conclude that the situation is one of compensated respiratory alkalosis. Surely these patients have a ventilation-perfusion imbalance, with a shunting effect in the collapsed areas. Consequent hypoxaemia has led to hyperventilation in unaffected lung with resulting hypocapnoea and alkalosis.

It is clear that it is unwise to make assumptions about acid/base status on the basis of bicarbonate values alone. Less than one third of the patients studied had pH values performed, and those were in the latter portion of the study when the stimulus to objective reappraisal of the initial premise cannot have been strong.

If the lowered bicarbonate is a compensatory change, then to infuse alkaline fluids is not a logical treatment. The apparent improvement in the treated group must be viewed with caution as radiography of lung bases in supine patients with raised diaphragms is not, alone, sufficiently accurate to assess the presence or absence of atelectasis. Further, for the comparison to be valid the untreated group should have had injections of an equivalent amount of similarly hypertonic saline (perhaps on a double-blind schedule)—the author ignores the effect of 300 mEq of sodium on the extracellular fluid which must be augmented in volume significantly with some change in pulmonary and systemic haemodynamics.

I hope these observations will prevent any premature use of bicarbonate in post-operative patients until the findings are checked in more detailed and objective studies. In a proportion of patients with ischaemic heart disease, often undiagnosed, the movements of body potassium induced by the recommended regimen must predispose to dangerous arrhythmias.—I am, etc.,

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Cataracts

SIR,—Mr. J. A. E. Primrose's letter (31 October, p. 308) well illustrates the diversity of opinion which exists within the ophthalmic profession on the management of cataract cases, especially the unilateral variety. He is prepared to extract the cataract (to improve the field of vision on this side), but makes no attempt to correct the focus and restore binocular vision by a contact lens or an intraocular lens implant. Mr. Primrose is fortunate to deal with such undemanding patients.

I agree that senile cataract tends to be a bilateral disease, but in my experience the development of significant opacities in the contralateral lens may be delayed for very much longer than a year or two (as Mr. Primrose expects) after the first cataract has been dealt with; 15 or 20 years or more may elapse without this happening. Indeed, I should be interested to know why one so often sees in practice advanced unilateral cataracts (at any age) with a clear contralateral crystalline lens when the usual causes of cataract—for example, associated congenital defects, trauma, heterochromic cyclitis, etc.—cannot be inculcated.—I am, etc.,

D. P. CHOYCE.

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Phentolamine in Heart Block

SIR,—Not all would agree with the implication by Dr. R. Haider and Dr. S. P. Singh (31 October, p. 307) that complete heart block with slow ventricular rate following acute myocardial infarction should

Continuous ECG strips showing the Wenckebach phenomenon. The block varies between 2:1 and 3:2; when 3:2 block is present there is progressive lengthening of the P/R intervals, culminating in failure of conduction.

always be treated with transvenous pacemakers—certainly not as a routine when this is due to inferior infarction, where Adam-Stokes attacks are rare and the block usually transient.^{1,2} They support the use of phentolamine as an alternative, but on the basis of a case of second-degree atrioventricular block, and, whatever the phentolamine did, it failed to stimulate the sino-atrial node (the theoretical basis for its use), for the atrial rate remained steady at 88 beats a minute and it was the degree of atrioventricular block that varied from 2:1 to 3:2.

It is difficult to see why increasing the atrial rate should be beneficial when the block is in or distal to the atrioventricular node. Had the block been of the Mobitz (Type II) variety (unusual in inferior infarction), where the P-R intervals of conducted beats are fixed, the danger of complete heart block might have been greater,³ but if their Figure 2 is representative their patient showed the Wenckebach phenomenon rather than "sinus beats alternating with 2:1 block beats." In Wenckebach (Type I) block changes from 3:2 to 2:1 block can occur spontaneously^{3,4} (see Figure), and there is no proof that the phentolamine was responsible for the effects they describe.—I am, etc.,

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Transmission of Buruli Disease

SIR,—Epidemiological evidence (in press) suggests that infections with *Mycobacterium ulcerans*, the organism causing Buruli disease, are usually acquired from some nonhuman source. Attempts to identify this source of infection have so far been unsuccessful. However, the epidemiological characteristics of the disease suggests that *Myco. ulcerans* may be a saprophyte on certain species of grass, and that Buruli disease is contracted by contact with grass and subsequent penetration of *Myco. ulcerans* through small scratches or other traumatic

lesions in the skin. This could explain the geographical distribution of the disease in Uganda, and the way in which the site of the lesion on the body differs with the sex and age-group of the patients.

During the past year we have obtained cultures of mycobacteria from different grasses collected within areas where the disease occurs.

Two cultures, from the grasses *Hyparrhenia rufa* and *Imperata cylindrica*, have yielded mycobacteria which have the characteristics of *Myco. ulcerans* obtained from human lesions in Uganda. The initial cultures were obtained on Lowenstein-Jensen medium containing sodium pyruvate after eight and fifteen weeks' incubation at 33°C. There was complete inhibition of growth on media containing sodium salicylate, nicotinamide, and paranitrobenzoic acid. At 37°C. there was delayed and diminished growth of one of the mycobacteria and complete inhibition of growth of the other. A suspension of the organism from *H. rufa* was injected into the footpads of three mice, and four weeks later one mouse developed an erythematous swelling macroscopically identical to the initial lesions which resulted from injection of *Myco. ulcerans*. Antigen tests are being carried out to determine whether these mycobacteria may be classified as *Myco. ulcerans*.

Twenty-four other cultures of mycobacteria have been obtained from specimens of *H. rufa*, *I. cylindrica*, *Panicum maximum*, and *Pennisetum catabasis*. All these cultures were obtained after incubation at 33°C., with no growth at either 25°C. or 37°C. Identification and pathogenicity tests have so far been carried out on four of them. They are dissimilar to *Myco. ulcerans* in the absence of inhibition of growth on special media or at 37°C., but they are similarly pathogenic to mice, producing erythematous footpad swellings which either regress after some weeks or progress to involve the entire leg.

Our observations show that grasses are an abundant source of mycobacteria, some of which are similar to *Myco. ulcerans* and cause local lesions when injected into mice. As far as we are aware this is the first time mycobacteria of this kind have been isolated from a non-human source.—We are, etc.,

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Genital Herpes

SIR,—Your leading article on genital herpes and cervical cancer (31 October, p. 256) is timely. You mentioned that herpes simplex virus (H.S.V.) may be associated with male smegma, but it is not yet clear whether this virus is carried asymptotically in the male urethra. Overt endourethral herpes is uncommon, and is usually associated with severe dysuria.^{1,2} Searches for H.S.V. in non-specific urethritis have been remarkably unsuccessful. In a study of 120 men with non-specific urethritis, using both rabbit kidney tissue culture and electronmicroscopy, no H.S.V. was found (Catterall, personal communication).

Recently in Portsmouth we have isolated H.S.V. in human embryo lung fibroblasts from the urethral swabs of two out of 12 men with scanty urethral discharge. The men showed no clinical signs or symptoms of herpes genitalis. From one of them T-mycoplasmas were also isolated. The swabs

were inserted into the fossa navicularis, approximately 10 mm. from the exterior. It may be significant that the swabs were transported in our mycoplasma transport medium, which contains yeast extract and 20% unheated horse serum,³ and were not frozen. We have frequently isolated H.S.V. from cervical swabs transported in nutrient broth, but it is possible that a higher number of isolations of H.S.V. might be achieved by using a transport medium enriched with serum.—We are, etc.,

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Sign in Gall-bladder Disease

SIR,—I think we should be thankful that Sir Zachary Cope has used his own ills to improve our knowledge of gall-bladder disease (18 July, p. 147).

The pathogenesis of acute cholecystitis is still far from clear, and there remains disagreement on the role played by bacteria versus noninfective inflammation. In addition to the clear account of the anamnesis it would be very much worth while, I believe, to know whether a bile culture was taken at surgery on this distinguished patient, and if so, what the bacteriologist reported.—I am, etc.,

RODOLFO HERRERA-LLERANDI.

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Calcium Metabolism in Patients with Epilepsy

SIR,—I was interested in the papers by Professor C. E. Dent and his colleagues (10 October, p. 69) and Mr. D. J. F. Richens and Dr. A. Rowe (p. 73). In 1968 when Kruse and Stecher¹ published their findings of rachitic changes in the bone and blood in nearly 15 out of 60 patients aged between 4 and 23 years of age examined at the Kinderklinik Heidelberg we were doing a survey of serum folates in 41 children. All these children had been under treatment with phenobarbitone or phenytoin, or both, for a duration of 1-11 years. X-ray films were taken of the wrist joints of these children and there was no evidence of disturbance of ossification in any child. The serum calcium estimations ranged from 9.0 to 11.7 mg./100 ml. (only one specimen was 11.7). The main range was from 9.0 to 11.1. However, the alkaline phosphatase levels all showed an increase ranging from 14.8 to 41.2 K.A. units. Despite the very high levels of serum alkaline phosphatase recorded it is interesting to note that no hypocalcaemia was demonstrated in any of these children.

The children were originally selected because their anticonvulsant treatment had been relatively stable over the years, and though other drugs had been given at times

this had been only for relatively short periods. At that stage only one of the children had had pheneturide and that only for a period of three months, a year before these investigations took place. His serum alkaline phosphatase was 16.6 K.A. units and calcium 10.7 mg./100 ml.

I am grateful to Dr. Albert Sachs, lately consultant pathologist to the Queen Victoria Hospital, East Grinstead, who did the estimations for us, and to Dr. William Campbell, radiologist to the Queen Victoria Hospital, who reported on the x-ray examinations.

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Wrong Operations

SIR,—I am not impressed by Dr. J. N. Thorpe's suggestion (24 October, p. 242) of using the patient's date of birth as an identity code. It is said that "there is one born every minute" so that it would seem that there are 1,440 of the less sapient members of the genus *Homo* with identical birthdays for every day of the year.

A friend of mine with a mathematical turn of mind assures me that if a room contains 20 people the chances are even that two will share a birthday. Perhaps they arrange things differently in Sweden.—I am, etc.,

Plymouth.

IAN W. PAYNE.

Oxygen and Myocardial Infarction

SIR,—Dr. A. L. Muir and his colleagues (31 October, p. 276) show that in patients with myocardial infarction mixed venous oxygen saturation is reduced when they are clinically in left ventricular failure or cardiogenic shock. The authors claim that these measurements correlate with cardiac output, and suggest they should be used when the apparatus for estimating cardiac output is not available as "an important aid to the rational treatment of patients. . ."

Inspection of the data in fact suggests that their clinical assessment gave a better guide to cardiac output than mixed venous oxygen saturation in cases complicated by cardiac failure. This may be because the most severely affected group were receiving oxygen when the measurements were made, whereas the group classified as left ventricular failure were not.

Although the data taken overall reveal a correlation the scatter between individual results is such that measurements of mixed venous oxygen saturation would be an unreliable substitute for measurement of cardiac output in monitoring a patient's progress.—I am, etc.,

RICHARD WALDRAM.

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Motoring

SIR,—In fairness to the British manufacturer may I enlarge on your Motoring Correspondent's comments (7 November, p. 374) concerning the Britax Twin-Lok seat belts which can be put on and adjusted with one hand? In fact, this type was originally designed for the Triumph 2000 Mk. II from the blue-print stage, and was shown

for the first time in London at the 1969 Motor Show. Since then it has featured on an increasing number of new cars. Just to remove any ambiguity, automatic retraction is not unique to the Volvo belt, as the Twin-Lok is also available in static and automatic forms.

On another point raised may I suggest that the smallest engine in any given body shell is by no means always the most economical—for example, the Ford Capri 1300 and 1600 respectively? In give-and-take driving the smaller engine has to work harder to yield any set performance and quite often therefore uses more petrol than its more lightly stressed bigger stable-mate. In addition, the larger engine often proves more durable for the same reason, and is usually less noisy and more pleasant to sit behind.—I am, etc.,

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Skin Disease and the Gut

SIR,—Those of us with children under our care have long been aware of the close link which exists between the skin and the gut (leading article, 1 August, p. 240). Could it be otherwise, when the prototype disorder linking both was recognized nearly 30 years ago and appropriately named acrodermatitis enteropathica by Professor Niels Danbolt, who still holds the chair of dermatology at Oslo? We ourselves^{1,2} were among the first to report enzyme histochemical and ultrastructural studies of this disorder, and later recognized genetic variants which had previously been considered to be didoquin-resistant forms of the disease. We were able to show that impaired tolerance of disaccharides and monosaccharides was responsible for this, and elimination of these from the infant's diet was followed by prompt response to didoquin (sugar-free), and all three children are now thriving and well. No gross or light microscopical changes were detected in jejunal or duodenal biopsy from any variants of the disease, but it would be quite misleading for changes in morphology to be considered the sole of this or indeed any disease, because gross functional disturbances may present without any detectable morphological change even at the ultrastructural levels.

As to the question of possible links between dermatitis herpetiformis and coeliac disease: I cannot recollect a single child with coeliac disease who had anything amiss with the skin during the several years that I shared a ward at the Hospital for Sick Children with Sir Wilfred Sheldon, who is prominent among the pioneers in the investigation of this disorder. During the same period, and subsequently, several children suffering from both forms of dermatitis herpetiformis (the bullous and small vesicular form) were under my care, but showed no signs or symptoms suggestive of any enteropathy. It would seem, therefore, that if the northern workers are correct, coeliac disease in adults is very different from that seen in children as we meet it in the south of England.

With regard to "regional" differences, surely a more profitable approach would be to study the distribution of both gluten enteropathy and dermatitis herpetiformis in different ethnic populations. We already possess sufficient information about the gene for gluten enteropathy throughout Europe, and we know too, that ethnic differences are reflected in the British population, shown by the appreciably higher incidence of this disease in children of Irish, Welsh, and Scottish