by the best doctors available. But why, oh why, does the Department disadvantage hospital doctors, the National Health Service, and the British public by confining expenses only to those receiving their *undergraduate* training in the United Kingdom? Much time, talent, and effort is spent in postgraduate training of overseas doctors. Why, I wonder, do we not capitalize on these efforts also?—I am, etc.,

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Return to Work

SIR,—It would appear that Frances E. Mace (25 August, p. 458) is confusing occupational therapy with occupational medicine.

In my many years in occupational health I have yet to meet a hospital occupational therapist visiting industry who will acquaint herself with the work or working conditions, or who has the necessary knowledge to advise an employer of what work a man is capable of in any given work situation.

Rehabilitation through a return to suitable work is "occupational therapy" as undertaken in industry, but is somewhat different to the occupational therapy usually provided in hospitals. Much more could be done to help rehabilitation by earlier return to work in a suitable occupation under suitable conditions, but advice on this has not yet been the province of the occupational therapist.—I am, etc.,

JAMES GREGORY

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Medical Association of South Africa

SIR,—I write to support Dr. G. W. Gale's accurate and comprehensive letter (29 September, p. 692) in which he sets out the case against the attempt to expel the M.A.S.A. at the forthcoming meeting of the World Medical Association. I have worked in the Republic of South Africa, including a non-European hospital in the Transvaal, and I also believe that for its policy and actions in the extremely complex racial and political situation which obtains in South Africa today the M.A.S.A. deserves support rather than stricture.

It is on account of the rigid political structure which reduces non-White medical graduates to a trickle that the White doctors of South Africa spend a considerable proportion of their time and energy dealing with the medical needs of the vast non-White community. Through sheer weight of numbers plus such factors as distance and the reluctance of the Bantu to abandon traditional medical methods until these are seen to have failed (which produces advanced disease undreamt of in Europe) White medicine in South Africa is presented with an enormous burden which it shoulders cheerfully and with compassion—and this often on top of what many doctors in the U.K. would call "a full working day."

To label these our colleagues collectively as a "tool of apartheid" is derisory and utterly unfair. They are in fact a tool of true medicine—*despite* apartheid.—I am, etc.,

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Association between Hypothyroidism and Abdominal Aneurysm

SIR,—Atheroma is a well-recognized complication of hypothyroidism, but a specific predisposition to abdominal aneurysm has not hitherto been reported. We here report such an association and comment on its significance.

This clinical correlation was noted during a study of random hospital records in an attempt to delineate hitherto unrecognized relationships between certain diseases. The only criterion for inclusion in the study was the recording of an adequate history. Twenty-six cases of myxoedema were compared with 324 random controls, making a total of 350 cases. There were four aneurysms in the 26 cases of myxoedema compared with one aneurysm among the 324 controls ($P < 0.001$).

This result suggests that aortic aneurysm is a specific complication of hypothyroidism. Aortic aneurysm is relatively uncommon even in patients with generalized atheroma, and the high incidence (14%) in myxoedema suggests that this cannot be explained solely on the basis of atheroma occurring as a complication of myxoedema. An alternative method of investigating this relationship would be to study the incidence of hypothyroidism in patients presenting with abdominal aneurysm. It is now recognized that there are various stages of preclinical myxoedema and the most sensitive screening procedure would be to look for thyroid antibodies in these cases. Such a study is in progress and to date we have studied five patients who presented with abdominal aneurysms and have found suggestive evidence of hypothyroidism in four of them.

If it be accepted that there is a specific relationship between hypothyroidism and abdominal aneurysm it is of interest to speculate on the underlying mechanism linking the two conditions. Hypothyroidism is associated with hypercholesterolaemia, which is known to predispose to atheroma which may antedate the onset of clinical hypothyroidism.[1 2] Atheroma of the aorta is extremely common, but aneurysm formation is relatively rare and this would argue that factors other than atheroma are necessary for aneurysm formation. Moreover, atheroma is primarily a disease of the intima, whereas aneurysm formation requires damage to the thick muscle coat of the aorta. The standard textbooks of pathology suggest that aneurysm formation is due to atheromatous ulcers which rupture into the media, but this explanation is not entirely convincing. It is therefore of possible relevance that hypothyroidism is an autoimmune disease in which circulating antibodies are regularly detectable. This situation could lead to immune complex deposition in the vasa vasorum, and the resulting damage may lead to occlusion of these small vessels with resulting damage to the muscle wall of the aorta. There is convincing experimental evidence to support this concept in that experimental atheroma is most readily produced by combining the effects of hypercholesterolaemia with an immunological insult in rabbits and baboons.[3 4] Autoimmune processes may play a part in the causation of degenerative diseases, including atheroma, and this might constitute a further link between atheroma and an autoimmune process like hypothyroidism.

Another field in which these two factors may operate together is renal disease, and it is of particular interest that Edwards and Charlesworth[5] have convincingly demonstrated that the outcome in renal transplants is related to the lipid level, again suggesting that the combination of hyperlipidaemia and an immunological reaction can cause vascular damage, in this instance to the small vessels of a transplanted kidney. In the nephrotic syndrome due to glomerular disease one has yet another clinical situation in which there is an immunological insult on the kidney acting in association with hyperlipidaemia. It is therefore interesting to speculate whether strenuous attemps to lower the serum cholesterol level in this condition would improve the long-term prognosis. We currently have such a trial in progress, but it will take some years to collect meaningful results because of the long and complicated history of the nephrotic syndrome.

We conclude that there is a specific association between hypothyroidism and abdominal aneurysm and advance the hypothesis that this is due to a dual attack on the aorta by immune complex deposition acting on a background of atheroma. Certain parallels between this association and renal disease have been drawn, together with the possible therapeutic implications of controlling hyperlipidaemia in certain renal diseases.—We are, etc.,

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RONALD FINN

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2 Fowler, P. B. S., Swale, J., and Andrews, H., *Lancet*, 1970, **2**, 488.
3 Hardin, N. J., Minick, C. R., and Murphy, G. E., *American Journal of Pathology*, 1970, **59**, 104a.
4 Howard, A. N., Patelski, J., Bowyer, D. E., and Gresham, G. A., *Atherosclerosis*, 1971, **14**, 17.
5 Edwards, K. D. G., and Charlesworth, J. A., *Lancet*, 1973, **1**, 1192.

Methyldopa and Depression

SIR,—I must take issue with Dr. C. J. Bulpitt and Professor C. T. Dollery (1 September, p. 485) regarding their findings that "there was no evidence that [depression] was affected by therapy" with hypotensive agents and that "depression . . . [was] not related to methyldopa therapy," though they do admit that "average daily dose levels [of methyldopa] in excess of 1,500 mg were associated with . . . depression."

Questionnaires to detect depression, to be significant, must be carefully designed and validated and should be administered under supervision of a worker trained in this field. The question asked cannot be considered an adequate assessment, nor can the results obtained be accepted as scientifically valid, particularly bearing in mind that we are dealing with a self-administered questionnaire.[1 2]

Depression as a side effect of methyldopa is well documented[3-8] and is a frequent finding at psychiatric outpatient clinics. On theoretical grounds there is reason to expect methyldopa to cause depression, as it depletes tissue store of biogenic amines, particularly noradrenaline;[4 5] it inhibits the decarboxylation of both dopa and 5-hydroxytryptamine (5-HT) and decreases the concentration of 5-HT in the central nervous system;[4 5] it is converted in the body to methylnoradrenaline, which is stored in the sympathetic endings and when released, is much less effective as a sympathomimetic, in fact, acting as a false neurotransmitter.[4 5] This action contrasts with that of antidepressants, which increase the levels of noradrenaline in the C.N.S.[4 5]

The authors are not entitled on the evidence presented to conclude that depression is not related to methyldopa. Psychiatric experience[3] would advise caution in dismissing methyldopa so lightly; physicians must remain aware of this side