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