been evaluating evening primrose oil in dye-free capsules in 14 patients with long-lasting MS, all of whom displayed pyramidal, cerebellar, and posterior column signs and symptoms. They were clinically relatively stable and no patient had had an obvious relapse in the year before starting evening primrose oil. They were assessed clinically using the Kurtzke disability score. Fine motor control was assessed using the B-M manual dexterity test, which involves putting small pins into holes in 60 seconds. Grip strength was assessed by a dynamometer. All the patients had had previous experience with the tests. Eight patients received four 0.6 ml evening primrose oil capsules/day while six received the same dose of the capsules plus 0.5 mg colchicine twice a day. Colchicine was given for two reasons¹: (a) it has proved successful in Behçet's syndrome,² a disease in which there may be MS-like neurological disorders; and (b) it appears to stimulate conversion of PUFAs to thromboxane A₂, a substance which may prevent accumulation of calcium in cells.⁴ Uncontrolled calcium entry may be a major factor in demyelination.⁷ The results were as follows:

Evening primrose oil alone—Five patients showed no change on the Kurtzke scale while three showed some improvement. There was no improvement in grip strength but there was a significant ($P < 0.05$) improvement in performance in the manual dexterity test.

Evening primrose oil plus colchicine—Four patients improved on the Kurtzke scale while two did not respond. Two of the patients regarded themselves as dramatically improved and one returned to work after being unemployed for eight years. In the manual dexterity test two patients who had been unable to insert pins at all for two years became able to insert some, and the overall improvement in the group was significant at the $P < 0.025$ level. Grip strength also improved but this improvement just failed to reach significance.

These results, we believe, justify a formal controlled trial and suggest that evening primrose oil packaged in dye-free capsules, especially when combined with colchicine, may be of value.

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Low-cholesterol diet in patients with gall stones taking chenodeoxycholic acid

SIR,—In a recent article (23 September 1978, p 851), Dr D P Maudgal and his colleagues concluded that a low-cholesterol diet resulted in an enhancement of the effect of chenodeoxycholic acid (CDCA) in patients with gall stones whose bile had already been rendered unsaturated by CDCA when taking a diet containing 600 mg cholesterol.

It should be noted that the difference obtained in the saturation index by the reduction of dietary cholesterol was very small (0.12 in experiment 1 and 0.05 in experiment 2) and we question whether these small differences are of any biological, as distinct from statistical, significance. There is certainly no evidence in the literature that the rate of gall-stone dissolution is related to the degree of lowering of the saturation index. The abstract

of Bell *et al*¹ cited by the authors states only that gall-stone dissolution in the rhesus monkey was associated with bile unsaturated with cholesterol. It is likely that the most important rate-limiting factor in dissolution may be the interfacial barriers² rather than the decrease of the saturation index.

We offer two other methodological criticisms of the paper. Firstly, the duration of "overnight fast" prior to bile sampling was not specified and yet it is critically important in studies where there is comparison of the saturation indices. A decrease in duration of fast from 16 to 9 hours, for example, can lower the index by as much as 0.36 in women and 0.19 in men.³ Secondly, the mean decrease in biliary saturation index of 0.12 in experiment 1 was produced in hospital on a 100 mg cholesterol diet, but a control group who entered hospital on a 600 mg cholesterol diet was not studied. There are many factors in the hospital environment, including the length of pretest fasting, which may have contributed to this change in saturation index.

In summary, we are not convinced that a low-cholesterol diet has been shown to have any place in the dissolution therapy for gall stones by CDCA.

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Vitamin E and malignant hyperthermia

SIR,—Last year (20 May, p 1345) I suggested that there might be an association between the consumption of excessive vitamin E and the development of malignant hyperthermia. The Malignant Hyperthermia Research Unit of the University of Leeds has now investigated the three children of the patient I previously described. Two of them have been proved by muscle biopsy to be susceptible to malignant hyperthermia.

It therefore seems likely that the episode of malignant hyperthermia I encountered was due to a genetically determined susceptibility, not to vitamin E.

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Effect of propranolol on plasma concentrations of HDL apoprotein and lipid

SIR,—The provocative reports by Dr A Helgeland and others (5 August, p 403) and by Drs D Streja and D Mymin (25 November, p 1495) associating low concentrations of high-density lipoprotein (HDL) cholesterol with propranolol therapy are further supported by the longitudinal study of Dr N Tanaka and others on 10 subjects before and after propranolol therapy.¹ In that study subjects were normolipaeamic and on no other medication. They were maintained on a standard hospital diet and no weight changes were observed during the study. After control blood samples had been collected, 60 mg/day of propranolol was administered orally for seven weeks and

120 mg/day for the eighth and final week. Throughout the period of propranolol administration serial plasma samples demonstrated a persistent fall in both HDL apoprotein and lipid concentrations. At the end of eight weeks of therapy only three-quarters of the concentrations of plasma HDL apoprotein and lipid remained.

It has been suggested that the apoproteins of HDL may have a key role in bringing about the anti-atherogenic effect of HDL.² The study's demonstration of a significant fall with propranolol therapy in both plasma HDL apoprotein and HDL lipid content heightens concern about a non-beneficial effect of chronic propranolol therapy in hypertensive and coronary artery disease patients.

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Cardiac arrhythmias and "epilepsy"

SIR,—You kindly published my comment on cardiac arrhythmias that can be confused with epilepsy (2 July, 1977, p 45). The long Q-T interval syndrome was mentioned as one of the possibilities, exemplified by the case history of a young woman. The patient died of ventricular fibrillation recently, at the age of 23, and the necropsy finding prompts me to correct my earlier report.

The pathologist found an extremely thin-walled right ventricle, almost devoid of heart muscle. A very small fibrotic area was seen in the left ventricle. The heart valves and the coronary arteries were intact. This unusual (congenital?) form of myocardiopathy is referred to as parchment heart¹ or *ventricule droit papyracé*² in the French literature. It can be regarded probably as a variant of endomyocardial fibrosis. Some of those patients who survive infancy are prone to have recurrent attacks of ventricular tachycardia. Only a few similar cases have been described.²⁻⁴

The prolongation of the Q-T interval was a secondary phenomenon in this case and due to the myocardiopathy. It might have had a contributory role, however, in the pathogenesis of ventricular tachycardia and fibrillation.

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Spontaneous pneumomediastinum complicating anorexia nervosa

SIR,—In their report of two cases of spontaneous pneumomediastinum complicating anorexia nervosa Drs Anita J Donley and T J Kemple (9 December, p 1604) found only one report of pneumomediastinum complicating starvation or wasting diseases.

We have recently managed a patient with spontaneous pneumomediastinum complicating severe hyperemesis gravidarum at 10