or compound fractures, to the Queen Alexandra; patients whose chances of survival might well have been prejudiced had it been necessary to take them all the way to Woolwich.

Now that the Defence Ministry has been functioning smoothly for some years, would it not be worthwhile to extend the Queen Alexandra on its existing site to serve as the main hospital for the Navy and Air Force as well as for the Army, as at present? The amalgamation and still closer integration of all three Medical Services would surely be the logical consequence of the Defence Ministry, and in the interests of patients and staff alike?

I am an ardent admirer of the Tate Gallery, and a frequent visitor to its exhibitions. I welcome the extension of accommodation so that many of the fine pictures in the vaults can be enjoyed by the general public. Great Britain possesses some of the world's most eminent architects, one of whom could surely devise a plan expanding the accommodation of the Gallery, vertically if need be, by utilizing the northern and eastern portions of the present site, parts of which would seem to be covered with nondescript "temporary" structures.

May I appeal to my colleagues, Sir, through the medium of the *B.M.J.*, to lend their support for the retention of the Millbank hospital?—I am, etc.,

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Bullous Lesions in Poisoning

SIR,—May I contribute to the recent correspondence on "Bullous Lesions in Poisoning" (7 August, p. 371) some points on the management of these lesions?

A 33-year-old female was referred to our plastic surgery unit on 23 June, having been comatose from an overdose of Sonalgin tablets (butobarbitone; phenacetin; and codeine phosphate) a week before (blood barbiturate on admission was 2·1 mg/100 ml). On examination she had patches of skin necrosis at the point of the right elbow, on the medial surface of the right lower thigh, over the right internal malleolus, on the medial surface of the left leg just below the knee, and on the dorsum of the left foot above the instep. These areas, which were initially blistered, had developed the appearance of full thickness burn eschar surrounded by erythema. There was also much induration surrounding the lesions below the left knee and on the right thigh, which was grossly swollen, but induration and swelling were absent from the other lesions.

Necrotic tissue was excised at operations on 29 June and 6 July. At the right elbow and ankle and the left foot necrosis was confined to the skin, but in the right thigh and left leg below the knee necrosis was found to have involved a wider area of subcutaneous fat and muscle. The extent of muscle necrosis was greater in the right thigh, such that most of the vastus medialis and some of th eadjacent rectus femoris muscles had to be excised. Healing was obtained by suture of the thigh wound and grafting of the others.

The finding of induration and excessive swelling in the two sites where necrosis had extended into muscle conforms with previous descriptions of muscle necrosis in similar cases.[1 2] The fact that these signs were absent from the other sites where necrosis was confined to skin suggests that they may be of diagnostic value in determining the depth of soft tissue necrosis.

The extensive necrosis of fat and muscle in the right thigh lay beneath a small patch of skin necrosis—a typical finding in electric burn injuries. I suggest that, as with electrical burns, treatment should be early excision of necrotic tissue with exploration of the muscular compartment if there is surrounding induration and excessive swelling. Failure to excise necrotic muscle may result either in early death from myoglobinuria[1 2] or in late contractures that follow healing by fibrosis.[3]

I thank Mr. R. D. P. Craig for permission to report on his patient.

—I am, etc.,

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Red Cell Size and Air Composition

SIR,—During routine haematological surveillance of personnel who spend intermittent prolonged periods in a controlled revitalized atmosphere, which among other contaminants contains around 20 p.p.m. of carbon monoxide, we have noted an apparent small but consistent increase in the mean corpuscular volume (M.C.V.) of red cells, as determined by the Coulter M.C.V. Computer, linked to a Model B Coulter cell counter. expected, the highest results are usually obtained from smokers, individual values in excess of 100 μ^3 being fairly common. Since even nonsmokers in the intermittently confined group have higher carboxyhaemoglobin levels than normal, further studies are in progress to correlate carboxyhaemoglobin levels with M.C.V. Meanwhile it would be of value to know if other workers using electronic cell counters and sizing methods have reported similar changes in groups of smokers.—We are, etc.,

	No. of Men		Mean M.C.V. (μ3)	S.D.
Intermittently Exposed Group	522	Before Exposure on this Occasion	90·6	5·6
		After Exposure	92·8	5·7
Control Group	78		86·0	7·0
Published Value [1]			87·0	7·0

The Table indicates the effect of a single period of confinement on these personnel, and compares these results with those of a similar group of men who had never been confined, together with some published normal values.[1]

Similar results but with a greater individual variance are obtained by determining the M.C.V. from the results of red cell counts and microhaematocrit readings, for which some published results are given as $87 \pm 5{\cdot}0\mu^3$ [2] and $78—94\mu^3$.[3]

It is also apparent in this work that, as

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Intermittent Claudication

SIR,—Mr. S. J. A. Powis and his colleagues (29 August, p. 522) give valuable information on the amount of improvement to be expected after division of the tendo Achillis in cases of intermittent claudication.

I do not want to go into the question of spontaneous improvement more than six months after the onset, but I would like to draw attention to simpler means of cutting out the action of the gastrocnemius muscle. This can be achieved by immobilizing the ankle joint during walking, which is easily done by a below-knee double iron set in a flat socket in the heel of the shoe. A rocker sole makes walking with a stiff ankle easier. Most present day shoes will require also a steel plate inserted into the sole to prevent it from breaking. The gait is, of course, different from that with a mobile ankle, but this hardly ever needs special training.

The effect is immediate, doubling or trebling the "claudicating distance." A great merit of this method is its reversibility.—I am, etc.,

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Reiter's Syndrome—Protean Symptomatology?

SIR,—It has been well argued in the past that ankylosing spondylitis and Reiter's syndrome are identical diseases.[1] In one series of 54 patients with classical ankylosing spondylitis 45 had definite evidence of prostatitis.[2] Ankylosing spondylitis has also been found in 2—5% of cases of ulcerative colitis and other inflammatory diseases of the intestine.[3] Reiter's original description was of a typical post-dysenteric case[4] and, after an outbreak of Flexner dysentery in Finland, 344 cases of Reiter's syndrome were described.[5]

Keratoderma blenorrhagica occurs in about 10% of patients with Reiter's syndrome, and keratoderma is considered by many to be a form of psoriasis:[6] ankylosing spondylitis has been reported in up to 33% of patients with psoriasis.[7]

Non-gonococcal urethritis is not a single

entity; Harkness, in his classic monograph, names more than 50 conditions known to cause urethritis.[8] One of these is post-dysenteric Reiter's syndrome.

The situation is obviously confusing, and your leading article (14 August, p. 386) carefully avoided the textbook statement that Reiter's syndrome is usually a complication of venereally acquired non-gonococcal urethritis. Nevertheless, in the statement that the incidence of the disease is estimated at about 1% of the patients with non-gonococcal urethritis it perpetuates the textbook teaching without mentioning any of the discordant facts.

A pedantic approach is often necessary to make a difficult subject intelligible to students; it is unfortunate that this sometimes carries over into scientific thought and clinical practice.—I am, etc.,

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Fluorescent Staining and Human IgM

SIR,—When antibody is united to an insoluble antigen, the class of globulin attached may be ascertained readily by the fluorescent antibody technique, using conjugated antiglobulins of appropriate class-specificity.[1]

In human serum, during and after acute infection, a rising and falling titre of specific IgM fluorescent staining against a virus seems to indicate reliably the amounts of virus-specific IgM,[2 3] but, according to the results shown in the Table, IgM-specific fluorescence given by random human sera not associated with recent virus infection is of more doubtful significance. The Table represents mostly the incidence of IgM-specific staining given by individual sera from rheumatoid arthritis and also by normal sera, when these are applied to HEp_2 cells, infected with herpes-simplex virus.

Source of Sera	Number Tested	Before Absorption with Aggregated IgG	After Absorption with Aggregated IgG
RF-positive Rheumatoid Arthritis	35	31 (1)	3 (5)
RF-negative Rheumatoid Arthritis	10	1 (2)	0 (1)
RF-positive non-Rheumatoid Patients	4	3 (0)	1 (1)
RF-negative non-Rheumatoid Patients	41	4 (8)	0 (6)
Acute Virus Infections (Herpes, Measles, Mumps, Rubella)	29	28	27*

Figures in brackets faint, but specific, additional reactions.

RF = rheumatoid factor. Tested by IgG-coated latex on serum diluted 1:3.

* One weakly positive rubella serum negative after absorption.

Sera, absorbed with human tissue powder and HEp_2 cells applied to acetone-fixed virus-infected cells at estimated dilution 1:5 followed by fluorescein-conjugated sheep and anti-human-IgM.

It is clear that much IgM-specific staining of herpes simplex virus by rheumatoid and by normal sera is caused by IgM antiglobulins, as is evident from removal of the staining property by absorption with aggregated human IgG. Moreover, many sera which do not have sufficient rheumatoid factor to show in a latex-globulin agglutination test do produce IgM-specific staining which also is removed by aggregated IgG. It is relatively easy to show that, as one would expect, a serum which contains rheumatoid factor will cause IgM-specific staining of cells infected with different viruses, provided virus-specific IgG is also present in the serum, and it can also be shown that rheumatoid factor itself is responsible for this staining. We do not give these experiments here.

In sera from rheumatoid arthritis and in normal sera there are, too, IgM antibodies to herpes simplex virus which resist absorption by aggregated IgG, and we show in the last line of the Table that virus-specific IgM found in acute herpes simplex, measles, mumps, and rubella is likewise resistant to absorption by aggregated IgG.

It is possible that some kinds of virus-specific IgM, like antinuclear factor,[4] may be associated with or may react also with denatured globulins and so explain our findings, but no other evidence of it has yet been found.

Since our results affect surveys of sera for virus-specific IgM, [3 5 6] we propose, when reporting on virus fluorescent antibody, to adopt the terms "primary" or virus-specific, IgM staining and "secondary" or anti-IgG virus-non-specific IgM staining, according to whether absorption of the staining factor by aggregated IgG is resisted or not. The terms primary and secondary IgM staining will avoid confusion with other terms already used in fluorescent antibody work such as direct, indirect, and non-specific staining. The type of IgM staining given by a serum may be important in the pathogenesis of virus diseases, especially when antiglobulins cannot be detected by other means. Associations between reacting components may be more complex than we assumed in a previous survey.[6]—We are, etc.,

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Future of the Species

SIR,—The review of *Sex and the Population Crisis* (4 September, p. 590) once again reminds us that "our whole species is in peril and time is very short." A similar conclusion was reached by Dr. S. R. Eyre at this year's British Association Meeting (*The Times*, 4 September).

Why then do we persist in refusing to allow this imminent peril to alter our current medical attitudes? It can only be folly of a most short-sighted kind that condones, for instance, the prescription of fertility drugs, elaborate procedures for saving spina bifida babies, or extravagant treatments for restoring the elderly.

If mankind is indeed in a swarming phase the practice of medicine must take the long-term ill effects of such therapies in aggravating the population crisis into account, however painful the denial of immediate satisfactions may be. Governments may soon be forced to interfere with personal reproductive habits. Meanwhile, there must be grave doubts thrown upon that meddlesome interference with nature which so easily goes by the name of therapeutic advance.—I am, etc.,

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Long-term Haemodialysis without Transfusion or Drugs

SIR,—We have read with interest the paper by Dr. Stanley Shaldon and others (24 July, p. 212) recommending the use of testosterone for the treatment of anaemia in patients on maintenance haemodialysis, as well as the disappointing results obtained by Drs. P. P. Mayer and B. H. B. Robinson (7 August, p. 373) using the same preparation and doses of testosterone. We should like to present here our relative observations indicating that improvement in haematocrit and haemoglobin can occur in the adequately nourished and dialysed patient, independent of any kind of therapy.

Six patients (age range 23-60 years) were undergoing long-term haemodialysis 12-14 hours weekly with a R.S.P. Travenol artificial kidney, using a coil type dialyser at a blood flow rate 200-300 ml/min. This was done with potassium-free dialysate, except when the patient was on digitalis, when potassium 2·5-3·5 mEq/l. was added. All patients received a liberal diet containing $\geqslant$1·25 g/kg first-class protein and 70-75 mEq sodium and potassium. No regular blood transfusions, haemopoietic drugs (iron, folic acid, androgens), or exchange resins were given. The patients' general condition improved rapidly. Blood urea and serum potassium were 0·60-1·75 g/l. and 2·25-5·75 mEq/l. respectively between two successive haemodialyses. Their initial anaemia of a mean value P.C.V. 18·3%; Hb 6·0 g/100 ml (range P.C.V. 12-23%; Hb 4·5-7·1 g/100 ml) was improved in 4-6 months to mean value P.C.V. 26·4%; Hb 8·5 g/100 ml (range P.C.V. 22-37%; Hb 7·0-11·8 g/100 ml) and it remained round this value for one to three years (12, 14, 17, 18, 23, and 33 months).

Adequate haemodialysis with liberal diet, perfectly possible by using a potassium-free