EFFECTS OF STEROID ANESTHESIA
UPON CARDIAC RHYTHM OF THE DOG

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The occurrence of cardiac arrhythmias during general anesthesia using various anesthetic agents has been reported (1-3). It is also known that endotracheal intubation will produce cardiac arrhythmias in a certain number of patients anesthetized with various anesthetic agents, and that certain agents like chloroform and cyclopropane will sensitize the heart to the action of epinephrine with consequent, and often fatal, irregularities (4-6). 21-Hydroxypregnane-3,20-dione sodium succinate (Viadril®) has been recently investigated and found deserving of widespread trial as a general anesthetic agent (7-10). It is the purpose of this paper to report some observations of the effects of this drug upon the cardiac rhythm of the dog.

METHOD

Twenty-eight mongrel dogs (average weight, 7 kg.) were divided into 3 groups. No dog received premedication. The first group (A) consisted of 5 dogs that received chloroform anesthesia by the open drop technique. Endotracheal intubation was performed as soon as relaxation permitted, and ten minutes later each dog received, intravenously, 140 μg. of epinephrine kg. of body weight. Smaller dosages of epinephrine, with this technique, did not produce consistent ventricular fibrillation. The second group (B) consisted of 3 dogs injected, intravenously, with 140 μg. of epinephrine/kg. of body weight. The third group (C) consisted of 20 dogs anesthetized with a single intravenous injection of 55 mg. of Viadril (2.5 per cent)/kg. of body weight. Eight minutes following injection of Viadril, the trachea of each dog was intubated under direct laryngoscopy. Five minutes after endotracheal intubation, epinephrine (140 μg./kg. of body weight) was injected, intravenously, in each dog.

Electrocardiograms (lead II) were recorded in all 3 groups of dogs during the various experimental procedures.

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Fig. 1. (A) Sinus arrhythmia before chloroform anesthesia. (B) Sinus rhythm during chloroform anesthesia. (C) Ventricular tachycardia following adrenalin injection. (D) Ventricular fibrillation (continuation of strip C). (Dog number 3.)

Fig. 2. (A) Sinus bradycardia in dog unanesthetized. (B) and (C) Sinus arrest with nodal rhythm and ventricular tachycardia following adrenalin injection. (D) Sinus arrest (continuation of strip C). (Dog number 8.)
Fig. 3. (A) Sinus arrhythmia before anesthesia. (B) Sinus arrhythmia following Viadril® injection. (C) Sinus arrhythmia during endotracheal intubation. (D) Sinus arrhythmia following adrenalin injection. (Dog number 15.)

Fig. 4. (A) Sinus arrhythmia before anesthesia. (B) Sinus arrhythmia following Viadril® injection. (C) Sinus rhythm during endotracheal intubation. (D) Sinus arrhythmia with a ventricular premature contraction following adrenalin injection. (Dog number 11.)
Results

Group A.—Intravenous injection of epinephrine in the dogs anesthetized with chloroform (open drop method without addition of oxygen) proved fatal in all instances. The terminal event was ventricular fibrillation. Figure 1 illustrates typical electrocardiographic changes in a dog so treated.

Group B.—The dogs in this group, receiving only epinephrine, exhibited changes in cardiac rhythm, but all survived. These changes consisted of sinus arrest, sinus arrhythmia, and ventricular tachycardia. Figure 2 illustrates some of the electrocardiographic changes.

Group C.—The dogs of this group, anesthetized with a single dose of Viadril, showed either sinus rhythm or sinus arrhythmias following the single injection of Viadril. Electrocardiograms recorded before the injection of Viadril showed also sinus arrhythmia. No oxygen was given to the animals during the experiments and anesthesia was kept for most of the experiments in plane 2, stage III. During and following intubation, though some bucking on insertion of the endotracheal tube occurred, no changes from previous cardiac rhythms were observed (fig. 3). Injection of epinephrine never proved fatal. Changes in rhythm following such injection consisted of sinus arrhythmias, ventricular premature contractions, and ventricular tachycardias. Figure 4 illustrates some of these changes.

There was no laryngospasm during the intubation of the dogs. Severe sloughs occurred in 2 dogs in which Viadril was accidentally extravasated into the surrounding tissues during its intravenous injection.

Discussion

The occurrence of sinus arrhythmia in the dog is very common and is not significant. It is relevant that this basic pattern never changed in our dogs during the injection of Viadril and endotracheal intubation. Now, if we assume that during endotracheal intubation the primary factor in determining cardiac arrhythmias is hypoxia with carbon dioxide retention, we must hypothesize that the Viadril in the dosage used was not able to depress respiration to an extent that hypoventilation occurred. We do not have evidence in favor of such hypothesis since, although we determined the respiratory rate, we did not record the tidal volume. Alveolar ventilation, which is important for gas exchange, cannot be calculated unless tidal volume values are obtained in addition to the respiratory rate.

If we agree with others that cardiac arrhythmias following endotracheal intubation are of a reflex nature, we may hypothesize that Viadril interferes with such reflexes. In consideration of the fact that laryngeal reflexes are less active during Viadril anesthesia, and this impression is also supported by our clinical observation of Viadril anesthesia on human beings, we favor this second hypothesis.
SUMMARY AND CONCLUSIONS

In our experimental observations in the dog, injection of Viadril in the dosage used produced nothing other than sinus arrhythmias. Also, endotracheal intubation under Viadril anesthesia, though occasionally performed in a light plane of anesthesia, produced only sinus arrhythmias. Viadril did not seem to sensitize the heart to the effect of epinephrine.

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