Hypothesis for low-energy transthoracic defibrillation

Sir,

The editorial on ventricular defibrillation (Adgey, *British Heart Journal*, 1978, 40, 1197-1199) is both timely and informative. The Belfast group has a prodigious experience in out-of-hospital resuscitation and their prospectively collected data have been invaluable in determining energy requirements for defibrillation. The evidence indicating the need for defibrillators storing more than 400 Ws is, at present, inadequate and unconvincing (Lown *et al.*, 1978).

In addition to the views expressed by Dr Adgey, I would like to submit some theoretical considerations which may help to explain the surprising efficacy of low energy shocks. The proponents of high energy defibrillation espouse the view that as body weight increases, the energy required to defibrillate increases, presumably, in a fixed mathematical manner. They have suggested that a log-dose-response curve based on body weight exists for ventricular defibrillation (Geddes *et al.*, 1974; Tacker *et al.*, 1974). This argument presumably presupposes, as did Wiggers in 1940, that complete depolarisation of the heart is necessary to terminate ventricular fibrillation. Though the animal studies are convincing, it is not clear that such a condition needs to be met to effect defibrillation clinically.

Evidence of the latter view derives from more recent studies involving intracavitary catheter defibrillation studies in animals and in man (Mirowski *et al.*, 1973; Ewy *et al.*, 1978). In this model, energies as low as 4 Ws may terminate ventricular fibrillation. It is extremely unlikely that such low energies are capable of depolarising the entire myocardium. Zipes *et al.* (1975) have also shown that selective infusion of potassium chloride into coronary arteries can also terminate ventricular fibrillation. They postulated that segmental depolarisation of the myocardium occurs with such selective infusions. It is likely that, with both methods, a critical mass of myocardium is temporarily disengaged by depolarisation from the remainder of the fibrillating heart. This effect is evidently adequate to abort ventricular fibrillation as the multiple re-entering wavefronts encounter either refractory or partially refractory tissue. There is no basis for making an *a priori* assumption that this effect relates to body weight. Even if body weight or heart weight were a factor, it may be expected that much lower energies than are required to depolarise the entire heart would suffice to

defibrillate it. If a similar mechanism operates during transthoracic defibrillation, it would account for the success of low-energy defibrillation.

In view of the clinical data provided by the Belfast (Pantridge *et al.*, 1975; Campbell *et al.*, 1977) and the Virginia (Crampton and Hunter, 1976; Gascho *et al.*, 1979) groups, it is necessary to evaluate critically the reasons for failure during defibrillation. It is likely that the most egregious miscreant is the user and not the device. It is distressing to see how frequently electrodes are malpositioned on the chest during defibrillation. It was, I believe, Dr Samuel Johnson's acerbic wit which informed us, in another connection, that a cigarette, 'is a device with a fire at one end and a fool at the other'. Each improper discharge of the defibrillator provides an opportunity to paraphrase this redoubtable sage.

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Editorial notice

References

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Full details of this new, uniform style, which was agreed at a meeting of the International Steering Committee of Medical Editors held in Vancouver, were published in the *Br Med J* (1979; **1**: 532–35) and *Ann Intern Med* (1979; **90**: 95–9). Reprints of this article are available from the *Br Med J* (price 50p).