with regard to a recent report on pulmonary infections in London addicts.[6 7] These are relevant considerations which I believe to be within the scope of the article by Dr. Bewley and his colleagues, and which deserve brief mention.—I am, etc.,

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## Blood Base Changes

SIR,—Dr. J. L. Anderton and others (4 May, p. 279) suggest that it is necessary to use arterial blood for the determination of a patient's metabolic acid–base state (base excess). I would dispute this. It is true that during pancreatic secretion venous blood from the gut should have a metabolic acidosis as compared with arterial blood, but the tissues of the arm normally neither produce nor utilize non-volatile acids, therefore there is no difference, due to this cause, between blood from the brachial artery and from an antecubital vein. However, because oxyhaemoglobin is a stronger acid than reduced haemoglobin, venous blood has a relative metabolic alkalosis due solely to its reduced haemoglobin content. When the Radiometer Astrup apparatus is used to determine the metabolic acid–base state, whether as base excess, buffer base, or standard bicarbonate, the blood is equilibrated with mixtures of carbon dioxide in oxygen, and the haemoglobin perforce becomes fully saturated with oxygen. The buffer line determined by this technique is therefore that of fully oxygenated blood, and, provided that the patient is not suffering from arterial hypoxaemia, it will give an estimate of his arterial metabolic acid–base state.

It is not disputed that the $P_{CO_2}$ of venous blood is higher than that of arterial blood from the same patient, but the Astrup technique determines the metabolic acid–base state of the blood whatever the $P_{CO_2}$. It is perhaps worth mentioning that the difference between the metabolic acid–base states of arterial and venous blood may result in errors when the Astrup technique is used for the determination of the carbon dioxide tension of desaturated blood. In this technique the *p*H which is used in the interpolation is that of the blood as it was taken from the patient, whereas the buffer line is that of fully oxygenated blood. If the blood is desaturated, therefore, the calculated $P_{CO_2}$ will be too low by an amount which depends *inter alia* on the concentration of reduced haemoglobin. Errors due to this discrepancy may be minimized by calculation of the position of the true—that is, venous—buffer line and the use of this for the interpolation process. The shift of buffer line may be determined by means of the approximate factor of Siggaard-Andersen[1] or the more precise factor of Kelman,[2] which takes account of the influence of *p*H and $P_{CO_2}$ on this parameter.—I am, etc.,

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## Noradrenaline in the Tropics

SIR,—The catecholamines adrenaline and noradrenaline are well known to be unstable in dilute solutions, being affected by many conditions, such as oxygen, alkaline media, high temperature, and the presence of heavy metal ions.[1 2] Although it has recently been reported that under temperate conditions noradrenaline is stable for up to four hours in infusion solutions as used clinically,[3] it was felt desirable to confirm that this stability would pertain in the tropics. The stability of noradrenaline in infusion solutions under local conditions was therefore investigated, and the preliminary findings are reported in this communication.

Solutions of noradrenaline in physiological saline (0.9% NaCl), in dextrose (5%), and in dextrose (5%) in physiological saline were investigated. The infusions were made up of 4 mg. noradrenaline (injection noradrenaline bitartrate, expressed as salt) in 540 ml. of the solution under test. All were prepared according to *B.P.* requirements. The flow rates of the solutions were adjusted so that each "drip" lasted four hours. The noradrenaline content immediately after mixing, at the second hour, and at the fourth hour was estimated fluorometrically.[4] Room temperature at which the "drips" were run was 29–30.5° C.

Noradrenaline was found to be stable in dextrose (5%) as well as in dextrose (5%) in physiological saline (three separate observations were made on each solution). However, with physiological saline there was considerable loss of activity (four separate observations). Activity remaining after two hours was $71 \pm 5.4\%$ (mean $\pm$ S.E.) and by four hours was $36 \pm 12.4\%$. These reductions in activity were statistically significant ($P < 0.02$ for two hours and $P < 0.01$ for four hours). The *p*H of physiological saline varied from 7.2 to 7.7, that of the other two solutions was constantly at 4.0. After the addition of 4 mg. noradrenaline bitartrate (contained in 1 ml.), the *p*H of physiological saline fell to 5.7. The *p*H values of dextrose and dextrose in saline were lowered by only 0.1.

These findings suggest that when noradrenaline is to be given in infusion solutions under tropical conditions the use of physiological saline is best avoided unless steps are taken to modify the *p*H.—I am, etc.,

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## Psychiatric Problems and General Practitioners

SIR,—Having just read the article "Participation of General Practitioners in Community Psychiatry" (20 April, p. 168) I feel that its conclusions could be damaging to the trend for integration of community health care. It is based on a small and unrepresentative sample, compared with the national statistics of general practice, and it is the work of two observers only, in collaboration. I hope, therefore, that these findings will not be taken to represent a true picture of general practice.

In the sample of general practitioners chosen 50% were over 50, and 60% had practice lists of over 3,000 urban patients. In the whole country only a third of general practitioners are over 50 and the average list is 2,470 (Ministry of Health figures 1967). The majority of these doctors, then, were hard worked, or ageing, or both. Many had trained before psychiatry was given any prominence (28% had *no* psychiatric undergraduate training at all) and before hardly any of the present psychiatric drugs had been developed. With the tripartite health service they would have indeed been detached "from the organization and operation of specialist psychiatric services." It is not surprising, therefore, that this survey found some indifference to psychiatry. Equally, future changes probably will not concern many of them—they will have retired.

Most of the implied criticisms in this article could be removed once the skills of the social and welfare workers are more directly a part of the general practice community—that is, reluctance to take on more psychiatric patients, or to give aftercare for the chronically disabled. The attitude of an older section of general practitioners should not be allowed to discourage such progress. It is a pity that this survey gave such little prominence to such enthusiasm as it did reveal. It would be interesting to compare this survey with another, representing the views of the younger generation of general practitioners. I suspect this would be much more encouraging.—I am, etc.,

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## Group A Streptococci Resistant to Lincomycin

SIR,—We were interested in the letter from Drs. J. Kohn, J. H. Hewitt, and C. A. M. Fraser (16 March, p. 703) describing an outbreak of infection in a burns unit with *Streptococcus pyogenes* resistant to lincomycin and erythromycin.

Since the isolation of erythromycin-resistant strains of *Str. pyogenes* in burns which we reported nine years ago[1] all strains of *Str. pyogenes* isolated from burns in this unit have been tested for sensitivity to erythromycin. No further strains resistant to erythromycin or to other macrolide antibiotics were found between 1959 and December 1967, in spite of the use of oral erythromycin as our standard treatment for infection of burns with *Str. pyogenes*. In 1963 we made a small controlled trial of lincomycin in patients with *Str. pyogenes* infection of burns; no streptococci resistant to lincomycin