VENTRICULAR FIBRILLATION DURING CEREBRAL ANGIOGRAPHY UNDER HALOTHANE ANAESTHESIA

Report of a Case

BY

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SUMMARY

Ventricular fibrillation supervened during cerebral angiography under halothane anaesthesia in a patient with a cerebral tumour related to the hypothalamus. Angiography was later carried out successfully under local analgesia. This was followed five days later by resection of the tumour under general anaesthesia. On this occasion halothane was not used.

A man aged 49 was sent for percutaneous cerebral angiography under general anaesthesia. He had bilateral papilloedema, paralysis of the external rectus and superior oblique muscles of the right eye, and was mentally sluggish. No abnormalities were found in his heart or lungs; his blood pressure was 130/80 mm Hg; a stool test suggested that he might be a porphyric, a condition not uncommon here. He had been given atropine 1.2 mg half an hour previously.

Thiopentone being contraindicated by the possibility of porphyria, anaesthesia was induced using nitrous oxide and oxygen with a flow rate of 5 and 2 l./min, supplemented by 0.5 per cent halothane from a Fluotec vaporizer, the concentration increasing gradually to 3 per cent. The second stage was marked by breath-holding, muscular rigidity and some struggling. When this ceased three doses of suxamethonium, each of 25 mg, were given at about 30-second intervals; the cords and trachea were sprayed with 4 per cent lignocaine and a nylon endotracheal tube inserted. Ventilation was assisted for about 2 minutes until it returned to normal. The concentration of halothane was then reduced to 2 per cent. At no time was cyanosis or other evidence of anoxia noted. The pulse remained regular, about 88/min, and the blood pressure was not palpably altered.

Two ml of 1 per cent procaine without adrenaline was injected round the common carotid artery. Before a syringe could be connected to the needle, blood ceased to flow back. Simultaneously the patient inspired deeply and the radial pulse became impalpable. Immediate thoracotomy was performed and the heart was seen to be in ventricular fibrillation. Normal rhythm was restored with some difficulty after 1 hour.

Six days later an electrocardiogram was taken and Dr. Geoffrey Dean reported: "... The chest lead shows inversion of the T wave, showing definite anterior myocardial damage but this is not the picture of a recent coronary thrombosis." On the eighth day angiography was carried out under local anaesthesia; there was no significant alteration in pulse rate or blood pressure during the procedure. On the thirteenth day a frontal lobe tumour the size of half a tennis ball was resected at an operation lasting 4½ hours. The tumour, a glioblastoma multiforme, was situated in the posterior part of the floor of the anterior fossa in the middle, and its posterior part was related to the hypothalamus. Anaesthetic agent used was paraldehyde and nitrous oxide with oxygen, d-tubocurarine being given in addition. No adrenaline was used. During the first half-hour there was a rise in systolic pressure from 110 to 190 mm Hg and a moderate tachycardia. This was followed by a slow fall to 140 mm Hg and during the resection of the tumour both pulse rate and blood pressure remained steady.

DISCUSSION

It is improbable that a coronary thrombosis caused the arrest. The patient did not at any time show
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signs of thrombosis; he withstood the craniotomy well; he has not since complained of anginal pain and the electrocardiographic report was negative. The “definite anterior myocardial damage” could well have been due to prolonged massage and repeated use of the defibrillator.

Raventós (1956) has shown that halothane sensitizes the myocardium to the action of adrenaline and it is recognized that administration of adrenaline during halothane anaesthesia can cause cardiac arrest. Two cases have recently been reported by Rosen and Roe (1963). The work of Black et al. (1959), showing that halothane, unlike chloroform, does not increase the release of catecholamines, suggests that induction of anaesthesia and surgical stimulation during light anaesthesia may be safer with halothane than with chloroform, and this assumption is supported by the few cases of arrest in these circumstances as yet reported. One case of ventricular fibrillation, occurring immediately after a stormy anaesthetic induction of pressure on the carotid sinus are said by Rovenstine and Cullen (1939) to be enhanced by chloroform and repeated use of the defibrillator. The heart in this case, however, was in fibrillation, not asystole, and it persistently returned to fibrillation when massaged after asystole had been produced by the defibrillator.

It seems probable that endogenous adrenaline acting on a sensitized heart, rather than vagal inhibition, is likely to have been the cause of arrest, but whichever mechanism was responsible halothane appears to have been a factor, for in its absence angiography was done without ill effect and also in its absence induction of anaesthesia led only to hypertension and tachycardia.

REFERENCES


FIBRILLATION VENTRICULAIRE AU COURS D'UNE ANGIOGRAPHIE CÉRÉbraLE FAITe SOUS ANESTHESIE PAR HALOTHANE

SOMMAIRE

Chez un patient atteint de tumeur cérébrale en rapport avec l'hipothalamus, une fibrillation ventriculaire survint au cours d'une angiographie cérébrale sous anesthésie à l'Halothane. L'angiographie fut néanmoins réalisée plus tard sous anesthésie locale. Elle fut suivie cinq jours plus tard de résection de la tumeur sous anesthésie générale — à cette occasion l'halothane ne fut pas utilisé.

VENTRIKULÄRE FIBRILLATION WÄHREND ZEREBRALER ANGIOGRAPHIE UNTER HALOTHAN-ANAESTHESIE

ZUSAMMENFASSUNG

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