to afford a sum of that size, and expect that the "coupon" candidates will be elected as usual. I said that I thought this was a bad system and that I was going to ask my Division to propose a different one, namely that the statements of all duly nominated candidates should be published in the *B.M.J.* and any other general medical journals, including the give-aways, that could be induced to print them. My objection to the present system is that it virtually puts the gift of these G.M.C. seats in the hands of the Representative Body, which is too large to act as an efficient selection committee, has little time to consider the matter, and is forced to act on inadequate information. My suggestions have now been embodied in a resolution for the A.R.M. from the Mid-Essex Division (*Supplement*, 23 May, p. 129).

I did not think you were likely to publish my letter. I therefore sent a copy to *World Medicine* inviting the Editor to use it if, but only if, it did not appear in the *B.M.J.* within four weeks. Early this month he phoned to check that I had received an acknowledgement from you and to ask if I was of the same mind. Receiving two affirmatives he has written a leading article on the subject (19 May, p. 13). You have now been so kind as to telephone to tell me that although my letter was received and acknowledged it missed consideration by accident, not design. You think I might perhaps have asked you why it was not printed rather than writing to someone else. I suppose I might have done so and shall make this my practice in future. My excuse is that I suffer from that distressing but almost universal malady, peripheral paranoia, of which the main diagnostic feature is an invincible belief that there is a Medical Establishment consisting of the teaching hospitals, the royal colleges, the G.M.C., the Department of Health, the B.M.A., and the *B.M.J.*, and that its members will use any device to stifle protest or opinion which might offend any of them. I have suffered from this disorder since I qualified in 1938 and have found a very large number of fellow-patients among doctors whom I have met professionally, socially and at B.M.A. meetings (including two A.R.M.s and an S.R.M.).

On the subject of the G.M.C. elections, my paranoia has been fed by two incidents: (1) In the last election all the independents did well and one of them came very close. The *B.M.J.* did not publish the voting figures for the unsuccessful candidates, and this paranoiac of course imputed a motive: not to let it be known that rebellion had come to the verge of success, lest other dissidents take heart for the future. (2) A candidate for a "coupon" may send the Secretary of the B.M.A. a personal statement for circulation to Divisions before the A.R.M. These statements are limited to *twenty* words, initials and dates to count as a word each. My paranoid guess is that the Establishment does not want the Divisions or the Representative Body to know too much about the candidates or their reasons for wanting to get on the G.M.C., lest these reasons should persuade *hoi polloi* to support some unsafe person. I discounted economy as the motive because the Association has sent each member in our constituency quite long election statements from two candidates for the B.M.A. Council.

I must add that, wanting to get on the G.M.C. myself, I am asking the Representative Body for a coupon, on the nomination of my Division. Under the present system there seems to be no other chance of election. But I very much hope that before the A.R.M. reaches this piece of business it will have adopted the Mid-Essex resolution and abolished coupons altogether, so that neither I nor anyone else will get one and we can all stand as independents on our own merits and our own views.—I am, etc.,

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**We greatly regret that Dr. Cargill's first letter was, and is still, mislaid.—ED., *B.M.J.*

Immunological Responses to Vascular Injury in Severe Hypertension

SIR,—Professor A. E. Doyle and Dr. A. Ebringer, in their paper on raised serum IgG levels in hypertension (18 April, p. 146), suggest that the raised levels of immunoglobulin G (1,568 ± 370 mg./100 ml.) which they found in 118 patients with severe hypertension, might be an index of hypertensive vascular injury. Their observations raise a number of important questions.

First, it can reasonably be assumed that their 11 patients with accelerated hypertension had widespread focal visceral arteriolar lesions. But what of the 98 patients with benign hypertension? Pathologically the small vessels of these cases would show no more than hyaline change. Can arteriolar hyaline provoke hypergammaglobulinaemia?

Second, do cases of polyarteritis nodosa, rheumatoid arthritis, hypersensitivity angiitis, and granulomatous angiitis with normal blood pressure, but with widespread focal vascular injury[1 2] provoke hypergammaglobulinaemia G? If not, there is a case against Professor Doyle's hypothesis and some other explanation for the origin of the raised IgG levels he has demonstrated must be sought.

Third, the demonstration of gammaglobulin in arteries in rats with deoxycortone hypertension[3] does not prove that the vascular injuries are caused by the protein accumulation, even if the globulin is antiartery IgG. The globulin present at such sites may not be antibody, or, if antibody, may not be antiartery antibody; it may aggregate nonspecifically or simply may escape from the circulation at sites of plasmatic vasculosis.[4]

Fourth, the suggestion that the elastic fragmentation associated with hypertension[5] is a sign that antigenic connective tissue components are released in accelerated hypertension is speculative. Even if this is so, and even if there is an accompanying change in antigenicity so that autoimmune inhibition breaks down, there is no microscopic evidence that immunologically instructed macrophages remove arteriolar cell debris in accelerated hypertension, a course of events necessary if Professor Doyle's hypothesis to account for raised IgG levels is to prove tenable.

Fifth, if the focal vascular injuries of accelerated hypertension are provoked by autoimmune antiartery antibodies and are mediated by complement, local polymorph infiltration, as in the Arthus lesion and as in hypersensitivity angiitis, would be anticipated. In accelerated hypertension in man, and even in the late arteritic lesion of rat deoxycortone hypertension, polymorph infiltrates are most unusual and their presence would, in fact, cast doubt on a diagnosis of hypertensive arteriolar disease. Their absence is evidence against the view that arteriolar necrosis in accelerated hypertension is caused by the accumulation of immune complexes.

I have recently approached the question of an immunological component in the acute arteriolar injury of accelerated experimental hypertension from an alternative direction.[6] The evidence of White and Grollman[7] made it seem possible that an antiartery immunological mechanism might play a part in causing or sustaining this focal vascular injury. Rats with accelerated deoxycortone hypertension were therefore treated by one of three immunosuppressive regimens. The effects of a rabbit anti-rat lymphocytic serum (A.L.S.) were contrasted with those of whole-body *x*-irradiation and of large doses of cyclophosphamide. In terms of histological response (plasma protein levels were not measured) A.L.S. was entirely ineffective in influencing the severity of arteriolar disease, while cyclophosphamide reduced the frequency of vascular injury. The drug however was highly toxic to non-hypertensive control animals as well as to hypertensives. *X*-irradiation, surprisingly, diminished the severity of arteriolar disease but (as would be expected from the known potency of irradiation in causing hypertension) did so without reducing the usual extent of left ventricular hypertrophy.

The accumulated evidence supported the hypothesis that selected forms of immunosuppression would ameliorate acute experimental hypertensive arteriolar injury, but it was clear that further work was needed to establish the mechanism of injury and the role of the immunological system, whether primary or secondary.—I am, etc.,

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Compulsory Clinical Attachment

SIR,—Judging from the absence of correspondence in your columns on this scheme for overseas doctors since it became compulsory from November last year (H.M. (69) 90), it might perhaps be assumed there is some apathy as regards its chances of success by comparison with a voluntary scheme that preceded it. This indifference, if existent, is regrettable, and the chairman of the Overseas Committee (*Supplement*, 11 April, p. 20) has stressed once more the main reason for which overseas doctors should be arriving here—namely, to engage in postgraduate study in preparation for one or other of our higher diplomas. Whether or not the Department of Health always thinks similarly is at times hard to discover, but its attitude gives strong reason for doubt.

It is, of course, easy to apportion blame,

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