Sudden death

Sudden unexpected death has frightened laymen and puzzled doctors for centuries. An apparent epidemic of sudden deaths led Pope Clement XI to ask Lancisi to find out their cause, and in De Subitae Mortibus the latter suggested that many were due to heart disease.1 When the clinical syndrome of myocardial infarction was recognised early this century2 doctors found that patients with this condition could die suddenly. The medical world then came to assume that because some patients with proved infarction died suddenly all patients with sudden unexpected death must have had underlying infarcts. When no cardiac necrosis could be found at necropsy, this was explained away by suggesting that the patient had died before histologically recognisable tissue changes had occurred.

The advocates of this theory also ignored the differing patterns of coronary artery disease in the two conditions: whereas 80-90% of hearts with transmural myocardial infarction have occlusive coronary thrombi,4 5 in cases of sudden death without cardiac necrosis no coronary thrombi are found.4 6 A pathologist is reluctant, particularly if he has to testify in court, to admit that he does not know what caused sudden death. Epidemiologically, this has led to great confusion, since labels for which there is no evidence have come into widespread use. As Crawford has pointed out, pathologists, being under pressure to give definite and immediate answers, often use ischaemic heart disease, coronary thrombosis, or some synonym as a convenient explanation for deaths that the necropsy has failed to explain.7

Quantitative methods for studying coronary artery and heart muscle disease should, however, give us a better understanding of sudden death.4 Other useful information is now beginning to emerge from clinical studies. In Seattle, Cobb and his colleagues8 have taught over a quarter of their population to keep victims of "sudden death" alive by cardiopulmonary resuscitation until highly trained back-up teams can intervene. During six years some 650 of these patients survived to leave hospital; when they were subdivided into those who developed signs of myocardial infarction during their convalescence and those who did not, an important difference in the pattern of death emerged. The patients who had no evidence of infarction had a much higher death rate (40%) at two years than those in whom "sudden death" occurred as part of the syndrome of infarction. This suggests that the mysterious mechanism that produced their initial episode had continued to operate and to place them at high risk.

If thrombotic coronary artery occlusion does not account for the non-infarct sudden-death syndrome, then what does? A profile of the underlying processes is beginning to emerge: severe disease of the walls of many of the coronary vessels9 10 and large hearts, scarred by previous myocardial infarction,11 seem especially common. Severe disease of the walls of subendocardial arteries is also a characteristic of patients with short-lived strokes (transient ischaemic attacks),12 where microembolism is a suggested explanation for the evanescent hemisphere or eye symptoms. The plaques of the neck arteries may be generating microthrombi, most of which are so small that they can traverse the cerebral circulation without any effect; but when one affects a vessel in the eye the clinical consequences are dramatic. Could microembolism from severe coronary disease be similarly traversing the coronary circulation with no discernible effect unless one hits a vessel supplying the rhythm-generating and conducting tissues? Platelet microthrombi in the coronary arteries have indeed been found after sudden death.13 If such patients were to be resuscitated, would they remain at risk from continuing microembolism, as suggested by the Seattle findings?8

If we could find drugs to modify this process we could improve the unfavourable natural outcome. Of the agents that can alter the behaviour of platelets, sulphinpyrazone (Anturan) appears to reduce the incidence of sudden death in those who have previously had infarctions.14 We need trials both to confirm this result in those who have survived an infarction and also to assess the effect of sulphinpyrazone and of aspirin15 and dipyridamole16 in the survivors of Seattle-type resuscitation procedures who have not had infarcts. If continuing microembolism puts them at risk from recurrent "sudden death" then the analogy with cerebral transient ischaemic attacks may provide a clue to treatment. Above all, we must now accept that unexpected sudden death and death after cardiac infarction are pathologically and clinically distinct entities that may need different preventive and therapeutic measures.

3 Parkinson, J, and Bedford, D E, Lancet, 1928, 1, 4.
11 Haerem, J W, Atherosclerosis, 1972, 15, 199.

Reliability and reversibility of female sterilisation

Younger women with smaller families are now asking for sterilisation. Economic pressures, adverse reports of the effects of the pill, and public awareness of improvements in sterilisation techniques have all contributed to this trend. Sterilisation has always been considered irreversible, but more recently attempts have been made to develop methods that are at least potentially reversible and which require minimal time in hospital. In a society with a younger sterilised population and a relatively high incidence of marital instability both reliability and reversibility will assume growing importance.
Before laparoscopy became fashionable in Britain sterilisation was generally performed by laparotomy. The failure rate of the Pomeroy operation using absorbable suture material was about 2 per 1000. The vaginal approach by culdoscopy and culdotomy never had the same popularity, and recent reports have again indicated its limitations.¹

The new technique of laparoscopy required the development of effective methods of occluding the tubes. Reliance on poorly controlled high-frequency unipolar electrical current has gradually been replaced by the more controlled bipolar system or even thermocoagulation, but debate continues about the optimal amount of tube requiring destruction and the relative merits of division or resection or both. Resection has been associated with higher complication rates and in one review² the pregnancy rate was 4.3 per 1000. Even bipolar coagulation is excessively destructive when several sites are coagulated. A surgeon attempting reversal after a Pomeroy operation finds a relatively clean surgical wound; he may find quite the opposite after electrocoagulation, when even microsurgery may be of little help if there is extensive tissue destruction. Add to this the other complications of electrocoagulation such as contact burns and its continued use becomes difficult to justify when less destructive and equally effective methods are available.

Of these alternatives, the most popular are the various occlusive devices. The tantalum (Wick) clip gave an unacceptable pregnancy rate of 7 per 1000 from tubal fistulous communications, and the results of early studies using the Hulka-Clemens spring-loaded clip showed a similar failure rate due to faulty manufacture. Nevertheless, those made in Britain (Rocket clip) have given a failure rate of only about 2 per 1000 and they destroy only 0·3 cm of tube. They have one advantage over a new snap-shut plastic clip (Hug): the applicator has a safety catch mechanism allowing removal before final closure if placement is in question. The silicone (Falope) ring causes infarction and obliteration of the lumen of the 2·3 cm of tube ensnared in the ring. Traumatic injuries to the tube and mesosalpinx have resulted from the need to draw a loop of tube into the instrument delivering the ring, but such complications have proved less frequent as operators gain experience. The failure rate is comparable with that of the Rocket clip. Successful reversal after the use of such devices has been reported.³

Any study of the reliability of a method of sterilisation, however, should take into account all the variables. These include operator experience, timing of the procedure, the route and technique used, the means of achieving occlusion, the site of tube selected, and the method of analysing results. Furthermore, as a recent report from Singapore has emphasised, expressing failure rates as the ratio of pregnancies to the total number of operations takes no account of the time of exposure.³ Pregnancies are most likely in the first two years, with a peak between three and six months, and should be expressed on a life-table cumulative basis.¹ Twelve-month life table pregnancy rates on data from 60 different centres have shown identical rates of 2 per 1000 for laparoscopy (using electrocoagulation or devices) and minilaparotomy. Ectopic pregnancies are most likely with electrocoagulation, with a rate varying from 19/0 to 59/0.⁶

There is now much to recommend effective procedures which give a good chance of reversibility. The narrowest isthmic portion of the tube is the best choice for both closure and reversibility. In underdeveloped societies minilaparotomy using a Pomeroy operation or a device probably has advantages over laparoscopy. Vaginal procedures are technically more difficult and may also limit accessibility to the isthmus. In Britain laparoscopy seems likely to remain the most popular approach, with laparotomy being reserved for sterilisation at caesarean section and in the immediate puerperium (some obstetricians believe an elective laparoscopy at six weeks is preferable to take account of the possibility of unexpected infant death). Now that devices have proved as effective as electrocoagulation or surgical occlusion they can be recommended by any route, and preference should be given to those destroying the least tissue. Whether operations should be performed under local or general anaesthesia or in inpatients or outpatients depends upon the type of case and the resources available. Even so, we should certainly make more use of day surgery.


Recurrent vaginal candida infection

Why is vaginal infection with Candida albicans and other yeasts so often recurrent? Candida infection, of course, is the most common cause of vaginitis;¹ it may lie dormant in the vagina without, or perhaps before, causing symptoms,² and intercourse and relapse are often related. The sexual route of transmission has been recognised for many years,³ but the physical effects of intercourse may also play a part by transferring yeasts from the adjacent epithelium or by damaging the vaginal mucosa. Statistical evidence suggests that yeast infection may often be acquired sexually,⁴ but frequent events will also occur together frequently by chance.

Many factors may increase the susceptibility of the vagina to yeast infection,⁵ but being aware of them rarely helps the doctor to deal with the practical problem. Autoinfection from the bowel is well known,⁶ and yeasts were detected in every faecal culture in a more recent series of patients with recurrent vaginal infection.⁷ We have no evidence, however, that simultaneous treatment of the bowel will prolong freedom from recurrence of the vaginal infection or, indeed, that the bowel itself will not be rapidly recolonised. Whether the patient’s susceptibility or some environmental factor is the main problem, in practice the effective solution usually is intermittent prophylactic treatment.

In a recent study of 40 recurrent cases Davidson and Mould⁸ compared prophylactic clotrimazole with a placebo. Whereas the group receiving clotrimazole had fewer symptoms subsequently, there was no difference between the two in the frequency with which candida alone was isolated. The authors suggest that the symptoms were not the result of candida alone and point out that 70% of the patients and their male partners had non-specific genital infection; a similar finding was reported by Rohatiner.⁹ Furthermore, when the trial ended after four months patients who proved free from candida had a recurrence of symptoms. In other words, there was a complete dissociation of symptoms and candida infection.