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 is 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 350 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 postoperative 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 inculpated.—I am, etc.,

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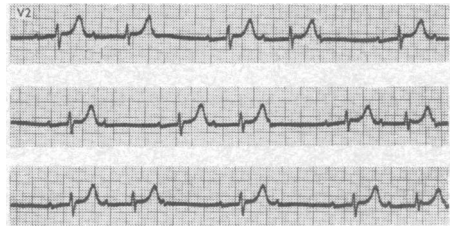
Phentolamine in Heart Block

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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2 Chamberlain, D., and Leinbach, R., *British Heart Journal*, 1970, **32**, 2.
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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. cylinderica*, *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 asymptomatically 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