has continuous access to his patients over many years and sees them both in health and in sickness, has far better opportunities for studying and recording the natural history of disease than any other medical man. It is, as Sir Cecil says, a great pity that their important observations are not handed down to future generations.

It seems to me, and I believe that I am not alone, that the clinical part of medical training is very poor in the teaching of those medical students who will become the general practitioners of the future. Medical training naturally omits at the beginning all the natural difficulties, by its simplification of the great complexities of the human body and the study of physiological and pathological changes and other specialized sciences. Yet the newly qualified young doctor must face the whole man as a patient, with all his individual variations within the limits of the normal and the physiological. It ought to be easier for him to face this whole man in the last year of his studying than at the beginning, and he ought to have heard near the beginning of his course in normal physiology of all the normal individual differences. In fact, he hears nothing of this, but only about an average type and precise laboratory findings. After 30 years of general practice one knows how wide the amplitude of normal differences is, and the great value of knowledge of what I call "the constitutional factor" and the "innate temperament" of everyone.

It is a great pity that this important experience cannot be handed on to future general practitioners.—I am, etc.,

Split, Yugoslavia. JURAJ KALINIĆ.

Vascular Changes During Anaesthesia

SIR,—In their recent paper (June 14, p. 1284), Drs. R. Shackman and I. G. Graber showed an increase in skin temperature and a concomitant diminution in rectal temperature during anaesthesia. One must, I think, accept their conclusion that the former denotes a rise in skin blood flow (see also correspondence, June 28, p. 1409, and July 5, p. 44). The significance of the fall in rectal temperature is not so clear. From independent experiments on animals I am convinced that anaesthetics do indeed produce a diminution at least in liver blood flow and probably also in rectal blood flow, but I very much doubt whether rectal temperature observations alone can be regarded as evidence of the same reaction in man.

I have never claimed that rectal temperature alone may be used as an index of blood flow. It is true that earlier experiments reported by me (*British Medical Journal*, 1951, **2**, 1379) showed that rectal temperature changes could be produced by rectal blood flow changes. This is a very different matter, and the conclusions were supported by direct recordings of rectal blood flow. Moreover, the maximum temperature changes which could be attributed to vascular reaction were less than 0.5° C. Without the use of special methods—the heated thermocouple technique was used in my work—I would hesitate to claim that any change in rectal temperature was primarily vascular in origin.

The interpretation of rectal temperature changes in anaesthesia is further complicated by the fact that anaesthesia produces a drop in all internal temperatures. Dr. Birnie and I showed that, in the rat, ether or "nembutal" anaesthesia lowered the liver temperature from mean values of over 39° C. to mean values of about 34° C. (circumstances compel me to quote from memory). In the rabbit we have recorded liver temperatures as low as 33° C. with rectal temperatures of 31.8° C. during nembutal anaesthesia. These changes were not primarily vascular, a point established by simultaneous blood flow records. They were the result of alterations in thermal balance; whether this was due to metabolic depression or increased heat elimination or a combination of both has not been established.

I cannot help feeling that the findings of Shackman and Graber may arise from similar causes. Diminutions in rectal blood flow probably contributed to the temperature drops, indeed they may have been the initiating factor. None the less, I consider that the full extent of the temperature drop reported was too great for so simple an explanation and must have been determined, in part at least, by alterations in heat equilibrium similar to those occurring in other species.—I am, etc.,

Bristol. J. GRAYSON.

Prolonged Respiratory Paralysis after Succinylcholine

SIR,—In view of the correspondence regarding the dosage of succinylcholine chloride and the duration of the ensuing apnoea (June 21, pp. 1342 and 1352), the following case may be of interest.

A man of 58 was to undergo electro-convulsive therapy as an out-patient. He was six feet tall and weighed 140 lb. (63.4 kg.); atropine, 1/100 gr. (0.65 mg.), was given subcutaneously half an hour prior to the treatment. Thiopentone sodium was administered until the patient was asleep, 0.3 g. being required. This was followed by 50 mg. succinylcholine. An airway was inserted and the patient inflated with oxygen, care being taken to avoid hyperventilation. The shock was administered one minute after the onset of muscular paralysis. Excellent modification was obtained. Inflation of the lungs was continued until spontaneous respiration was re-established. This occurred seven and a half minutes after the disappearance of the initial muscular contractions following the injection of succinylcholine.

Further treatments were considered essential. Accordingly, in view of the report of Hurley and Monro (*Journal*, May 11, p. 1027), a smaller dose of succinylcholine was employed. The technique was similar in every detail except that only 20 mg. relaxant was used. Again an airway was inserted into the mouth with ease and oxygenation of the lungs carried out. Modification of the fit was inadequate. The duration of apnoea was three minutes. Subsequently, six treatments have been carried out using 40 mg. succinylcholine. With this dose apnoea persisted for six minutes and satisfactory modification was obtained. In this case, using a small constant sleeping dose of thiopentone, the length of apnoea depended on the dose of succinylcholine used.

I am grateful to Dr. George Edwards and Dr. Desmond Curran for permission to record this case.—I am, etc.,

Department of Anaesthetics, St. George's Hospital, S.W.1. B. L. DAY.

Ultra-short-acting Relaxants

SIR,—I was most interested in the report by Drs. H. Richards and H. R. Youngman (June 21, p. 1334) on the short-acting relaxants, particularly in their use in electroplexy, in which it seems likely they will supplant the longer-acting compounds. One of the routine relaxants used at this hospital during the past six months has been a heteropolymethylene-bis-trimethylammonium di-iodide preparation (Ro 3-0386), which is somewhat similar in its effects to succinylcholine iodide. So far relaxation has been adequate, recovery rapid, and no ill effects have been observed.

The technique employed here has been to give a 3-gr. (0.2-g.) capsule of sodium amytal one hour prior to the induction of the fit. Immediately before applying the electro-convulsive therapy 5 mg. Ro 3-0386 is given by intravenous injection over a period of 10 seconds. The required depth of relaxation is produced in one to two minutes and passes off within four minutes.

There has been the occasional complaint of slight constriction in the throat. Assisted respiration has not been found necessary, although oxygen has been given in about 25 of a total of 50 administrations. No changes in the blood pressure were observed except in one case, an arteriosclerotic, where a transient reduction occurred.

I am grateful to Dr. F. Wrigley and Roche Products, Ltd., for the Ro 3-0386 used in these cases.—I am, etc.,

Preston, Lancs. R. B. TAYLOR.

Post-operative Brachial Plexus Paralysis

SIR,—Dr. F. G. Wood-Smith (May 24, p. 1115) does a great service in bringing this important subject forward. I am sure that the hazards of the extreme positions on the operating table which he mentions are not sufficiently recognized or widely enough known. One could almost write a small book on the hazards of the steep Trendelenburg position.

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