Cardiac arrhythmias and epilepsy

SIR,—I should like to add another, probably less well known, cardiac abnormality to the arrhythmias which can be confused with epilepsy, as exemplified in the paper of Dr G D Schott and others (4 June, p 1454). The Q-T interval syndrome,¹ or the long Q-T syndrome,² described by Romano and Ward as a heritable disorder, can occur without familial background occasionally, and the patient is threatened by serious rhythm disturbances.

We have recently seen a young woman who underwent a thorough neurological examination for having fits in her home a few weeks after delivery. Her ECG abnormality, prolonged Q-T interval with T-negativity in the right precordial leads, was not correctly interpreted at that time and its significance was recognised somewhat later, when an ECG could be taken during a syncopal attack: a short burst of ventricular tachycardia was seen. Emotional factors may play a role in provoking arrhythmia in patients with the long Q-T interval syndrome. The best treatment of this disorder is still open to question; beta-blockers are probably the most promising to prevent serious ventricular tachycardia or fibrillation.

Although this syndrome is regarded as a rarity, it has been recognised more often in recent years, and since young people are mainly involved its significance cannot be overestimated. If undetected and untreated it may have disastrous consequences for the patient.

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¹ Vincent, G M, Abildskov, J A, and Burgess, M J, *Progress in Cardiovascular Disease*, 1974, **16**, 523.

² Schwartz, P J, Periti, M, and Malliani, A, *American Heart Journal*, 1975, **89**, 378.

Anoxic seizures or epilepsy?

SIR,—The paper by Dr G D Schott and others (4 June, p 1454) is most welcome, drawing attention as it does to the neurological disguises of cardiac arrhythmias, disguises which may be even more difficult to penetrate in paediatric practice.^{1 2} But it is also a worrying paper, because it implies that neurologists still regard convulsions as epileptic, whether they occur in the child or the adult. Schott *et al* write that "any cause of cerebral anoxia may result in an unmistakable *epileptic seizure*" (my italics), but Sharpey-Schafer, to whom they refer, actually wrote "a full *epileptiform* convulsion is common . . . if a syncopal attack is severe and prolonged."³ Epileptiform attacks are those which look like epileptic fits but are not epileptic.⁴ If neurologists are still not recognising the difference between epileptic and anoxic seizures,⁵ then the vagal-mediated convulsive syncope,⁶ which is surely more common than are cardiac arrhythmias, will be regularly misclassified epilepsy. In paediatric practice it has long been known that ocular compression under combined EEG and ECG control may reproduce such reflex anoxic seizures, with cardiac standstill and EEG flattening.^{7 8} In order to monitor the ocular compression effect a single-channel ECG must be recorded along with the EEG routinely, and an occasional spin-off is the recognition of prolonged Q-T interval, bradycardia, and so forth. However, I accept the need for proper

cardiac investigation and Holter monitoring in all unexplained anoxic seizures.

Dr J H Pearn's survey of epilepsy and drowning in childhood (11 June, p 1510) is a valuable and encouraging contribution, but as a paediatrician he also appears to equate convulsions with epilepsy. Constrained by the Short Report format he does not give full details of each drowned child, but from what he says I suspect that the girl who died (case 8) had syncope when leaning over the bath and that her previous "idiopathic grand-mal epilepsy" was instead a tendency to reflex anoxic seizures.^{7 8} The search for a trigger mechanism in water-immersion seizures will be in vain so long as these are assumed to be epileptic.⁹ Anyone who reads Keipert's paper¹⁰ must conclude from his vivid descriptions that the triggered fits were anoxic; this could have been confirmed by combined ECG/EEG monitoring (with ocular compression) and if necessary prevented by atropine 0.01 mg/kg/day.¹¹ To imagine that febrile convulsions (cases 1 and 2) are of necessity epileptic rather than anoxic is more understandable because the Gastauts' evidence to the contrary⁶ has been ignored by most authors since.

Although once upon a time epilepsy encompassed any kind of seizure, syncope cut loose several centuries ago. Please let us keep it that way, and not blur the fundamental distinction between epileptic and anoxic fits.

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¹ Scott, O, Macartney, F J, and Deverall, P B, *Archives of Disease in Childhood*, 1976, **51**, 100.

² Radford, D J, Izukawa, T, and Rowe, R D, *Archives of Disease in Childhood*, 1977, **52**, 345.

³ Sharpey-Schafer, E P, *British Medical Journal*, 1956, **1**, 506.

⁴ Temkin, O, *The Falling Sickness*, 2nd edn, p 341. Baltimore and London, Johns Hopkins Press, 1971.

⁵ Gastaut, H, in *Handbook of Clinical Neurology*, vol 15, p 815, ed P J Vinken and G W Bruyn. Amsterdam, North-Holland Publishing Company, 1974.

⁶ Gastaut, H, and Fisher-Williams, M, *Lancet*, 1957, **2**, 1018.

⁷ Lombroso, C T, and Lerman, P, *Pediatrics*, 1967, **39**, 563.

⁸ Gastaut, H, and Gastaut, Y, *Electroencephalography and Clinical Neurophysiology*, 1958, **10**, 607.

⁹ Gastaut, H, and Tassinari, C A, *Epilepsia*, 1966, **7**, 85.

¹⁰ Keipert, J A, *Medical Journal of Australia*, 1972, **2**, 1124.

¹¹ Swaiman, K F, and Wright, F S, *The Practice of Pediatric Neurology*, vol 2, p 876. St Louis, Mosby, 1975.

Apples and the teeth

SIR,—Now that we have been able to share with the writer of your leading article (30 April, p 1116) the privilege of assessing the experimental data on the eating of apples after sugar¹ it is immediately evident that this work has been misquoted.

It is suggested that the final blow to the apple story has been given by showing that eating apples when plaque pH is already low after a sugar food does not lead to a protective rise in pH. In fact Dr Geddes and her colleagues have shown that in 13 out of 18 experiments the plaque pH did rise in response to apple eating and in less than a third did it fall. In the discussion part of their paper these authors suggest that apples may be slightly beneficial in stimulating a high salivary flow and thus buffering plaque acids produced from a previous sugary food, especially in subjects with a low plaque pH minimum—that is, those most at risk. At the same time they point out that in some subjects the sugar content of the apple may lead to a further fall in pH.

I do not think that too much should be read into these limited experimental data and such emotive statements as "not without hazard to the teeth" should be avoided until we have accurate clinical evidence that the reasonable consumption of apples does in fact damage teeth significantly. In the meantime I would suggest that the apple is still a fruit that can be recommended to children as a substitute for sugary snacks in the belief that it will do less harm than the latter to the teeth and indeed may do no real harm at all.

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¹ Geddes, D A M, *et al*, *British Dental Journal*, 1977, **142**, 317.

* * * There is no real conflict between the statements made in our leading article and the data referred to by Professor Winter. The slight rise (statistically non-significant) in plaque pH which occurred in 11 out of 16 (not 18) experiments could not be construed as "protective," especially as compared with the dramatic effects of peanuts described in the same article. The mild opinion expressed in our article, and shared by the authors in the article referred to, that the data suggest that apples "are not without hazard to the teeth" is immediately followed by the qualification that they are probably "not so damaging as other traditional dental enemies in the diet." The evidence may come from "limited experimental data" but, together with clinical evidence that excessive consumption of fruit, including apples, may be associated with dental erosion,¹ they do not lend support to Professor Winter's belief that apples "may do no real harm at all."—Ed, *BMJ*.

¹ Eccles, J D, and Jenkins, W G, *Journal of Dentistry*, 1974, **2**, 153.

Insurance companies' attitude to psychiatric illness

SIR,—As a member of the lay public I am probably guilty of some ethical breach by merely reading the *BMJ*, let alone writing to its Editor. Nevertheless, being engaged in the business of life assurance I feel constrained to do what I can to correct the dangerous advice which is implicit in the letter of Dr D T Maclay (4 June, p 1471). He seems to have encouraged patients of his not to disclose personal histories of "outpatient attendances for psychotherapy and perhaps for mild drug medication" in applications for life assurance. In doing so he could find himself partly responsible for prejudicing the validity of life assurance contracts entered into by his patients.

Insurance contracts generally are in certain respects quite unlike the vast range of ordinary commercial contracts. Leaving aside certain protections for buyers arising from the "consumer age" legislation of the last decade or so, the common law says that in an ordinary contract for a tangible good each contractor must look out for himself; the legal maxim is "let the buyer beware." Given the absence of bad faith on either side, both contractors are on an equal footing. In insurance contracts only one party, the "buyer" or applicant or proposer, knows all there is to know about the risk which he is asking the insurer to run. This is particularly so in life assurance, where the quality of the risk hinges upon factors such as personal