"safe and effective prophylactic," and because "50% of acute infarcts develop major ventricular dysrythmias."

(4) Consider intravenous atropine for bradycardia.

(5) If the pulse is irregular with runs of ectopic beats 50 mg. of lignocaine slowly intravenously and the rest of the syringe pack intramuscularly.

(6) Finally, beseech the ambulance men to give a smooth, steady, clammer-free ride to Treliske's coronary care unit.—I am, etc.,

R. D. MARTIN.

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Bell's Palsy and Herpes

SIR,—In your leading article on Bell's palsy and surgery (17 October, p. 126), you twice say that surgical decompression for the relief of the condition is controversial. I agree that this is so, and would indeed echo Professor Ritchie Russell's teaching that if you want to succeed with a decompression operation you must operate before the onset of the lesion. There is no magic moment after the paralysis has occurred.

Where the controversy comes in, however, is in relation with imperfect diagnosis. Too often a herpetic lesion is wrongly categorized as a Bell's palsy, and creeps into the statistical surveys on one side or the other. Bell's palsy is characterized by sudden and only slightly progressive paralysis, without much pain. In facial nerve herpes there is severe faceache which often precedes the paralysis by as much as 24 hours. The paralysis at its onset is usually incomplete, but it progresses over the next 24-48 hours.

My experience has been that this latter is the kind of case which has a bad prognosis if left alone and which rewards decompression. The time to do this is while the paralysis is progressing. Although the lesion may well be ganglionic basically, the nerve sheath becomes very inflamed, with vascular engorgement and an almost shaggy appearance. This is different from the "swollen spaghetti" look of the Bell's palsy nerve.

While agreeing that many herpetic cases do recover on masterly inactivity, I would urge that surgery should be thought about early and undertaken if the paralysis is progressing. But the differential diagnosis is the main issue, and here the virologist may help.—I am, etc.,

RONALD MACBETH.

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Children in Intensive Therapy Units

SIR,—Drs. J. R. Harper and S. Varakis (13 June, p. 670) have pointed out that any hospital dealing with acutely ill children must solve its problems according to the local conditions. I strongly support the view that in a peripheral hospital the nursing of children in general intensive care units is a forward and not a retrogressive step.

My own service looks after a population of over 300,000. During the past 14 years we have been able to engage only two nurses with paediatric nursing qualifications; we would have been glad of others, but they are just not available.

The intensive care unit at Wigan Infirmary has now been in operation for just over three years. A total of 700 patients have been admitted and of these 50 (7%) have been children aged 12 and under. A very wide range of ages and of clinical conditions have been handled. We are fortunate that the unit is situated a few yards from the main children's ward, so that a mother living-in with her child in the hospital can use one of the rooms available on the children's ward. The transfer of a child to or from the intensive care unit at the appropriate time is also very easy.

I have not inquired in detail from the families involved what they thought about their experiences. This would be interesting, but in my case largely irrelevant. I am certain that the intensive care unit has saved lives which would otherwise have been lost, and the doubts of any of the families would pale into insignificance compared with the certainty of the medical and nursing staff of the hospital that the intensive unit has been invaluable.

While I agree with Drs. W. J. Appleyard and M. C. Joseph (16 May, p. 423) that the trend should be towards nursing children in children's units, it must never be at the expense of safety and technical efficiency. In the average peripheral hospital the staffing situation would seldom reach the levels to allow these standards of intensive care on the children's ward itself, and the paediatrician would find it very hard to justify such a concentration of nurses and equipment in competition with his colleagues in other disciplines, with whom these rare and expensive facilities can easily and effectively be shared.—I am, etc.,

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Prophylaxis of Postoperative Pulmonary Atelectasis

SIR,—Space permits us to comment on only a few of the points raised by Mr. M. O'Driscoll (3 October, p. 26). In brief, we find the design of his experiment inadequate, confused, and based on a false premise, the methods unsound, the results and their interpretation contradictory, the discussion untenable, and consequently the summary misleading.

Of the eight references that Mr. O'Driscoll cites in his first paragraph five have been misrepresented. Three of these authorities did not make the statements ascribed to them; two are quoted out of context and without those authors' reservations.

We are told that the study group consists of 181 patients divided into two unmatched groups. In Table I a further 116 patients mysteriously appear. 235 of Mr. O'Driscoll's patients received halothane as an anaesthetic while a further 52 were given Fluothane (halothane).

The latter part of this paper concentrates on those patients who did not receive bicarbonate and in whom chest complications were said to have occurred. It is claimed that a "particularly pronounced" metabolic acidosis was seen in these patients. Table III shows this group to have been alkalotic with a mean pH of 7·43. To describe a mean base deficit of less than 4 mEq/l. as diagnostic of marked metabolic acidosis shows a lack of appreciation of the normal variations in acid-base state. Deductions made on the basis of isolated serum venous bicarbonate levels (presumably performed by an autoanalyser technique) are totally meaningless. These patients had a mean P_{CO_2} of 27·6 mm. Hg. Mr. O'Driscoll is most reluctant to ascribe this respiratory alkalosis to increased alveolar ventilation. He believes that this hypocarbia demonstrates a reduction in physiological dead space and a probable reduction in carbon dioxide production. This is not so. Alveolar dead space is normally small and even its reduction to zero would have an insignificant effect upon arterial P_{CO_2}. Anatomical dead space could hardly decrease by the necessary order implied in the data. A reduction in CO_2 production of the order of 73% would be required in order to produce the changes in P_{CO_2} seen. In any event falls in both CO_2 production and in dead space are usually accompanied by hypoventilation in order to maintain normal alveolar P_{CO_2} values.

Because of the known difficulties in measuring ventilation with a respirometer and face mask, the known errors of blood-gas analyses performed on arterialized venous blood, the lack of details of any correction of acid-base data for changes in temperature or oxygen saturation, and the apparent confusion between Astrup and venous bicarbonates, we feel that the most likely explanation of Mr. O'Driscoll's findings is a methodological error.—We are, etc.,

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Allergy to Iprindole (Prondol) with Hepatotoxicity

SIR,—We wish to add four further cases of iprindole-induced hepatitis to those previously reported in your correspondence columns (7 February, p. 367, and 25 April, p.238).

Case 1.—Female aged 48 years. Iprindole prescribed for mild depressive symptoms. Hb and E.S.R. normal. Many years ago she had taken amitriptyline for similar symptoms without ill effects.

Seven days after starting iprindole noticed definite improvement in mood. On the eighth day sudden onset of "influenzal" symptoms—shivering, back pain, malaise, anorexia, and headache. The patient took her own temperature and found it to be 104°F. (40°C.). At this point she stopped the drug. The following day she developed pain in the right hypochondrium and passed dark urine.

When examined on the tenth day after starting iprindole the patient was found to be mildly jaundiced and tender beneath the right costal margin. The temperature was not raised. Urine contained bile on inspection, and this confirmed by Ictotest.

Liver function tests taken the following day were as follows: Serum bilirubin 1·1 mg./100 ml.; S.G.O.T. 36; S.G.P.T. 93 units/ml.; cephalin cholesterol flocculation positive; colloidal gold flocculation negative; alkaline phosphatase 20 K.A. units. Albumin 3·4; globulin 3·1; total serum protein 6·5 g./100 ml.; zinc sulphate turbidity 5 units.

Jaundice rapidly disappeared and bilirubin was no longer present in the urine four days after its first detection.

The patient made a rapid and complete recovery. Cholecystography showed a normal gall bladder.