Electro-convulsive Therapy

SIR,—The following method is used for giving electro-convulsive therapy at Cork Mental Hospital. The patient receives 1/100 gr. (0.65 mg.) atropine sulphate ten minutes before stimulus, followed by thiopentone sodium, 0.15–0.25 g. intravenously. The syringe is disconnected, but the needle remains intact, and through it is injected succinylcholine, 50 mg. As soon as fibrillation ceases the patient's lungs are inflated 3–4 times with oxygen, using a mask and rebreathing bag. The headpiece is then applied and the electrical stimulus is given.

We would like to record the following observation which may be of interest. If the pupil contracts immediately after the stimulus we have noticed that a convulsion very rarely occurs. Owing to the short time factor involved before the succinylcholine wears away, we now feel it safe to give another stimulus straight away, as there seems to be little danger of a delayed convulsion. We have found this sign very useful and wondered if other readers have had any similar experience.—We are, etc.,

E. THOMAS.
B. F. HONAN.

Cork.

Christmas Disease

SIR,—The correspondence which has followed publication of the article by Dr. Rosemary Biggs and colleagues on "Christmas Disease" (*Journal*, December 27, 1952, p. 1378) has raised several important questions, including that of nomenclature. Dr. A. S. Wiener's suggestion that this disease be called haemophilia II or B (*Journal*, March 7, p. 559) has merit because it focuses attention on the genetic similarity to classical haemophilia. His proposal, however, is based on the assumption that the mutant genes for classical haemophilia and Christmas disease occupy the same locus on the X chromosome—that is, they are alleles. We believe this assumption to be incorrect. There are well-defined differences, biological and chemical, between the normal plasma components which prevent haemophilia and Christmas disease. For example, the factor preventing haemophilia (AHF) is not absorbed by BaSO_4 , and its activity diminishes during clotting. On the other hand, the "Christmas factor" is absorbed on to BaSO_4 , and its activity increases during clotting.

It was suggested that the same allelic relationship exists between Christmas disease and classical haemophilia as exists between mild and classical haemophilia. This analogy, we believe, is unsound. The two forms of haemophilia, resulting from different degrees of reduction of the identical plasma protein, AHF, are due to the mutant genes h and h^m ; Christmas disease, resulting from diminution of an entirely different protein, is probably due to a mutant at a different locus on the X chromosome. The facts that normal plasmas, even in males with the single X chromosome, possess both AHF and Christmas factor, that haemophilic plasma contains "Christmas factor," and that AHF is present in Christmas disease can hardly be interpreted in any other fashion. Linkage studies would be most valuable.

As for the problem of nomenclature, we suggest that haemophilia and Christmas disease be grouped with the other haemorrhagic diatheses which are characterized by absence or malfunction of a specific plasma protein required for normal clotting, and a resultant clotting defect due to impaired conversion of prothrombin to thrombin. Haemophilia is the archetype of this group of diseases. The name "haemophilia" is hallowed by tradition and deeply ingrained in the literature; we suggest that its use be limited to those haemorrhagic states due to inadequate AHF. For the other diseases meeting the above criteria we suggest the term "haemophilioid states."

The following table indicates the presently described members of this group of haemorrhagic diseases. The proposed classification has the merits of simplicity and elasticity. When the exact chemical impairment in each disease is

known, more meaningful terms, neither eponymic nor arbitrary, can be applied. An elaboration of this classification, along with diagnostic criteria, will be published soon.

Disease State**Synonyms**

Haemophilia	
Haemophilioid state A	Parahaemophilia, accelerin, or AcG deficiency
Haemophilioid state B	Hypoproconvertinaemia, spca-deficiency
Haemophilioid state C	"Christmas disease," PTC-deficiency
Haemophilioid state D	PTA-deficiency

—We are, etc.,

JOHN B. GRAHAM.
K. M. BRINKHOUS.

Chapel Hill, N. Carolina.

Controlled Respiration and Apnoea in Anaesthesia

SIR,—Dr. Gordon M. Wyant (*Journal*, June 13, p. 1334) recommends anaesthetists not to lose sight of the fact that apnoea "is essentially an unphysiological state of affairs." Now what does Dr. Wyant mean by "unphysiological"? How, for example, would he achieve, in his sense, "physiological anaesthesia"? Surely it must be admitted that anaesthesia essentially is unphysiological, in that it involves interference with the normal mechanisms of maintaining the internal environment. It is, for example, true to say that under general anaesthesia it is impossible to produce relaxation of the abdominal muscles without some involvement of the respiration. It is under these conditions that "the lesser of the two evils" is to have ventilation of the lungs which is artificial but efficient, rather than that which is natural and impaired.

Professor E. A. Pask (*Journal*, May 9, 1948), writing on the same subject, believes that "many physiologists will be surprised to learn that positive pressure ventilations can be carried out at all without causing some disturbance in the cardiovascular system." It is not clear in this context whether he necessarily implies by the term "disturbance" a resultant reduced efficiency. We are sure he would join us in wishing to refer these physiologists to the all-important work on human subjects carried out by Cournand and his colleagues.¹ These workers showed that, given the correct intrapulmonary pressure curve, artificial inflation of the lungs did not, in fact, impair cardiac function.

We would conclude with two points: (1) It appears that the term "unphysiological" is being too loosely used. In artificial circumstances, for example, during anaesthesia, the maintenance of the internal environment to comply with cellular requirements—that is, normal physiology in Claude Bernard's sense—may imply the use of artificial methods. (2) Many in the vanguard of modern anaesthetic progress still maintain a regard amounting almost to a superstition for the authority of the—to quote Professor Pask—"old-fashioned" anaesthetist. We are not, however, prepared to believe that the virtue of being old-fashioned gives any doctrinal authority in respect to the matters under discussion.—We are, etc.,

G. JACKSON REES.
T. CECIL GRAY.

Liverpool.

REFERENCE

¹ *Amer. J. Physiol.*, 1948, 152, 162.

The Post-operative Chest

SIR,—Mr. V. J. Downie's remarks regarding the treatment of the "post-operative chest" (*Journal*, June 20, p. 1388) cannot be allowed to pass without comment.

During the past 25 years or so, many workers have investigated this problem very carefully; if would be a great pity if their work were to be wasted because it came to be accepted that prevention and treatment based on a proper understanding of the pathology of this condition was of little or no importance. Mr. Downie suggests that a good anaesthetic technique will prevent these complications. This is certainly not the case. A bad anaesthetic technique may certainly increase the incidence of post-operative chest com-