

A STUDY OF THE TOLERANCE OF THE CANINE HEART TO VENOUS AND AORTIC OCCLUSION UNDER HYPOTHERMIA.

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(Received on July 22, 1960.)

Cold lowers the tissue metabolism. The oxygen requirement of the tissue is decreased and so the blood flow to an organ can be stopped completely for some time or decreased to a considerable degree for a long period without there being any ischaemic damage to the organ concerned. This rationale of hypothermia is used in intracardiac surgery when inflow and outflow occlusion is done and a dry heart is opened up for the repair of the septal defects.

However, hypothermia is not quite a safe procedure and the mortality due to ventricular fibrillation is considerable. The cause of this fibrillation is not yet known and consequently no measures could be taken to prevent its onset.

The incidence of ventricular fibrillation under hypothermia varies from animal to animal. In the same animal it varies with the anesthetic agent used (Kapoor, 1960). Further it becomes more frequent if venous inflow and aortic outflow occlusion are done for certain operative interferences. Here we have studied the tolerance of the canine heart to inflow and outflow occlusion under chloralose anesthesia.

METHODS

Healthy mongrel dogs were anaesthetised by chloralose (BDH; 80 mgm. per kilogram body weight intravenously). Tracheal intubation was done and dogs were hyperventilated throughout the experiments by air through an artificial respiratory pump (200-250 c.c. of air per stroke at a rate of 28/min was given and the volume was increased to 400-500 c.c. when the chest was opened). Rectal temperature was recorded by a centigrade thermometer. All EKG recordings were taken by Burdick cardioscribe on standard Lead II.

Cooling was done by immersing the animal in ice cold water (1°C to 5°C). The animal was cooled upto a temperature of 26°C to 27°C in the bath and then taken out of the bath. Rewarming was done by immersing the dog in the water at a temperature of about 45°C.

The experiments were planned in the following groups :

Group I : 12 dogs were cooled and the lowest temperature from which they could be rewarmed back safely to normal temperature was determined.

Group II : In 13 dogs at 23°C chest was opened by a lateral incision in the 4th intercostal space. Vena azygos was ligatured. Superior vena cava was occluded and thirty seconds after this inferior vena cava was also occluded and the duration of complete venous occlusion that the heart tolerated was observed.

Group III : In 10 dogs the venous occlusion was done and an exploration of the right auricle was done by making a transverse incision between the two stay sutures and the wound was subsequently stitched by a continuous suture.

Group IV : In 26 dogs the venous occlusion was done and one minute after the venous occlusion aortic occlusion was done by Potts clamp after opening the chest through a left sided incision. The aorta was clamped at its root.

RESULTS

Group I : The normal body temperature of the dog varied from 36°C to 40°C. In 30 to 40 minutes the temperature came down 26°C to 28°C by immersion cooling. A fall of 3 to 5°C was observed within 10 minutes in all dogs outside the cooling bath. It was found that 23°C was the safest limit from which the animal could be rewarmed back to normal temperature (Table I).

TABLE I

Tolerance of dogs to simple cooling and rewarming.

No. of Dogs Experimented upon	Range of temperature to which the cooling was done	No. of dogs expired	Survival
2	28° - 26°C	Nil	100%
3	25° - 23°C	Nil	100%
4	22° - 20°C	1	75%
3	Below 20°C	3	Nil

Group II : Venous occlusion was tolerated upto 15 minutes safely and the dogs could be revived back to normal by rewarming.

TABLE II

Tolerance of dog to venous occlusion:

No. of Dogs Experimented upon	Period of occlusion in minutes	No. of dogs expired	Survival.
2	0 - 5	Nil	100%
2	5.5 - 10	Nil	100%
5	10.5 - 15	1	80%
4	15.5 - 20	3	24%

Group III: Venous occlusion for 5 minutes followed by auricular exploration in 2 cases was done successfully and the dogs were revived afterwards by rewarming. Out of the two dogs in whom occlusion period was 5.5 to 10 minutes, one tolerated occlusion but expired during rewarming. In 6 dogs where occlusion period was increased above 10 minutes the survival was only 33.3%

TABLE III

Tolerance of dogs to venous occlusion with Auriculotomy.

No. of dogs Experimented upon	Period of occlusion	No. of dogs expired	Survival.
2	0 to 5 mts.	Nil	100%
2	5.5 to 10 mts.	1	50% one dog tolerated occlusion but died during rewarming.
6	10.5 to 15 mts.	4	33.3%

Group IV: Venous and aortic occlusion was tolerated by 6 dogs for 1 to 5 minutes; other six dogs for 5.1 to 10 minutes and two dogs for 10.1 to 20 minutes

TABLE IV

Tolerance of dogs to venous and aortic occlusion.

No. of dogs Experimented upon	Time of occlusion	No. of dogs expired	Survival
6	1 - 5 mts.	Nil	100%
13	5.1 - 10 mts.	7	46.1%
7	10.1 - 20 mts.	5	28.5%

DISCUSSION

The tolerance of the animals to hypothermia is variable. Materius and Maison (1948) reported lethal temperature ranging from 16.8° to 11.7°C in thirteen dogs. Crimson (1945) revived dogs under pentobarbital sodium from a normal temperature level below 24°C provided this temperature was only maintained for 20 minutes. Under chloralose anesthesia we were able to revive all animals from a temperature of 23°C.

The occlusion studies were done at this safer temperature of 23°C and the animal was maintained in hypothermia at 23°C for a period ranging from 7 to 70 minutes.

Tables II, III & IV show that on an average the heart tolerated simple venous occlusion upto 15 minutes, venous occlusion with auricular exploration from 5 to 10 minutes and in majority of cases venous and aortic occlusion from 5.1 to 10 minutes, but the survival percentage was less as compared to simple venous occlusion with auricular exploration.

Similar studies by Scott *et al.* (1954) revealed 75% survival in dogs with venous occlusion lasting for 10 minutes, 80% survival in dogs with venous and aortic occlusion lasting for six minutes and 14.3% survival with venous and aortic occlusion lasting for 10 minutes. As they did auriculotomy in all their experiments, we cannot compare our results with theirs.

However, it is quite apparent from these observations that simple venous occlusion is less detrimental than venous and aortic occlusion combined together and specially if such an occlusion is allowed to last for a period longer than 10 minutes. Furthermore if simple occlusion is associated with any operative trauma to the heart muscle, heart failure sets in early as is evident from the fact that the heart tolerated simple venous occlusion upto 12 minutes while if venous occlusion was associated with auriculotomy, the

tolerance was reduced to 5.5 to 10 minutes. It is possible that injury to the myocardium is increased considerably by venous occlusion and operative interferences and both factors combined together are more detrimental than one alone.

The lowered tolerance of the heart to venous and aortic occlusion can be explained by the fact that venous occlusion by decreasing the cardiac output reduces coronary blood flow leading to myocardial ischaemia. The venous occlusion if associated with aortic occlusion increases the degree of ischaemia still further because if the aorta is clamped near its root it may narrow down or close completely the openings of the coronary arteries.

SUMMARY

The tolerance of the canine heart to venous and aortic occlusion was studied under hypothermia. Dogs were cooled by immersing in cold water and it was observed that the dogs under chloralose anaesthesia can be revived from a temperature of 23°C safely. They can tolerate under chloralose anaesthesia venous occlusion upto 15 minutes; venous occlusion combined with aortic occlusion for 5.1-10 minutes; though ventricular fibrillation under total occlusion may occur as early as one minute and as late as 20 minutes but the survival rate in the latter is definitely lower.

ACKNOWLEDGEMENT

This research work was financed by Indian Council of Medical Research.

We thank Dr. R.C. Shukla, Head of the Physiology Department, K. G. Medical College, Lucknow, for giving facilities for the work.

REFERENCES

1. Grimson, J.M. (1945) : *Mem. Rep. No. TEEAA 695-56A Eng. Div. A.A.F.* 21 Dec.
 2. Haterius, H.O. and Maison, G.L. (1948) : *Am. Jour. Physiol.* **152**, 225.
 3. Kapoor, S. R. and Agarwal, H. C. (1960) : *Ind. Jour. Med. Res.* **48**, 433.
 4. Scott, *et al.*, (1954) : *Am. Surgeon* **20**, 799.
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