cancer were fewer than those of Professor Cooper and his colleagues but we included only selected cases. Patients with bacterial infection, confirmed by the nitroblue tetrazolium method, were excluded. Measured serum muramidase levels may in some cases of untreated cancer be due to local bacterial infection cannot be ruled out, particularly in colorectal cancer, which often infiltrates the superficial layer of the mucous membrane. This may explain why only some patients have raised serum muramidase levels.

The observation that bacterial infection is associated with raised serum muramidase levels prompted us to measure these in 40 patients, of whom 28 had a local and 12 a systemic infection. Some of the patients had cancer and others did not. The serum muramidase level was raised in all of the patients with systemic infections but in only five of the 28 with local infections. All of these five had cancer and their local infection was in tissue surrounding the neoplasm. The nitroblue tetrazolium test was positive in five patients.

Our findings lead us to believe that a raised serum muramidase level in some cancer patients is only a secondary phenomenon and is of no diagnostic or prognostic value.—We are, etc.,

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Ischaemic Heart Disease in Young Women

Sir.—Dr. F. M. Oliver (1 February, p. 267) has offered further information in support of his claim to have found hypertension in 34% of a group of young women suffering from ischaemic heart disease. He now tells us that in some of those whom he describes as hypertensive the clinical recognition was made after blood pressure measurement on more than a single occasion. Indeed, of those "with myocardial infarction" 28 out of 81 had a "diastolic blood pressure of 100 mm Hg or more recorded on three or more separate clinical examinations.

What we still do not know is how many of these patients were found to have hypertension before their infarction. Does the phrase "with myocardial infarction" mean that in all of them it was found retrospectively? It would seem so, to judge by Dr. Oliver's reply to my comments on the influence of ischaemic heart disease upon the electrocardiographic diagnosis of left ventricular hypertrophy, his only remaining index of hypertension since in his original paper (2 November 1974) he said, "...nothing of retinal or necropsy findings. He agrees that "previous myocardial infarction" virtually excludes the contribution that S-T and T-wave changes can make to the recognition of such hypertrophy while maintaining nonetheless that increases in the relevant S and R waves "showed" the condition in 23% of his infarction patients. No doubt all R waves so robust as to withstand anterior infarction deserve respect, but are not these values also of doubtful significance in the presence of a damaged and perhaps dilated myocardium?

Certainly, if Dr. Oliver found some of these women to have diastolic levels of 100 mm Hg on repeated examinations spread over weeks this would suggest that most of them had been mildly hypertensive before their infarction even though the recordings were made a year later. But the association between hypertension and angina cannot justify his inference that "hypertension is an important pre-infarction risk factor in young women." This is the view long nourished by the Framingham studies. It derives, however, from a process of reasoning that equates symptoms with ischaemic heart disease regardless of morbidity change. My own feeling is that there is no evidence to suggest that hypertension commonly provokes angina (and infarction rarely but rather more readily in women than men) in subjects whose coronary vessels have already been damaged by atheroma and moreover that it plays no more than a slight and occasional part in the genesis of that atheroma.—I am, etc.,

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