Student Suicides

SIR,—Since Sir Alan Rook presented his valuable study of student suicides (*Journal*, March 7, p. 599), correspondents have continued his discussion of the aetiology of these and other psychiatric breakdown among students, and of the steps which can be taken by the student health services to prevent them, or to treat them in their early stages.

But preventive measures should begin much earlier. The great majority of these students have vulnerable personalities and succumb to stresses which others surmount unscathed. The seeds of their breakdown have been sown long before. In interviews with such patients it is usually found that a demonstrable degree of maladjustment has been present for many years. There is a special opportunity for valuable preventive work in the pre-university years, when the intelligent adolescent is commonly in an introspective phase, preoccupied with himself and with the problems of growing up, ready to talk and not unreceptive in his attitude towards professional help, once he is convinced that the psychiatrist has something to offer.

Opportunity comes in the first instance to the family doctor and to the school medical officer. It is for them to recognize that an intelligent adolescent who, for example, loses his or her capacity to work and does not know what to do, rows endlessly with the family or develops psychosomatic symptoms, may be showing manifestations of potentially serious illness which should be investigated, not dismissed as tiresome "teen-age" behaviour. These young people are often profoundly unhappy. It is for the general practitioner or school medical officer to persuade the patient, the parents, and sometimes the headmaster that something can and should be done to help them. Opportunity comes in the second place to our psychiatric clinics. There is a special responsibility on them to provide adequate services for these gifted adolescents and to present them in an acceptable form.—I am, etc.,

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London, W.1. DUGMORE HUNTER.

Intermittent Suxamethonium Injections

SIR,—I have read with some consternation in the *Journal* of March 21 (p. 786) the letter by Dr. J. Bullough on the alleged dangers arising from the intermittent administration of succinylcholine chloride.

I am convinced that anaesthetists who have been using this relaxant for years—whether as a single dose, intermittently, or as a continuous drip—will not agree that it should be held responsible, *per se*, for the phenomena imputed by your correspondent. Bradycardia, cardiac arrhythmia, and hypertension occurring during general anaesthesia were known long before the advent of the muscle relaxants. They are now recognized as conditions arising from the ill effects of hypoventilation[1]—namely, hypoxia and hypercapnia. It is also known that bradycardia and hypertension can occur when there is a build-up of intra-alveolar tension resulting from incorrect pumping of the re-breathing bag. Furthermore, there is no scientific proof whatsoever that succinylcholine has any deleterious effect on the cardiovascular system.

In the anaesthetic death reported, the doses of the inhalation narcotics recorded were: approximately 0.7% trichlorethylene, and 75% nitrous oxide with 25% oxygen. I find this statement not only of little value but misleading. Surely it is the amount which the patient *absorbs* which counts? And this depends mainly on the degree of respiratory ventilation—the *tidal volume*. Yet this all-important factor of respiration is not even mentioned by your correspondent, who merely states that at one stage the patient breathed by herself. One is left to assume that inadequate gaseous interchange took place, and therefore the possibility of hypoxia must be considered, although the anaesthetic machine might have been registering 25% oxygen. Again, with inadequate ventilation, one must assume also that insufficient carbon dioxide was excreted. In the absence of post-mortem data, the fatality appears therefore to be the sequel of hypoventilation, plus, more than likely, the poisonous effects of trichlorethylene which, having been absorbed but not excreted, exerted accentuated toxicity. The patient, who apparently was not a good risk, was unable to compensate adequately. There seems to be no valid evidence to indict succinylcholine as the scapegoat; nor is there any necessity to seek for an antidote in atropine. Groundless postulations, such as "the heart becomes sensitized," appear to be used far too glibly in general anaesthesia to cover a multitude of sins and can only bring the specialty into disrepute as a science. May I remind your correspondent that practically every weird, untoward, and so-called inexplicable phenomenon associated with general anaesthesia can be attributed to the concomitant state of hypoventilation which the anaesthetist has inadvertently allowed to occur?

It would appear more than possible that the 214 cases referred to by your correspondent are all examples of side-effects of hypoventilation occurring during the anaesthesias. I refer Dr. Bullough, and others who think along similar lines, to *The Man Behind the Bag*, by Lester Rumble, Jr.[2]—I am, etc.,

Johannesburg, South Africa. H. H. SAMSON.

REFERENCES
[1] Samson, H. H., *S. Afr. med. J.*, 1956, **30**, 470.
[2] Rumble, Lester, *Sth. med. J. (Bgham, Ala.)*, 1956, **49**, 368.

SIR,—In the correspondence on this subject I have not as yet seen a plain statement of the fact, which is surely well recognized, that suxamethonium exerts a muscarine-like action during its phase of depolarization. This is not surprising, for there is no reason why stimulation should be confined to the motor end-plates alone. The unwanted effects commonly noted by the anaesthetist are bradycardia and salivation, and both are abolished or reduced by atropine. It seems likely that all muscle relaxants of the depolarizing type would have a similar effect, but my own experience is mainly confined to suxamethonium.

The marked effect of a second dose alone, reported by Dr. J. Bullough (*Journal*, March 21, p. 786), is rather a different matter, and would require another explanation which I can make no attempt to give. My own experience suggests that any single dose of suxamethonium may produce marked bradycardia or transient vagal arrest in a susceptible patient.—I am, etc.,

London, S.W.5. T. A. BOLISTON.

SIR,—I have been much interested in the letters by Dr. J. Bullough (*Journal*, March 21, p. 786) and Drs. I. Verner and Christina Comty (*Journal*, May 9, p. 1239) on this subject; since both letters mention animal experiments, it may interest your readers to know that the Universities Federation for Animal Welfare (UFAW) is at present sponsoring a research programme on the effects of relaxants on animals, hoping thereby to gain information as to the most humane ways of using these drugs in veterinary medicine.

I have used suxamethonium bromide ("brevidil E") extensively in the rabbit as (1) single intravenous injection,

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