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

medical history and family history which do not permit of easy discovery by the insurance company.

The insurer's position of disadvantage has been recognised by the courts for something like 150 years by fixing the applicant with the duty to disclose all facts material to the risk which he knows or could reasonably be expected to know. This is a higher duty of disclosure than the "ordinary" good faith which applies to other transactions and is called *utmost* good faith. A "material fact" has been judicially defined as a fact which would affect the judgment of a prudent underwriter in deciding on the quality of the risk to be run. The legal remedy for non-disclosure of a material fact is that the aggrieved party (usually the insurance company) is permitted to avoid liability under the contract.

Dr Maclay probably knows all this full well. The point I urge him to consider is that the testing of a fact for its materiality is not a matter for the applicant or his medical adviser, nor indeed even for the insurer (although the latter must be considered a better judge). Whether or not a fact is material as defined is ultimately a matter for the courts alone. Thus Dr Maclay has to be sure that, say, any random High Court judge would agree that a personal history of "outpatient attendances for psychotherapy and for mild drug medication" was not a fact that would "affect the judgment of a prudent underwriter." This particular point, so far as I know, has never been tested and there is no precise legal precedent. With all respect to Dr Maclay he really should not take upon himself the judge's role.

As it happens, I incline to agree with him that the kind of personal history he quotes should not disqualify an applicant from obtaining life assurance on special terms, or even perhaps ordinary terms according to individual circumstances. But as an underwriter I would sooner *know* about such a history and make up my own mind about the quality of the risk. Better by far is it for Dr Maclay to advise his patients to make a full disclosure even of psychiatric histories he regards as trivial and then seek a life office—there are enough of them—which is prepared to take a "sensible" view.

I should not like to leave anyone with the impression that life offices frequently seek solutions in the courts to problems arising from non-disclosure. You can, Sir, count on your thumbs the number of times in the last 15 years that life offices have gone into court to seek to avoid liability following breaches of utmost good faith. What I do say is that an applicant ignores this important principle only at his peril.

Private medical attendants could help their patients, especially those with personal histories of physical ailments, by taking care over the completion of reports requested by life offices. Overworked GPs have no time to write essays, of course, yet a sketchy report grudgingly given can do great disservice to an anxiety-prone patient, whose psyche may be further disturbed by an underwriting decision that could have been more favourable on better evidence.

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Suicide and life insurance

SIR,—The consequences for life insurance of mental illness have been the subject of recent correspondence. A related matter is the approach adopted by life insurance companies to suicide. That suicide precludes payment on a life insurance policy seems to be a widely held belief. The purpose of this letter is to make known some facts bearing on this point

and to propose that the time has come to abolish insurance penalties for suicide.

A booklet published by Stone and Cox¹ summarises the policy conditions of 100 life insurance companies; 52 of them have suicide exclusion clauses, with varying time restrictions. For 26 companies it is one year, for 16 13 months, and for 10 companies two years. The payment to which the excluding companies commit themselves in the event of suicide varies. Twelve companies undertake to refund the premiums paid or the surrender value of the policy, three indicate that payment will be less than the full amount, and one states that all premiums will be forfeited; the remaining 36 simply say that the policy is voided. This presumably does not preclude discretionary part payments. Most companies with exclusion clauses state that suicide within the defined period renders the policy void except for third-party interests. We understand this qualification to mean that policies used as cover for some commercial transaction are not subject to the exclusion clause and are thereby protected.

As part of our inquiry to ascertain the effects of conjugal suicide on 44 surviving spouses^{2,3} we included questions concerning life insurance. Of the 44, 20 were insured, 13 were not, and in 11 cases we could not ascertain whether the life had been insured. Some payment was made in all 20 cases known to have been insured; in six of the 20 (30%) a proportional sum was paid and in one case the widow could not remember whether she had received the full amount or a proportion.

Suicide exclusion clauses are presumably inserted to discourage or prevent a fraud on the insurance company. Does the law not already provide a remedy whereby payment can be refused on proof of such a fraud, making suicide exclusion clauses unnecessary? The 45 companies which do without a suicide clause must resort to such means of protecting other policyholders. The only other explanation for suicide exclusion clauses is that such clauses are an expression of moral disapproval of suicide, surviving from the days when suicide was a crime. It is of interest that five companies also exclude death "at the hands of justice."

Fraud by suicide must be excessively rare in England. A broad provision to avoid the necessity of detecting a rare event may have unfair financial consequences for the families of suicides, and for that reason we believe all life insurance companies should now follow the lead of the large minority and remove suicide exclusion clauses from their policies.

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¹ *Life Assurance Policy Conditions*. London, Stone and Cox, 1976.

² Shepherd, D M, and Barracough, B M, *British Medical Journal*, 1974, 2, 600.

³ Barracough, B M, and Shepherd, D M, *British Journal of Psychiatry*, 1976, 129, 109.

Serological test for tuberculosis

SIR,—I would like to comment on the paper "Evaluation of a serological test for tuberculosis" by Dr D A Mitchison and others (28 May, p 1383).

Firstly, I would agree that the results obtained reflect the true titre of agglutinin antibody and cannot be explained on the basis of technical differences between the respective laboratories.

Secondly, the finding that there is no relationship between agglutinin levels, Man-

toux reaction, or BCG vaccination history is consistent with our own experience.

Thirdly, it is undoubtedly true that the agglutination test will not provide a yes-or-no answer in the diagnosis of tuberculosis. For the past 12 months in this laboratory titres have been correlated to culture results and tables showing "probability of culture-positive disease" generated. These have been distributed to laboratories requesting serology as an aid to the interpretation of results. Although disease has been found in patients with all levels of titre, it is apparent that the probability of disease rises with titre. In an analysis of the first serum sample received from each of 8000 patients it is apparent that some significance still attaches to the value of 1/120, though 44% of patients at that level do not have tuberculosis (see table). Analysis

Correlation between mycobacteria isolations and agglutinin titre

Titre	No	No with positive culture	% With positive culture
2400 (2500)	28	28	100
1200 (1250)	27	25	93
600 (500)	119	103	87
300	378	321	85
240 (250)	419	326	78
120 (125)	1980	1116	56
60 (50)	2403	48	2
30 (25)	2207	25	1
<30 (<25)	400	1	0.25
Totals	7961	1993	

has also revealed that women have higher titres than men and that the majority of false-positive results occurred in patients with elevated IgM levels. This information can all help in the interpretation of a result, but eventual assessment of the test must depend on the co-operation of requesting centres in supplying details of culture results and diagnoses. This information is obtained for just over 70% of sera sent to Midhurst, and our analysis figures naturally exclude the other 30% "unknown" quantity.

The study reported by Dr Mitchison and his colleagues refers to an assessment of the test in Hong Kong patients. Any attempt at correlation of these figures with the Midhurst figures obtained from patients in Britain fails to recognise the influence of disease prevalence and ethnic origin on the validity of any serological test. Indeed, the lack of correlation is to be expected; Professor J G Cruickshank, working in Rhodesia, was unable to record titres below 1/120 in any Black Rhodesian worker, while great differences exist in the titres observed in the new immigrant and second-generation immigrant of this country.

It is also of some relevance that our figures refer to culture-positive disease with *Mycobacterium tuberculosis*. It is possible that smear-positive patients are affected with other mycobacterial strains, and this may account for some of the seronegative results. Titres in contact patients are often elevated, and this may well reflect contact with the antigen. Titres in our experience, however, fall rapidly in these patients, and it is unfortunate that the Hong Kong group were not reassessed serologically as well as radiologically. It is disappointing that Hammersmith patients group 1, who more accurately reflect the type of sample received at Midhurst, were not subjected to closer analysis in terms of diagnosis. I am unable to perform this analysis