Desferrioxamine and Acute Iron Poisoning

SIR,—A reference to the iron-chelating agent desferrioxamine occurs in the *British Medical Journal* of 17 October, p. 993, under the heading "Current Practice—To-day's Drugs." The author correctly describes desferrioxamine as a water-soluble compound having a strong avidity for iron, and reproduces a table of chelating agents for heavy metal poisoning, in which the compound is again correctly described as the antidote of choice for iron poisoning, being remarkably non-toxic.

However the further statement that desferrioxamine needs to be given by intramuscular or intravenous injection requires modification in the light of clinical experiences in several countries. Intramuscular injection is entirely safe and provides effective blood levels within 20 to 30 minutes. Rapid intravenous injections have been reported to cause anaphylactic-type hypotensive reactions in several cases, and we therefore recommend that this method of administration should be avoided.

For the benefit of casualty officers and others who may be required to deal with iron-poisoning emergencies, we suggest the following procedure:

- (1) Inject 2,000 mg. desferrioxamine dissolved in Water for Inj. B.P. intramuscularly.
- (2) Perform gastric lavage followed by the oral administration of 5,000 mg. desferrioxamine in 50–100 ml. water (by stomach tube if necessary).
- (3) Set up an intravenous drip and administer desferrioxamine by continuous intravenous infusion at a rate of not more than 15 mg./kg. per hour. Maximum dosage 80 mg./kg. in 24 hours.
- (4) Repeat intramuscular injections of 2,000 mg. desferrioxamine at 12-hourly intervals, if necessary.

Used as described, hypotensive reactions are not to be expected.—I am, etc.,

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Anticoagulants: Another View

SIR,—Professor John McMichael's comments on long-term anticoagulant therapy (17 October, p. 1007) seem to reflect undue animus, and his manipulation of the data appears to be inconsistent. He first removes one group (deaths in the first three months), and later says that such selection (in this case the grouping into sexes) is "not permissible." It is difficult to see why he makes one selection and disallows another, particularly when separate consideration of the results in men and women would seem almost obligatory in a disease in which the incidence and mortality differ in the sexes. The fact that 80% of the treated group of men returned to work compared with 64% of the control group is surely a result worth striving for.

Professor McMichael calls the incidence of haemorrhage ominous. In my experience this word in well chosen. I have seen eight instances of haematuria among patients on long-term anticoagulant therapy. In one this was due to an unduly low prothrombin level, associated with the use of Atromid (clofibrate).

In the remainder the control was regarded as satisfactory. Four cases were found to have a silent renal stone, one a renal-tract infection, one a hypernephroma, and one case was not explained. The report criticized by Professor McMichael makes no mention of possible factors predisposing to haemorrhage, but my experience is that haematuria in a patient whose prothrombin index is 2 to 2.5 is an indication for careful renal-tract investigation.

The subject of this trial is an important one. The conclusions appear to be based on an experience with less than 400 patients. Would it not be reasonable to extend the observations if the existing facts do not suffice to convince? It may show that what is good for the gander is also good for the goose.—I am, etc.,

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Treatment of Xanthomatosis

SIR,—I was most interested in Dr. B. W. Roper's report (17 October, p. 990) of two cases of essential hypercholesterolaemic xanthomatosis treated with Atromid. I have treated nine cases in this way, together with three cases of essential hyperlipaemia, and reported my findings earlier this year.¹

These patients have now been treated for periods varying from eighteen months to two years. The blood cholesterol is normal in all of them, and the skin lesions have resolved in all but one. This patient, who had gross cutaneous involvement, continues to progress satisfactorily, improvement to date being assessed at 75%.

There seems little doubt that at present Atromid is the best treatment for essential hypercholesterolaemic xanthomatosis.—I am, etc.,

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PETER BORRIE.

REFERENCE

- ¹ Borrie, P., *Brit. J. Derm.*, 1964, 76, 53.

Functional and Imagined

SIR,—Dr. Ronald Gibson's paper (17 October, p. 965) was full of stimulating thought and will surely be widely read and deeply considered. It is unfortunate, therefore, seeing what influence it may well have, that some of the references therein are so evidently symptomatic of an essential misunderstanding of the nature of psychosomatic illness.

"I am quite sure in my own mind that the symptoms are real and that there is no emotional overlay here" are the words that, with approval, he puts in the mouth of an exemplary G.P.

"I can find no cause for this pain; therefore I do not believe that it exists; therefore it must be imagined. . . ." This, one understands, is the G.P.'s interpretation of a letter received from a specialist colleague, which letter presumably read something like this. "I can find no organic cause for this pain, which I believe is due to anxiety or stress (or to be functional, or to be psychosomatic in origin)."

I do not feel that there is any other conclusion to be drawn from these two quota-

tions than that Dr. Gibson believes a functional pain (or a psychosomatic ailment, or a symptom due to stress or emotion) to be an *imagined one*. This is unfortunately all too common an interpretation both by patient and by doctor, and for as long as the misunderstanding exists so long will the doctor who has a predominantly, if not totally, organic orientation towards disease fail to understand the ideas and opinions of the doctor who attributes at least some symptoms to psychological causes, and indeed so long will the former's patients be without real support and help.

I would urge Dr. Gibson to reconsider the matter in this light—to picture for a moment a novice soldier on the battlefield, with his underlinen soiled by the effects of uncontrollable intestinal hurry. Such diarrhoea and such catastrophe are not imagined. They are both psychosomatic in aetiology and very real. Perhaps more commonplace is the medical student awaiting a viva voce examination. He does not *imagine* that he has frequency; he *has* frequency of micturition, but the cause is not organic. I feel sure that if Dr. Gibson were to dissociate in his mind the words "functional" and "imagined" he would find opinions attributing symptoms to tension and anxiety and emotional difficulty, in the absence of organic disease, and in the presence of a suitable aetiology, far more acceptable.—I am, etc.,

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HUJOHN RIPMAN.

Laevadosin for Muscular Dystrophy

SIR,—In the *B.M.J.* of 10 October (p. 915) the preliminary results of a trial of therapy (Laevadosin) in Duchenne-type muscular dystrophy have been reported, and the conduct of this trial appears more interesting than its results. So far from being "purposely designed to replicate closely" our earlier assessment,¹ very different approaches and methods make comparison impossible.

When serum-enzyme values are used as a parameter, one must remember that physical activity in health causes corresponding variations in those of aldolase and the transaminases,² that this effect is greatly magnified in muscular dystrophy,³ and that continuous serial assays of very numerous specimens successively obtained under standard conditions are thus obligatory for valid statistical analysis of the disease state.⁴ Serum creatine-kinase (C.K.) activity, also a sensitive index of the disease process, likewise shows similar variations not only in health⁴ but also in muscular dystrophy⁵ by assay methods more dependable than those used here.⁶ This present trial seems founded, however, on previous work by the same collaborators, who, failing to detect an immediate increase in serum C.K. after very brief exercise in a few normal individuals,⁷ then supposed that similar findings in dystrophic patients were thereby proved without actual investigation⁸—thus conferring validity on casual outpatient assay.⁹ This erroneous assumption is matched by the incidental statement that elevated serum-C.K. values are found in hepato-cellular disease⁸ despite the dearth of C.K. in liver tissue.¹⁰ On that basis, instead of our standard morning pre-ambulatory blood specimens thrice weekly for some four months in the hospital ward, specimens once a week only for about a month have been