exaggerating the situation, but, as Dr. Scott and Mr. Malcolm Morrison in a recent article[1] have emphasized, it seems possible that requests for paraclinical investigations are not always entirely rational. Dr. Scott and Mr. Morrison have indicated some questions which the clinician ought to ask himself when requesting an investigation. We would suggest that the clinician asks himself two questions: (1) What information do I expect to gain from this investigation? (2) Will this knowledge affect the management of the patient? If he does not know the answer to the first question, or the answer to the second question is "no," then it would seem likely that the examination should not be carried out.

We hope Dr. Scott's letter will provoke further correspondence as we believe that investigation into these problems is long overdue.—We are, etc.,

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REFERENCE
1 Morrison, M., *World Medicine*, 1969, 5, No. 6 p. 42.

Pathogenesis of Pre-eclampsia

SIR,—I would agree with Mr. A. T. Coopland (13 December, p. 688) that much of the controversy and conclusion regarding the pathogenesis of pre-eclampsia is due to the difficulty in differentiating patients suffering from this disease and those with a chronic renal lesion. The difficulty is most apparent when the patient exhibits proteinuria with some oedema and mild or no hypertension. As has been shown[1] if alpha$_2$macroglobulin can be demonstrated in the urine the patient is suffering from an organic lesion of the kidney—in our cases, Type II (Ellis) nephritis. In pre-eclampsia this large molecule protein does not appear until the condition is severe, with marked proteinuria and a diastolic pressure above 100 mm. Hg.

This relatively simple test, carried out by the aid of immuno-electrophoresis, serves to distinguish the two types of patient, and will ensure that the population for study is clinically homogeneous.—I am, etc.,

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REFERENCE
1 McEwan, H. P., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1969, 76, 809.

Screening Blood Donors

SIR,—Drs. J. E. P. Fitzpatrick and C. C. Kennedy (1 November, p. 299) state that the only means of excluding donors carrying hepatitis virus is by careful questioning. However, Gocke and Kavey recently described a close correlation between transmission of hepatitis by blood donors and the presence of Australia (Au) antigen in their serum.[1]

In two cases of hepatitis, both presumably transmitted by blood transfusion, we have therefore retrospectively studied the donors involved. In both cases one of the donors was found to carry the Au antigen in the blood.

The first patient was a 50-year-old woman who in May 1969 received seven units of blood during removal of a cerebral tumor. Three months later she developed icteric hepatitis. Au antigen was demonstrated in her serum by means of an immune precipitation reaction in agarose.[2] Four weeks later the test was negative. In October the seven donors were tested for the presence of Au antigen. One donor was found to be Au positive. He had normal liver function tests and a negative history regarding icterus or contact with icteric patients.

The second patient was a 54-year-old woman who in February 1969 received three units of blood during operation for an arterial aneurysm. Four months later she developed icteric hepatitis. Her serum was tested only when she had practically recovered; no Au antigen could be demonstrated. In October the three donors were tested for the presence of Au antigen and here also one of the donors was found to be positive. Liver function tests could not be performed. This donor had suffered from icterus eight years previously.

The finding of two Au-positive donors out of 10 contrasts sharply with the results obtained in an unselected series of 800 consecutive donors, where Au antigen was only found in two. In both hepatitis cases therefore we assume the Au-positive donor to have transmitted the causal agent. It is striking that the donors still carried the antigen five and eight months respectively after having donated the infective blood. They are probably to be regarded as healthy carriers and have been excused from donating blood.

It is unlikely that it will be possible in all cases of hepatitis after blood transfusion to find the infective donor by retrospective testing for Au antigen. Nevertheless it seems worth while to follow this procedure until a time when prospective screening for viraemia has become a matter of routine.—We are, etc.,

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REFERENCES
1 Gocke, D. J., and Kavey, N. B., *Lancet*, 1969, 1, 1055.
2 Prince, A. M., *Proceedings of the National Academy of Sciences of the United States of America*, 1968, 60, 814.

Glue Ear

SIR,—The leading article on glue ear (6 December, p. 578) perpetuates the uncertainty and confusion of thought on this subject to such a degree that I feel compelled to contradict several of the statements made.

To quote a few passages: "The viscosity seems to be due to mucus . . ." There is no seems about it. It has been proved by histological staining methods (periodic acid Schiff stain, toluidine blue in polarized light) that the viscosity of the middle ear exudate is due to its high content of mucopolysaccharides. Once this fact has been recognized and understood, one principle of treatment at least becomes clear—namely, that we ought to attempt to liquify the mucus (or break up the polysaccharide molecule) by a suitable mucolytic agent—for example, urea. The high viscosity of mucus interferes with the action of cilia of the Eustachian tube and of the middle ear mucosa just as much as it does in the bronchi, nose, and sinuses. That this is a fact and not conjecture must again be clearly stated, instead of using the vague phrase "dysfunction of the Eustachian tube." But the uncertainty continues: "... this dysfunction may as well be due to mucosal swelling in the tympanic orifice as to some failure at the pharyngeal end." It is also an unequivocally established fact that the Eustachian tube is not blocked at either end.[1-3]

Even the writer of your leading article acknowledges that removal of adenoids does not prevent or mitigate glue ear. I wholeheartedly agree with this statement. In a large series of patients I have found adenoids in only 20% of cases.

Once the fact that the Eustachian tube is patent has been comprehended, it becomes illogical trying to ventilate the middle ear through the tympanic membrane by the insertion of a grommet. Fortunately Nature knows better and "the grommets are generally extruded before six months, the membrane healing behind them. They are left in position until they are extruded or deliberately removed because they have become blocked" (*sic*!).

The leading article rightly states that diagnosis of glue ear is difficult and "in some cases it cannot be excluded without diagnostic myringotomy." A far better method is a diagnostic aspiration with a short-bevelled wide bore needle (a Harris lumbar puncture needle). If viscid exudate is present in the middle ear, on attempted aspiration a vacuum will form in the syringe and will tend to bring the plunger back into the barrel, because the "glue" will not go through the needle. I have found this to be a most valuable diagnostic sign of glue ear. The puncture in the tympanic membrane heals quicker than a formal myringotomy incision, in 24-48 hours.

In my experience the most suitable mucolytic agent for the treatment of glue ear is a solution of urea (2 g. of urea/4 ml. of water) prepared immediately before use and sterilized by filtration through a Millipore filter. Boiling would cause hydrolysis. The urea solution is injected under general anaesthesia through the tympanic membrane into the middle ear.[3] This injection also automatically increases the hydrostatic pressure in the middle ear which overcomes the additional physical forces causing retention of the exudate (surface tension, capillarity, negative intratympanic atmospheric pressure etc.). This treatment is far more physiological than the use of the grommet, because it aims at re-establishing aeration of the middle ear by the only natural way, namely the Eustachian tube. The children (and the parents) must then be taught how to inflate their Eustachian tubes by the Valsalva manoeuvre to maintain the aeration of the middle ear.

The last but not least confusion of thought is to compare the success or failure of the various methods of treatment of glue ear by the number of relapses. These are a matter of the child's immunity to upper respiratory tract infections. Immunity increases with advancing age and at puberty is usually high enough to prevent further recurrences. It has nothing to do with hormones and nothing whatsoever with the various methods of treatment under discussion. The success of treatment should therefore not be judged by the number of relapses but by the improvement in hearing.

The concluding advice of your leader writer that it is "sometimes wiser to prescribe a hearing-aid to tide the patient over rather than to persist in repeated myringotomies and