in the seat with the back well rounded, but this is the very position which predisposes to prolapsed intervertebral disk and causes so much discomfort to countless motorists.

Having eventually squeezed yourself into a car, you find that the steering wheel is in the wrong place, and there is nothing that you can do about it, for the pre-war steering wheel of adjustable length and rake is a thing of the past. In some cases the pedals are so set that they cause a marked strain on the muscles of the lower leg. Finally, what amounts to the almost criminal is the crowding of the pedals. My own car is one of the cheapest models and yet one of the roomiest; its performance is indifferent, but I can sit upright in the seat without getting backache and yet have 4 in. (10 cm.) clearance between my head and the roof, but the seat, alas, lacks any pretence at design; it gives no support to the back anywhere near where it is required: yet even in this roomy car I find that the inside of my right shoe gets worn by the brake pedal while the outside of the shoe is wearing away the panelling of the bodywork. I have driven cars in which I could not use the foot-brake without also depressing with the same foot either the accelerator or clutch: could anything be more dangerous or more likely to cause accidents? I must add that I am large but no giant, being a mere 6 ft. 1½ in. (1.8 m.) and taking size 10 shoes.

If present tendencies continue, our cars will only be fit for midgets. Let us hope that next year there will be more cars for men and more consideration for some elementary points of safety and simple anatomy.—I am, etc.,

Huntingdon. R. T. D. FITZGERALD.

Neostigmine-resistant Curarization

SIR,—The dangerous and not uncommon condition of persistent post-operative oligapnoea after the use of relaxants in patients distended by ileus has been described by Dr. A. R. Hunter (*Journal*, October 20, p. 919) and subsequent correspondents under the resounding title of "neostigmine-resistant curarization."

So far I have thought of this state all too simply as a diaphragm-resistant ileus and have tried to relieve an enfeebled and captive diaphragm by adding a slight foot-down tilt of the bed to the routine measures of resuscitation. I see now that this humdrum mechanical concept will not do; but I shall continue to worry about how the diaphragm, even were it not bedevilled by low plasma-potassium levels and curare-synergistic bacterial toxins, can ventilate the lungs when splinted by an abdominal balloon.

Many of these patients arrive, even without the aid of relaxants, neostigmine-resistant or not, at the stage of respiratory inadequacy well described by Dr. J. F. Rickards (*Journal*, November 10, p. 1114), where one may well ask:

> "Can storied Urn or animated Bust
> Back to its Mansion call the fleeting Breath?"

and Dr. Rickards answers specifically: "Drugs and hyperventilation have no effect." I feel that we should fix the more familiar causes of apnoea well in front of our eyes before we plunge deeper into biochemical speculations.—I am, etc.,

Exeter. W. J. WALTER.

SIR,—I have read with interest, not unmixed with horror, the recent correspondence on neostigmine-resistant curarization. Surely the solution of the problem lies in its prevention—that is, in the avoidance of the use of any relaxant drugs whatsoever in cases of intestinal obstruction. No doubt it is extremely interesting to speculate on the possible causes of death in these cases, but our work should aim at the preservation of life rather than in the production of stimulating, scientific post-mortem discussions.

As has been shown in the recent survey of anaesthetic deaths,[1] our main worry is vomiting, and the obstructed patient is very prone to vomit during induction, even where an oesophageal tube has been inserted and suction applied to empty the stomach of its contents. To use thiopentone and relaxant drugs on such patients is asking for trouble at the start, quite apart from any question of neostigmine failing to do the trick later on. I suggest that a safer though less spectacular method is the nitrous oxide–ether sequence with intubation. This may appear to some to be old-fashioned and unprogressive, but, if it results in a diminution in the number of fatal accidents, then I think there is something to be said for it.—I am, etc.,

Glasgow. ALBERT CHRISTIE.

REFERENCE

1 Edwards, G., Morton, H. J. V., Pask, E. A., and Wylie, W. D., *Anaesthesia*, 1956, **11**, 194.

Cholesterol and Vascular Disease

SIR,—In a recent letter (*Journal*, November 17, p. 1172) Sir John McNee mentions that cholesterol dissolved in sunflower oil when fed to rabbits causes atherosclerosis, whereas similar experiments in cats caused no such lesions. Recent work by Bronte-Stewart[1] and others would suggest that the use of sunflower oil as a solvent in the above experiments was unfortunate.

It has been shown that vegetable oils (especially sunflower and peanut oils), by virtue of their unsaturated fatty acid content, protect the human subject from the rise in serum cholesterol which normally accompanies a cholesterol-rich diet. For the same reason, marine oils have a similar cholesterol lowering effect; whereas all fats of animal origin (including eggs and milk products) and saturated vegetable oils (as artificially produced by hydrogenation in the manufacture of margarine and most cooking fats) cause a sharp rise in serum-cholesterol.—I am, etc.,

London, W.1. J. NICKSON.

REFERENCE

1 Bronte-Stewart, B., Antonis, A., Eales, J., and Brock, J. F., *Lancet*, 1956, **1**, 521.

SIR,—Sir John McNee's reminiscence (*Journal*, November 17, p. 1172) on Anitchkov's early experiments is a charming contribution to the history of experimental atherogenesis. However, his remark regarding the role of cholesterol in human vascular disease, that feeding experiments were only successful in one herbivorous animal, the rabbit, calls for some comment.

While for many years rabbits were used almost exclusively in these feeding experiments, further investigations in this line on other herbivorous animals showed surprisingly poor results. Experimental atherosclerosis could be produced only in two more herbivorous animals, in the guinea-pig and hamster,[1] and later on most of the experimental work was done on mixed-fed animals. Perhaps the most extensive studies were performed on chicks, mainly in Chicago, either by feeding large amounts of cholesterol alone[2] or adding stilboestrol to the cholesterol-rich diet.[3,4] Steiner and Kendall[5] produced typical vascular changes in carnivorous dogs by adding thiouracil to the cholesterol feeding, and after many failures atheromatous changes could be caused in "middle-aged" obese rats on diets "modelled on that of patients who developed coronary thrombosis."[6] Incidentally, even in ducks and geese similar lesions could be effected by dietary means.[7] Lastly, in an adult rhesus monkey extensive atherosclerosis, together with cutaneous manifestations of xanthomatosis, could be produced recently by prolonged high cholesterol feeding.[8]

Perhaps of even greater importance is the experimental production of extensive atherosclerosis by, not necessarily extensive, deficiency of different dietary factors. In rhesus monkeys widespread atheromatosis was produced by pyridoxine-deficient diet,[9] and these findings were confirmed quite recently not only in monkeys but also in dogs.[10] Furthermore, in cebus monkeys a cholesterol-supplemented diet, deficient in, but not completely devoid of, sulphur-containing protein produced extensive atherosclerotic changes.[11]

In this short letter this vast subject cannot be discussed in detail. I only want to reiterate the statement that at the

present state of research little doubt exists about the intimate relationship of cholesterol metabolism and human atherosclerosis. It would appear that the most promising approach to this problem lies in more experimental and clinical studies on the *interrelationship* which influences the production and maintenance of high blood lipid levels in men; even more important is their permanent reduction, possibly by physiological means. Such work is in progress in several centres, and advances may be expected in the near future.—I am, etc.,

London, W.1. Z. A. LEITNER.

REFERENCES

1 Altschul R., *Amer. Heart J.*, 1950, **40**, 401.
2 Dauber, D. V., and Katz, L. N., *Arch. Path.*, 1942, **34**, 937.
3 Horlick, L., and Katz, L. N., *J. Lab. clin. Med.*, 1948, **33**, 733.
4 Chaikoff, I. L., Lindsay, S., Lorenz, F. W., and Entenman, C., *J. exp. Med.*, 1948, **88**, 373.
5 Steiner, A., and Kendall, F. E., *Arch. Path.*, 1946, **42**, 433.
6 Wissler, R. H., Eilert, M. L., Schroeder, M. A., and Cohen, L., *Fed. Proc.*, 1952, **11**, 434.
7 Wolffe, J. B., *et al.*, *Amer. Heart J.*, 1949, **38**, 467.
8 Mann, G. V., and Andrus, S. B., *J. Lab. clin. Med.*, 1956, **48**, 533.
9 Rinehart, J. F., and Greenberg, L. D., *Amer. J. Path.*, 1949, **25**, 481.
10 Mushett, C. H., and Emerson, G. H., *Proc. Fed. Amer. Soc. exp. Biol.*, 1956, **15**, 526.
11 Mann G. V., Andrus, S. B., McNally, A., and Stare, F. J., *J. exp. Med.*, 1953, **98**, 195.

SIR,—Sir John McNee is to be thanked for referring (*Journal*, November 17, p. 1172) to his early demonstration of atheroma developing in rabbits fed with cholesterol, thus confirming the work of Anitchkov. Sir John emphasizes that this method was only successful in giving rise to atheroma in herbivores, and he states that he is unaware that atheroma has ever been produced in carnivorous or mixed-feeding animals by similar means. In this connexion it may be of interest to recall the more recent results of Steiner and Kendall.[1] These workers in fact succeeded in causing pronounced atheroma in dogs by feeding cholesterol *along* with thiouracil. In these experiments intense hypercholesterolaemia and also hyperlipaemia preceded the development of atheroma. Apparently, without simultaneous depression of thyroid activity, cholesterol in the diet cannot alone produce sufficient hypercholesterolaemia in dogs to cause atheroma; Steiner and Kendall used thiouracil so as to inhibit thyroid activity.

Sir John McNee suggests that the failure to produce atheroma, in any but herbivorous animals, by feeding cholesterol, diminishes the importance of the results with rabbits in their bearing on human disease. The work of Steiner and Kendall, however, indicates that excessive cholesterol in the diet, when *coupled* with depression of thyroid activity, may indeed be a potent factor in the generation of atheroma in omnivorous animals like man.—I am, etc.,

Cambridge. KENDAL C. DIXON.

REFERENCE

1 Steiner, A., and Kendall, F. E., *Arch. Path.*, 1946, **42**, 433.

Degree in Nursing

SIR,—The Royal College of Nursing is convinced that nursing to-day is a truly academic discipline, and it recommends university training lasting perhaps five years. Dear, oh dear! As such graduates are intended eventually to become matrons and holders of other senior posts, does this mean that the less scholastic State-registered nurses will be bracketed with the State-enrolled assistant nurses and auxiliary helpers, and will be debarred from more responsible and lucrative posts?

Would it not help the recruitment of nurses if the syllabus of subjects for State-registration could be made less academic, and the course shortened by a year? As the evolution from student nurse to sister has repeatedly shown that in many cases "the ugliest duckling turned out to be the fairest of the swans," and vice versa, would it perhaps be a better plan to have a scheme by which each year a certain number of sisters could be sent to the universities to study politics, philosophy, and economics, if there are some posts for graduate nurses at home and abroad?—I am, etc.,

Crowthorne, Berks. RUDOLPH PAYNE.

Irradiation-induced Malignant Hypertension

SIR,—The letter by Dr. R. W. Luxton (*Journal*, November 17, p. 1176), in which he states that we "appear to have made a somewhat limited examination of the literature" (*Journal*, October 20, p. 910), cannot go unanswered. Dean and Abels[1] did not describe a similar case, since their patient did not have malignant hypertension, nor did they claim it. Both Dr. Luxton's own patients who did have malignant hypertension died within seven weeks of the onset of symptoms, without nephrectomy.

Dr. Luxton regards as more serious that we did not mention that both kidneys may be badly damaged by radiation. It appeared so self-evident that when both kidneys have been exposed to the same dose of radiation they may suffer the same fate that it did not seem necessary to mention it, and in our particular patient the fact that there was a significant difference in the dosage sustained by the two kidneys was recorded. We believe that when, years after irradiation, one kidney is found after full investigation to be functioning normally the possibility that it has suffered serious damage can reasonably be excluded. In our case, we recorded that intravenous pyelography, chromocystoscopy, and micro-urea concentration all indicated normal function in the right kidney.

Finally, Dr. Luxton overlooks the fact that, where there has been an extensive upper abdominal deposit from a testicular growth, this is usually in close relation to one kidney and may by pressure or actual infiltration predispose to unilateral radiation damage. We did, in fact, mention this possibility in our patient. We are not quite certain as to the lesson which Dr. Luxton intends to point in his letter, unless it is that renal function tests are not to be relied upon in this condition. If, indeed, we had proceeded on that view, our patient would not have had his nephrectomy, and his life would not have been saved.—We are, etc.,

W. M. LEVITT.
London, W.1. S. ORAM.

REFERENCE

1 Dean, A. L., and Abels, J. C., *J. Urol.*, 1944, **52**, 497.

Medical Relief Appeal

SIR,—I am very distressed to find political letters, or at least letters with a strong political bias, being published in the correspondence columns of the *Journal*.—I am, etc.,

London, W.1. LIONEL TAYLOR.

Treatment of Iron-deficiency Anaemia

SIR,—Dr. A. Piney (*Journal*, November 10, p. 1118) and Dr. M. E. Lampard (p. 1119) take me to task on the subject of iron stores. Dr. Lampard says that my "statements are hardly credible, as 50% or more of the nation would be anaemic, or on the verge of anaemia." The credibility of a statement does not turn upon the size of the problem it purports to reveal. And 50% is, of course, Dr. Lampard's own figure: I quoted none because I know of none. However, here is an extract from a recent publication,[1] chosen at random: "Due to the periodic menstrual blood loss in woman during her reproductive life, the female enters pregnancy usually in a rather precarious state of iron balance, and may spend her entire life prior to the menopause in a state of greater or lesser iron deficiency." I do not know what one of Dr. Lampard's female patients would say about "having her hide pricked," as Dr. Lampard puts it; but I think I know what alternative she would suggest if told to take one iron tablet a day for the rest of her life. I think, too, that the Chancellor of the Exchequer might be interested in any such ingestion on a wide scale.

Dr. Piney objects to my statement that "at a haemoglobin level of 100%, iron absorption from any oral preparation is very small." I mentioned two supporting papers in my original letter. I will now quote from another of the many papers[2] on this topic. It says: "Although the intestinal uptake in iron-deficiency anaemia may be 2–10 times that occurring in the normal individual, it has been noted that once the anaemia is corrected, uptake slows down and tissue stores are only slowly reconstituted. Has Dr. Piney some