

to the underlying disease than to the treatment, and in these sick children risks must be taken. In supplementary or short-term parenteral feeding, however, unnecessary risks are unjustifiable. Careful monitoring and strict aseptic control may reduce such risks to a minimum, but in every case the advantages of the treatment must clearly outweigh its hazards.

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Learnt voluntary control of heart rate and rhythm

Normal people become aware of the autonomic control of the heart and blood vessels only when this becomes extreme, when it has ceased to be purely automatic—for example, tachycardia of fear or the hypotension of a faint induced by the sight of blood. Nevertheless, there are so many ways that this automatic regulation can be reset by the interference of other parts of the nervous system—the fall in heart rate and blood pressure during sleep, the rise while doing mental arithmetic—that it is autonomic only in the sense that it is not usually under direct conscious control. Since activity in the higher centres so profoundly influences the control systems, many investigators have speculated that these could be brought under voluntary control if the individual was provided with a signal recording the state of the system. The use of such a signal has become known as biofeedback.

We now have convincing evidence that trained individuals can achieve small, short-term changes in both heart rate and blood pressure if they are provided with a visual or auditory signal to show them the results of their efforts. Weiss and Engel¹ first used biofeedback to treat a group of eight patients with cardiac arrhythmia, five of whom learnt to decrease the frequency of the abnormal beats. More recently Pickering and Miller² used the technique to investigate and treat two patients with frequent premature ventricular contractions. They justified the time spent on this approach on two grounds:

firstly, that the drugs commonly used to treat premature ventricular contractions are potentially dangerous myocardial depressants; and, secondly, that drug treatment is inappropriate because of the evidence that the nervous system can influence the onset and disappearance of premature ventricular contractions.³ Certainly the frequency of premature beats falls during sleep and increases during psychological stress.^{4 5}

Their first patient had bigeminal rhythm and was provided with an oscilloscope signal from a cardiac monitor. Gradually, over 25 training sessions spread over several months, he acquired some conscious control. Eventually he was in sinus rhythm for 3% of the time when he was just resting and 27% of the time when he was trying consciously to influence the rhythm. This patient's suppression of bigeminal rhythm was associated with an increase in heart rate, though it was not wholly due to this change. The second patient had premature ventricular beats and was provided with a ratemeter signal. He quite quickly learnt to increase his heart rate by 20-25 beats minute by conscious effort. Once he achieved a sinus rate of about 117 beats minute the arrhythmia was usually suppressed.

The use of biofeedback to control cardiac arrhythmias raises two questions. Firstly, how does the patient achieve the effect? Secondly, is the effect large enough and well enough sustained to be of any therapeutic value? In both the cases described an increase in rate seems to have been important, though not the sole explanation. Clearly if the method is to be of practical value the patient must be able to apply the training he has received in the laboratory with the biofeedback signal when he no longer has that signal available. The second of Pickering and Miller's patients seemed to be able to do this, but the first could not. Possibly, too, the increase in the heart rate achieved to suppress arrhythmias might have had other adverse consequences if it had been maintained over long periods. Just as investigators who make therapeutic claims for new drugs have to prove their safety and efficacy in long-term use, so must advocates of biofeedback.

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Motorcyclists' injuries and crash helmets

Motorbikes are dangerous: a motorcyclist is eight times more likely to be fatally injured than the occupant of a car per unit distance travelled.¹ Despite this, the number of motorcycles on our roads is rising and probably will continue to do so as the cost of transport goes up. Accompanying the increase in motorcycle use the Royal Society for the Prevention of Accidents² has noted an increase in the number of casualties among users of "powered two-wheelers" in the first nine months of 1976—part of a general upward trend over the last five years. The implications of this trend are serious, affecting not only motorcyclists but also other road users; clinicians concerned with patient care; design engineers of safety equipment, such as crash helmets; and the rest of the community, who share the costs (nowadays put at around