F.I.M.L.T., and Miss Diana Cox, R.T., for technical assistance.

—I am, etc.,

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2 Evans, J. R., Artenstein M. S., and Hunter, D. H., *American Journal of Epidemiology*, 1968, **87**, 643.
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Angina Pectoris

SIR,—I read with interest "Angina Pectoris—I" (27 February, p. 501) and "Angina Pectoris—II" (6 March, p. 545) and I thoroughly enjoyed the discussion.

May I be allowed to make a comment regarding "Mechanism" of angina pectoris? The causes of angina, brilliantly listed in the diagram, embrace everything but aortic valvular disease, which may be listed under provoking factors (B) and sub-heading—reduced coronary blood flow (5). Aortic stenosis may result in angina pectoris because of interference with coronary blood flow in the small vessels during systole due to high intraventricular pressure, and in aortic regurgitation coronary perfusion may be impaired by low diastolic pressure. In both forms of aortic valvular disease increased size and work of the heart may contribute to coronary insufficiency by augmenting the need for blood.

May I also have the opinion of the readers regarding the treatment of angina pectoris with β-blockers. At present three β-blockers are available: propranolol, practolol, and oxprenolol. Dr. J. C. Petrie has rightly mentioned that practolol is less likely to cause bronchoconstriction, but practolol is less potent than the other two and it has been used more for arrhythmias than for angina. Oxprenolol, as the manufacturers claim, has got intrinsic sympathomimetic activity and is less likely to cause cardiac failure. In our Unit during the last three months we have used oxprenolol in ten patients with angina pectoris. The results of treatment are extremely satisfactory, but three patients developed intractible cardiac failure even on very low doses of oxprenolol. How common is this unwanted side effect in others' experience?—I am, etc.,

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Methyldopa and Associated Thrombocytopenia

SIR,—In their paper on methyldopa and associated thrombocytopenia (27 February, p. 494) Dr. S. M. Manohitharajah and others comment on the lack of reports of platelet antibodies in patients on methyldopa with a positive direct antiglobulin test.

I should like to report such a case recently admitted to this hospital with haemolytic anaemia. Although she had as yet not developed thrombocytopenia complement fixation test for incomplete platelet antibodies was weakly positive. Cessation of methyldopa has not yet been long enough for us to recheck her platelet antibodies. It is perhaps of interest that she is also a case of treated pernicious anaemia.—I am, etc.,

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Blood Pressure and Bone Cement

SIR,—Since the correspondence last year on the subject of bone cement (22 August, p. 465; 29 August, p. 523; 5 September, p. 528; 19 September, p. 710; and 17 October, p. 176) we have been carrying out arterial blood pressure recordings during total hip replacement operations in which prostheses fixed with acrylic cement (Simplex Opaque) have been used. An arterial needle leading to a pressure transducer and amplifier with continuous recorder (Devices M.2) has been employed. So far, records of 36 insertions of cement have been obtained, and a fall in blood pressure has been seen in 34. The fall has averaged 7% of the pre-cement blood pressure after the insertion of cement into the acetabulum, and 11% in the femur. No catastrophic falls have been seen. The cement mixes have been prepared by a mixing technique known to eliminate up to 15% of the monomer by evaporation, and decompression of the femoral medulla with a polyethylene catheter has been routine. Maximum blood pressure fall occurred on average 165 seconds after the commencement of insertion of the cement into the femur. Other manoeuvres—for example, the use of rotating cutting tools in the femur and the acetabulum, or the reduction of the hip—often produced blood pressure changes as great as those following the insertion of the cement. This work suggests that a blood pressure fall occurs in nearly every case, but will be noted only by using continuous recording techniques.

Absorption of monomer from the cement surface has usually been regarded as being responsible for the blood pressure falls which have been noted in the past. This concept derives primarily from the experimental work of Homsy *et al.*[1] who showed that, in dogs, a fall of blood pressure was a regular sequel to the intravenous injection of monomer. Monomer is very insoluble in water or saline and a biphasic suspension is produced when monomer and blood or saline are mixed. In repeating some of Homsy's experimental work we have confirmed a regular fall in mean arterial blood pressure after the intravenous injection of monomer, but this has in every case (11 injections) been associated with a rise of central venous pressure which has started at least as soon as the blood pressure falls, and considerably outlasted it. Homsy commented on the appearance of "haemorrhagic lesions" in the lungs of his experimental animals and the lack of lesions in the other organs.

It is thus quite possible that the haemodynamic effect of monomer given by intravenous injection is due to pulmonary monomer embolism, and not to any specific pharmacological action of the monomer. It is by no means certain that the monomer in the free state is absorbed from the surface of the cement mix after implantation. Surprisingly, no attention has so far been given to the activation reaction as a possible source of compounds of monomer with local components of blood, fat or bone. Activated monomer (which is not present following the direct intravenous injection of monomer) is a highly unstable molecule, readily combining with a variety of substances (including, of course, polymer). It is possibly highly important with respect to the local toxic effects of the cement mix, and the experiments of Hulliger[2] suggest this may be so. Absorption from the cement surface after implantation is probably much more complicated than has been thought in the past, and at the present time it is not possible to say categorically that blood pressure changes following implantation of cement are *definitely* a consequence of toxic absorption from the cement surface.

We would suggest that more orthopaedic units using acrylic cement should monitor their cases by continuous arterial blood pressure recording, with, if possible, central venous pressure recording and E.C.G. This should be combined with a record of the method of the preparation of cement, the exact time of the commencement of the insertion of the cement, the duration of the insertion process, the time of the insertion of the prosthetic components, and the weight of cement inserted. This type of record is especially important in interpreting the exceedingly rare case in which cardiovascular collapse may take place: the absence of such data in this situation may lead to the misinterpretation of the cause of the collapse.—We are, etc.,

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1 Homsy, C. A., Paper presented at Joint Workshop on Total Hip Replacement and Skeletal Attachment of American Academy of Orthopaedic Surgeons, 6 November 1969.
2 Hulliger, L., *Archiv für orthopädische und Unfall-Chirurgie*, 1962, **54**, 581.

Dyspareunia

SIR,—The article on dyspareunia by Mr. W. T. Fullerton (3 April, p. 31) has interested me. However, Mr. Fullerton seems to have overlooked an important type of dyspareunia in geriatric patients. Such patients are very prone to conceal this symptom. It is frequently owing to a perineal transverse skin ridge that may appear red and denuded, and becomes stretched during intercourse. Simple incision or excision of this tag cures this symptom.—I am, etc.,

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Streptococcal Meningitis

SIR,—I should like to contest the statement made by Drs. S. M. Hempling and M. de L. P. Coutinho (17 April, p. 166) that streptococcal infections are nowadays decreasing in frequency and severity.

I recently had occasion to survey streptococcal infections in patients attending the casualty department of the Leeds General Infirmary. The results of this study[1] confirmed an earlier subjective impression that, far from decreasing, these infections had increased in incidence by a factor of three over the previous five years. The lesions produced were of a wide range, and were not confined to minor skin sepsis.[1] On at least one occasion one of these patients almost succumbed to the infection and de-