HALOTHANE SENSITIZATION A CASE REPORT

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THERE HAVE BEEN several cases of anaesthetists having become sensitized to halothane as a result of the inhalation of the vapours discharged from the gas machine. The cases so far have all involved the liver and resulted in decreased liver function after exposure. The following case report involves myself, and in this case the organ involved is the heart.

I was born in 1914, and have done a fair amount of anaesthesia since 1947. My health has always been good; there have been no known allergies in self or family. For several years I have had occasional attacks of cardiac arrhythmia. These were self-terminating and the nature undiagnosed at the time. However, in the Autumn of 1965, this change in cardiac rhythm appears to have failed to terminate itself, as a result of which I suffered from dyspnea on exertion. In the Spring of 1966, I had a cardiogram done and discovered that I was in auricular fibrillation. This was reversed with quinidine after a trial of digitalis, and once reversed remained fairly stable. I had attacks of fibrillation once or twice a month and occasionally at longer intervals. These attacks were easily reversed by quinidine (200-400 mgm) and exercise tolerance while in sinus rhythm was very good. I discovered that too much tea would provoke an attack, but no other precipitating causes were discovered at this time. Quite suddenly in the autumn of 1969 I began having attacks of fibrillation nearly every day and sometimes twice in one day. These attacks required more quinidine than heretofore for reversal.

At the beginning of 1970, I accordingly went to see a cardiologist, who on investigation could find no evidence of arteriosclerosis, hyperthyroidism or other predisposing cause. He confirmed a good exercise tolerance while in sinus rhythm. About this time, I noticed that whenever I was away from anaesthesia for a few days, i.e. over Christmas or a long weekend, things improved. I naturally suspected halothane, which we started using in our hospital in 1963, so I switched to using methoxyflurane instead. Attacks of fibrillation ceased. Holidays followed this and during the subsequent two months I had only one attack which occurred on top of a 9,000 foot mountain which I had climbed.

On returning from holidays, I gave methoxyflurane for three weeks and then tried using halothane again. After two busy days, I got an attack of fibrillation at 4 a.m. which needed a larger total dose of quinidine than average to reverse. Since then I have used no more halothane and had no more attacks. I think there are two lessons to be drawn from this experience. First, cases of paroxysmal arrhythmia with no apparent cause may be due to chemical sensitization. Second, that as we are really very ignorant of the effects of long continued exposure to small amounts of anaesthetic agents, it is foolish to continue to allow the effluent from gas machines to blow off into the air of operating rooms, especially as operating rooms are seldom well ventilated. The effluent could easily be led outside.