of postmenopausal women cannot be excluded—for example, stress, parity, and diet, also the persistence of enzymic activity in the ovaries^{2,3} or possibly in the uterine body.

I found that hysterectomized and oophorectomized women tolerate continuous treatment for a longer period than "normal" women. Intermittent therapy (thrice weekly) with all forms of oestrogenic substances is the treatment now used in this clinic. This therapy is based on empirical rules.

It would be a great asset if we could evaluate the menopause in a more precise manner to establish or confirm oestrogen lack as the cause of the various postmenopausal symptoms, and research is proceeding along these lines in this clinic.—I am, etc.,

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¹ Dove, G. A., Morley, F., Batchelor, A., and Lunn, S. F., *Journal of Reproduction and Fertility*, 1971, **24**, 1.

² Yin, P. H., and Sommers, S. C., *Journal of Clinical Endocrinology and Metabolism*, 1961, **21**, 472.

³ Novak, E. R., Goldberg, B., Jones, G. S., and O'Toole, R. V., *American Journal of Obstetrics and Gynecology*, 1965, **93**, 669.

Decline of the Necropsy

SIR,—The writer of your leader "Decline of the Necropsy" (24 April, p. 181) must surely have had his tongue in his cheek. In compliment to my clinical colleagues it should be made clear that the decline of the hospital necropsy has arisen since surgeons now send their specimens to the pathologist while the patient is still alive, and advances such as antibiotic therapy have converted necropsies on medical patients to studies in degeneration rather than infection.

As for "hack operations"—firstly, these frequently involve the honour and liberty of the subject; insurance, compensation, and legacies; and the reputation of one's colleagues. The same cannot be said of routine hospital necropsies.

Secondly, the principle envisaged by your writer could be extended further. Surgeons might be released from removing normal appendices in nursing homes, gynaecologists from performing routine abortions in private clinics, and physicians from fussing over private neurotics anywhere; these gentlemen would not then be "condemned to neglect more important hospital duties." Moreover there is no reason why a barrister should waste his valuable time attending court for a plea in mitigation in respect of some peculiarly revolting villain; solicitors should not be asked to conduct conveyancing; indeed one can continue ad infinitum.

Although admittedly I must now eschew the higher flights of chemistry and viro-bacteriology nonetheless a fundamental interest in haematology has not prevented me from considering myself a better "pathologist" because on occasion I look at sections and even make postmortem examinations. Indeed, I have come to the view that apart from the occasional (very occasional) surgical biopsy it is the coroner's necropsy alone in which the full skill, learning, and responsibility of the pathologist (as distinct from technical and scientific procedures) is seen to full advantage.—I am, etc.,

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Blue Valve Syndrome

SIR,—Your leading article "Blue Valve Syndrome" (8 May, p. 294) gives a rather incomplete picture of a condition which must, by now, be familiar to all pathologists interested in the heart. This is presumably the condition originally described in 1958 by Fernex and Fernex,¹ which is not uncommon in general hospital necropsy material, at least in this region.² Apart from the familial cases, this abnormality is seen mainly in the elderly and was found in 1% of necropsies on patients over 50 in this hospital.

It may well be that the condition proceeds to intractable heart failure, but this process probably takes many years. Histories of loud mitral systolic murmurs for 20 years before the final illness are not uncommon, and about a third of the cases that I have seen died of non-cardiac disease. Clinicians should be aware that mucoid degeneration predisposes to "spontaneous" rupture of the chordae tendinae as well as to endocarditis (both infective and non-bacterial thrombotic), but apart from these complications the mitral incompetence seems to be relatively well tolerated in the age group in which it is most often found.

Finally, may I protest against perpetuating this new term "blue valve syndrome". It seems to have been coined on the basis of a single case by authors³ whose review of the literature was confined to four American papers. In my experience of over 50 cases the colour is more accurately described as pearly-grey and emphasis on the occasional blue tinge would only add to the number which are incorrectly labelled rheumatic valvular disease. There is undoubtedly a place for a short, recognizable name for this comparatively common condition but Fernex and Fernex's original term "mucoid degeneration" is as short, and considerably more accurate than the term "blue valve".—I am, etc.,

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¹ Fernex, M., and Fernex, C., *Helvetica Medica Acta*, 1958, **25**, 694.

² Pomerance, A., *British Heart Journal*, 1969, **31**, 343.

³ McCarthy, L. J. and Wolf, P. L., *American Journal of Clinical Pathology*, 1970, **54**, 852.

Beta-adrenergic Blocking Drugs

SIR,—Your leading article on "Beta-adrenergic Blocking Drugs" (30 January, p. 243) raises some fundamental questions in this rapidly expanding field. The first is the distinction between intrinsic sympathomimetic activity and selectivity. There is good pharmacological evidence, in both animals and man, that practolol has a cardio-selective action. However, it is quite possible that an agent with intrinsic sympathomimetic activity could achieve a selective effect if sympathetic tone were higher in the heart than in the bronchial smooth muscle. It has been shown for agents with intrinsic sympathomimetic activity that where sympathetic tone is low, no change in function results, but where tone is high beta blockade occurs.¹ This is supported by the comparative study on airway function in asthmatics done by Connolly and Batten.² It would appear from their study that a drug does not have to be cardioselective to have less effect on airway resistance. In any case

whether drugs are cardioselective and/or have intrinsic activity, they may still provoke asthmatic attacks in sensitive subjects, and at present there is no beta-blocking drug which is free from side effect.

The leading article comments on a paper in the same issue by Sandler and Pistevos (p. 254). In this paper, considerable falls in blood pressure were seen after intravenous oxprenolol given for the treatment of dysrhythmias after myocardial infarction. It is unfortunate that oxprenolol was administered as bolus injections in amounts up to 6 mg. As the drug is approximately equipotent with propranolol, it is not surprising that a bolus injection might cause hypotension. The main therapeutic conclusion is that any effective beta-blocking drug when given intravenously should be injected slowly with extreme caution, as there is no known beta-blocking drug which would be free from the risk of provoking hypotension in such a situation.

Another major problem in evaluating the newer beta-blockers is obtaining an accurate estimate of their beta-blocking potency versus propranolol in man. This is especially true of the cardioselective drug practolol, where the standard test, which is prevention of isoprenaline tachycardia, becomes difficult to interpret.

In the therapeutic situation practolol has been evaluated extensively in angina. In only one published study so far³ where practolol has been compared with propranolol in the same patients it seems that practolol was approximately one tenth as potent as propranolol. In the same symposium, Prichard⁴ also stated that the maximum effect that could be obtained in angina with practolol was less than that which could be obtained with propranolol. Therefore it becomes difficult to interpret the frequency of side-effects such as bronchospasm and heart failure, because part of the apparent therapeutic advantage of practolol may be due to the fact that it is being used in doses which do not produce the same degree of beta blockade.

It would seem to me that there is a strong case for doing double-blind cross-over trials in angina, using the newer drugs oxprenolol and practolol, in an attempt to assess their relative potency, and side effects due to beta blockade, such as hypotension and bronchospasm.—I am, etc.,

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¹ Dollery, C. T., Paterson, J. W., and Connolly, M. E., *Clinical Pharmacology and Therapeutics*, 1969, **10**, 765.

² Connolly, C. K., and Batten, J. C., *British Medical Journal*, 1970, **2**, 515.

³ Prichard, B. N. C., Lionel, N. D. W., and Richardson, G. A., *Postgraduate Medical Journal*, 1971, **47**, Supplement, p. 59.

⁴ Prichard, B. N. C., *Postgraduate Medical Journal*, 1971, **47**, Supplement, p. 112.

Breath-activated Aerosol

SIR,—I should like to report a small study of a new breath-activated, pressurized inhaler. This device (Autohaler) has been developed in an attempt to overcome the problems which some patients experience in synchronizing the delivery of a metered dose of bronchodilator drug with the beginning of a deep inspiration.¹ To obtain maximum benefit from a pressurized inhaler, the dis-