# A STUDY OF THE EFFECTS OF EPIDURAL ANESTHESIA ON HEART RATE, RESPIRATORY RATE AND BLOOD PRESSURE IN THE DOG

by

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# INTRODUCTION

Epidural anesthesia was first suggested by Corning in 1885. He noted that the injection of cocaine solution into the spinal canal of the dog was followed by paralysis of, and loss of sensation in, the animal's hind limbs (Hall, 1966). The practical application of epidural anesthesia in veterinary surgery was first demonstrated in 1925 by Retzgen in Germany and in 1927 by Frank in the United States. Since then numerous compounds have been investigated as epidural anesthetics: procaine, tutocaine, lidocaine, hexylcaine, quinine, urea hydrochloride, mepivacaine, and others. At the present procaine, hexylcaine, and lidocaine are probably used more extensively in veterinary medicine for epidural anesthesia than the others.

Although epidural anesthesia has been used extensively in small animal surgery the effects of an epidural anesthetic on the cardiovascular and respiratory systems has been subject to controversy. The literature contains numerous conflicting reports regarding the effect of an epidural anesthetic on these systems.

The purpose of this project is to study the effect of an epidural anesthetic in dogs on the blood pressure, heart and respiratory rate.

## LITERATURE REVIEW

Mode and Location of Action: The exact mode and location of action of a local anesthetic injected into the epidural space is subject to controversy. According to Venugopalan (1962); Cuille and Chelle, in 1931, were of the opinion that the anesthetic penetrated the spinal nerves. Since then three principal areas for the site of action have been postulated: extension of the anesthetic through the intervertebral foramina and effecting a paravertebral block, penetration of the anesthetic through the dura mater with anesthesia of the spinal nerves and spinal cord and penetration of the spinal nerves within the epidural space (Collins, 1966).

Earlier experimental evidence seemed to indicate that the most likely way epidural anesthesia was accomplished was by producing an extensive bilateral paravertebral block (Bromage, 1954). India ink and radiopaque media have been injected into the epidural space of dogs. The flow of these compounds indicated that local anesthetics would probably spread in the same manner and in so doing, would anesthetize the spinal nerves.

The possibility of the anesthetic diffusing across the dura mater and affecting the spinal cord and nerve roots has been investigated. Rudin (1951) showed that the epidural injection of 10 ml. of 2 or 3% procaine hydrochloride, in the

dog, resulted in ultimate concentrations of procaine in the subarachnoid space varying between 0.3 and 0.8 mg. per ml. of cerebrospinal fluid. Spinal cord function in the dog was depressed by subarachnoid procaine concentrations varying from 0.1 to 1 mg. per ml. (Jones, 1966). The presence of a standard solution of procaine and radioactive lidocaine and mepivacaine has been demonstrated (Bromage, 1963) in the thoracic and lumbar spinal cord after epidural injection. Lessor amounts have been demonstrated in the cervical cord and medulla.

Earlier explanations concerning the site of action during epidural anesthesia were dominated by the assumption that passage of the anesthetic into the cerebrospinal fluid precedes neural fixation and blockade. Bromage (1963) contended that if this assumption was dropped in favor of the idea that passage into the cerebrospinal fluid followed or accompanied neural involvement after sub-dural and sub-pial spread, then all the available clinical and experimental findings would agree. He stated that anesthetic solutions reach the sub-perineural spaces by diffusion around the capillary and lymphatic channels of the vasa nervorum, at and beyond the dural "ink cuff" areas. Once inside the endoneural spaces, longitudinal capillary networks provide tissue interfaces along which the anesthetic solutions can follow the spinal roots and extend into the sub-pial spaces of the cord itself. From there, the concentration gradient allows

a gradual diffusion into the cerebrospinal fluid with the nerve block having already occurred. In 1954 Bromage demonstrated in rabbits that the area of the dural cuffs were particularly favorable to the passage of particulate matter. India ink suspensions with a particle size of 0.4 to 1.5$\mu$ readily passed out of the subarachnoid space through the dural (ink) cuff, into the surrounding tissue, and thence into the regional lymph glands. With colloidal carbon this passage took less than half-an-hour. He stated it seemed reasonable to suppose that if coarse particulate matter could pass with such ease through the "ink cuff" area, the relatively tiny crystalloid molecules of a local anesthetic solution should be able to migrate, in reverse direction, through the same region.

Selective Blockade of Spinal Nerves: Sarnoff (1947) demonstrated that there was a differential susceptibility of nerve fibers to the blocking action of a local anesthetic, which depends on the fiber size. His work was aided by other authors (Collins, 1966) who showed that nerve fibers could be divided into three major groups (A,B,&C) depending on fiber size which in turn correlated with fiber function. The A group of fibers ranged from 3 to 20$\mu$ in diameter, were myelinated and carried both motor and sensory impulses. The B group of fibers were smaller, myelinated, and ranged from 1 to 3$\mu$ in diameter. The third or C group of fibers were the smallest. They were non-myelinated, preganglionic

autonomic fibers.

Generally, local anesthetics block nerve transmission most easily in the smaller fibers (autonomic and sensory). The larger motor fibers are the slowest in onset and the shortest in duration (Collins, 1966). It has been shown, in man, that the subarachnoid exposure of mixed nerves or spinal nerve roots to appropriate dilutions of a local anesthetic can produce a selective blockade. The concentrations needed to produce this selective blockade are known as the "critical concentrations" (Table 1). The discrepancy between the concentrations which must be injected

Table 1. Concentrations of Local Anesthetics Required to Block Various Types of Nerve Fibers in the Subarachnoid Space (Collins, 1966).

| Type of Fiber | Procaine (mg/cc) |
|---|---|
| Vasomoter | 0.2 |
| Sensory | 0.33-0.5 |
| Motor | 0.5-0.75 |

and the much lower concentration in the spinal fluid when anesthesia exists has not been fully elucidated. It has been postulated that the intrathecal nerve elements avidly take up the anesthetic agent and when saturated an equilibrium is established with the spinal fluid bath (Collins, 1966). In 1966 Goodman & Gillman reported that selective blockade may also be obtained with different concentrations of a

local anesthetic injected epidurally. Sympathetic block may be obtained with 0.5 to 1% lidocaine solution, while 1.0 to 1.5% lidocaine will cause sensory loss. Motor blockade will result from the injection of 2% lidocaine solution.

Factors Affecting the Spread of Solutions Injected into the Epidural Space: The extent of anesthesia after the injection of a local anesthetic into the epidural space is determined by a number of variables. Some of the variables are intrinsic to the animal and some are extrinsic and due to variations in technique and drugs employed. Hall (1966) contended that the intrinsic variables governing the spread of solutions in the epidural space were perhaps best understood if the space was considered as a cylindrical "reservoir." He notes that the volume of the "reservoir" may be determined by factors such as the length and diameter of the cylinder and the size of the structures which it containes.

Hall (1966) states that the epidural "reservoir" of the dog contains the spinal cord, cerebrospinal fluid, spinal nerves, meninges, a variable amount of fat, and blood vessels. The "reservoir" exits through which injected solutions can escape were the intervertebral foramina, the extradural blood vessels and lymphatics, the dura mater into the cerebrospinal fluid, and diffusion into the epidural fat.

According to Hall (1966) workers such as Harthoorn and Brass relate the dosage of local anesthetic solution to the length of the vetrebral column (occipital-tail root measurement). Gravity has been reported to affect the spread of solutions injected into the epidural space (Bromage, 1954). In 1935 Brook reported that it would be advantageous to keep the patient in dorsal recumbency for sometime immediately after injection. His contention was that this would faciliate greater contact between the anesthetic solution and the dorsal roots of the spinal nerves which are sensory in nature and proportionately less contact between the solution and the ventral roots which contain motor fibers and the connecting fibers of the sympathetic trunk. It has been postulated that changes in the epidural pressure affects the spread of an epidural anesthetic (Bromage, 1954). In 1963 Tufvesson reported that an epidural anesthetic had a tendency to spread rapidly during pregnancy, at least at term and during labor. The cause for the increased spread was not fully understood but was thought to be related to changes in the intra-abdominal pressure (Collins, 1966) and venous plexuses in the epidural space (Hall, 1966).

Effect of Epidural Anesthesia: Most epidural injections in the dog are given at the lumbo-sacral space (Hall, 1966). As the anesthetic progresses forward, provided it is in sufficient concentration, the various nerves become

anesthetized. Sensory and/or motor function to the areas supplied is interrupted. Table 2 lists various spinal nerves and the regions and organs that they supply.

(a) Autonomic Nervous System: Miller (1964) has defined the autonomic (general visceral efferent) system as that portion of the nervous system concerned with motor innervation of smooth muscle, cardiac muscle, and glands. He explained that some authors preferred to include sensory innervation of the viscera under the general heading of autonomic nervous system. He preferred not to include the visceral afferent fibers in his classification. On anatomical, pharmacological, and functional bases the autonomic nervous system has been subdivided into sympathetic (thoracolumbar) and parasympathetic (craniosacral) portions (Miller, 1964). The ganglia of the sympathetic system have been divided into two classes: vertebral and prevertebral (Dukes, 1955). He reports that the vertebral ganglia occur in a double chain, the sympathetic trunks, which extend along the ventral surface of the vertebral column from the base of the skull to the tail. They are segmentally arranged except in the cervical region, where the number is reduced to two or three. The prevertebral ganglia are scattered among the viscera and comprise chiefly the celiac, anterior mesenteric, and posterior mesenteric ganglia.

Table 2. The Distribution of the Sensory and Motor Fibers of the Spinal Nerves which are Usually Affected During Epidural Anesthesia (Hall, 1966 and Miller, 1964)

| Spinal Region | No. of Nerve | Structures Supplied | |
|---|---|---|---|
| | | Sensory | Motor |
| Coccygeal | 4-7 pairs | Greater part of the tail | Coccygeal muscles |
| Sacral | 1, 2, & 3 Pudendal N. | Base of tail, anus, vulva, perineum & adjacent parts | Anus, terminal rectum, vagina, penis, bladder, uretha |
| Sacral Lumbar | 1, 2, 5, 6 & 7 | Dorsal br.--sensory to region of croup<br>Ventral br.--enter into the formation of the lumbosacral plexus | |
| Sacral Lumbar | $S_{1,2}L_{5,6,7}$ Sciatic N. | Lower parts of limb below mid-tibia | Flexors of the stifle (in part), flexors of the hock and digit |
| Sacral Lumbar | Caud. gluteal N. | Lateral aspect of thigh | Flexors & abductors of hip |
| | Cran. gluteal N. | Lat. & post. parts of hip and thigh | Extensors of hip (in part) |
| Lumbar | 5 & 6 Obturator N. | Medial aspect of thigh | Abductors of the hip |
| | 4, 5 & 6 Femoral N. | Ant. & med. aspects of limb as low as hock | Flexors of hip (in part), extensors of stifle |

| Spinal Region | No. of Nerve | Structures Supplied: Sensory | Motor |
|---|---|---|---|
| Lumbar | 3 Ilio-inguinal | Loins & croup ant. stifle, scrotum, prepuce, inguinal area & mamary gland | Sublumbar group (in part), post. abdominal muscles |
| | 2 Caud. iliohypo-gastric N. | Loins, flank, ant. & lat. thigh, scro-tum, prepuce, mam-mary gland. | Sublumbar group abdominal muscles. |
| | 1 Cran iliohypo-gastric N. | Loins, post. abdominal area, lat. thigh | Post parts of abdominal muscles. |
| Thoracic | Last two | Abdominal wall & flank | Abdominal muscles |
| | Mid-thoracic region to last pair | Ant. & vent. parts of abdominal wall | Intercostal mus-cles ant. parts of abdominal muscles. |

Dukes (1955) described the sympathetic chain connecting with the spinal nerves from the first thoracic to the fourth lumbar segment via the white rami which consist of preganglionic fibers that originate in the spinal cord. After these fibers enter the sympathetic chain they traverse up or down the chain and make synaptic junction with nerve cells in several ganglia. The action of an anesthetic solution on preganglionic fibers originating from a particular segment of the spinal cord has been reported to influence ganglia of the sympathetic trunk situated caudal and cranial to the level of the spinal segment and the splanchnic nerves emerging from then (Hall, 1966). It has been reported in man that during subarachnoid anesthesia the sympathetic block is further forward by one to three segments than the level of somatic sensory block; in epidural block the upper level of sympathetic block is four to five segments further forward than that of the sensory fibers (Defalque, 1962).

The parasympathetic nerves in man that have been described to be involved during epidural anesthesia are the vagus and pelvic nerve (Hall, 1966). The vagus nerve is not directly affected by the anesthetic solution, but vagal stimulator effects manifested by increased peristalsis in the small intestines, ascending and descending colon and relaxation of the ileocecal valve are produced as a result of depression of the antagonistic sympathetic system (Brook,

1935). During subarachnoid anesthesia in man a cardiac and bronchial effect may be seen (Collins, 1966). He states that if the cardioaccelerator nerves are blocked, vagal slowing of the heart and bronchial spasm will result.

The pelvic nerve supplying parasympathetic fibers to the descending colon, rectum, and bladder has been reported to be directly affected by an epidural anesthetic solution (Hall, 1966). The sphincter of the bladder does not relax until the sympathetic system is also depressed, because the sphincter of the bladder receives tonic impulses through the sympathetic system and relaxation impulses from the parasympathetic system (Brook, 1935).

(b) Action on Blood Pressure: A decrease in blood pressure has been reported to occur during epidural anesthesia and the extent of the decrease dependent on the number of spinal sympathetic preganglionic fibers that are blocked (Hall, 1966). It has been reported in man that the degree of epidural hypotension is not as great as with subarachnoid anesthesia (Defalque, 1962). However, he demonstrated that in careful clinical evaluation of similar groups of patients anesthetized by subarachnoid and epidural methods a greater decrease occurred (10%) during epidural anesthesia. Due to the decrease in blood pressure vasoconstrictor agents have been employed in local anesthetic solutions to provide some pressor response on absorption (Collins, 1966). However, Ward et al (1965) reports that

a more severe decrease in blood pressure occurs when vasoconstrictor agents are used than when they were not.

The literature concerning this aspect of epidural anesthesia in dogs is subject to much controversy. One of the principal reasons for this confusion is that extensive clinical investigations in this respect seem to have not been carried out in domestic animals (Tufvesson, 1963). He reported that "occasional blood pressure measurements" on dogs indicated that the decrease in blood pressure, when present, will occur in five minutes. Lumb (1963) reported that in the dog the greater splanchnic nerve arises from the twelfth thoracic sympathetic ganglion and if the anesthetic solution passes cranially past the first lumbar segment a serious fall in blood pressure may occur. At the same time it was reported though the anesthetized area extends as far forward as the seventh thoracic segment vasodilation in the anesthetized part will be compensated for by a vasoconstriction in the unaffected regions so that the blood pressure remains unchanged (Tufvesson, 1963). He also reported that should the anesthetized area reach the fourth thoracic segment a moderate fall in blood pressure will occur and if the anesthetic reaches the second thoracic segment the blood pressure will fall suddenly to 40-50% of the initial level.

Eckenhoff (1948) demonstrated hypotension in dogs following epidural anesthesia. The studies were related to

the effects of hypotension on cardiac work and efficiency. In dogs previously anesthetized with a general anesthetic (pentobarbital sodium) hypotension was produced by the epidural injection (2nd or 3rd lumbar interspace) of procaine hydrochloride in doses ranging from 1 to 2 mg/lb. bd. wt.. In 10 to 15 minutes a drop of approximately 20-40% in the blood pressure occurred.

(c) Action on the Heart: Eckenhoff (1948) demonstrated that the cardiac output was reduced about 42% during procaine induced epidural anesthesia. It has been reported in man during epidural anesthesia using 2% lidocine hydrochloride with epinephrine (1/200,000) the heart rate increased 15.8% and when epinephrine was omitted the heart rate increased 6.7% (Ward et al, 1965). He also noted that following subarachnoid anesthesia the cardiac output fell 17.7% while during epidural block the cardiac output increased 30.2%. Epidural block without epinephrine produced a fall of 5.4% in the cardiac output. Brook (1935) reported that if an anesthetic solution reaches the level of $C_7$-$T_5$ and the majority of the preganglionic sympathetic fibers in this area are anesthetized, a reflex slowing of the heart will occur due to unopposed vagal action. He also reported that cardiac venous return is also decreased due to a decrease in inspiratory activity and to a visceral pooling of blood.

(d) Action on Respiration: As an epidural anesthetic spreads cranially the nerves supplying the abdominal and intercostal muscles (Miller, 1964) and the phrenic nerve supplying the diaphragm would be anesthetized (Brook, 1935). It has been reported that if extreme hypotension results, a drastic reduction in medullary blood flow will result; this, in turn can produce respiratory arrest (Goodman and Gillman, 1965). They also reported that such arrest is not caused by direct action of the local anesthetic on the medulla since an injection of 1 ml. of 5% or 0.5 ml. of 10% procaine solution into the fourth ventricle of dogs has no effect on respiration.

## MATERIALS & METHODS

Thirteen mongrel dogs of various ages, sexes, and weights were divided into three groups. Epidural anesthesia was produced in all of the dogs using 2% lidocaine hydrochloride with epinephrine (1:100,000)*. The dogs in group A received 1 ml/5 pounds body weight at an interval rate of 1 ml/30 sec., the dogs in group B received 1 ml/5 pounds body weight at a more rapid rate and group C received excessive dosages of the anesthetic (Table 3). The dogs in group B were given 2% Xylocaine as rapidly as possible to determine if this method would have a greater effect on the blood pressure than the slower injection. The dogs in group C were given excessive dosages because it has been reported that anesthesia up to the foramen magnum has been achieved without serious effects (Jones, 1966).

All of the dogs were examined clinically and were found to be in good health. No laboratory examinations or vaccinations were given because of the short term of the study. The dogs were prepared and handled in the same manner until the epidural anesthetic was administered. The hair over the spinal column was clipped. The prominence of the dorsal spinous processes in the caudal thoracic and lumbar area was marked on the skin. The dogs were

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*Xylocaine, Astra Pharmaceutical Products, Inc., Worchester, Mass.

Table 3. The Sex, Weight, and Dosage of 2% Xylocaine with 1:100,000 Epinephrine Administered Epidurally to the Dogs in Groups A, B, & C.

| Dog Number | Sex | Weight (lb) | Dose of 2% Xylocaine (ml) |
|---|---|---|---|
| Group A | | | |
| 10 | Fe | 15 | 2.5 ml Xylocaine @ .5 ml India ink * |
| 11 | M | 20 | 4 |
| 12 | M | 25 | 5 |
| 13 | M | 25 | 5 |
| 14 | M | 25 | 5 |
| Group B | | | |
| 15 | M | 25 | 5 |
| 16 | Fe | 30 | 6 |
| 17 | M | 50 | 10 |
| 18 | M | 40 | 8 |
| Group C | | | |
| 19 | M | 35 | 17 (died) |
| 20 | M | 25 | 15 |
| 21 | M | 25 | 10 |
| 22 | Fe | 30 | 10 |

* Dog number 10 was given 2.5 ml. 2% Xylocaine @ .5 ml. India ink in order that the cranial spread of the anesthetic could be determined at necropsy.

preanesthetized using a morphine (1.2mg/kg)-atropine (0.032 mg/kg) combination.

One hour following preanesthetic medication the dogs were positioned in right lateral recumbency and the medial aspect of the right thigh was prepared for surgery. Local anesthesia was produced over the femoral artery approximately four centimeters distal to the inguinal area using 2% Xylocaine. The area was draped and the skin incised. The femoral artery was exposed by blunt disection. The artery was catheterized approximately to the origin of the deep femoral artery using polyethylene tubing (Intramedic PE 320)*. Following catheterization the dogs were placed in sternal recumbency and the legs were tied to the table. The head was supported with sandbags. The catheter was connected to a pressure transducer** and flushed with approximately 10 mg. heparin diluted in 100 ml. saline. Systolic and diastolic blood pressure were recorded on a multichanneled oscillograph (Physiograph-Four) via the pressure transducer and a carrier preamplifier† .

The heart rate, determined by the electrocardiogram (ECG), and respiration were monitered by attaching a needle electrode to either side of the chest over the fifth

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*Intramedic (PE 320) Polyethylene tubing, Clay-Adams, Inc., New York.

**Statham (P23H) pressure transducer, Hata Rey, Puerto Rico.

†E & M Instrument Co., Inc., Houston, Texas.

intercostal space midway between the vertebral column and sternum. The electrodes were connected to the Physiograph through an impedance pneumograph* and preamplifier*. A base line for blood pressure, heart rate, and respiration was established.

Local anesthesia was produced over the lumbo-sacral foramen using 2% Xylocaine. After two to three minutes the epidural space was entered using a 20 ga. 3 inch needle with stylet. The needle was inserted almost perpendicular to the spine with the bevel of the needle directed caudally.

A syringe was attached to the needle and aspirated to determine if the subarachnoid space or the lateral venous plexus had been entered.

All of the dogs were given a test dose of one cubic centimeter of 2% Xylocaine to determine if the epidural space had been entered which was indicated almost immediately by dilation of the external anal sphincter. The remainder of the anesthetic was then injected into the epidural space. The dogs in group C, except No. 19, were intubated with an endotracheal cannula after effective respiration had ceased. They were then resuscitated using AMBU resuscitation bag** until spontaneous respiration had begun.

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*E & M Instrument Co., Inc., Houston, Texas.

** Air-Shields, Inc., Hatboro, Pa.

the cranial spread of the anesthetic was noted in all of the dogs. The loss of the following reflexes was used to determine the extent of anesthesia: anal and tail reflex, flexor pinch reflex in the pelvic limbs and the loss of skin crawl and sensitivity over the dorsal and lateral sides of the dogs. These reflexes were tested by pinching the dogs with a hemostat while they were monitered on the Physiograph. The dogs were observed for foreleg extension and after they were removed from the table the patellar reflex was checked.

The dogs in group A were monitered on the Physiograph from 30-50 minutes, group B from 50-60 minutes, and group C from 30-90 minutes. Following the monitering period the catheter was removed from the femoral artery, the artery ligated, and the skin sutured before the dogs were removed from the table. All of the dogs except No. 10 & 19 were allowed to recover from anesthesia.

Within 24 hours following epidural anesthesia the dogs were anesthetized using pentobarbital sodium (13mg/lb). The dogs were positioned on an roentgenographic table in sternal recumbency with the pelvic limbs flexed and the head supported on a styrafoam block. Like needle insertions and positioning as with Xylocaine infussion were utilized.

An aqueous radiopaque solution* (sodium diatrizoate) was injected into the epidural space at the same dosage and

*Hypaque 50%, Winthrop Laboratories, New York.

Table 4. The Dosages of 2% Xylocaine and Hypaque 50% the Dogs in the Various Groups Received.

| Dog Number | 2% Xylocaine (ml) | Hypaque 50% (ml) |
|---|---|---|
| Group A | | |
| 10 | 3 | none* |
| 11 | 4 | 5 |
| 12 | 5 | 5 |
| 13 | 5 | 5 |
| 14 | 5 | 5 |
| Group B | | |
| 15 | 5 | 5 |
| 16 | 6 | 6 |
| 17 | 10 | 10 |
| 18 | 8 | 8 |
| Group C | | |
| 19 | 17 | none** |
| 20 | 15 | 15 |
| 21 | 10 | 10 |
| 22 | 10 | 10 |

*See Table 3.; page 17 for explanation.

**Dog number 19 died approximately 30 minutes after the epidural anesthetic had been given.

rate as the previous anesthetic (Table 4). Dorsal-ventral and lateral radiographs were taken of the entire spinal column as soon as the injection was completed. The aqueous radiopaque media was used to establish a characteristic flow pattern of an aqueous solution in the epidural space.

The dogs were euthanized after the radiographs were completed and necropsies were performed.

## RESULTS AND DISCUSSION

Epidural anesthesia was produced in dogs using 2% Xylocaine. The blood pressure, heart, and respiratory rates were monitered on a Physiograph preceding and during anesthesia.

Level of Anesthesia: The anesthesia produced in the dogs of group's A & B was similar to the type of anesthesia noted during work on clinical cases at Dykstra Veterinary Hospital. Epidural anesthesia using 2% Xylocaine is prompt and lasts from 2 to 2½ hours. The anus begins to dilate almost immediately followed by relaxation of the tail. The flexor pinch reflex in the pelvic limbs is absent in 8 to 10 minutes and the cranial extent of anesthesia is usually achieved in 12 to 15 minutes.

The cranial extent of anesthesia was generally greater in group B than in group A (Table 5). The level of anesthesia was ascertained by pinching the dogs with a hemostat and noting the loss of pain perception or reflex movement. The level of anesthesia in dogs No. 11, 12, and 14 was almost identical. The cranial extent of anesthesia was in line with a transverse plane through the second lumbar vertebra. The level of anesthesia in dogs No. 10 and 13 was similar. It extended to a transverse plane through the third lumbar vertebra. Complete epidural anesthesia was not established in dog No. 10. Thirty-five minutes following

Table 5. The Level of Anesthesia Achieved in the Dogs of Groups A and B. The Level of Anesthesia Corresponds to a Transverse Plane Drawn Through the Indicated Vertebra.

| Dog Number | Level of Anesthesia |
|---|---|
| Group A | |
| 10 | $L_3$-$L_4$ |
| 11 | $L_2$ |
| 12 | $L_2$ |
| 13 | $L_3$ |
| 14 | $L_2$ |
| Group B | |
| 15 | $L_3$ |
| 16 | $T_{10}$ |
| 17 | $T_2$-$T_3$ |
| 18 | $T_9$-$T_{10}$ |

induction of anesthesia the patellar and flexor pinch reflex were present. This dog was given 2.5 ml Xylocaine plus 0.5 ml India ink so that the cranial extent of the anesthetic could be determined at necropsy. Brook (1935) has reported that it is not uncommon to have incomplete or asymmetrical anesthesia occur. The India ink may have caused the incomplete anesthesia and for this reason it was not further used.

A more cranial level of anesthesia was expected in group B than in group A. One of the factors governing spread of a local anesthetic in the epidural space is the speed at which it is injected (Collins, 1966). He reports that the fast spreading anesthetic is not absorbed as quickly by the epidural fat nor does it become fixed with the spinal nerves as quickly. Due to the rapid spread the anesthetic comes in contact with a greater number of spinal nerves and produces a larger area of anesthesia. The level of anesthesia in the dogs of group B was variable. In dog No. 15 the level was in line with a transverse plane through the third lumbar vertebra, in No. 16 the tenth thoracic, in No. 17 the second thoracic, and in No. 18 the ninth thoracic vertebra. The level of anesthesia in dog No. 15 did not compare with the levels of the other dogs in the group. When this dog was given Hypaque 50% the media spread quite far laterally through the intervertebral foramina. This is probably the reason for the level of anesthesia achieved in this dog.

The level of anesthesia noted in the dogs of group C was much further cranially than in the other two groups. It appeared as if complete anesthesia of the spinal nerves from the second cervical segment caudal was obtained. These dogs were given excessive dosages of 2% Xylocaine ranging from 10 to 17 ml. Within 10 to 15 minutes following the induction of anesthesia the dogs were not responsive to being pinched with a hemostat over the entire spinal column. The flexor pinch reflex in the forelegs was absent. Dog No. 19 died approximately 30 minutes following induction of anesthesia due to respiratory arrest. A severe reaction was noted in this dog immediately following the injection of the anesthetic. The dog developed opisthotonus and appeared to loose consciousness. This reaction lasted for approximately 3 to 4 seconds and was possibly due to spinal cord irritation or to a sudden increase in pressure in the epidural space. Dogs No. 20, 21, and 22 were intubated following cessation of respiration and were artificially resuscitated until spontaneous respiration had begun.

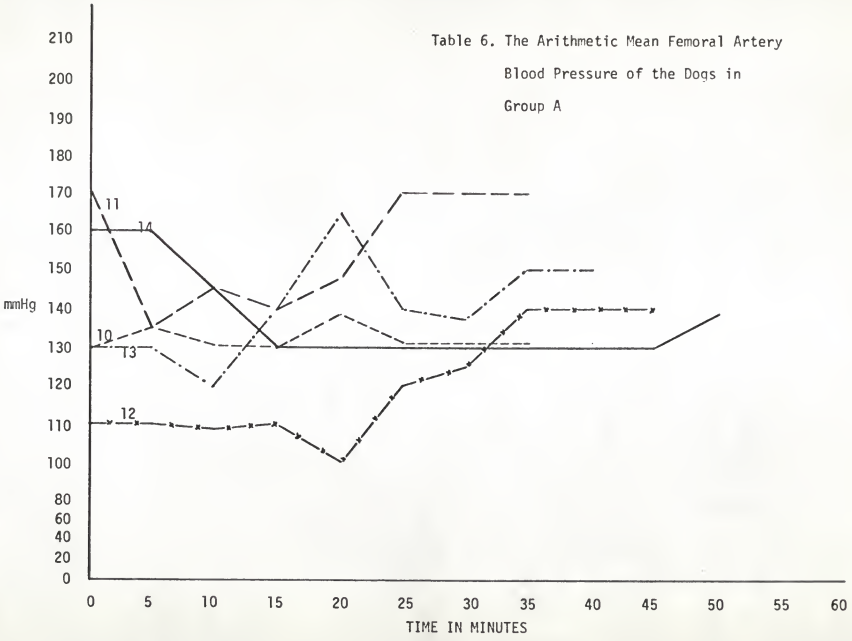
The dogs in group A were quite restless and moved much more than the dogs in group B. They were responsive to sounds and movement by persons they could see. The development of foreleg extension in the dogs of group A (No. 11, 12, 13 and 14) made them uncomfortable and they tried to achieve a position in order to relieve the discomfort. Dog No. 15 (group B) did develop foreleg extension but it was not as

severe as what was noted in group A. The foreleg extension produced in these dogs resembled the Schiff-Sherrington phenomena noted during mid-thoracic injuries to the spinal cord. Foreleg extension has also been noted during routine clinical work at Dykstra Veterinary Hospital. Due to the restlessness and moving by the dogs in group A they were not monitered as long as the dogs in group B. The dogs in group A (No. 10, 11, 12, 13 and 14) were monitered from 35 to 50 minutes, those in group B (No. 15, 16, 17, and 18) from 50 to 60 minutes.

Blood Pressure and Heart Rate: The systolic and diastolic blood pressure of the dogs was monitered via a femoral artery catheter preceding and during epidural anesthesia. The arithemetic mean femoral artery (AMFA) blood pressure was calculated by determining the mid-point between the systolic and diastolic blood pressure on the Physiograph recordings.

The AMFA blood pressure of the dogs in group A (Table 6) decreased an average of 10.4% (range, 0-21%) from the preanesthetic level in an average of 10 minutes (range, 0-20 minutes). Following the initial decrease the AMFA blood pressure increased to above the preanesthetic level in all of the dogs in group A except one (No. 14). The AMFA blood pressure of dog No. 10 approximated 130 mmHg during the recording period. The AMFA blood pressure in dog No. 11 increased from 134 mmHg to 170 mmHg within 25 minutes, dog

Table 6. The Arithmetic Mean Femoral Artery Blood Pressure of the Dogs in Group A



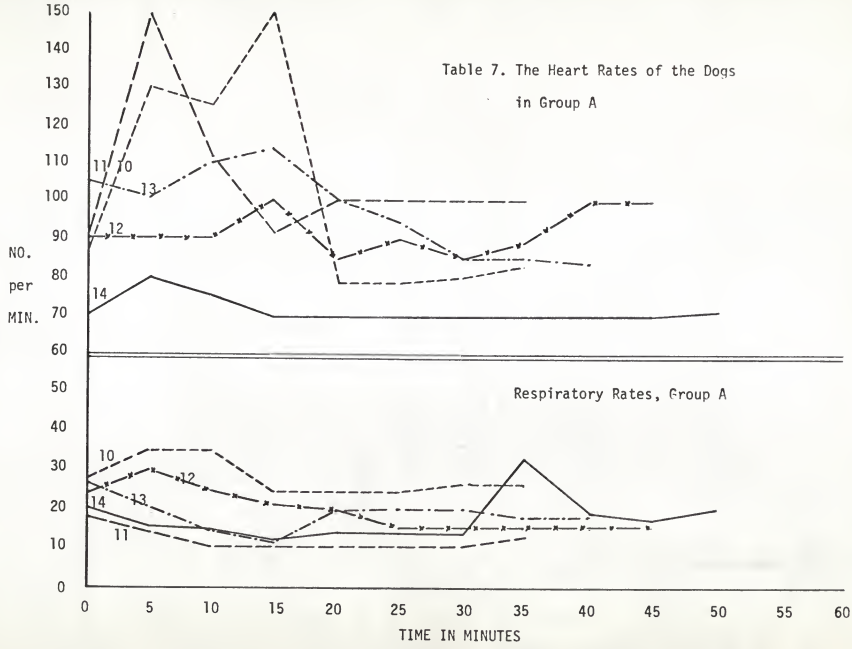


Table 7. The Heart Rates of the Dogs
in Group A
Respiratory Rates, Group A
NO.
per
MIN.
TIME IN MINUTES
11
10
13
12
14
10
12
13
14
11
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
0
5
10
15
20
25
30
35
40
45
50
55
60

No. 12 increased from 100 mmHg to 140 mmHg within 35 minutes, dog No. 13 increased from 120 mmHg to 164 mmHg within 20 minutes. The AMFA blood pressure of dog No. 14 had started to increase when the dog was removed from the table.

The heart rates of the dogs in group A (Table 7) increased an average of 41.6% (range, 9-68%) above the preanesthetic level in an average of 11 minutes (range, 5-15 minutes). Following the initial increase the heart rates decreased to approximately the preanesthetic levels. The initial response in heart rate generally indicated a change compatible with the corresponding blood pressure. The heart rate should decrease as the blood pressure increases.

The AMFA blood pressure of the dogs in group B (Table 8) decreased an average of 13.5% (range, 4-20%) from the preanesthetic level in an average of 10 minutes (range, 5-15 minutes). There was no marked increase in blood pressure following the initial decrease in this group.

The heart rates of the dogs in group B were quite erratic. The heart rate of dog No. 15 demonstrated two sharp increases 5 and 30 minutes following the induction of anesthesia. The heart rate increased from 90/minute to approximately 100/minute. The increases in heart rate were followed by decreases to approximately 65/minute. The heart rate of dog No. 16 increased from 78/minute to 110/minute in 20 minutes, decreased to 90/minute and returned

Table 8. The Arithmetic Mean Femoral Artery Blood Pressure of the Dogs in Group B

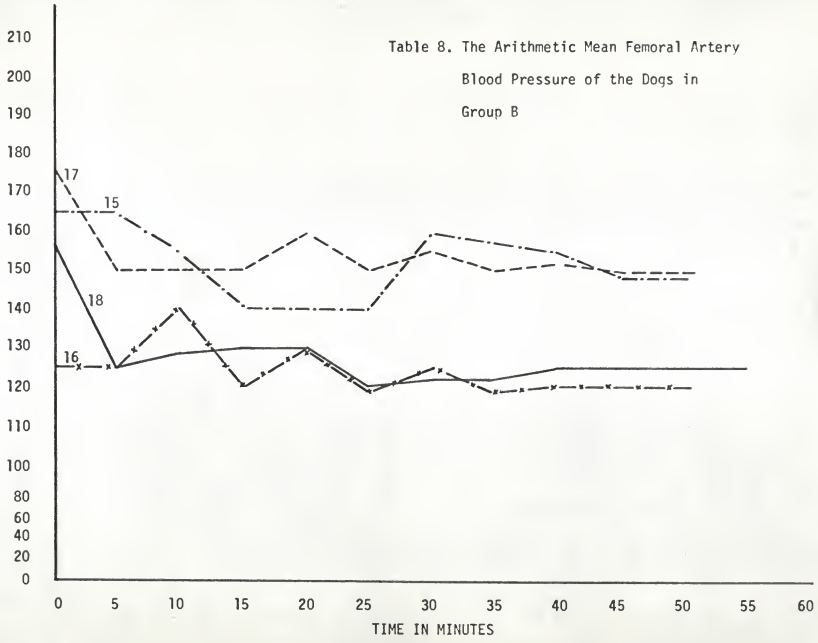
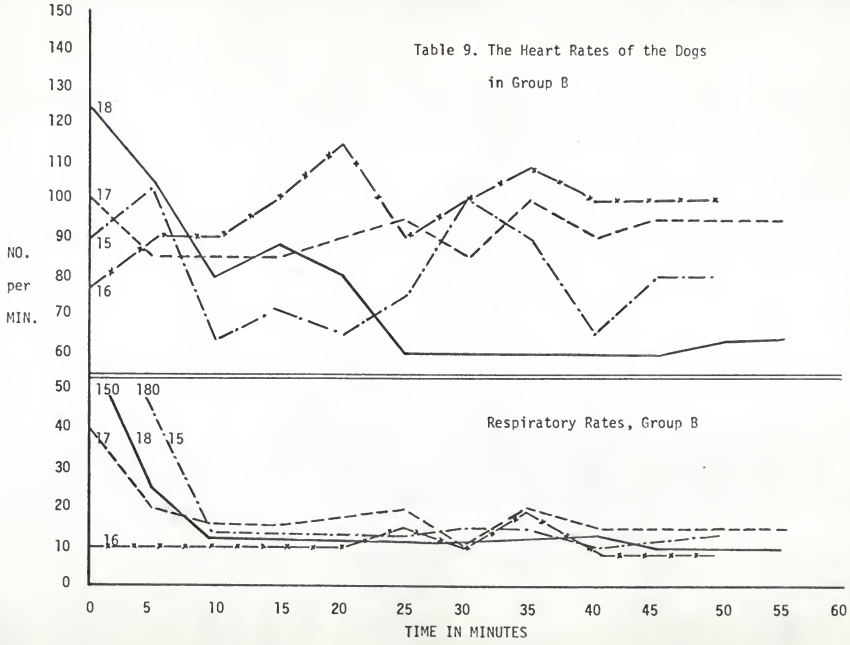


Table 9. The Heart Rates of the Dogs in Group B



to approximately 100/minute. The heart rate of dog No. 17 remained fairly stable. The heart rate of dog No. 18 decreased from 124/minute to 60/minute within 25 minutes. The dog was excited at the start of the study and this would account for the high initial heart rate.

An initial decrease of 8.2% (range, 2-15%) was noted in the AMFA blood pressure of the dogs in group C (No. 19, 20, 21, and 22). The initial decrease occured within 5 minutes in all of the dogs (Table 10). Within 2 to 3 minutes following the cessation of respiration a precipitious drop in blood pressure occurred. Dog No. 19 died within 3 minutes after cessation of respiration due to anoxia. The blood pressure decreased from 170mmHg to 0mmHg within 2 minutes. Following cessation of respiration there was a definite increase in the height of the T wave on the ECG (Appendix). The increase in the height of the T wave is probably indicative of cardiac anoxia. The change in the T wave pattern of the ECG was noted in all of the dogs in this group following cessation of respiration. Dogs No. 20, 21, and 22 were intubated after respiration had ceased and were artificially resuscitated until spontaneous respiration had begun. Dogs No. 21 and 22 were resuscitated for 20 and 30 minutes respectively. Dog No. 20 was resuscitated for approximately 60 minutes. Dogs No. 21 and 22 received 10 ml of 2% Xylocaine while dog No. 20 received 15 ml. The longer length of time that dog No. 20 had to be

Table 10. The Arithmetic Mean Femoral Artery Blood Pressure of the Dogs in Group C

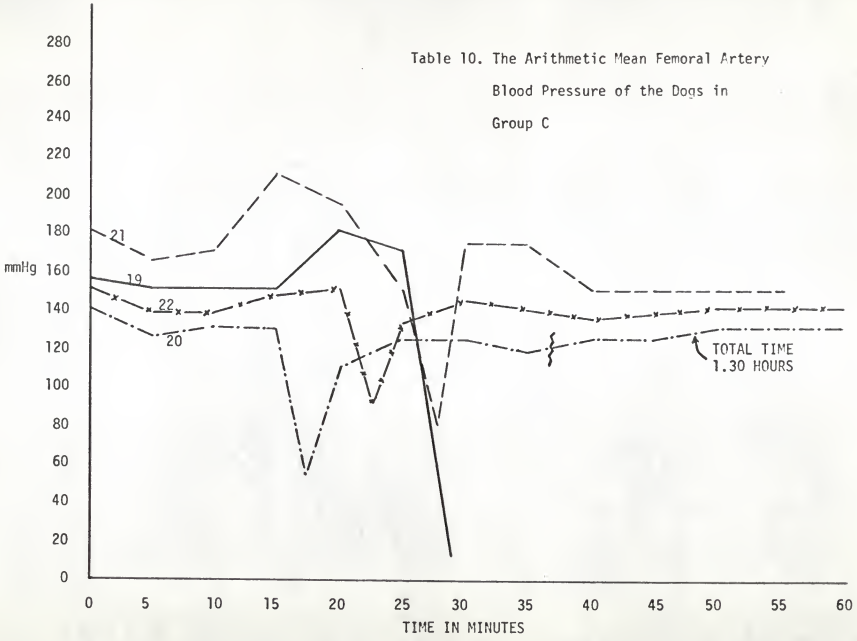
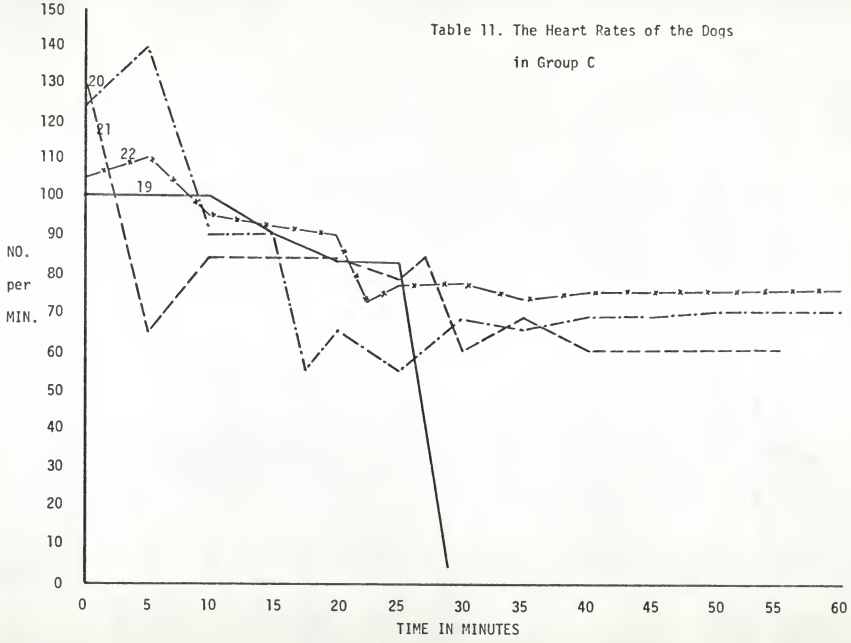


Table 11. The Heart Rates of the Dogs in Group C



resuscitated was probably due to more complete anesthesia obtained from the larger dose of 2% Xylocaine. Following resuscitation the blood pressure returned to approximately the preanesthetic level in dogs No. 20, 21, and 22. The ECG T wave decreased to approximately its initial height following artificial resuscitation.

The heart rates of the dogs in group C decreased following cessation of respiration (Table 11). The heart rate of dog No. 19 decreased from 88/minute to 0 within 2 minutes after respiration had ceased. The heart rates of dogs No. 20, 21, and 22 decreased an average of 45% after respiration had ceased; before artificial resuscitation was started. Following resuscitation the heart rates stablized, however, they were approximately 50% below the preanesthetic levels. The decrease of 50% below the preanesthetic levels is probably due to anesthesia of the cardioaccelerator fibers that originate in the cranial thoracic region allowing for reflex vagal slowing of the heart.

Respiration: The respiratory rates of the dogs in group A (Table 7) decreased an average of 30% (range, 14-44%) from the preanesthetic level in an average of 16 minutes (range, 10-25 minutes). The rates were relatively stable following the initial decrease ranging from 10-30/minute. The respiratory rates of the dogs No. 15 and 18 (group B) were above 150/minute at the start of the study (Table 9). Dog No. 17 initial respiratory rate was 40/minute. Within

10 minutes following induction of anesthesia the rates of these three dogs decreased to 10-12/minute and remained relatively stable through-out the study. The initial rate of dog No. 16 was 10/minute and remained relatively stable until the dog was removed from the table. Respiration ceased within 18 to 26 minutes following the induction of anesthesia in the dogs of group C (No. 20, 21, and 22). The initial respiratory rates were comparable to the rates noted in the other groups. They ranged from 30-18/minute. Dogs No. 21, 22 and 20 were artificially resuscitated for 20, 30, and 60 minutes respectively until spontaneous respiration had begun. Following spontaneous respiration the rates ranged from 10-15/minute.

Radiographic Studies: In order that the flow of a local anesthetic in the epidural space might be studied all of the dogs (except No. 10 and 19) were given Hypaque 50% epidurally. Numerous solutions have been used in order that the flow of an epidural anesthetic might be studied (Moore, 1958). Xylocaine (2%) and Hypaque 50% are both aqueous solutions and their spread in the epidural space should be similar. The radiopaque media was given at the same dosage and rate as the previous anesthetic (Table 4).

In all of the dogs in group A and B (except No. 10, 14, and 15) the radiopaque media spread into the caudal cervical and cranial thoracic region ($C_6$-$T_2$). India ink was noted in the area of $T_1$-$T_2$ at necropsy of dog No. 10.

The cranial extent of the radiopaque media in dog No. 15 was at $T_6$. The media extended to $C_4$ in dog No. 14. The media appeared to fill the epidural space in two of the dogs (No. 21 and 22) in group C (Appendix). The cranial extent of the media in dog No. 20 was $T_3$.

The radiopaque media spread laterally through the intervertebral foramina in all of the dogs in group B and in dogs No. 11 and 20. The majority of the spread was in the lumbar and cervical-thoracic region (Appendix). In dogs No. 11 and 20 the spread in the lumbar area was quite extensive. The lateral spread in this area would reduce the cranial extent of the media. The anesthetic solution would probably spread in a like manner and this would result in a lower level of anesthesia being achieved.

In 5 of the dogs (No. 11, 12, 13, 16, and 17) the radiopaque media did not spread evenly in the epidural space (Appendix). In all of these dogs, except No. 16, there was unilateral spread of the media on the left side of the epidural space. The spread was on the right side in dog No. 16. When the dogs in this study were necropsied it was noted that the epidural fat was not distributed uniformly in the epidural space. The fat may be denser in one area then in another. However, a correlation between the unilateral flow and the location of the epidural fat could not be made at necropsy.

## SUMMARY AND CONCLUSIONS

In this study epidural anesthesia with 2% Xylocaine is prompt and lasts for approximately two hours. The level of anesthesia in the dogs of group A was similar to what has been noted during routine clinical work at Dykstra Veterinary Hospital. The levels of anesthesia in the dogs of group A extended to a plane through the second to fourth lumbar vertebra. These dogs were given 2% Xylocaine at a dosage of 1 ml/5 pounds body weight (1 ml/30 sec). Collins (1966) reported that an anesthetic will spread further if it is given at a fast rate. The dogs in group B were given 1 ml/5 pounds body weight at a rapid rate and the levels of anesthesia extended to a plane through the second thoracic to third lumbar vertebra. The dogs in group C were given 1 ml/2 pounds body weight. It appeared as if complete anesthesia of the spinal nerves from the second cervical, segment caudal was obtained. Based on the radiographic studies and India ink injection the anesthetic solution probably spread as far cranially as the junction of the cervical-thoracic vertebra in 10 of the 13 dogs. The spread was similar in groups A and B. The lower levels of anesthesia produced in the dogs of group A was probably due to slower filling of the epidural space and the anesthetic becoming diluted in the cranial thoracic region.

In 1935 Brook reported that convulsions and unfavorable side reactions may occur if an epidural anesthetic is given rapidly. These reactions were not observed during this study when the routine dosage of 2% Xylocaine was used. Dog No. 19 did evidence opisthotonus and appeared to loose consciousness for 3 to 4 seconds following the rapid injection of 17 ml of 2% Xylocaine. This reaction was probably due to spinal cord irritation or a sudden increase in epidural pressure caused by the injection of the large amount of anesthetic.

Five of the dogs in this study developed foreleg extension 15 to 20 minutes following the induction of anesthesia. The foreleg extension noted in these dogs resembled the Schiff-Sherrington phenomena occuring during the mid-thoracic injuries to the spinal cord.

Tufvesson (1963) reported that during "occasional blood pressure measurements" under epidural anesthesia in dogs a decrease in blood pressure will appear in 5 minutes. The AMFA blood pressure of the dogs in group A decreased an average of 10.4% from the preanesthetic level in an average of 10 minutes. Following the decrease, the blood pressure increased to the preanesthetic levels or above. The increase corresponded to the dogs becoming restless and moving on the table. The heart rates of the dogs in group A increased an average of 41.6% from the preanesthetic levels in an average of 11 minutes. Following the initial increase the heart

rates decreased to approximately the initial levels.

The AMFA blood pressure of the dogs in group B decreased an average of 13.5% from the preanesthetic levels in an average of 10 minutes. The blood pressures of this group then remained relatively stable throughout the monitering period. The dogs in this group were not as restless and did not move as much as the dogs in group A. The heart rates increased to slightly above the initial levels in two of the dogs in this group. The rates decreased in the other two dogs.

There was an initial average decrease of 8.2% in the AMFA blood pressure in the dogs of group C which occurred in an average of 5 minutes. A dose of 1 ml/2 pounds body weight was given to the dogs in this group. One dog in this group died from respiratory arrest. The three other dogs were intubated after respiration had ceased and were artificially resuscitated until spontaneous respiration had begun. Following the cessation of respiration the blood pressure was allowed to decrease 40-50% before resuscitation was started. The blood pressure increased to approximately the previous levels following resuscitation.

The heart rates of the dogs in group C decreased approximately 45% following the cessation of respiration. The rates stabilized following resuscitation, however, they were approximately 50% below the preanesthetic levels.

Blood pressure will decrease during epidural anesthesia. These studies indicate that if a normal dose of anesthetic is used the decrease will possibly be no more than 20% of the preanesthetic level. The speed at which the anesthetic is given should be considered. It would appear, from these studies, that if the anesthetic is given at a fast rate the blood pressure is not able to respond to external stimuli. A serious decrease in blood pressure will likely not occur until anesthesia of the phrenic nerve has produced respiratory arrest. In 1963 Tufvesson reported that a drop of 40-50% in the blood pressure will occur if the anesthetic area extends as far cranial as the second thoracic vertebra.

During epidural anesthesia the heart rate usually increases as the blood pressure decreases. However, if the anesthetic spreads cranially to involve the cardioaccelerator fibers to the heart a reflex vagal slowing of the heart will occur.

In 1966 Jones reported that he had obtained anesthesia to the foramen magnum of a dog without any evidence of toxic symptoms. This observation did not coincide with observations in this study. If the anesthetic spreads cranially and anesthesia of the phrenic nerve is produced respiratory arrest will result. If the dog is artificially resusciated until the effect of the anesthetic has worn off spontaneous respiration will resume.

A radiopaque media and India ink were used to determine the flow of an aqueous solution in the epidural space. Based on radiographic and necropsy evidence these aqueous solutions spread to the caudal cervical and cranial thoracic area in 10 of the 13 dogs. The possibility of an aqueous anesthetic spreading into this area must be considered.

The radiopaque media spread laterally through the intervetebral foramina in 6 dogs while in 5 dogs the media evidenced a unilateral spread in the epidural space.

## ACKNOWLEDGMENTS

The author wishes to express his sincere appreciation to Dr. J. L. Noordsy for his guidance and assistance and to Dr. J. E. Mosier for his advice during this study.

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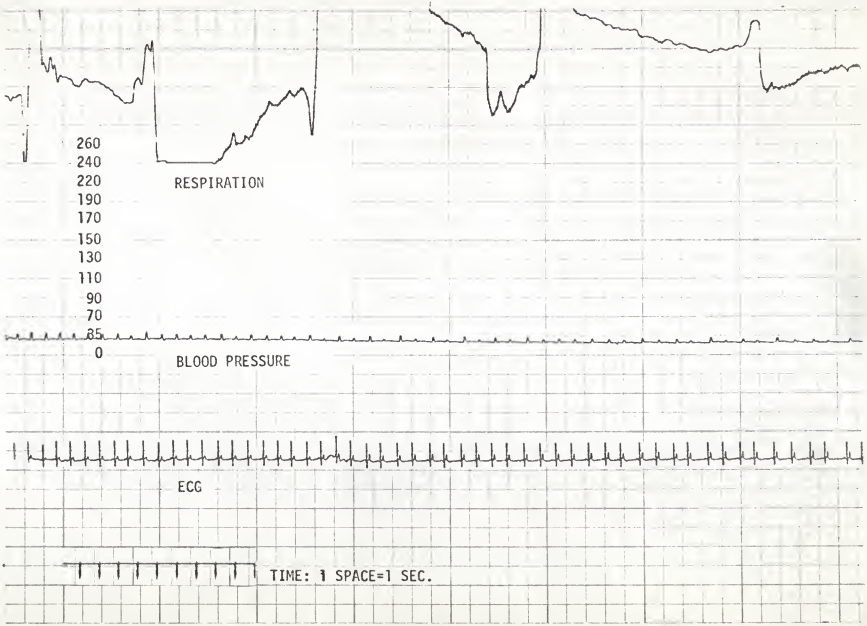
# APPENDIX

EXPLANATION OF PLATE I

Physiograph recording of dog No. 19 two minutes following cessation of respiration. The deflections of the respiratory tracing are due to movement by the dog. The blood pressure is approximately 35 mmHg and the height of the T wave of the ECG tracing has increased. Within 30 seconds following this recording the dog died as a result of respiratory arrest.

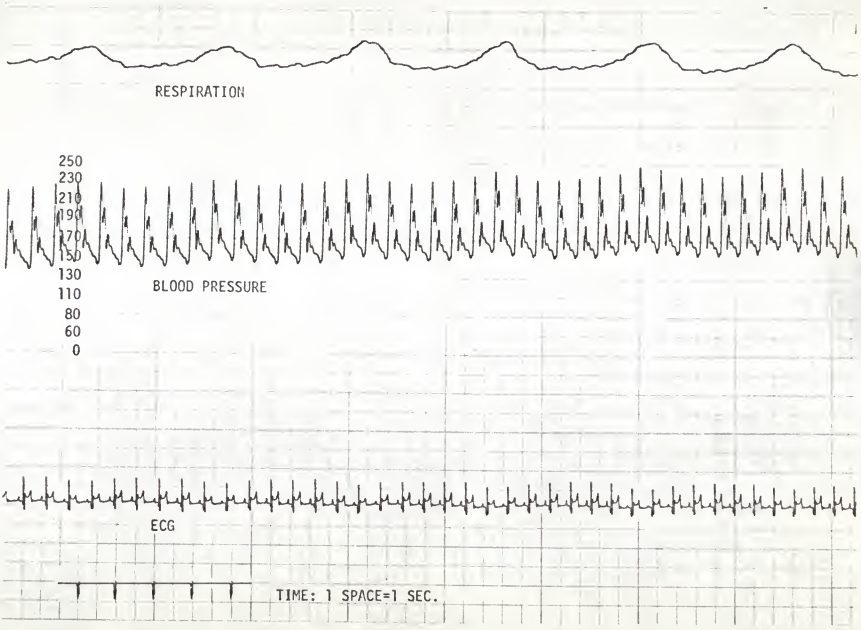
PLATE I


260
240
220
190
170
150
130
110
90
70
85
0
RESPIRATION
BLOOD PRESSURE
ECG
TIME: 1 SPACE=1 SEC.

EXPLANATION OF PLATE II

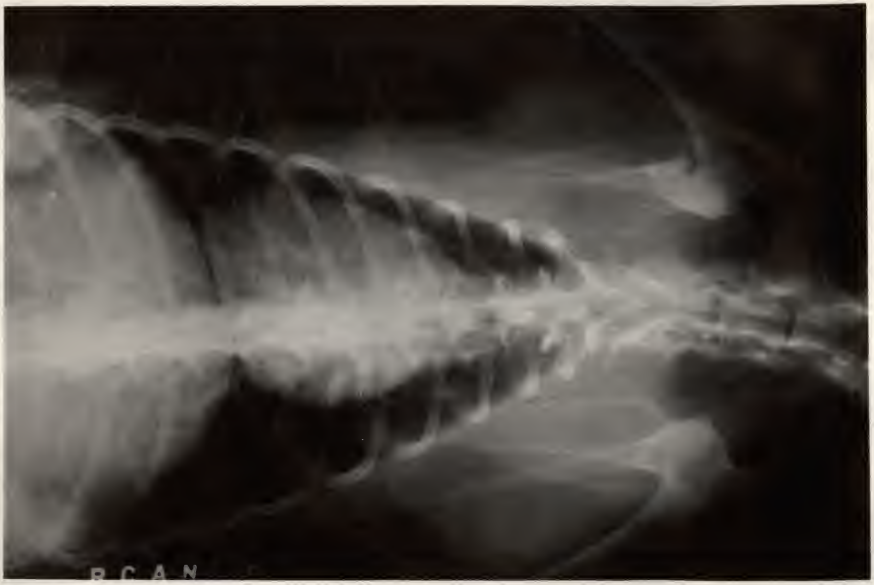
Physiograph recording of dog No. 21 two minutes before effective respiration had ceased. The amplitude of the respiratory tracing has been increased and the blood pressure has increased. The height of the T wave of the ECG tracing has increased.

PLATE II


RESPIRATION
250
230
210
190
170
150
130
110
80
60
0
BLOOD PRESSURE
ECG
TIME: 1 SPACE=1 SEC.

## EXPLANATION OF PLATE III

A dorsal-ventral radiograph of dog No. 14. The radiopaque media has spread as far cranially as the sixth cervical vertebra and there is lateral spread of the media through the intervertebral peramina.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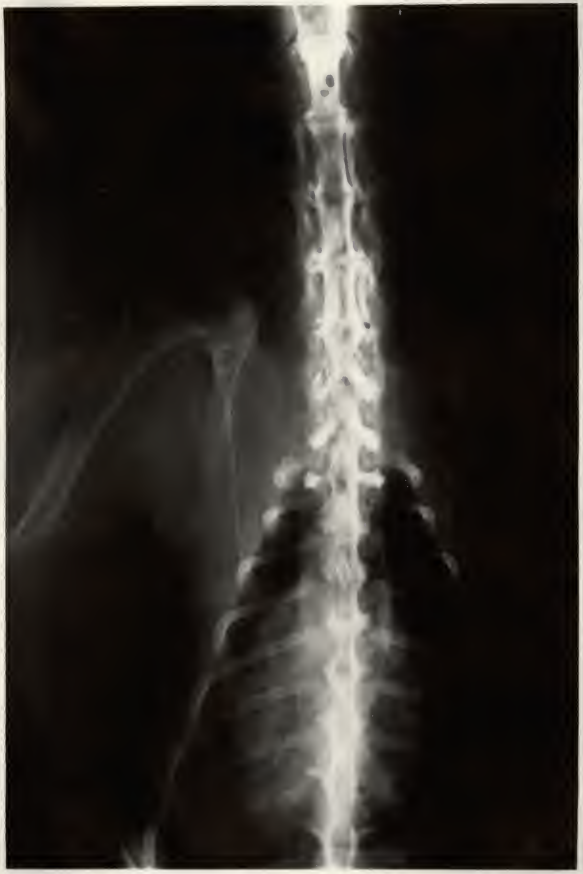
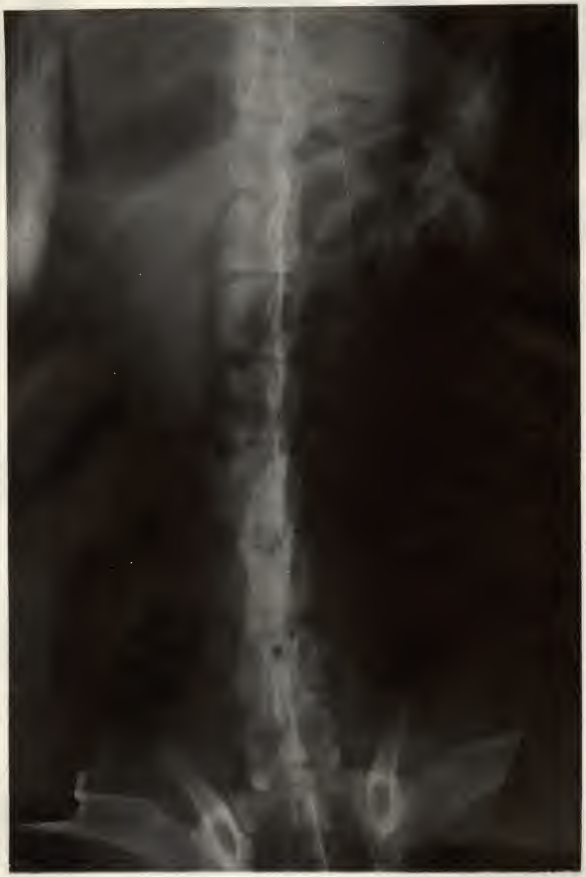
EXPLANATION OF PLATE IV

A dorsal-ventral radiographic of dog No. 21. The radiopaque media has extended as far cranially as the second cervical vertebra and there is lateral spread of the media through the intervertebral foramina.



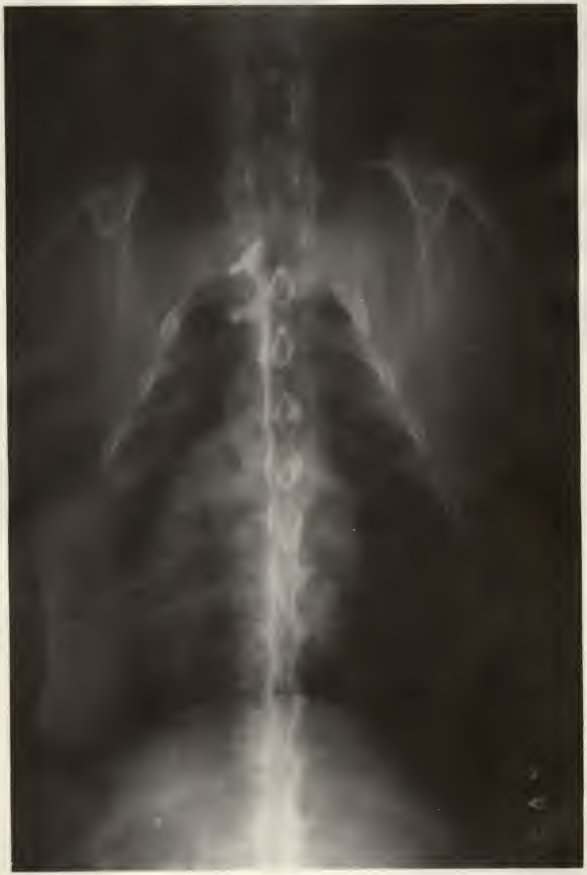
EXPLANATION OF PLATE V

A dorsal-ventral radiograph of dog No. 12. The radiopaque media has spread uniformly from the seventh to fourth lumbar vertebra. From the fourth lumbar to the twelth thoracic vertebra the media is on the right side of the epidural space.



EXPLANATION OF PLATE VI

A dorsal-ventral radiograph of dog No. 11. The radiopaque media has spread as far cranially as the seventh cervical vertebra. From the third thoracic vertebra cranially the media is on the left side of the epidural space. There is lateral spread through the intervertebral foramina.



# A STUDY OF THE EFFECTS OF EPIDURAL ANESTHESIA ON HEART RATE, RESPIRATORY RATE AND BLOOD PRESSURE IN THE DOG

by

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B.S., Texas A&M University, 1964
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AN ABSTRACT OF A MASTER'S THESIS

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requirements for the degree

MASTER OF SCIENCE

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Manhattan, Kansas

1967

Epidural anesthesia has been successfully employed in dogs for a number of years. The indications for an epidural anesthetic are numerous, however, they generally are limited to surgical procedures involving the tissues caudal to the umbilicus.

The effect of anesthesia on blood pressure, heart, and respiratory rates in man has been reported. However, the effect in dogs has been subject to much controversy with veterinary literature containing conflicting reports.

Thirteen mongrel dogs were used to study the effects of an epidural anesthetic on blood pressure, heart, and respiratory rates. The dose of 2% Xylocaine with epinephrine (1:100,000) ranged from 1 ml. per 2 to 5 pounds body weight. The blood pressure, heart, and respiratory rates were monitered for periods ranging from 30 to 90 minutes using a Physiograph-Four following induction of anesthesia.

The average decrease in blood pressure ranged from 8.2 to 13.5% in the period from 5 to 10 minutes following induction of anesthesia. Serious decrease in blood pressure did not occur until respiration had ceased. Following resuscitation the blood pressure returned to a level which approximated the level prior to respiratory arrest.

The effect on heart rates was erratic. The rate ranged from an initial increase of 41% to an initial decrease of 50%. As the level of anesthesia extended cranially the

heart rates decreased. Respiratory arrest was followed by a rapid decrease in heart rate; within 2 minutes the rate dropped to a level which averaged 45% below the rate recorded prior to respiratory failure. Artificial resuscitation produced prompt return of the heart rate to the level immediately preceding respiratory arrest.

The averaged decrease in respiratory rates ranged from 10 to 45% of the preanesthetic levels. The spread of the anesthetic agent into the caudal aspect of the cervical region produced anesthesia of the phrenic nerve and respiratory arrest. Artificial resuscitation prevented serious consequences to the animal.

Aqueous radiopaque media (1 ml. per 2 to 5 pounds body weight) was used to determine the spread of an aqueous solution in the epidural space. In 10 of 13 dogs the media moved cranially to $T_2$ with one instance of spread to $C_4$ and 7 instances of spread to $C_6$. Lateral spread occurred through the intervertebral foramina in 6 dogs. Unilateral spread occurred in 5 dogs.

Conclusions based on this study are: (a) serious decrease in blood pressure and heart rate did not occur until respiration had ceased, (b) in instances where the level of anesthesia did not extend cranially to the mid-thoracic region the blood pressure and heart rate showed positive response to external stimuli, and (c) if

respiratory arrest occurred during epidural anesthesia artificial resuscitation prevented serious effects to the dog.