treatment is indicated when retrograde venous pressure is not very marked. With both types of therapy the patient should be ambulatory since this is the best way to prevent complications. If each patient is individualized and treated according to specific indications, excellent results will be obtained both from injection therapy and from surgery.—We are, etc.,

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Suicidal Attempt with Propranolol

SIR,—With reference to the letter from Drs. P. Karhunen and G. Härtel (21 April, p. 178) dealing with the effect of a massive dose of practolol on a healthy heart we would like to report a somewhat similar case of suicidal attempt with propranolol.

We have recently observed a man aged 45 with ocassional bouts of supraventricular dysrhythermia which had been treated with small doses of propranolol. In a suicidal attempt he took approximately 50 tablets of propranolol (2,000 mg) and arrived in hospital two hours later in good general condition. A vial which had contained 50 tablets two days previously was found empty in his lodgings and fragments of tablets readily identifiable as propranolol were recovered on washing out his stomach. The blood propranolol level was not measured. His heart rate was 80/min, pulse normal, blood pressure 120/80 mm Hg. E.C.G. showed normal sinus rhythm. He stayed in hospital for five days without any signs of cardiac disturbance.

Propranolol is known to possess the most potent negative inotropic and chronotropic activity of all beta-blockers. Nevertheless in our case a massive dose of propranolol produced no signs of depressive action on the heart. It seems therefore that the question of the effect of propranolol on the healthy heart needs to be reconsidered.—We are, etc.,

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Formylation of Folate

SIR,—We would like to comment on the paper of Drs. Janet Perry and I. Chanarin (9 June, p. 588) regarding folate absorption.

The synthesis of H_4PteGlu by dithionite reduction of PteGlu at 75° C does not give a pure product; it always contains H_2PteGlu, and possibly pteridine decomposition products.[1] Column chromatography does not permit unequivocal separation of the complex mixtures reported. In their own figures, the peaks for PteGlu and H_2PteGlu overlap considerably and those for 5-CH_3-H_4PteGlu are not in the same place on the two chromatograms.

The reduced folates were incubated for 1 hour at 37°C at very low concentrations and in the absence of an antioxidant. At 37°C in aqueous solution, with a partial O_2 pressure of 104 mm Hg, the half life of H_4PteGlu and tetrahydrobiopterin is 20-50 min.[2]

The principal serosal material 10-formyl folate,[3] fed to rats, showed no 5-CH_3-H_4 PteGlu-(2-^{14}C) in the first three days in liver, faeces, or urine, starving the animals for three days prior to the dose made no difference.[4] Neither 10-CHO-PteGlu nor 10-CHO-H_2PteGlu is reduced by dihydrofolic acid reductase.[5] Thus the alleged principal transport product is not converted to the major storage form of folates.

No mention is made of specific activity of the radioactive compounds isolated. In the absence of this information the microbiological assay reported means little since the large contribution of endogenous folate[6] to the total cannot be calculated.

Smith *et al.*[7] showed that folic acid transport occurred without metabolism as a prerequisite and this has been confirmed in a more recent paper.[8] The results quoted by the authors are due to the oxidation of the endogenous tissue folate 10-formyl tetrahydrofolic acid to 10-formylfolic acid and have no relevance to the mechanism of transport of folic acid in the rat small intestine.—We are, etc.,

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1 Blakely, R. L., *The Biochemistry of Folic Acid and Related Pteridines*. Amsterdam and London, North-Holland Publ. Co., 1969.
2 Blair, J. A., and Pearson, A. J., *Tetrahedron Letters*, 1973, No. 3, 203. *Journal of the Chemical Society*. In press.
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Detection of Hypertension in Childhood

SIR,—I beg to question the assumption, upon which your leading article (18 August, p. 365) seems to rest, that essential hypertension is a distinct disease entity of which the early preventive treatment may justifiably be discussed.

Essential hypertension is diagnosed by exclusion of all known causes of hypertension. We may well have to accept that it may be impossible to discover all the factors which produce it. The causes may vary with each individual case; they may be diverse and interrelated (genetic, biochemical, psychological, etc.). At present we can but consider essential hypertension as a clinical sign of some disturbance of normal homoeostasis whose nature we do not understand. It follows that what your article implies by prophylaxis and treatment really relates to the modification of symptoms and signs of a complex morbid process which cannot be totally avoided or healed. The only available treatment is symptomatic. Symptomatic treatment will often leave the causes of the illness unaffected. It can be considered prophylactic only if it be maintained indefinitely in order to effect some degree of reduction of the potential morbidity of the disease process.

It seems to me that whereas the treatment of the causes of illness may be a matter for public concern and for screening, the treatment of symptoms can be ethically justified only by the specific request of the individual. Symptomatic treatment does not heal; it only pretends to do so. To offer symptomatic treatment in the later years of life seems humane and may even be considered good medicine; but I find horrifying the thought of advising a hypertensive young person to "improve" the rest of his life with drugs—or even special dieting—without the full cognizance and active request of that individual or his legal guardian.

Your article gives the impression that the persuasive introduction of symptomatic-prophylactic measures against hypertension may in future be justified in terms of economics and acceptance as beneficial by the medical profession. I submit that there is more to it than that.—I am, etc.,

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SIR,—In your leading article (18 August, p. 365) you state that "if the treatment of hypertension in young adults comes to be accepted as beneficial . . . the screening of 20-year-olds will be easily justified." Screening of that age group would be difficult. Assuming that raised blood pressure in childhood means hypertension in adult life, then it would be easy to identify children with a raised blood pressure prior to leaving school. School medical staff could fit this into the present medical examinations. The names of those found at school to have a raised blood pressure would be sent to the relevant family doctor. N.H.S. records of these children would be identified by a suitable symbol on the envelope with the year that the patient attains the age of 20. In that year, the family doctor, or possibly the health visitor in a practice with such an attachment, would re-check the blood pressure and treatment would be instituted if necessary.

I feel this to be a practical way in which potential hypertensives could be identified without the difficulty of examining every young adult, and one in which the school health service could make a valuable contribution. I suggest that a pilot scheme be begun in the area of one health authority.—I am, etc.,

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Aspirin in Prophylaxis of Ischaemic Heart Disease

SIR,—It is now five years since O'Brien[1] published his paper on the "Effects of Salicylates on Human Platelets" which showed that aspirin inhibited adenosine diphosphate (ADP) release from platelets. This release contributes to the adhesion of platelets and is increased by adrenaline. If Duguid's hypothesis that an intimal tear and platelet aggregation are the first steps in the development of the atherosclerotic plague is correct, aspirin might protect against the development of atherosclerosis. Aspirin might also be expected to reduce the likelihood of thrombosis occurring in an atherosclerotic artery. Adrenaline plays a part in inducing platelet aggregation; in particular it induces a second wave of closer and firmer aggregation. Aspirin abolishes this second