VENTRICULAR FIBRILLATION DURING HYPOTHERMIA SUCCESSFULLY TREATED BY REWARMING AND ELECTROSHOCK

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Ventricular fibrillation is one of the major complications which may arise during induced controlled hypothermia. In the absence of cardiac manipulations this condition may arise spontaneously at temperatures below 25°C, but even at temperatures around 28°C ventricular fibrillation often follows intracardiac manipulations (Bigelow, Lindsay, Harrison, Gordon, and Greenwood, 1950).

Various methods have been applied to prevent and treat ventricular fibrillation. Bigelow and his colleagues, operating on dogs, realized the importance of maintaining a normal blood pH, and attempted to obtain this by adding 5% CO₂ to the anaesthetic mixture. Cookson, Neptune, and Bailey (1952) found that adding CO₂ was of no importance. They found that adrenaline often produced ventricular fibrillation in dogs during hypothermia, but that by using the adrenolytic substance “benodaine” the onset of ventricular fibrillation was prevented when adrenaline was necessary for the treatment of cardiac arrest.

Swan, Zeavin, Holmes, and Montgomery (1953) demonstrated experimentally the importance of hyperventilation and its accompanying high blood pH in the prevention of ventricular fibrillation. This has been confirmed by many other investigators.

Niazi and Lewis (1956), during experiments on dogs, confirmed that spontaneous ventricular fibrillation did not arise so frequently when normal blood pH was maintained by the addition of 5% CO₂.

Prevedel, Montgomery, and Swan (1954) described the use of intravenous prostigmine for the prevention of ventricular fibrillation, and state that even if the condition is established the use of this drug will increase the chances of successful defibrillation.

Ribert (quoted by Shumacker, 1955) suggested blocking the sino-auricular node with procaine in order to prevent the onset of ventricular fibrillation, and the effectiveness of this was confirmed by the work of Radigan and Morrow (quoted by Schumacker, 1955).

Senning (1955) produced ventricular fibrillation deliberately in intracardiac operations in order to lessen the incidence of pulmonary air embolism. Senning and Kaplan (1956) have published experiments which indicated that the use of intravenous alcohol gives a certain protection against ventricular fibrillation arising during hypothermia, and also makes electroshock therapy more effective.

It has been generally recommended that in electroshock treatment current values of 2 amperes and 200 volts in 0.1 second should be used for ventricular defibrillation. Swan and Zeavin (1954) prefer to inject potassium into the aorta just distal to the aortic valve, then after clamping the descending aorta perform cardiac massage, so forcing the potassium into the coronary circulation.

Senning recommended the following practice in the treatment of ventricular fibrillation: cardiac massage until the tone of the heart muscle is good, then 25 mg. procaine per kg. body weight is injected into the right auricle, followed by 30 μg. per kg. body weight of adrenaline half a minute later. Then after a further two minutes the defibrillator is used with a current of 2.5 ampères in 0.1 second.

All authors agree that rewarming should be carried out as speedily as possible, because it is easier to defibrillate a normothermic heart. Diathermy is a practical method of carrying this out.

Brock and Ross (1955) use veno-venous cooling, and therefore can rewarm the patient quickly if ventricular fibrillation arises.
PERSONAL OBSERVATIONS

Recently we have seen three cases of ventricular fibrillation during intracardiac operations performed under induced hypothermia. These were successfully treated by electroshock therapy combined with rapid rewarming of the patient by pouring hot saline solution into the thoracic cavity. Hypothermia had been induced in all cases by surface cooling.

After the induction of anaesthesia the patients were covered by thin wet sheets, and plastic bags containing crushed ice were placed on and around the body. Cooling was made more effective by pouring ice water over the patients and turning on to them an electric fan. The temperature was recorded in the rectum and oesophagus with a thermocoupler (model "electrolab"). The electrocardiogram was checked at intervals. The anaesthetic varied in the three cases, but all patients were intubated and hyperventilated.

CASE 1.—This was a 17-year-old girl with an atrial septal defect (Fig. 1). Anaesthesia was induced and intubation was accomplished with intravenous barbiturate ("enbomal natrium" (300 mg.)) and succinylcholine (100 mg.). Anaesthesia was maintained with N₂O/oxygen and relaxation was obtained by the administration of "laudolissin" (40+10 mg.). Intravenous "phenergan" was given to produce peripheral vasodilatation, and thus accelerate cooling.

The atrial septal defect was closed by the Søndergaard-Bjørk circular suture (circumclusion) and ventricular fibrillation started immediately following the tying of the suture. Without delay cardiac massage was started and details of further treatment are shown in Fig. 1. When electroshock and various injections were ineffective rewarming was started by pouring hot saline at a temperature of 40°C. into the open thoracic cavity; in all 125 litres were used. The heart muscle tone remained excellent throughout the whole procedure. As may be seen from the temperature curve, the oesophageal temperature rose rapidly while the rectal temperature remained constant. When the temperature in the oesophagus had risen to 34.2°C. electroshocks resulted in the heart regaining its normal rhythm. The heart had then been in ventricular fibrillation constantly for one hour and

Fig. 1.—Chart showing events during operation of Case 1. Rectal temperature O-O, oesophageal temperature ——, systolic blood pressure V, diastolic blood pressure A, pulse ——. E. S. = electric shock and figures in brackets are the numbers of shocks given.
CASE 2.—A 10-year-old girl was admitted with a diagnosis of an isolated pulmonary stenosis. Anaesthesia was induced and intubation was accomplished by N₂O/₃/O₂ and cyclopropane. Anaesthesia was maintained by cyclopropane and oxygen with 20 mg. "laudolissin" for relaxation. Cooling was uneventful.

Operation was begun and the thorax and the pericardium opened. It was intended to isolate the main pulmonary artery from the aorta in order to clamp the pulmonary artery, and also to insert a circular suture around the pulmonary conus, which could then be closed by a tourniquet. During the attempts to isolate the pulmonary artery the posterior wall was damaged and forceps had to be applied to the perforation interrupting the circulation. Then the circular suture around the conus was inserted rapidly and a clamp applied to the artery distal to the perforation. Arteriotomy was now performed and the pulmonary valve partially excised with scissors under open vision.

Five minutes after the circulation had been interrupted ventricular fibrillation began. Cardiac massage was delayed until 10 minutes 50 seconds following the interruption of the circulation, because both the perforation and the arteriotomy had to be closed before the circular suture and the clamp could be removed. When repeated use of the defibrillator had proved ineffective rewarming was begun by pouring hot saline into the thoracic cavity. The oesophageal temperature rose rapidly, and when 32.4°C. were registered defibrillation was successful and normal rhythm was resumed. The heart had been in ventricular fibrillation for 50 minutes, and 65 litres of saline had been poured into the chest. In this case no medicaments had been injected because the tone of the heart had remained excellent during cardiac massage. This patient also awoke without psychic disturbances, and her post-operative recovery was uneventful.

CASE 3.—This was a 23-year-old man, with a diagnosis of isolated pulmonary stenosis. The anaesthetic was intravenous barbiturate ("eniumal" 300+100 mg.), "laudolissin" (60 mg.), and succinylcholine (50 mg.). Phenergan, 50 mg., was given to increase the vasodilatation.

The operation began when the temperature had reached 30.7°C. and 27.3°C. in the rectum and
oesophagus respectively. A clamp was placed on the pulmonary artery and a circular suture around the pulmonary conus. At this point ventricular fibrillation began. The valvotomy and dilatation were performed and cardiac massage begun as soon as possible. In order to make certain that the valvotomy was effective the circulation was stopped again and the valve explored with the finger. The opening in the pulmonary artery was closed by sutures and heart massage was begun again, and then heating with saline started. The circulation had been interrupted twice—first for five minutes, then for two minutes. The rising temperature curve corresponded to those from the first two cases.

During rewarming defibrillation was repeatedly attempted but was not successful until a temperature of 35.2° C. had been reached. The heart had now fibrillated for 109 minutes 20 seconds, and 150 litres of saline had been used for rewarming.

Electroshock therapy had been given 16 times, using two to three shocks a time. A current of 2 to 2.5 amps. was used, the time varying from 0.1 to 0.75 seconds. At a certain time when the heart seemed weak, 700 mg. calcium chloride was given intracardially.

Recovery was normal, but post-operatively there have been some psychic disturbances. His mental state 14 days after the operation is such that specific events have only a transitory impression upon his brain.

**DISCUSSION**

Blades and Pierpont (1954) have recommended intrapleural lavage of the chest for the cooling and rewarming of patients undergoing hypothermia. In a discussion following their contribution Muller mentioned a case of hypothermia where ventricular fibrillation was treated by intrapleural lavage with hot saline. The heart rhythm became normal by treatment with hot saline and cardiac massage alone. The patient, however, died shortly after being transferred to bed.

As the combination of rewarming and electroshock proved so effective in our three cases we have considered it of sufficient importance to call attention to this treatment.

Brock and Ross maintain that the pharyngeal temperature is an accurate measurement of the body temperature. Swan and others measure rectal temperature. Many authors do not indicate where the temperature is measured.

In a series of 22 patients cooled by surface cooling we found the rectal temperature in small children to be lower (1–1½° C.) than the oesophageal temperature. In older children and adults the rectal temperature during cooling has been constantly 2–3° C. higher than the oesophageal temperature. In heating the thorax with the hot saline the oesophageal temperature has risen more rapidly than the rectal temperature, which remained low. Only when the heart has resumed normal rhythm has the rectal temperature begun to increase significantly, and then the oesophageal temperature has dropped a little followed by an increase parallel to the increase in the rectal temperature. We presume that the oesophageal temperature gives a more accurate indication of the temperature of the heart than does the rectal temperature and therefore is a better guide to the progress of treatment.

The main interest of these cases lies in the fact that periods of cardiac massage of 114 minutes, 50 minutes, and 109 minutes’ duration respectively can be undertaken without visible damage to the heart and none or very few post-operative psychic disturbances. We conclude that this is due to the fact that an efficient circulation by cardiac massage can be maintained more effectively during hypothermia.

While too far-reaching conclusions should not be drawn from three cases, we feel our results justify the recommendation of rewarming with hot saline in cases in which the defibrillator is ineffective at low temperatures.

**SUMMARY**

Three cases of ventricular fibrillation during hypothermia and cardiac operation are reported. Defibrillation by electroshock was successful after rewarming the patients by pouring hot saline into the thoracic cavity.

The ventricular fibrillation lasted for 114 minutes, 50 minutes, and 109 minutes respectively.

Recovery of the patients was perfectly normal in two cases, in the third case there were some symptoms of cerebral damage.

**REFERENCES**


