

THE ROLE OF NERVES IN THE PRODUCTION OF CARDIAC ARREST DURING SURGICAL ANAESTHESIA

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Summary: The role of cardiac nerves in the production of cardiac arrest during surgical anaesthesia on coronary ligated hypoxic heart has been studied. When atropinised coronary ligated dogs were exposed to hypoxia the terminal event was a cardiac asystole in 88% of the dogs. In propranolol treated dogs, or in dogs where sympathetic ganglia upto T6 were bilaterally removed earlier, coronary ligation and hypoxia produced ventricular extrasystoles, ventricular tachycardia and repeated sinus arrest followed by ventricular fibrillation. The possibility of the origin of arrhythmia from the damaged myocardium, and the presence of an intact vagus in the production of ventricular fibrillation has been discussed.

Key words: heart arrest

anoxia

myocardial infarct

INTRODUCTION

Of the various factors leading to cardiac arrest (either asystole or ventricular fibrillation) during surgical anaesthesia, hypoxia seems to be the common denominator. Besides hypoxia, the other factors could be undue cardiac sensitisation to vagovagal reflex effects and asphyxia with an alteration of the normal conducting mechanism(1).

Kumar and Srivastava (9) exposed coronary ligated dogs to progressive hypoxia and observed that dogs showing evidence of infarction when exposed to hypoxia developed primary cardiac arrest. Wollenberger and Sahab (18) demonstrated the release of noradrenaline by anoxia in the rabbits heart. Levy (11) observed that ventricular fibrillation was brought about by the action of adrenaline on a myocardium sensitised by hypoxia or chloroform anaesthesia. Gesell and Charles (5) showed that vagal effects on the heart were increased by hypercapnia and by mild hypoxia whereas severe hypoxia paralysed the effect of the vagus. Sloan (15) pointed out that vagovagal reflex could be the causative factor for sudden cardiac arrest leading to death during surgery. During normal oxygenation of the blood vagal stimulation rarely causes cardiac arrest but hypoxia or hypercapnia seems to complement vagal inhibition and the effects may be added to cause a temporary or permanent cardiac arrest. These vagovagal reflexes could be mitigated by the use of atropine as a premedication(16).

The present study was undertaken in an endeavour to find out the effect of cardiac denervation (surgical or chemical) on coronary artery ligated dogs under surgical (plane III) anaesthesia during progressive hypoxia.

MATERIALS AND METHODS

Experiments were performed on 18 healthy mongrel dogs of both sexes weighing between 8.5 kg to 17 kg. They were anaesthetised with chloralose 85-100 mg/kg body weight given intravenously. An additional dose of 20-25 mg/kg was often needed after about two hours to maintain a smooth plane of anaesthesia. A midline incision was made in the neck to expose the trachea and the carotid artery. The trachea was cannulated for giving positive pressure respiration during thoracotomy and later for inducing progressive hypoxia. Mean arterial blood pressure was recorded from the right carotid artery. Diaphragmatic excursions as an index of respiration were recorded by hooking the diaphragm to a starling lever through a pulley. Femoral artery and vein were cannulated on one side for drawing blood for gas analysis and administration of drugs respectively.

Intermittently lead I electrocardiogram was taken at 25 mm/second speed of paper and blood drawn for gas analysis. Thoracotomy was done in the 5th left intercostal space while the animal was maintained on a positive pressure respiration. The anterior descending branch of the left coronary artery was ligated in the interventricular groove as high as possible. A raised ST segment and T wave reversal were taken as indication of infarction. The chest was then closed in layers after fully inflating the lungs and drawing out all the air from the pleural cavity through a rubber tube. The dog started breathing spontaneously, and it was then left for half an hour to stabilise itself.

Hypoxia was induced by making the dog rebreathe air in a 7 litre spirometer fitted with a sodalime tower to absorb CO₂ till death supervened. Arterial blood was analysed for its O₂ content by the method of Peters and Vanslyke(12).

The dogs were divided into three groups. Group 1, consisted of 8 dogs atropinised with 0.3 mg/kg given intravenously before subjecting them to hypoxia. Group 2, consisted of 7 dogs, given propranolol 1 mg/kg body weight intravenously to cause adrenergic receptor blockade (4) before hypoxia was induced. Group 3, consisted of 3 dogs, sympathectomised previously under pentobarbital sodium anaesthesia given intravenously in dose of 30 mg/kg body weight. The lateral sympathetic chains comprising of the stellate ganglion down to the sixth sympathetic ganglion were removed by bilateral thoracotomy. The chest was closed in layers after fully inflating the lungs under positive pressure to draw out all the air from the pleural cavities. Procain penicillin 4 lac units and streptomycin 1/2 gm were given intramuscularly post-operatively daily for 8 days. These dogs were experimented upon at least 15 days after sympathectomy.

RESULTS

Twenty dogs of Kumar and Srivastava (9) in which a coronary ligation and hypoxia were done under the same laboratory conditions were treated as control for this study. On

TABLE I : Showing successive cardiac events with progressive hypoxia.

Dog Nos.	Whether myocardial infarction present after coronary ligation	Successive cardiac events with progressive hypoxia after coronary ligation	Terminal cardiac event
<i>Group 1.</i>			
1.	Absent	Brady.,* Incomplete heart block, complete heart block.	Asystole
2.	Present	Brady., Sinus tachy.**	Ventricular fibrillation
3.	Absent	Tachy., Brady., Idioventricular beats.	Asystole
4.	Present	Brady., Tachy., Incomplete heart block, complete heart block.	Asystole
5.	Present	Progressive brady., Tachy., Brady.	Asystole
6.	Present	Brady., Tachy., Brady., ventricular extrasystole.	Asystole
7.	Present	Brady., Tachy., Incomplete heart block., complete heart block.	Asystole
8.	Present	Progressive Brady.	Asystole
<i>Group 2.</i>			
1.	Present	Brady., sinus arrest, junctional (Nodal) Rhythm, Ventricular tachy.	Ventricular fibrillation
2.	Present	Brady., Junctional (Nodal) Rhythm.	Asystole
3.	Absent	Brady., Sinus arrest, Idioventricular beats, Junctional Rhythm.	Asystole
4.	Present	Progressive brady., junctional (Nodal) Rhythm.	Ventricular fibrillation
5.	Absent	Brady, Tachy.	Ventricular fibrillation
6.	Present	Brady., Sinus arrest, Sinus Rhythm, Junctional ectopics Nodal Rhythm, Ventricular Tachy.	Ventricular fibrillation
7.	Present	Brady., Sinus arrest. Sinus Rhythm.	Ventricular fibrillation
<i>Group 3.</i>			
1.	Present	Brady., Ventricular extrasystole.	Ventricular fibrillation
2.	Present	Brady., Supraventricular extrasystole, ventricular extrasystole, Auricular flutter, Sinus Rhythm, Ventricular extrasystole.	Ventricular fibrillation
3.	Present	Brady., Ventricular tachy.	Ventricular fibrillation

* = Bradycardia
** = Tachycardia

exposure to hypoxia those animals showed no change in the blood pressure and heart rate till the oxygen saturation of blood was 75%. As hypoxia progressed hypertension and tachycardia occurred followed by bradycardia and hypotension, till the oxygen saturation of the blood fell to 8-16% when a respiratory failure occurred very suddenly which was followed by a fall of blood pressure to zero and then a cardiac asystole. The terminal event was either a ventricular fibrillation or a cardiac asystole. Out of those 20 dogs myocardial infarction occurred in five dogs. In these five dogs the arrest was due to a fibrillation. In three of these five a primary cardiac arrest was observed, as respiration continued for about one minute thereafter.

In group I of the present study myocardial infarction occurred in six out of eight dogs. When arterial oxygen concentration dropped to 50% hypertension and bradycardia was observed in six dogs. In seven dogs the terminal cardiac event was a cardiac asystole, only in one dog a ventricular fibrillation was observed (Table I). Cardiac asystole occurred from 0-6 min after the fall of blood pressure to zero and in one dog the heart stopped as late as 14 min after the drop in blood pressure to zero.

In group II of the present series myocardial infarction occurred in five of the seven dogs. In all the dogs after induction of hypoxia the blood pressure was maintained till the oxygen saturation of the blood came down to 60-70%. Then the blood pressure started falling and came down to zero as suddenly as in the other groups preceded by respiratory arrest. A progressive bradycardia was observed within five min of the induction of hypoxia. The terminal cardiac event was a ventricular fibrillation in five dogs and asystole in the other two (Table I). In all these dogs bradycardia was followed by junctional rhythm, sinus arrests, ventricular extrasystoles, ventricular tachycardia and low voltage bizarre complexes before the final fibrillation or asystole (Table I).

In the last group myocardial infarction occurred in all the three dogs. On induction of hypoxia, hypotension and bradycardia developed when O₂ saturation came down to 48%. Ventricular fibrillation preceded by ventricular extrasystole supraventricular extrasystole or auricular flutter was observed 0-4 mins after the fall of blood pressure to zero.

DISCUSSION

The present study was carried out in dogs anaesthetised with chloralose without any premedication. The level of anaesthesia was plane III of stage 3 of the type maintained in human beings during surgery. Chloralose was chosen as anaesthetic since it has least effect on central neural regulation and cardiovascular system (17), and chloralose does not produce significant alteration in E.K.G. pattern (14).

In the control group of hypoxic dogs (9) ventricular fibrillation developed terminally in only those animals that showed electrocardiographic evidence of infarction, the rest of the dogs developed asystole. Ascanio *et al.* (2) observed that 50% of their dogs in whom myocardial

necrobiosis was produced by an injection of Hexa, died of ventricular fibrillation, but their vagotomized Hexa treated dogs developed many extrasystoles but no fibrillation. 88% of the completely atropinised animals of group I of this study, too did not develop ventricular fibrillation. It appears likely therefore, that an intact vagus is essential for fibrillation to develop in a damaged myocardium exposed to hypoxia.

Kumar *et al.* (10) studied the response of the heart to efferent vagal stimulation under hypoxia and observed irregularities in the rhythm of the heart in mildly hypoxic dogs during vagal stimulation. No such irregularities occurred in completely atropinised dogs even under severe hypoxia. In atropinised hypoxic dogs of group I of this study however, partial heart block, complete A-V dissociation and idioventricular beats were observed. Such irregularities could be due to atropine itself (8). In group II, asystole was observed in two dogs only, and in one of them repeated sinus arrests were observed. If this sinus arrest had continued a primary cardiac arrest could have occurred. Ruzicka and Nicholson (13) observed outpouring of epinephrine in any excitement, which in a sensitised myocardium can produce cardiac arrhythmias (11). It has been mentioned that experimental arrhythmias induced by ligation of a coronary artery in dogs are not generally susceptible to β -adreno receptor antagonist except when deliberately worsened by adrenaline injection (3). This fact may account for the persistence of various ventricular arrhythmias namely ventricular extrasystole, ventricular tachycardia and ventricular fibrillation even after propranolol administration in animals of group II. It is obvious that β -adrenoreceptor antagonists could only mitigate the effect of circulating catecholamines either endogenous (released by anaesthetic and hypoxic stress) or parenteral. The damaged myocardium could therefore be the possible site for origin of arrhythmias (ventricular fibrillation) either by setting up reflexes mediated by intact vagus (2) or by releasing nor-adrenaline under anoxic condition (18) which would act on α -receptors (6). A vagal over activity due to depression of the sympathetic tone is a possibility. A progressive bradycardia supports this hypothesis. Johnstone (7) also suggested this possibility when he observed a bradycardia and hypotension with fluothane anaesthesia.

In group III dogs since the sympathetic chain had been removed and the β -receptors were intact, endogenous catecholamines which must have been released due to hypoxic and anaesthetic stress could be responsible for these arrhythmias.

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