interference by quinidine or a metabolite of quinidine in the digoxin assay was excluded by the following control studies. (1) Digoxin was sought in the plasma of patients undergoing cardioversion in the same way while not on digoxin treatment: no measurable concentrations were found. (2) Quinidine was added to samples of whole blood from patients on digoxin alone until the quinidine concentrations fell within the therapeutic range: this did not affect the digoxin concentrations. (3) Digoxin added to blood samples from one group of patients who were taking quinidine alone,

and from another group of patients not taking quinidine: the digoxin concentrations did not differ between the two groups.

### Comment

The mechanism of this drug interaction is under investigation, particularly the possibility that quinidine affects the binding of digoxin in tissues. Until the clinical importance of the phenomenon is understood the increased risk of digitalis intoxication should be considered in patients taking this combination of drugs.

<sup>1</sup> Cramér, G, and Isaksson, B, *Scandinavian Journal of Clinical and Laboratory Investigation*, 1963, 15, 553.

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## SHORT REPORTS

### Biochemical changes associated with intractable pain

The "holy grail" of most medical practitioners who specialise in intractable pain is the objective assessment of pain. Various biochemical changes have been suggested<sup>1-4</sup>; terminal cancer with intractable pain has been associated with metabolic alkalosis,<sup>1</sup> and, conversely, Lindahl<sup>2</sup> suggested that acidosis was either the cause or the result of intractable pain. Some years earlier he had described a condition that he called "varalgia," in which acidosis was the concomitant of intractable pain, and if these patients were treated with an acid-free diet the pain was relieved.

Lascelles *et al*<sup>3</sup> measured the plasma cortisol concentration in a diurnal variation in two groups of patients with intractable pain, the cause of which was organic in one group and psychogenic in the other. There were no significant differences in the plasma cortisol concentration in these two groups; indeed, the results were within the normal range throughout the day. Plasma cholesterol and beta-lipoprotein concentrations have fallen after upper abdominal surgery<sup>4</sup> and the fall in these two criteria postoperatively could be equated with a decrease in pain. The plasma cholesterol and beta-lipoprotein concentrations, however, were within a normal range.

### Patients, methods, and results

The 52 patients admitted to the pain relief unit, Abingdon, with intractable pain\* were 32 women and 20 men with a mean age of 52.7 years (range 16-82). Fourteen had low back pain, 21 had cancer of various sites, and 17 suffered from various conditions such as arthritis, postherpetic neuralgia, and phantom limb.

On admission all patients had a full physical examination and any who failed Allen's test for the adequacy of the ulnar circulation to the hand were excluded from the study. After informed consent, the radial artery of the non-dominant wrist was cannulated percutaneously under local anaesthesia.  $\text{PaO}_2$ ,  $\text{PaCO}_2$ , and pH were measured with an electrode system (Radiometer PHM 27) every two hours from 0800 to 2200. Blood samples were taken for routine haematological (haemoglobin and white cell count etc) and biochemical tests (electrolytes and sequential multiple analyses 12/60). In addition, 13 patients had blood drawn at 0900 for plasma cortisol estimations.<sup>3</sup>

Thirty of the 52 patients obtained pain relief after treatment. This was assessed by asking the patient and confirmed by the withdrawal of analgesics or a substantial reduction in the strength and amount needed. Only 10 of these 30 patients, however, were available for follow-up  $\text{PaO}_2$ ,  $\text{PaCO}_2$ , and pH measurements. Repeat arterial cannulations were performed in seven of them, while the remaining three had a single arterial puncture.

The mean  $\text{PaO}_2$ ,  $\text{PaCO}_2$ , and pH together with the diurnal range for each of these three groups are shown in the table. After successful treatment

\*Intractable pain was defined for this study as pain of at least one month's duration that had not responded to conventional treatment.

Diurnal mean and range of  $\text{PaO}_2$ ,  $\text{PaCO}_2$ , and pH for each clinical group and mean  $\text{PaCO}_2$  and pH after successful treatment

	Diurnal mean and range of $\text{PaO}_2$ , $\text{PaCO}_2$ , and pH for each clinical group						Normal range at Oxford
	Low back pain		Cancer		General		
	Mean	Range	Mean	Range	Mean	Range	
$\text{PaO}_2$ (kPa)	13.2	7.3-18.4	11.6	7.1-16.0	12.1	8.7-17.5	8.0-16.0
$\text{PaCO}_2$ (kPa)	4.1	2.6-5.3	4.1	2.4-5.5	4.2	3.0-5.3	4.8-5.9
pH	7.42	7.37-7.46	7.42	7.33-7.52	7.42	7.32-7.49	7.36-7.45

Mean  $\text{PaCO}_2$  and pH after successful treatment

Sex	Age	Group	Pretreatment $\text{PaCO}_2$ (kPa)	Post-treatment $\text{PaCO}_2$ (kPa)	Pretreatment pH	Post-treatment pH
F	56	LBP	4.6	4.8	*	7.46
F	33	LBP	3.6	4.5	7.43	7.42
M	49	LBP	4.3	4.8	7.40	7.41
F	22	LBP	4.0	4.6	7.43	7.38
M	64	Cancer	3.9	4.9	7.41	7.40
M	60	Cancer	4.0	4.8	7.43	7.43
F	34	Cancer	4.4	5.0	7.44	7.43
F	79	Cancer	4.2	5.0	7.41	7.42
F	73	Cancer	4.5	5.1	7.45	7.44
M	57	Cancer	4.1	5.0	7.43	7.41

LBP = Low back pain.  
\*No result owing to technical fault.

there was a significant rise in the  $Paco_2$  ( $P < 0.001$ ) in ten patients (table) but no significant change in the other two values. Mean plasma cholesterol concentration was 5.6 mmol/l (range 2.6-7.7) (216 mg/100 ml (100-297)) (normal 4-8 mmol/l (154-308 mg/100 ml)) while the mean plasma cortisol concentration in the 13 patients studied was 472 nmol/l (range 110-1090) (17  $\mu$ g/100 ml (range 3-39)). Normal at 0900 was 280-690 nmol/l (10-25  $\mu$ g/l). The routine biochemical and haematological tests showed no unexplained abnormalities.

### Comment

These results do not confirm an association between alkalosis<sup>1</sup> or acidosis<sup>2</sup> and intractable pain. In the 13 patients studied the plasma cortisol concentrations were similar to those reported.<sup>3</sup> Plasma cholesterol concentrations were within the normal range. Nevertheless, the low  $Paco_2$  found in all groups associated with a normal pH confirms the chronicity of the hyperventilation observed. All but chronic anxiety and noradrenaline of the documented ventilation stimuli<sup>4</sup> could be excluded as the stimulus to this hyperventilation. But whatever the ventilatory stimulant, it is almost certainly secondary to intractable pain. This is confirmed by the significant rise ( $P < 0.001$ ) in  $Paco_2$  found in the 10 patients who had obtained pain relief. It has been possible to use the rise in  $Paco_2$  after pain relief as objective evidence of successful treatment.

I thank Dr Charles Michel of the University Department of Physiology for his advice and encouragement and also Dr Wilkinson of the Biochemistry Department of the Radcliffe Infirmary for his help in this study.

<sup>1</sup> Evans, R J, *Canadian Journal of Surgery*, 1972, **15**, 34.

<sup>2</sup> Lindhal, O, *Advances in Neurology*, vol 4, p 45. New York, Raven Press, 1974.

<sup>3</sup> Lascelles, P T, *et al*, *Brain*, 1974, **97**, 533.

<sup>4</sup> Keele, K D, and Stern, P R S, *Journal of the Royal College of Physicians*, 1973, **7**, 319.

<sup>5</sup> Hey, E N, *et al*, *Respiration Physiology*, 1966, **1**, 193.

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## Extensive retinal haemorrhages in infancy—an innocent cause

Extensive retinal haemorrhages in babies other than neonates are recognised as an important sign of child abuse,<sup>1</sup> being associated with violent shaking and the development of subdural haematomas,<sup>2</sup> and sometimes with thoracic compression.<sup>3</sup> We report such haemorrhages in a baby who had patently not been a victim of abuse.

### Case report

A previously well 2-month-old boy was left asleep in his pram in the garden protected by a cat net. After 30 minutes his father discovered him lying undisturbed but not breathing. He picked him up and found him pallid and limp, and thought he was dead. He called his wife, who came at once and held the baby to her shoulder and slapped him repeatedly on the back to try and revive him. Eventually he choked and spluttered, and at last began to breathe again. There was a little blood staining his face and the pillow, and some bloody mucus was expelled from his nostrils.

On arrival in hospital 10 minutes later he was still gravely ill, being barely conscious, shocked, and cyanosed, with erratic and inadequate respiration and a pulse rate of 160/min. He was afebrile. He was well-nourished, with no evidence of neglect or trauma, but there was blood caked in his nostrils. The eyes had no external injury and the media were clear, but we could see extensive fresh haemorrhages in the nerve-fibre layer of both fundi. There was bilateral macular oedema but no swelling of the optic discs.

His haemoglobin was 8.2 g/dl and chest radiography showed patchy bilateral perihilar shadowing. All other investigation results were negative

or normal (blood film; leucocyte and platelet counts; clotting studies; concentrations of serum electrolytes, calcium, and urea; cultures of blood and cerebrospinal fluid (CSF); CSF biochemistry and cytology; viral culture of nasopharyngeal secretions; skeletal survey; and subdural taps).

He rapidly improved after treatment with oxygen, intravenous fluids, and antibiotics. He remained irritable and hyperreflexive at first but after three days he was back to normal. He could fixate and follow a light, and optokinetic nystagmus was readily elicited. Electroretinography and measurement of visually evoked responses showed satisfactory function of both retinas and of the higher pathways. A repeat chest radiograph was clear. When reviewed two months later he appeared entirely unscathed by the experience. His eyesight seemed normal, and the retinal haemorrhages had completely resolved.

Because of initial suspicion of child abuse the parents were questioned with particular care on the child's admission and were later interviewed by a senior paediatrician. Their answers throughout were frank, consistent, and entirely convincing, and the family doctor and health visitor attested to their excellent parenthood. Not a single feature emerged from the social background, the history, or the physical findings to support this suspicion.

### Comment

Retinal haemorrhages can be produced by thoracic compression that is insufficient to cause detectable damage to the chest itself.<sup>4</sup> The retinal vessels of infants may be particularly vulnerable, for many babies have extensive fundal haemorrhages after an apparently normal birth.<sup>5</sup> We think that this baby became apnoeic after aspirating blood from an epistaxis, and that his mother's life-saving measures transmitted pressure from the thorax to retinal veins that were already compromised by hypoxia.

To avoid accusing innocent parents of battering their babies vigilance for child abuse must be balanced by open-mindedness to alternative explanations for its typical features: extensive fundal haemorrhages are not invariably diagnostic.

We thank Professor J K G Webb for his encouragement in reporting this case.

<sup>1</sup> Gilkes, M J, and Mann, T P, *Lancet*, 1967, **2**, 468.

<sup>2</sup> Caffey, J, *American Journal of Diseases of Children*, 1972, **124**, 161.

<sup>3</sup> Tomasi, L G, and Rosman, N P, *American Journal of Diseases of Children*, 1975, **129**, 1335.

<sup>4</sup> Morgan, O G, *Transactions of the Ophthalmological Society of the United Kingdom*, 1945, **65**, 366.

<sup>5</sup> Baum, J D, and Bulpitt, C J, *Archives of Disease in Childhood*, 1970, **45**, 344.

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## Needle tracheostomy for acute upper airway obstruction

Emergency tracheostomy is seldom needed, but when it is speed and simplicity are important. The introduction of a Medicut intravenous cannula (Sherwood Medical Industries Ltd) into the trachea to provide a temporary airway while preparations are being made for a formal tracheostomy has been described but has not attracted the attention that it deserves. I describe a patient on whom needle tracheostomy was used successfully.

### Case report

A 64-year-old man with a history of chronic sputum production (peak expiratory flow 150 l/min) was admitted with a five-week history of hoarseness and a one-week history of increasing shortness of breath. No obvious cause for his symptoms could be found, and there was no evidence of an acute exacerbation of his chronic lung disease. Radiography showed a hyperinflated chest, but the lung fields were clear and there was no bronchial neoplasm. Indirect laryngoscopy was arranged because of a possible laryngeal neoplasm. Before this could be done, however, he developed acute stridor