Family Doctor Team

SIR,—Mr. R. Crossman commences his recent Green Paper on the future of the National Health Service by stating that "the service should be centred on the family doctor team." Is this just another piece of political hypocrisy, employing a sentimental term which the public will like, or can Mr. Crossman really be so misinformed as to be unaware that the family doctor concept will soon have disappeared entirely from British medicine, and the centre will therefore have fallen out of his wonderful future health service.

Already in the industrial areas one's only chance of seeing the doctor of one's choice—presumably a member of this "family doctor team"—is to make sure of confining one's illnesses to office hours. Unfortunately most serious illnesses seem to have a bad habit of occurring outside these hours, when one will be seen by some totally disinterested stranger, who can hardly be regarded as a member of the team, even as a reserve. The Monday morning ward round in hospital, such as I have just done, consists very largely in discharging quite unnecessary admissions sent in by these doctors during the weekend.

Am I criticizing the doctor? Not at all. I am criticizing a system which encourages real or imaginary illness in every possible way. Not only does it cost nothing to visit the doctor but to be "off sick" is often a considerable financial advantage. Add to this the fact that satisfaction in a day's work well done seems to be a dying philosophy, and one can see why a doctor in an industrial area can retain his sanity only by farming out his patients for a large part of the time. The Welfare State is gradually strangling itself with cords of its own making, and Mr. Crossman's new deal will, in my opinion, have no effect but to draw these cords tighter.—I am, etc.,

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Drugs for Dysentery

SIR,—I should like to comment on the article on drugs for dysentery in the series "Today's Drugs" (4 April, p. 36). This article presumably advises specific drug therapy in every case of bacillary dysentery. There is, however, a school of thought, with an increasing number of adherents, which considers specific, as opposed to symptomatic, treatment quite unnecessary in a high proportion of cases.

Sonne dysentery, at least as seen in Britain, is generally a mild and self-limiting disease, and probably any favourite non-specific remedy such as chalk and opium mixture or a drug like Lomotil (diphenoxylate hydrochloride and atropine), though I agree not bactericidal, comforts the patient if required. Indeed in such cases the administration of sulphonamides or antibiotics may be quite useless therapeutically, may even prolong excretion of the causative pathogen as in salmonella infections, and may well stimulate the production of more and more multiply drug-resistant enterobacteria. In the severe cases, particularly in young babies and elderly patients, it would

seem wise to have in vitro drug sensitivity tests done as soon as possible as a guide to the drug of choice.

Here is a brief summary of our findings in in vitro sensitivity tests of *Shigella sonnei* strains of British origin in 1969. Of 509 strains tested, 59 (11.6%) were resistant to sulphonamides alone, 39 (7.7%) resistant to sulphonamides and streptomycin, 102 (20.0%) resistant to sulphonamides, streptomycin, tetracycline, and ampicillin, 75 (14.7%) resistant to sulphonamides, streptomycin, and ampicillin. Other drug-resistant combinations were found.

The drug resistance pattern does vary from area to area and from time to time within an area. Strains in other countries behave similarly and this applies equally to *Sh. flexneri*. These findings would emphasize to me that in vitro tests with representative cultures in any geographical area should be the main guide to specific drug therapy. I personally find it increasingly difficult to recommend any one drug as a panacea for bacillary dysentery.

So far as supportive treatment is concerned, in moderate dehydration when the patient does not require the intravenous control of electrolyte balance, I wonder if giving dilute normal saline and potassium chloride has any advantages over simply increasing the quantity of any palatable thirst-quenching fluid. In the tropics, if oral potassium is considered necessary, a very suitable acceptable substitute is coconut milk, as has been shown for the supportive treatment of cholera.

I would ask two questions arising from the article. It is stated that sulphonamides are widely and *successfully* (the italics are mine) used in the tropics as their cheapness is an advantage. What are the criteria of success? Is nalidixic acid an antibiotic—I am, etc.,

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Compression and Limb Blood Flow

SIR,—We read with interest the study by Mr. M. Spiro and others (21 March, p. 719) on the effects of externally applied pressure on femoral venous blood flow.

Their findings are of relevance to those concerned with postoperative management, especially in relation to the role of stasis in the development of deep vein thrombosis. There is however, a technical inaccuracy in their introduction which is probably the result of trying to condense into a few words the published findings of others. The authors state "... with a 15° head-down tilt on the table stasis was largely eliminated. Venous flow velocity (Wright and Osborn, 1952) and possibly total limb blood flow were increased (Beaconsfield and Ginsburg, 1955) by this manoeuvre." We did not say this in the article quoted. In our study¹ the body was maintained at the horizontal; when one limb was raised to 15° peripheral flow increased, but when the leg was further raised to 45° flow decreased. This reaction is the response of vessels to an alteration in limb posture, and is a local phenomenon since the flow in the contralateral limb is unaltered. Such local reactions must be distinguished from circulatory reflexes which

occur on both sides and are induced by change in body posture. These changes are secondary to baroreceptor activity, as instanced by the reduction in limb flow which occurs when the upright posture is assumed, or the increase in peripheral blood flow which is observed when the body is tilted from the vertical to the horizontal position.^{2,3}

While the article by Mr. Spiro and his colleagues is an interesting piece of investigation its application to clinical practice is, at the best, of extremely limited value. The authors themselves state: "... that if external compression is to continue to be used in the treatment and prevention of deep vein thrombosis its application must be carefully controlled. The optimum pressure usually lies between 5 and 12mm. Hg but may well vary from patient to patient. . . ." We can affirm from years of experience that in a busy surgical unit this type of control, while possibly desirable, is certainly not practical, and the prophylaxis against deep vein thrombosis depends much more on good preoperative preparation, avoidance of inadvertent peri-operative trauma, gentle surgery, immediate postoperative ambulation, and, of course, "a little bit of luck."—We are, etc.,

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SIR,—I read with interest the article on femoral vein blood flow by R. M. Spiro and others (21 March, p. 719). I would be interested to know whether the authors had considered the effect of posture on bandaging and venous flow, for it may well be that their conclusions about bandaging are only valid for the horizontal posture. Venous flow is affected by venous resistance. This, in turn, is affected by the calibre of the veins, which is itself a function of the transmural pressure. The addition of a further hydrostatic pressure within the veins (when assuming an upright position) to the normal dynamic pressure tends to dilate the veins and therefore to lower the resistance they offer to flow. It is interesting to note that in their results maximum flows occur when the bandage and hydrostatic intravascular pressures are approximately equal; the fall off in flow, with increased pressure, is presumably due to compression of the veins and therefore a rise in venous resistance. One would expect, theoretically, in the author's experimental model that this fall in flow would be delayed in the vertical position until the bandage pressure equals the new (now increased) venous pressure.

This prediction is supported by my own research involving the clearance of ²⁴Na from the subcutaneous tissues of the foot. In these experiments external bandaging pressure was applied in a similar fashion to that described by your authors. Figure 1 demonstrates that in dependent posture the

FIG. 1.—Three experiments in which the external bandage pressure was varied over a subcutaneous injection of ²⁴Na, in the dorsum of the foot.

FIG. 2.—Schematic drawing of Hydrostatic Stocking (After Wood, J. E.).

FIG. 3.—Effect of Hydrostatic Stocking. Vertical axis clearance of ²⁴Na expressed as clearance constant K. Horizontal axis denotes posture. The application of the hydrostatic bandage has prevented the usual slowing of clearance which occurs when a limb is lowered. (Chant, A. D. B., *Brit. J. Surg.*, 1968, 55, 860).

bandage pressure needed to obtain maximum clearance is much higher than in the horizontal position. It would appear (at any rate from these results) that for maximum clearance or venous flow to occur bandage pressure must approximately equal the intravascular hydrostatic pressure. In any posture this pressure is graded infinitely from toe to right atrium. For conventional bandage pressure to be equally well graded

is impossible, for the old teaching "tight turns at the bottom and loose at the top" is not only impracticable but also only relevant to the posture in which it was applied. This problem may be overcome by use of the hydrostatic stocking described by Wood¹ (Fig. 2). Using this, the hydrostatic pressure in bandage and vessels is kept equal and the clearance therefore maximum in all postures. The effect of such a bandage on the clearance of ²⁴Na is shown in Figure 3. This contrasts with the usual slowing in the clearance of ²⁴Na when the limb is lowered.²

Finally, it would be interesting to know the exact time course of the events which your authors described. It may well be that the increase in bandage pressure drives tissue fluid into the circulation; this, together with a diminution in the volume of blood in the limb consequent on compressing the veins, could account for the increase in flow which was observed, rather than the reflex they hypothesize.—I am, etc.,

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Phage Treatment of Severe Burns

SIR,—On reading Dr. G. Shera's reference to the use of phage in the treatment of infected burns (28 February, p. 568), I recalled our attempt 18 years ago to use locally applied staphylococcal phages for prophylaxis against infection of burns.

During the trial, swabs from burns were found to contain an increasing proportion of staphylococci which were recorded as non-typable or which showed diminished sensitivity to a phage included in the prophylactic application. Phage-resistant secondary growth of *Staph. aureus*, associated with acquired lysogenicity, appeared readily in cultures of staphylococci of groups I and III grown with phages to which they were initially sensitive.¹ Even if phage might have some value in treatment of infections by sensitive bacteria, it seemed likely that the emergence of resistance to phage, as to some antibiotics, was likely to deprive us very quickly of this potential therapeutic agent.—I am, etc.,

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Aldosterone and Antinatriuresis

SIR,—In a review of renal control of sodium reabsorption, Professor H. E. de Wardener (13 September, 1969, p. 611) marshalled evidence which suggested that the source of a postulated hormone promoting sodium excretion is not the kidney or adrenal. It is perhaps relevant to review some evidence which may go some way to resolve some difficulties about the nature of salt-handling by the kidney.

Experimentally, after unilateral adrenalectomy¹ the homolateral kidney excreted salt and water more rapidly than the contralateral kidney, and this was exaggerated during 0.9% NaCl diuresis. The glomerular filtration rate and effective renal plasma flow were not altered. A hormone following its proper course through the general circulation could not produce unilateral effects. We proposed,¹ therefore, before the crystallization of aldosterone, that an intimate adreno-renal relationship existed whereby an adrenal hormone effected adjustments of salt reabsorption by local diffusion directly into the kidney via renal capsular vessels, as had been demonstrated for adrenaline.² Eggleton and others³ agreed that the diuresis was due to diminished proximal tubular reabsorption. Previous explanations of the salt-losing phenomenon were denervation,⁴ and increased glomerular filtration rate.⁵

Why then postulate a natriuretic hormone when the removal or inhibition of a hormone promoting salt reabsorption would better fit the facts? Natriuresis following a saline load can be adequately explained by the expansion of the extracellular volume diluting the aldosterone plasma concentration in addition to an increase of intrarenal hydrostatic pressure⁶ and a decreased fractional proximal reabsorption.⁷

Natriuresis from both kidneys in a unilaterally adrenalectomized dog subjected to saline loading can be explained by a bilateral increase in hydrostatic pressure and dilution of aldosterone plasma level. The further relative increase in natriuresis from the adrenalectomized kidney may be explained by the removal of a salt-retaining hormone normally gaining direct access from an adjacent adrenal gland. The above factors are probably those which render it difficult or impossible to demonstrate aldosterone antinatriuresis in the normal dog.—I am, etc.,

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Propranolol in Hypertension

SIR,—We feel that the trial of Drs. F. J. Zacharias and K. J. Cowen on the effect of propranolol in hypertension (21 February, p. 471) requires some comment. Although under the heading "Method" it is mentioned that the patients were almost all receiving diuretics, in the Summary, Tables, and Results of the paper the term "placebo" group is used which conveys the impression that when the patients were not on propranolol they were receiving no other treatment. The Tables would have been more correctly labelled "propranolol plus diuretic" under the heading "Drug," and "diuretic alone" under the heading