AIR EMBOLISM OCCURRING

DURING ROOT CANAL THERAPY

by

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INTRODUCTION

Preliminary studies and a case report of air embolism occurring during root canal therapy indicated that pressurized air may enter the general circulation through the vessels around the apices of teeth and eventuate in death. This danger has probably increased with the widespread increase in office air pressures which are necessary to operate air rotohandpieces. Engorgement of the periapical vasculature due to pre-existing inflammation in the periapical area is intensified during root canal procedures. The chances of pressurized air entering the general circulation through these vessels are thus increased. A review of the literature reveals that factors such as the amount of air entering the circulation, air pressure, and the position of the individual play an important role in the consequences due to air embolism. The present study is aimed at obtaining more detailed information on these factors.

REVIEW OF THE LITERATURE

Air gains entrance into the vascular system when it is introduced into a vascular channel under a greater amount of pressure than that in the vessel. Air entry is also possible if the pressure within the vessel is less than the atmospheric pressure, and the patency of the opening is maintained. These conditions are favored in the veins of the neck and root of the arm, such as the internal and external jugulars, the subclavian and the axillary veins. The oblique passage of these veins through several layers of fascia and the firm attachment of their walls to the fascia prevents their immediate collapse even if they are severed completely. During inspiration, pressure within these vessels is negative and air may be sucked in through the open vessel. (1,12,17,18,32) Coles, Richardson and Hall (5) experimentally tested the occurrence of air embolism by exposing the jugular veins of dogs. When these vessels were catheterized, air was seen to enter into the vessels and later caused the death of these animals due to air embolism.

The topic of air embolism has been subjected to experimental work for the last three centuries. Wepfer was probably the first to conduct experiments on air embolism. He is known to have killed an ox of a stupendous size by blowing air with his mouth into the jugular vein. Redi, in 1667 killed two dogs, a horse, a sheep, and two foxes, by using the same technique. Similar studies were pursued by Heydius, in 1683, Camararius, in 1687, Morgagni and many others. All of them concluded that air collects

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in the heart and distends it extensively enough to prevent any further contractions. Bichat, in 1829, injected air into the carotid arteries of animals and concluded that small amounts of air were able to cause cerebral changes leading to death. Rudolph Virchow, in 1846, introduced air into the external jugular veins of cats and a dog, and observed air bubbles in the major veins, including the coronaries, the right heart, and the pulmonary artery. Cerebral vessels were completely free of any air bubbles. He concluded that the air had caused circulatory blocks leading to the death of the animals. Both Virchow, in 1846 and Erichsen, in 1844, noted that the heart continued to contract for a short time even after death. (3,18)

The hazard of air embolism is occasionally seen in some instances where air enters into the circulation due to a traumatic incident or during its intentional introduction into the potential cavities of the body for diagnostic procedures. Goddard and Moffit (15) in their review, list the following specific procedures which have occasionally caused death from air embolism: 1) vaginal insufflation and douching during pregnancy, 2) cesarean section, 3) antral irrigation, 4) attempted abortion, 5) tubal insufflation, 6) urethroscopy with air-inflating urethroscope, 7) introduction of air into cavities or potential cavities, such as the pleural cavity, peritoneal cavity, retroperitoneal or perirenal spaces, bladder, joint cavities, paranasal sinuses and eustachian tube, 8) operations on the breast, thyroid, lungs and prostate, 9) blood donation, 10) blood transfusion, 11) high altitude flying and 12) deep sea diving.

A special mention of air embolism occurring during antral irrigation would be appropriate since the antral area shares the same vasculature as

other areas of the maxilla. Cornfield and Laszo (6) stated that irrigation by puncture of the inferior meatus and injury to the nasolacrimal duct by faulty placement of the irrigation canula might cause temporary blindness due to occlusion of the retinal vessels. The authors mentioned eleven cases reported by Boenninghaus, where air was seen to be carried to the heart via facial and internal jugular veins. They further suggested that the air probably caused death by affecting the breathing centers in the medulla oblongata; however, this was difficult to determine at autopsy. Thickened mucous membranes and increased vascularity facilitated the possibility of such accidents.

In the literature, no cases relating death due to air embolism occurring during a dental procedure are reported; however, Magnin (23) commented on emphysema around the eye of a patient who had air introduced into the root canal of a maxillary anterior tooth. Shovelton (30) has mentioned thirteen cases of emphysema following the use of compressed air to dry root canals. The teeth involved were mainly in the anterior region of the maxilla. Emphysema in the various cases occurred in the cheek, lower eyelid and sometimes in the neck region.

Case Report Leading to this Investigation

A case involving a young patient who died during root canal therapy about two years ago came to the attention of the University of Oregon Dental School Pathology Department. A few minutes prior to death, pressurized air had been extensively used to clear debris and to dry the canal of one of the patient's lower anterior teeth; during this procedure, the air tip was inserted into and held in the canal according to procedures employed by some dentists. Although upon initial consideration it seemed most

unlikely that air sufficient in amount to cause embolic death could gain entrance into the general circulation through the small periapical vessels, this very mechanism was indeed confirmed at autopsy. The pathologist tied off the major vessels, and under water, opened into the right ventricle from which a large quantity of air bubbled forth.

Types of Air Embolism

Two major types of air embolism have been described in the literature. These differ from each other in the site of entrance of the air, its distribution within the blood vessels and its effects. The two types are: 1) arterial air embolism and 2) venous air embolism. In addition to these two a third type, paradoxical air embolism, is occasionally seen to occur. It is a combination of the two major types.

Arterial air embolism. Air enters the pulmonary vein, passes through the left ventricle and is then carried to the systemic arteries which supply the superiorly located portions of the body. A small amount of air may block important arteries like the coronary or cerebral artery. Neurologic manifestations follow cerebral air embolism whereas myocardial infarction occurs if the coronary arteries are involved. This type of air embolism occurs as a complication of the surgical procedures described previously. (4,9,10,13,31)

Venous air embolism. Air enters a systemic vein and passes into the right side of the heart. A bubbling sensation is felt in the chest and, concomitantly, the air present in the right ventricle produces a loud churning sound which is called a "mill-wheel murmur". An air trap is formed in the right ventricle, which obstructs the outflow of blood. Although venous air embolism is a rare complication during surgical pro-

cedures, it may occur if a vein with negative pressure is cut accidentally or when pressurized air is introduced into a vein. (4,9,10,13,31)

Paradoxical air embolism. Air enters a systemic vein and is later carried to the arterial system. It causes manifestations of both arterial and venous air embolism. This type of air embolism may occur in an individual with a patent foramen ovale or open ductus Botalli. Paradoxical air embolism also occurs when the pressurized air is carried to the arterial system through pulmonary arterio-venous shunts. (24,28,31)

Factors Involved in Fatal Air Embolism

Durant, Long and Oppenheimer (9) have stated the following four factors which are closely related to mortality from air embolism. These factors are: 1) the amount of air injected 2) the speed of injection 3) the position of the individual and 4) the effectiveness of the pulmonary excretory mechanism.

Amount of air injected. Injections of large amounts of air into peripheral veins are tolerated by experimental animals, whereas small quantities of air are rapidly fatal when injected into arteries. Repeated experiments by many authors demonstrated that large amounts of air could be injected into veins without fatal consequences. Dr. Hare in 1889 injected 60 ml of air into the jugular vein of a twelve pound dog and concluded that an enormous amount of air was necessary to cause death. (25) Dr. Hare (18) writes "Magendie states that he has thrown, with all the force and celerity of which he was capable, 40 to 50 pints of air into the veins of a very old horse without his dying immediately and Cormack in 1837 blew the contents of his chest, twice filled, into the jugular vein of a horse before the animal exhibited signs of uneasiness.

Barthelmy has found that in six horses, weakened greatly by the withdrawal of blood, as much as four to six litres of air must be introduced intravenously to cause death and estimates, in consequence, that a man weighing 136 pounds would be killed only by two-thirds of a litre." Richardson et al., (29) injected air into a dog at a slower rate --- 3,910 ml of air over an 87-hour period before the animal was found dead. Van Allen, et al., (32) have mentioned the experiments of many authors who injected air into the peripheral veins of various animals. A dog could withstand as much as 2,000 ml of air when injected into a jugular vein, in the experiment of Ilyin; Jehn and Nageli noted that in twenty five rabbits, the maximum tolerance of air was 0.89 ml per kilogram of body weight; in six cats, it was about 2.5 ml per kilogram of body weight. They reported a dog dying from 7.7 ml of air per kilogram of body weight. The authors concluded that different species present widely different tolerances and that among individuals of a single species some variation exists. Harkins and Harmon (19) injected air into the femoral vein of dogs and found the minimal fatal dose to be about 8 ml per kilogram of body weight. Wolffe and Robertson (33) injected air into the femoral vein of rabbits and dogs with lethal doses of air estimated to be about 0.5 and 15 ml per kilogram of body weight, respectively. The authors state that the amount necessary to produce death, when injected intravenously differs with each animal and further, that it seems to be directly proportional to the size of the pulmonary artery and its branches.

In general it seems that large amounts of air are needed to be injected intravenously to cause death; however, crossing over of pressurized air from the venous to the arterial circulation may be possible.

Under such circumstances, the arteriovenous shunts may open to aid such transport. Durant et al., (12) pointed out that deaths due to venous air embolism are occasionally reported in the literature. The reported cases probably constitute a relatively small proportion of the actual number of such accidents and the importance of this hazard should be well understood. The authors further state that relatively small amounts of air, probably as small as 40 ml, can be fatal to a person in poor health.

In arterial air embolism a small amount of air can effectively block a medium sized artery and serious consequences may result if a few ml of air are carried into either the cerebral or coronary circulation, or both. Studies of Durant and Oppenheimer have shown that as much as 0.05 ml of air may cause death in a dog. Van Allen et al., (32) found that the dog's maximum tolerance of air injected into the pulmonary vein was only 1.5 ml per kilogram of body weight. A total of a few ml of air caused dramatic changes in the vital and neurological signs. (22) Geoghegan and Lam (14) injected air into the left ventricles of dogs, with the animals dying of venticular fibrillation after the injections of 1.5 ml of air per kilogram of body weight. Van Allen et al.,(32) stated that Wever introduced air into the carotid arteries of dogs and monkeys which resulted in ischemic necrosis of the cerebral cortex. That air may be carried from the arterial to the venous system has been shown in the retina, pia mater, coronary circulation, lower trunk and lungs. (13)

Speed of injection. Death may not occur if large amounts of air are injected slowly over a long period of time. Durant, Long and Oppenheimer (9) introduced intravenously a total of 1,000 ml of air into dogs in amounts of 100 ml every five to ten minutes. They state that not all

animals, however, survive such procedures. Fries et al., (22) compared slow with rapid injections of air into the carotid arteries of dogs --- the immediate changes following the slow injections were often negligible, whereas the vital and neurologic changes were more dramatic after rapid injections. Richardson et al., (29) likewise found that the rate of injection was of great importance. They injected 1,377 ml of air into a 46 pound dog over a period of 460 minutes before it was found dead. Conversely, Wolffe and Robertson (33) felt that the speed of injection was of little significance, since, no matter how slowly injected, the air would accumulate in the pulmonary artery and its branches to eventually cause death. They felt also that the amount of air necessary to cause death is proportional to the size of the pulmonary artery and its branches.

Position of individual. Durant, et al., (12) were the first to advocate the vital importance of body position during air embolism. They found that during venous air embolism, the animal can tolerate more air when lying on the left side as compared to its tolerance in any other position. Furthermore, if the animal lying in any other position with severe circulatory disturbances, were changed to the left lateral position, the chances of its recovery were immensely increased. Under these circumstances, a dramatic return of forceful right ventricular contractions was noted. According to the authors, right lateral position is by far the worst of all. The pulmonary artery is then superiorly located and buoyant air lodges in the right ventricle just before it exits to the pulmonary artery causing an effective blockade to the outflow tract of the right ventricle. Oppenheimer, et al., (26,27) injected air in the amounts of 5.0 ml/kg and 7.5 ml/kg each to two groups of dogs in right lateral

position, with a survival rate of twenty two per cent respectively for the two groups. In the open chest experiments performed by these authors, they noticed that the right ventricle labors against the obstruction of an air trap in its outflow tract when the animal is lying on its back or right side. When the animal is turned onto the left lateral position, the outflow tract assumes a position inferior to the body of the right ventricle. The air trap disappears from its inferior position and is presumably churned into a froth which is mixed with the blood in the right ventricular cavity. The obstruction to the circulation is thus relieved and blood can be propelled into the pulmonary vessels by the right ventricular contractions. Presumably the froth is gradually carried from the right ventricle to the lungs where its excretion can take place. (9) Oppenheimer, Durant and Lynch (26) presented the favorable effect of the left lateral position in their experiments on the dogs. The mortality rate in dogs injected with 7.5 ml of air per kilogram of body weight was almost twice as high in the animals in supine position as compared to those in left lateral position. The favorable influence of left lateral position was also noticed within a very short time when the animals injected initially in the supine position were turned to the left lateral position. Durant et al., (12) and Dotter (8) have offered roentgenographic evidence of two accidental cases of air embolism which occurred during an angiocardiac procedure. The patients regained consciousness when they were turned to the left lateral position. Thus the left lateral position appears to be an important therapeutic measure if a case of venous air embolism is swiftly diagnosed.

The position of the body is much less important as a preventive

measure in cases of arterial air embolism. The head down position will definitely prevent the cerebral involvement if the patient assumes this position while embolism occurs; however, clearing of the cerebral manifestation would not occur even if he were to assume a head down position after emboli lodge. Furthermore, such a position can never prevent the involvement of the coronary arteries. (10

Effectiveness of the pulmonary excretory mechanism. Durant et al., (10) state that tachypnea (rapid and shallow breathing) which accompanies air embolism, acts as a protective mechanism and tends to prevent death since it helps effectively in clearing the pulmonary gases. It is interesting to note that tachypnea does not occur when the animals are placed in the left lateral position. The authors have given no explanation of the absence of tachypnea with animals in the left lateral position.

Effects of different types of gases as embolic agents. Gases such as air or oxygen are often deliberately introduced into the potential body cavities in clinical diagnostic roentgenology for contrast purposes. Procedures such as pneumoperitoneum, pneumothorax, and insufflation of the urinary bladder may be specifically mentioned in this regard. The hazards of gas embolism are particularly present in such procedures. Harkins and Harmon (19) injected air and oxygen into the femoral veins of dogs to compare the minimum fatal dose of these gases. Their results indicated that the minimum fatal dose of oxygen was no greater than that of air. No marked difference in toxicity was noted. Moore and Braselton (25) injected air and carbon dioxide directly into the pulmonary veins of cats with coronary artery embolism occurring in every animal. Injections of air measuring up to one-fourth ml per pound of body weight were tol-

erated without severe disturbance of normal heart functions. The authors stated that this would amount to 37.5 ml of air into a pulmonary vein of a man weighing 150 pounds. Doses of air exceeding ½ ml per pound of body weight were regularly fatal, while repeated injections of carbon dioxide in doses as large as 3.0 ml per pound of body weight were well tolerated. Carbon dioxide did not cause stable coronary emboli and was dissolved in the blood within fifteen to twenty seconds because of its great solubility. The authors, therefore, suggest the use of carbon dioxide for practical applications. Kunkler and King (22) compared the minimum lethal doses of air, oxygen and carbon dioxide by injecting these gases into the left ventricles of dogs and mice. The comparison of air, oxygen and carbon dioxide embolization revealed oxygen to be tolerated about two times and carbon dioxide about five times as well as air. Stauffer, et al., (31) found that intravenous injections of carbon dioxide in large amounts (7.5 ml/kg) caused only mild transient physiological alterations in dogs. They advocated the use of this gas for diagnostic roentgenology. Oppenheimer et al., (27) injected carbon dioxide a) intravenously, b) into the left heart and c) into the peripheral end of the carotid artery. Minimal cardio-respiratory changes were noted when the gas was introduced on the right or the left side of the circulation. Body position was not a factor in mortality and morbidity when carbon dioxide was used for contrast visualization. Graff et al., (16) found that air was approximately five times as toxic as carbon dioxide as an intravenous embolic agent. It seems that the differences in toxicity of these gases are due to their differential solubilities.

Signs and Symptoms and Causes of Death

Venous air embolism. Totally different clinical pictures are seen in the venous and arterial types of air embolism. In the venous type, the air in the right ventricle produces a loud churning sound which can be heard without using a stethoscope, and it is known as "mill-wheel murmur". The murmur appears immediately after the entrance of air into the circulation. Venous pressure rises to a marked degree and cyanosis becomes rapidly apparent. These symptoms are followed by a fall in blood pressure, rapid thready pulse, and syncope due to cerebral ischemia. In addition to the above mentioned changes, pallor of the tongue, petechial hemorrhages of the skin, air hunger, and chest discomfort are seen to occur frequently in patients with venous air embolism. (9)

Arterial air embolism. In the arterial form, the clinical picture depends upon ischemia in vital organs especially the heart and brain when the head is at a higher level than that of the arch of the aorta. Air usually enters the cerebral circulation and causes various neurological manifestations, such as aphasia, blindness, hemiplegia, monoplegia, nystagmus, loss of eyelid reflexes, dilatation of pupils, complete flaccidity and convulsions. (9,10,31) Fries et al., (13) noted similar neurological manifestations in dogs who received 1 to 3 ml of air per kilogram of body weight into the carotid artery over a period of thirty to sixty seconds. Two of the sixteen dogs who survived this procedure for three months remained neurologically defective, apathetic, and lethargic and had varying degree of paresis. These changes were also noted in the dogs who received 0.33 to 2 ml of air per kilogram of body weight over a period of one hour. Some of these dogs in addition, walked in circles and were apparently blind.

Moore and Braselton (25) injected air into the pulmonary veins of cats and concluded that death occurred mainly due to the obstruction of the coronary arteries. 0.5 ml per pound of body weight caused a typical coronary death. Smaller doses were followed by complete recovery. Body position was seen to have no relation to the causation of coronary emboli. Within a matter of two to four minutes after coronary obstruction, dilatation of the heart and ventricular fibrillation leading to death was noted in these animals. The heart behaved exactly as it does when the coronary arteries are ligated. Kent and Blades (21) noticed similar changes occurring after injecting 1 ml of air into the pulmonary veins of dogs.

After the entrance of air into the coronary vessels, weak and rapid contractions of the heart and dilatation of the ventricles were followed by ventricular fibrillation and death in all the animals. (21) Air gaining direct or indirect entrance into the coronary arteries produces ischemia of the myocardium in the areas supplied by the involved vessels. This ischemia is demonstrable grossly and on electro-cardiographic examination. Durant et al., (10) have mentioned about five specific features associated with arterial embolism. These are: a) Presence of air in the retinal vessels by opthalmoscopic examination, b) Liebermeister's sign -- sharply defined areas of pallor in the tongue. Liebermeister believed that this sign was not only an early one but also a constant one in cases of arterial air embolism. He further stated that the presence of this sign was almost a certain indication of air embolism and conversely its absence was a clear indication that an air embolism was absent., c) Marbling of the skin caused by air in the skin vessels

especially seen in superiorly located areas, d) Air bleeding -- air bubbles out when the affected skin areas are incised, and e) roentgenologic demonstration of the presence of air in the cerebral vessels (rarely seen).

Treatment

Various therapeutic measures have been advocated by many authors. Although immediate institution of such measures may save the patient's life, prevention is the best procedure. Some authors advocate only careful and limited use of air as a diagnostic tool with the use of more soluble gases such as oxygen or carbon dioxide whenever possible. Further, the ingress of air may be prevented by compression or ligation of the major blood vessels. (2,7) Blair and McGuigan suggested five important remedies for accidental cases of air embolism. These are: 1) prevention of admission of air by compression and ligation or both 2) venesection 3) cardiac stimulants 4) direct aspiration of heart and infusion of saline solution and 5) catheterization and aspiration of the right atrium. (2) Dible et al., (7) stated that air should be contra-indicated for use during inflammatory stages. Durant et al., (11,12) and Oppenheimer et al., (27) have shown the importance of left lateral position as a life saving maneuver. These above mentioned measures are described almost invariably in papers on the subject of air embolism.

MATERIALS, METHODS AND FINDINGS

Selection of the Animal

For carrying out experiments on air embolism through the root canal, animals with fairly large teeth are preferable. Considering the clinical applications of the problem, animals such as dogs, monkeys, or pigs should be acceptable. The dog seems to be especially suitable since most published investigations on air embolism were performed on dogs. It is thus easier to compare the results of this research with the many previous experimental findings. Also, dogs are less expensive than monkeys or pigs, they are readily available and need no elaborate care. And finally, the circulatory system of the dog is fairly similar to that of the human.

It is necessary at this stage to make a brief comment on the dentition of the dog and related portions of its blood vascular system which is important from the experimental point of view. The dog has two dentitions, with the first or temporary teeth appearing at the age of three to five weeks. These are replaced at varying intervals from six to eight months by permanent teeth. Dogs have a total of 42 teeth including twelve incisors, four canines, sixteen premolars and ten molars. Their dental formula may be expressed as: 2(I 3/3,C 1/1,P 4/4,M 2/3)=42. The lower teeth of a dog are supplied by the inferior alveolar artery and its branches; the maxillary teeth are supplied by the maxillary artery and its branches. The venous system more or less corresponds to the arterial system. Ultimately all the veins in the region of head and neck join to form the jugular vein on either side which opens into

the superior vena cava. The dog's heart is a four-chambered organ and is very similar to the human heart. (20)

Anesthesia

The dog was placed on a table and the hind legs were shaved with the animal hair clipper. The course of a leg vein was noted and a rubber tourniquet was applied on the proximal part of the thigh. The dog was held firmly for ease of intravenous injection. The tourniquet was released just prior to the injection of the anesthetic, Veterinary Nambutal, into the vein. During the initial phases of the experiment, considerable difficulty was encountered with anesthesia and required much trial and error before arriving at an adequate working dosage. On some occasions it was found that two dogs of the same body weight would need different amounts of Nembutal to attain the same level of anesthesia.

Adequate anesthesia was usually obtained by injecting 60 mgm of Nembutal per five pounds of body weight. This dose has been recommended for both males and females for routine surgical procedures. Sometimes it was necessary to administer additional amounts of anesthetic during the experimental procedure. This surgical anesthesia was established within four to eight minutes and lasted for about one hour, which allowed adequate time for the experimental procedure. In the event that more anesthesia was required a second injection, approximately one-quarter of the first dose, was given as soon as the animal showed signs of recovery during the experimental procedure.

Experimental Design and Findings

Group 1 (procedures 1-7) The procedures followed in these initial experiments were hopefully designed to afford the greatest possibility of air entering into the systemic circulation from the periapical regions of dogs' teeth. Seven experiments were performed in this series each with essentially the same procedure, unless otherwise noted. In each instance the anesthetized animal was placed in the right lateral position, since the reports in the literature indicate that this position is extremely hazardous. The dog's mouth was kept wide open by means of a mouth prop and the tongue retracted. The upper half of the crown of the selected tooth was removed with a rotating carborundum disc under a continuous water drip. Care was taken not to traumatize the adjacent teeth or the soft tissues around the tooth. The pulp, which usually bled freely, was removed with broaches. The root canal was widened with a reamer and was occasionally flushed with water. A canula was prepared by blunting a twenty-one gauge hypodermic needle, which was connected to an air valve by a plastic tube. The air pressure was measured by means of a pressure gauge. The canula was then inserted into the root canal and the initial respiratory and cardiac rates recorded. Air under 40 to 50 pounds of pressure per square inch was blown into the root canal through the canula. Any changes, as well as the cardiac and respiratory rates, were recorded by observation, palpation and auscultation. If the dog expired, an autopsy was performed in an attempt to ascertain the cause of death. As part of the autopsy, the thoracic and coronary vessels were observed in-situ, then the great vessels of the heart were tied off and the heart removed and placed

under water, and the right ventricle was then punctured. If the respiratory and cardiac changes were not remarkable for a period of 35 to 45 minutes, the experiment was discontinued. The animals were either allowed to recover or were sacrificed.

Summary of findings of Group 1 (procedures 1-7) The variable findings of Group 1 are summarized in Table 1. Four dogs died due to air embolism, revealing marked cardio-respiratory changes before death, together with varying degrees of emphysema in areas such as the tongue and sublingual tissues, the facial, and the periorbital region. Electrocardiographic and femoral arterial blood pressure tracings were recorded during experimental procedure Number 5, in which a dog died due to air embolism. These tracings were consistent with a typical embolic death.* Two animals which did not die revealed variable emphysema in similar locations as occurred in those animals which did die. These dogs also revealed mild cardiac and respiratory changes. During experimental procedure Number 6, air was simply blown over the cleaned root canal and here only minimal increase in the cardiac and respiratory rates was noted. The dog in procedure Number 1 was the only animal not demonstrating any detectable changes. The dog used in procedure Number 3 was fairly old and the experimental tooth had a narrow root canal. The minimal amount of air which entered into the periapical area however, did cause an extensive emphysema of the tongue and floor of the mouth. (Figures 7,8)

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* Diagnosis confirmed by Dr. Kurt R. Straube, Department of Radiology, University of Oregon Medical School

TABLE 1. SUMMARY OF THE EXPERIMENTAL DESIGN AND FINDINGS OF THE SEVEN PROCEDURES IN GROUP 1

1) All the animals were placed in the right lateral position.
2) The mandibular canines were used (except in experiment 7 -- upper canine was used, and in experiment 3 -- left lower first premolar was used).
3) Air pressure varied between 40 to 50 pounds per square inch.
4) Air was injected continuously into the root canal (except in experiment 6 -- air was blown intermittently).
5) Air seals to the teeth were fair to poor.

Experimental Procedure Number	Time of air Administration (minutes)	Body Weight of the Dogs (pounds)	*Multiple Clinical Changes	Death or no Death	Pallor followed by cyanosis of the tongue
1	45	35	Absent	No Death	Absent
2	8	29	Present	Death	Present
3**	35	39	Present	No Death	Absent
4	6	26	Present	Death	Present
5	11	25	Present	Death	Present
6	40	28	Present	No Death	Absent
7	34	28	Present	Death	Present

* Includes emphysema (facial, periorbital, lingual, or floor of mouth) and/or cardiac and respiratory changes.

** appeared older than most of the other youthful animals.

Essentially, the variables in this preliminary group covered a wide exploratory range and used the body position described as most dangerous in the literature. For details on each individual procedure, see Appendix 1.

Autopsies performed on the dogs dying of air embolism revealed large air bubbles in the major thoracic vessels. (Figures 13,14) The heart was distended. A frothy mixture of air and blood was visible through the thin wall of the right atrium. When the right ventricle was punctured under water, air bubbled to the surface. Minute air bubbles were noted in the coronary vessels. (Figure 12)

Group 2 (procedures 8-16) This group differed from Group 1 in that the parameters such as body position, tooth sectioning, air pressure and techniques of sealing and administration of air were standardized and refined and tended to simulate the most hazardous conditions during which dental treatment could lead to air embolism.

Of the various possible body positions, a sitting position was selected and each animal in this group was seated in a specially constructed chair. This chair consisted of a base, movable platform, and a back rest. (Figure 4) The animals were carefully tied to the chair and care was taken to prevent any abnormal pressure over the thorax and abdomen which would disturb normal function. The head of the animal was kept vertical by winding adhesive tape around the snout and the metal support of the chair. The ears were also attached to the support with adhesive tape for additional stability.

A constant air pressure of 35 pounds per square inch was employed

in each case following a survey of air pressures used in twenty local dental offices in which a mean pressure of 35 pounds per square inch was determined to closely simulate the average office condition.

The amount of air under 35 pounds per square inch delivered through the canula within a ten second period was measured by a water displacement method. The air conducting system, consisting of a pressure gauge, pressure regulator, a one way valve and a canula was connected to a bottle containing water. The air under 35 pounds per square inch pressure was introduced into the bottle for ten seconds. The air displaced the identical volume of water which passed through a side arm and was collected in a measuring jar. This volume of displaced water was measured by repeating the same procedure ten times. The average amount of water displaced was found to be approximately 1,000 ml varying between 990 to 1,010 ml. It was therefore inferred that a volume of about 1,000 ml of air was delivered through the canula within ten seconds at a pressure level of 35 pounds per square inch.

The left maxillary third incisor was selected arbitrarily for this portion of the study. Technically this tooth was thought to be more suitable for root canal procedures than the mandibular teeth used in Group 1. In addition, the maxilla offered a more stable working area than did the mandible, and also provided the hazards mentioned by Cornfield and Laszo in regard to antral air embolism and damage to the eye and related structures. (6)

The crown of the selected tooth was removed by a rotating carborundum disc with water spray. After removing the pulp, the root canal was initially widened by a reamer, and further widened with a small drill

mounted on a brest drill. The walls of the root canal were then threaded by using a tap mounted on a tap handle. Care was taken to prevent fracturing the tooth and clogging the root canal. A canula with the tip threaded externally was screwed into the root canal. (Figures 2,3,6) A tight fit between the canula and tooth was thus obtained and an additional precaution, quick-setting acrylic was used in an attempt to prevent leaks at the juncture of the canula and the tooth.

The beginning respiratory and cardiac rates were recorded and then air, under 35 pounds of pressure per square inch, was blown into the root canal through the canula initially for ten seconds. This was followed by repeated ten or fifteen second periods of administration of air at five to ten minute intervals depending upon the clinical reaction of each animal. The total time of air administration was recorded. Any clinical changes, as well as the respiratory and cardiac rates, were noted. The animals which survived the experimental procedure were sacrificed, and routine autopsies were performed. Various organs including the brain, heart, lungs, and jaws were grossly examined and placed immediately into fifteen per cent formalin solution for fixation. After fixing the tissues for three days, they were sectioned. The anterior portion of the maxilla was decalcified and split in the mid-line. One of the halves contained the experimental tooth while the other half was used as a control. All the sections were stained with hematoxylin and eosin. Kodachromes and x-ray records were made whenever these were deemed appropriate.

Summary of findings of Group 2 (procedures 8-16) The findings of Group 2 are summarized in Table 2. These findings are similar to those

TABLE 2. SUMMARY OF THE EXPERIMENTAL DESIGN AND FINDINGS OF THE NINE PROCEDURES IN GROUP 2

1) All the animals were placed in the sitting position.
2) The maxillary third incisors were used in all cases.
3) The air pressure was kept constant at 35 pounds per square inch.
4) Air was injected into the root canal for ten seconds, the animal observed for five minutes. If no death, the sequence was repeated five times. Then air was injected for fifteen seconds at five minute intervals until conclusion of the experiment.
5) The air seals to the teeth were generally good.

Experimental Procedure Number	Total Time of Air Administration (minutes)	Body Weight (pounds)	*Clinical Changes	Result of the Experiment	Pallor followed by Cyanosis of the Tongue
8	0.167 minutes (10 seconds)	24	Present	Death	Present
9	3 minutes	30	Present	No Death	Absent
10	2 minutes	28	Absent	No Death	Absent
11	10 minutes	31	Present	No Death	Absent
12	1.167 minutes	28	Present	Death	Present
13	10 minutes	34	Absent	No Death	Absent
14	0.167 minutes (10 seconds)	23	Present	Death	Present
15	0.333 minutes (20 seconds)	26	Present	Death	Present
16	15 minutes	35	Absent	No Death	Absent

* Includes emphysema (facial, periorbital, lingual, or floor of mouth) and/or cardiac and respiratory changes.

of Group 1 with the striking exception of the time necessary to cause death. Again, four dogs died due to air embolism, revealing marked cardio-respiratory changes together with varying degrees of emphysema in areas such as the tongue and floor of the mouth, the facial and periorbital regions. Two animals which did not die did, however, demonstrate mild cardiac and respiratory changes and emphysematous swellings in areas similar to those animals which died. The remaining three animals revealed no significant objective physical changes. Small amounts of air were sometimes noted leaking at the juncture of the canula and the tooth. In addition, air was occasionally and unpredictably seen bubbling out through the periodontal pockets around the teeth. During procedure Number 10, the left maxillary sinus was accidentally perforated during the reaming of the root canal. Air injected into the root canal escaped into the maxillary sinus. The sinus perforation was clearly seen on histological examination.

The gross autopsy findings on the dogs which died during these procedures were similar to those observed on the dying animals in Group 1. In addition, the brains of these animals revealed the presence of air emboli in the superficial vessels. (Table 3)

The histological sections of the heart revealed moderate engorgement of the blood vessels. The superficial, larger coronary vessels revealed the presence of red blood cells intermixed with the plasma. Some of the vessels revealed minute round empty areas around the periphery, while in some, large vacuolated areas occupied a major portion of the lumina. Similar vacuolated areas were noted in the sections of the lungs. Sections of the brain revealed the presence of minute and

TABLE 3. GROSS AUTOPSY FINDINGS ON ALL ANIMALS SACRIFICED.

Group	Procedure	Presence of air in			Emphysema of facial and Periorbital Tissues
		The Right Atrium and Ventricle	Major Thoracic & Coronary Vessels	Superficial Cerebral Vessels	
1	2	Present	Present	The brains of these animals not removed during autopsy	Minimal
	4	Present	Present		Minimal
	5	Present	Present		Minimal
	7	Present	Present		Present
2	8	Present	Present	Present	Present
	12	Present	Present	Present	Present
	14	Present	Present	Present	Present
	15	Present	Present	Present	Present
3	17	Absent	Absent	Absent	Absent
	18	Absent	Absent	Absent	Absent
	19	Absent	Absent	Absent	Absent

large empty areas in the lumina of the superficial vessels. The deeply located vessels also revealed the presence of minute vacuolated spaces. In addition, the separation of the endothelial layer from the adjacent nervous tissue was often noted. (Figures 18,20,22)

The decalcified sections of the maxillary bone, including the experimental tooth, revealed the presence of dentinal particles and frequently the remnants of pulp in the root canal. The periapical areas revealed a moderate number of hyperemic vascular channels. Emphysema and edematous fluid were seen in the fibrous connective tissue.

Group 3 (procedures 17,18,19 for control evaluation) This group consisted of three healthy young dogs sacrificed in order to obtain normal gross and histological observations of the heart, brain and lungs. The histologic sections were used as a control against the sections of these tissues (heart, brain, and lungs) from the experimental animals in Group 2.

Two dogs were dissected in an identical manner. The thoracic cavity was exposed and the heart and lungs were removed. The brain was carefully dissected in one piece. All the organs were fixed in fifteen per cent formalin for three days and were later sectioned for histological examination.

A slight modification was made while dissecting out the tissues of the third dog. After exposing the thoracic cavity, all the major vessels around the heart and lungs were carefully ligated. The brain was carefully removed in one piece. Attempts were made to prevent access of air into these organs. All the organs were immediately placed in fifteen per cent formalin for fixation. The organs were fixed for three days and were sectioned for histological preparations.

Gross examinations before removal of tissues demonstrated no air emboli in any organs or vessels examined, including those in which large amounts of air were found in Groups 1 & 2. (Table 3)

All the sections were stained with hematoxylin and eosin. Histologically the sections of the heart, lungs and brain of all the three animals closely resembled those of the experimental animals in Group 2, which died of air embolism. (Figures 19,21,23)

DISCUSSION AND SUMMARY

Differences & Comparisons Among Groups

Group 1 (procedures 1-7). This group in reality consisted of a series of pilot studies, most of which sought optimal methods for causing death in dogs by introducing air into the root canals of their teeth. The only constant in this series was the right lateral position of the dogs during the procedures, since this placed the animal in maximal danger as far as position was concerned. The teeth employed varied from mandibular to maxillary cuspids and a mandibular premolar. Sealing of the canals (simulating the clinical air tip) was not attempted in two cases, and in the others, only crude seals to help prevent backflow of air were constructed. The air pressures introduced into the animals varied from 40 to 50 pounds per square inch.

Group 2 (procedures 8-12). In contrast to Group 1 an attempt was made here to maintain standard conditions throughout to mimic the most dangerous conditions which would be likely to occur in the dental office. Thus, each animal was operated upon in a seated position. The air pressure was kept constant at 35 pounds per square inch. A specially threaded canula was used which blocked backflow of air much more efficiently than the crude seals of Group 1.

The maxillary third incisor was used in all Group 2 procedures. This tooth was selected for the following reasons: a) the size, length and shape of the root canal was optimal, b) the anatomical relationship to antrum and orbit was of possible significance, c) the working stability

was good, and d) root canal procedures are more commonly performed on maxillary than mandibular teeth in humans.

Comparison between Group 1 and 2. The experimental design for the two groups was different. The animals in Group 1 were placed in a right lateral position whereas those in Group 2 were seated in a specially prepared chair. In Group 1 the tooth used was variable, in Group 2 the maxillary third incisor was always used. A constant air pressure was used in Group 2, a variable one in Group 1. Attempts to seal the needle canula into the root canal in Group 1 (five procedures) were unsuccessful. In Group 2 a threaded canula was used and inserted into a threaded root canal. Only minimal amounts of leakage occurred in this group whereas extensive leakage occurred in Group 1.

All of the animals dying of air embolism in Groups 1 and 2 showed varying degrees of emphysema of the face and periorbital tissues.

In Group 1 autopsies were performed only on the animals which died during the procedures. The brains of these animals were not removed. Conversely, all the animals in Group 2 were autopsied and the brains were removed. The autopsy findings in comparable organs and tissues of the animals which died of air embolism in Groups 1 and 2 were similar. (Table 3)

Comparison between Group 2 and 3. Although the animals in Group 1 were examined grossly (with similar findings to those in Group 2) only the organs and tissues in Groups 2 and 3 were examined both grossly and histologically, and therefore only these two latter groups are compared here. Grossly, the hearts and brains and blood vessels of the dogs dying of air embolism in Group 2 demonstrated these air emboli in-situ before

removal of any of these tissues. However, in Group 3 none of these organs and tissues demonstrated air emboli grossly.

Sections of heart, lungs, and brain from the animals of Group 2 and 3 (control)were compared microscopically. It was noted that the tissues from both groups contained small vacuoles in the lumina of the blood vessels. (Figures 18,19,20,21,22,23) Initially such vacuoles in the tissues of the experimental animals were considered to be the result of air embolism. However, due to the similar findings in the control group it was concluded that vacuoles were not the result of air embolism, but were due to artifacts occurring during the sectioning of the organs or vessels, or during the processing of the tissues.

Patho-Physiological Changes in Air Embolism

A possible interpretation for the patho-physiological changes begins with the introduction of air under pressure into the vascular channels of the periapical regions of the animal. The introduced air forms bubbles in the venous channels leading to the jugular and internal mammary veins, thence to the superior vena cava and into the right atrium and ventricle. (Figure 11) If the animal is supine, seated, or in the right lateral position, the exit from the right ventricle to the pulmonary arteries is located superiorly. (Figures 15,16) The buoyant air forms a froth with the blood and rises to the surface, effectively blocking the normal flow of blood into the lungs. This causes a reflex increase in the systemic blood pressure at first, followed by a gradual lowering, with the blood leaving the systemic circulation and pooling in the right heart. Eventually the heart ceases functioning, the blood pressure drops to zero, and respirations cease. It is possible that some of the air may

pass through the pulmonary circulation via the arterio-venous shunts from the pulmonary artery to the pulmonary vein, then into the left atrium and ventricle, and through the aorta into the coronary arteries and/or cerebral vessels as it was seen in the four fatal cases in Group 2. This passage of air, particularly to the cerebral vessels, was probably more rapid due to the increased buoyancy factor in the seated position. It is possible that cerebral embolism may have played a role in the death of some of these animals by causing anoxia to one or more vital centers such as that involved with respiration.

Initial pallor followed by cyanosis of the tongue was invariably seen in all the animals which died of air embolism. Liebermeister stated that this sign was not only an early one but a constant one in cases of arterial embolism. (10) This observation physiologically confirms the gross findings of cerebral and coronary air emboli. This sign could be an important one for a dentist in diagnosing a case of air embolism swiftly.

Estimation of Lethal Dose of Air

Van Allen et al., (32) found that different species present widely different tolerances and among individuals of the same kind there exists some variation. Comparing the ratio of heart size to body weight of the dog and the human, it can be said that the human has a relatively smaller sized heart than the dog. This means that somewhat smaller amounts of air in proportion to body weight may prove fatal to the human. A review of the literature indicates that the estimated lethal dose of air for the human would be much smaller than for an experimental animal such as the dog. For example, one investigation estimates that 40 ml of air

could be fatal to an average human male. (12) This would imply that air under 35 pounds per square inch pressure could be fatal to a human when introduced into a root canal for a very short period. For example, by extrapolating the water displacement determinations it can be estimated that in three seconds it is possible that a maximum of 300 ml of air could be introduced into the vasculature.

In order to estimate the actual amount of air entering the periapical vasculature of the dogs, attempts were made to achieve an air-tight seal between the canula and the tooth by a variety of methods, with much better success in Group 2 than in Group 1. However, it was impossible to perfectly seal the canula in the bleeding root canal. Moreover, even if a perfect seal could have been obtained between the canula and the tooth the volume of introduced air (through the canula) might not have entered the systemic circulation since one or both of the following occurred during the procedures: rupture of the periodontal membranes with resultant escape of air through the gingival sulcus and/or localized accumulations of air in facial and periorbital tissues. Thus, it would appear that the minimal lethal dose of air introduced into the root canal cannot be established by the methods employed in this study, even though the maximum possible introduction of 300 ml of air per second was clearly demonstrated.

Possible Implications in Dental Practice

Eight out of sixteen dogs in this study died due to air embolism occurring as a result of root canal therapy. All of these were young dogs, the one older dog did not succumb. It is possible that in children, as in other younger animals, the wider apical foramina, thinner-

walled blood vessels, and greater possibilities of hamartomatous vascular proliferations, all increase the danger of fatal air emboli as described in this investigation. These, of course, are in addition to the engorgement and fragility of the increased open capillaries in the inflamed area being treated.

Since the probabilities are remote of a perfect seal occurring by the insertion of an air tip in a root canal in dental practice, the conditions of this study closely resembled the possible varying degrees of obstruction. Furthermore, the much more rapid deaths of the dogs in Group 2, in which better seals were present, indicates the increasing danger of similar situations in clinical practice.

It is therefore suggested that pressurized air not be used in clinical practice to dry or to debride root canals. If this situation should occur, however, the alert clinician might suspect air embolism by either local emphysema or initial pallor and subsequent cyanosis of the tongue. A procedure capable of saving some lives would be the placement of the patient in the left lateral position lying down.

CONCLUSIONS

1. Death may result in dogs when pressurized air is introduced into a mechanically enlarged root canal. Air may cause death due to its excessive presence in the right ventricle, large thoracic vessels, coronary vessels and cerebral vessels.

2. It is impossible to estimate the lethal dose of air when introduced via root canals. Variable amounts of air may not enter the systemic circulation, by either rupture of the periodontal membrane and escape through the gingival crevice, or by forcing its way into the facial tissues, or by variable amounts of backflow from the root canal. Thus, the amount of air entering the systemic circulation is extremely variable but always less than the amount delivered to the root canal.

3. Death due to air embolism can best be diagnosed by gross autopsy observations of bubbles of air in thoracic, coronary, and cerebral vessels in-situ. This is further demonstrated by tying off the great vessels, placing the heart under water, and observing the emanation of air bubbles from the punctured right ventricle. These findings may be confirmed by electro-cardiographic and blood pressure recordings consistent with death due to air embolism. Histologic demonstrations of air in vessels are not proof of death due to air embolism, since this finding may also occur in control animals, probably due to artifacts resulting from the severing of the vessels and/or processing of the tissues.

4. Based on the findings of this study it is recommended that pressurized air not be used to dry or remove debris from the root canals

of patients receiving endodontic treatment.

5. If for some reason a patient receiving dental treatment, in which pressurized air is being used, shows signs or symptoms of air embolism he should be placed in a left lateral position with the head lower than the feet. Emergency cardiac and respiratory resuscitation measures should be performed when indicated.

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APPENDIX A

Group 1

Procedure Number 1 The needle canula was inserted two ml into the exposed root canal. Air was blown into the root canal for 45 minutes. Since the canula was not tightly adapted to the root canal, the air was felt blowing back out of the canal. This and the absence of changes in the respiratory and cardiac rates indicated the probability that air did not enter the vasculature around the apical foramen in significant amounts. The animal was later sacrificed. The autopsy findings were unremarkable.

Procedure Number 2 Two modifications were made in this experiment. The needle canula was inserted more deeply into the root canal approaching the vicinity of the apical foramen. Also cold-cure acrylic joined the canula with the tooth surface resulting in a crude seal. This arrangement mimicked the clinical situation in which the tip of the air syringe is tightly wedged into the canal and prevents or minimizes the backflow of air. Air under pressure was introduced into the root canal for eight minutes, during which time a gradual tachycardia and polypnea followed by hyperpnea were observed. At the end of eight minutes respiration had stopped completely. An autopsy was performed with the dog in a supine position. Large air bubbles were found in the superior vena cava and the internal mammary and right jugular veins. The heart was enlarged and a frothy mixture of air and blood was seen in the right atrium and ventricle. When the right ventricle was punctured under water, air bubbled to the surface.. Minute air bubbles were also noted in the coronary arteries.

Procedure Number 3 The experimental procedure in Number 2 was repeated. However, an old dog (exact age not known) was used for this experiment whereas, the dog in experimental procedure Number 2 was about one year old. A carbide but was used to widen the narrow canal of the lower left premolar producing much ground dentinal powder which may have partially blocked the air from the periapical tissues. After 35 minutes of forcing air into the root canal, only a very slight increase was noted in the respiratory and cardiac rates. These changes were considered insufficient to continue the experiment any further. However, the minimal amount of air which did enter the vasculature passed into the lingual vessels, resulting in extreme emphysema of the tongue and floor of the mouth as detected by typical crepitation. (Figures 7,8) The experiment was terminated and the dog was sacrificed. The autopsy findings were unremarkable.

Procedure Number 4 Routine procedures were performed on a young dog. The canula was imperfectly sealed into the root canal of the right lower canine with cold-cure acrylic after which air was introduced for a period of six minutes. Tachycardia was observed, although to a lesser extent than in experiment procedure Number 2. Respiration gradually increased in rate and depth and at the end of six minutes had stopped completely. The autopsy revealed almost identical findings to those found in experimental procedure Number 2. The mandible, with the canula in place, was sectioned revealing the needle to be 2 mm from the apex of the tooth.

Procedure Number 5 Besides the standard procedures, continuous electro-cardiographic and blood pressure tracings were obtained. A

canula had been inserted into the right femoral vein to record the blood pressure. Prior to the introduction of air into the lower right canine, a control radiograph of the thorax was taken. The root canal, imperfectly sealed with acrylic, was not air-tight. At the end of ten to eleven minutes of air introduction, the dog was dead. It exhibited much the same symptoms as the dog in experimental procedure Number 4. Radiographs of the thorax were taken intermittently during and after the period of air introduction. A radiograph of the mandible with the canula in place was taken at the conclusion of the experiment. Electrocardiographic, blood pressure, and radiographic records follow:

Electro-cardiographic record revealed the time for one cardiac cycle to be 0.5 seconds during the control recording. For the first five minutes of air introduction, the cardiac cycles were normal, after which they were reduced from 0.5 to 0.3 seconds for 40 seconds. Following this, the time of the cycles increased from 0.3 to 1 second, from 1 second to 1.5 seconds, and from 1.5 seconds per cycle to 2 seconds per cycle. The dog died during the last recording of 2 seconds per cycle. The amplitude of the cycles gradually decreased up to the end of the experiment.

The control (pre-experiment) blood pressure was 138/122 and remained constant during the first four minutes of air introduction. It then increased rapidly to 210/135 and remained constant for the next five minutes. This was followed by a gradual fall in the blood pressure to 128/120. For a short period, there was an alternate rise and fall in the blood pressure followed by a gradual decrease up to the end of the experiment.

The control radiograph, taken prior to air introduction, revealed a normal thoracic picture. The radiograph, taken after five minutes of air induction, showed air in the heart chambers and the main veins; although the animal did not expire for another five minutes. Greater amounts of air in the heart chambers and the large vessels were seen on the radiograph taken after the cessation of air induction. The mandibular radiograph revealed the canula to be within one millimeter of the apical foramen. The findings at autopsy here were essentially those described for experimental procedure Number 2.

Experiment Number 6 The standard procedures were not followed in this experiment. An attempt was made to blow air into the root canal of the lower right canine without inserting or partially sealing the canula. In this manner, air was blown intermittently for 40 minutes with only a slight increase in the respiratory and cardiac rates. Since these changes were not remarkable, the experiment was discontinued.

Experiment Number 7 Experimental procedure Number 6 was repeated without sealing the canula for 35 minutes with the same results. Then an upper right canine tooth was prepared and a canula inserted into the root canal with an imperfect acrylic seal and the standard procedures were followed. During the first 45 seconds of air introduction, the respiratory and cardiac rates increased rapidly and after three minutes the dog died. An autopsy was performed with findings similar to those in experimental procedure Number 2. In addition, a prominent emphysematous bulging of the right eye was noted.

APPENDIX B

Group 2

Procedure Number 8 The canula was adjusted into the previously threaded root canal. An attempt was made to achieve an air tight seal between the tooth and the canula. Air under 35 pounds per square inch pressure was administered. The passage of air into the facial tissues was immediately identified by a progressive distention which extended to the neck region. Rapid and shallow breathing was noted within three seconds. These changes progressed rapidly and the animal stopped breathing at the end of ten seconds, at which time air administration was immediately stopped. A sudden increase in the heart rate was noted during the time of air administration, which gradually decreased. An autopsy was performed with the dog in the supine position. The right face was emphysematous with demonstrable crepitus and the right eye was seen bulging prominently. (Figure 5) The tongue was pale and cyanotic. Large air bubbles were found in the superior vena cava, internal mammary, and jugular veins. On gross examination, the heart was enlarged and minute air bubbles were seen in the coronary arteries. The presence of air in the heart was confirmed by its bubbling out from the heart when the latter was incised under water. Small air bubbles were noted in the superficial vessels of the brain.

The histological sections of the heart revealed moderate engorgement of the blood vessels. The superficial, larger coronary vessels revealed the presence of red blood cells intermixed with plasma. Some

of the vessels revealed minute round empty areas around the periphery, while in some, large vacuolated areas occupied a major portion of the lumina. These areas were interpreted to be due to the presence of the air injected through the root canal. This assumption was based on the gross finding of air emboli in these regions.

Sections of the brain revealed the presence of minute and large empty areas in the lumina of the superficial vessels. The deeply located vessels also revealed the presence of minute vacuolated spaces. In addition, the separation of the endothelial layer from the adjacent nervous tissue was often noted.

The decalcified sections of the maxillary bone, including the experimental tooth, revealed the presence of dentinal particles and frequently the remnants of pulp in the root canal. The periapical areas revealed a moderate number of hyperemic vascular channels. Emphysema and edematous fluid was seen in the fibrous connective tissue. This caused the distention and rupture of the connective tissue bundles. The control sections of the maxilla revealed moderate engorgement of the vascular channels in the periapical areas. All sections revealed a thin wall of fibrous connective tissue between the maxillary antrum and the apices of the teeth.

Procedure Number 9 The procedure in experiment Number 8 was repeated. Air was administered for ten seconds and the cardiac and respiratory rates were recorded. A gradual, slight change of rapid breathing was noted. This change persisted for about five minutes. Air was again introduced for ten seconds and the cardiac and respiratory changes were noted for five minutes. The air was blown ten seconds each time

and the animal was carefully observed for any changes in the cardiac and respiratory changes. The air was blown for a total time of three minutes. Since no significant changes were seen, the experiment was discontinued. The animal was sacrificed by a fatal dose of anesthetic solution injected directly into the heart. A moderate emphysema of the face and periorbital tissues was noted. No significant tissue changes were seen at autopsy.

Procedure Number 10 The procedure in experiment Number 9 was repeated. The air was introduced into the root canal at ten second intervals, for a period of two minutes. The observations of heart and respiratory rates were recorded. A loud, hissing sound was heard during the air introduction and a thin watery fluid was seen exudating from the nostrils. The experiment was discontinued as no significant changes were observed. It was suspected that the maxillary sinus was perforated during the reaming of the root canal and air was passing directly into it causing a hissing sound. The postmortem finding of perforation of the maxillary antrum was confirmed by examination of histological sections of the decalcified maxilla. The remaining autopsy findings was not significant.

Procedure Number 11 Routine procedure was followed. The pressurized air was blown into the root canal for ten seconds each time for a total of ten minutes. A slight change of rapid respiration was recorded in the latter part of the experiment, which persisted for about one hour and thirty minutes. The air was found to be leaking extensively around the canula. The experiment was discontinued. The animal was sacrificed by a fatal dose of anesthetic injected directly into the

heart. No gross tissue changes were seen at autopsy.

Procedure Number 12 Routine procedures were performed on a young dog. A fairly air tight seal was achieved and a very little amount of air was seen leaking around the canula. After introducing air for ten seconds, a slight increase in respiration was noted. After recording this change for ten minutes, the air was introduced for ten more seconds. The respiratory change still persisted without any deviation. Later the animal developed tachypnea (rapid and shallow breathing) with increasing doses of air. The air was introduced for 70 seconds at ten and fifteen second intervals. At the end of 70 seconds, the animal revealed severe tachypnea and a cardiac murmur was faintly audible. The change persisted for about four minutes and was followed by slow and deep respiration, and finally the cessation of breathing. Diffuse emphysema of the face and periorbital tissues was noted. (Figures 9,10) The tongue revealed severe cyanosis. An autopsy was performed with the dog in a supine position. Small amounts of air were noted in the superior vena cava, internal mammary, and right jugular veins. The heart was enlarged and a frothy mixture of air and blood was seen in the right atrium and ventricle. This was confirmed by puncturing the heart under water. Minute air bubbles were seen in the coronaries. Very small amounts of air were noted in the superficial vessels of the brain. The histological findings of the heart, brain, and lungs were similar to those noted in experimental procedure Number 8.

Procedure Number 13 The standard procedure was followed in this experiment. Air under 35 pounds per square inch pressure was blown into the root canal at five intervals of ten and fifteen seconds respectively. Later, the air was continuously injected for a prolonged time.

Between intervals the animal was observed carefully for any changes. Air was found to be leaking around the canula. Even after prolonged blowing of air, the animal failed to reveal any cardio-respiratory changes. On autopsy, the tissues were found to be unremarkable. Slight emphysema of the face was noted in the vicinity of the site of operation.

Procedure Number 14 After performing the preliminary procedures, the canula was inserted into the threaded root canal. A fairly air tight seal was achieved. Air was blown into the root canal for ten seconds and the animal was carefully observed for any cardio-respiratory changes. A progressive tachypnea was noted immediately during and after the time of air introduction. The dog was dead within three minutes. Vague heart contractions persisted for a short time after the complete cessation of respiration. Bulging of the eyeball was prominent and the tongue developed cyanosis. An autopsy was performed with the animal in the supine position. Minute amounts of air were noted in the major blood vessels, the coronary arteries, and the superficial vessels of the brain. A small amount of air bubbled out of the heart when it was incised under water. The tissues were saved for sectioning. The histological findings of the heart, brain, and lungs were similar to those noted in experimental procedure Number 8.

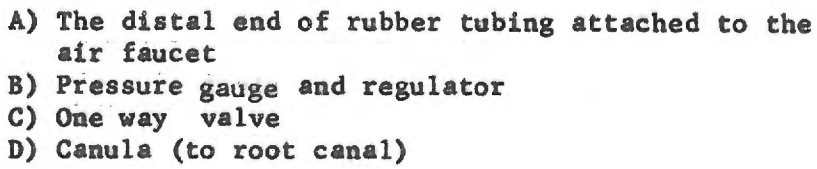
Procedure Number 15 Routine procedures were performed on a very young, healthy dog. The canula was well fitted into the root canal and the air was blown for ten seconds. A slow yet progressive change of rapid and shallow breathing was noted. This change was recorded for five minutes following the time of the administration of the air. It was thought that the amounts of air which entered into the tooth were

insufficient to cause death, and the air valve was turned on for another dose of air for ten seconds. Within a few seconds of the second dose, the changes of respiration became suddenly severe leading to the death of the dog. The amount of air blown into the root canal for about thirteen seconds was enough to kill the dog. The autopsy findings were very similar to those in experimental procedure Number 14. The histological findings of the heart, brain, and lungs were similar to those noted in experimental procedure Number 8.

Procedure Number 16 The routine preparatory surgical procedure was being performed. The air was blown for ten seconds. No changes were seen to occur. The time intervals of the air introduction were later increased to fifteen seconds each. The cardiac and respiratory rates were recorded. Air was blown for about fifteen minutes. Since no apparent changes occurred, the experiment was discontinued. The animal was sacrificed by injecting a massive dose of anesthetic intravenously. The tissues appeared unremarkable on autopsy.

APPENDIX C.

Figure 1. Air Conducting System



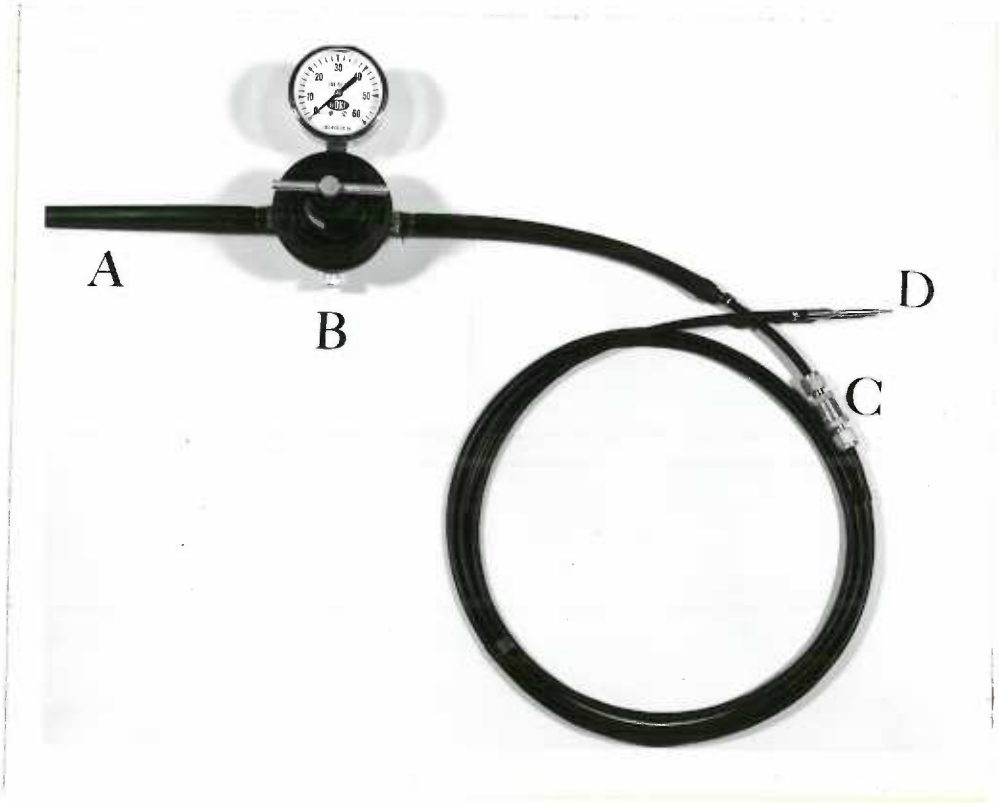
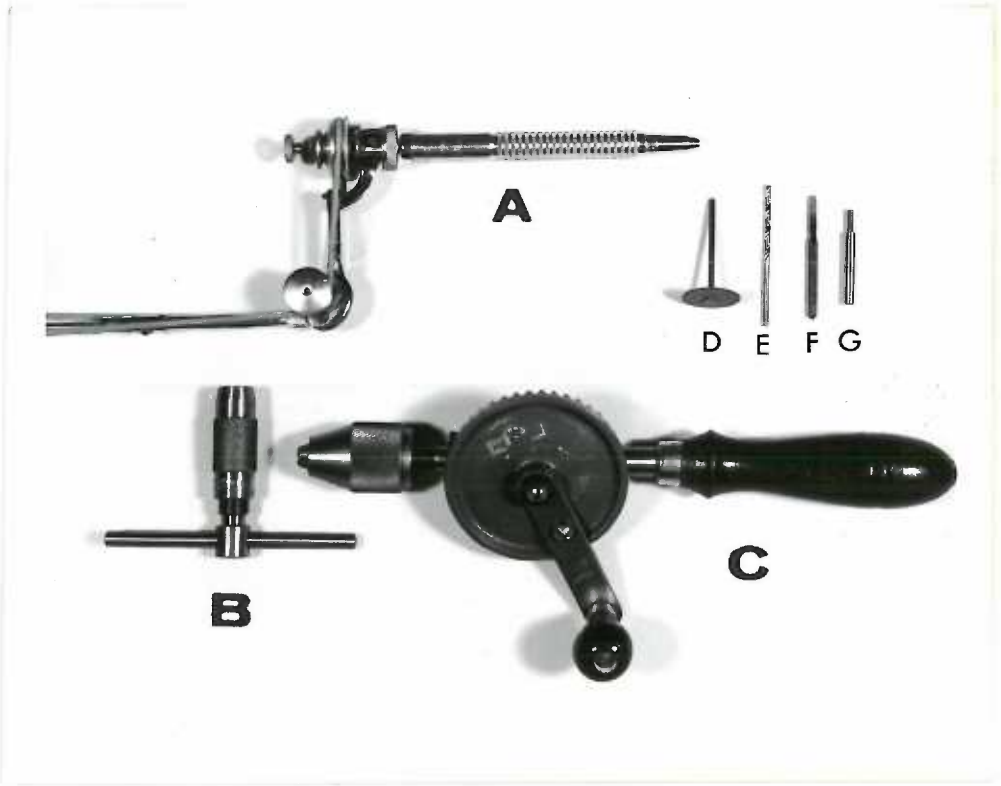

A
B
C
D

Figure 2. Instruments used for the preparation of the root canal prior to adjusting the canula

A) Straight hand piece
B) Tap handle
C) Brest-drill
D) Carborundum disc
E) Drill
F) Tap
G) Air Canula

Figure 3. Instrumentation of root canal for the removal of pulp and adjustment of the canula

A & B) Broaches used for removal of pulp tissue
C & D) Reamers used for widening the root canal
E) Drill used for further widening of the root canal for threading
F) Tap used for threading the canal
G) Canula with threaded tip


A
D E F G
B
C

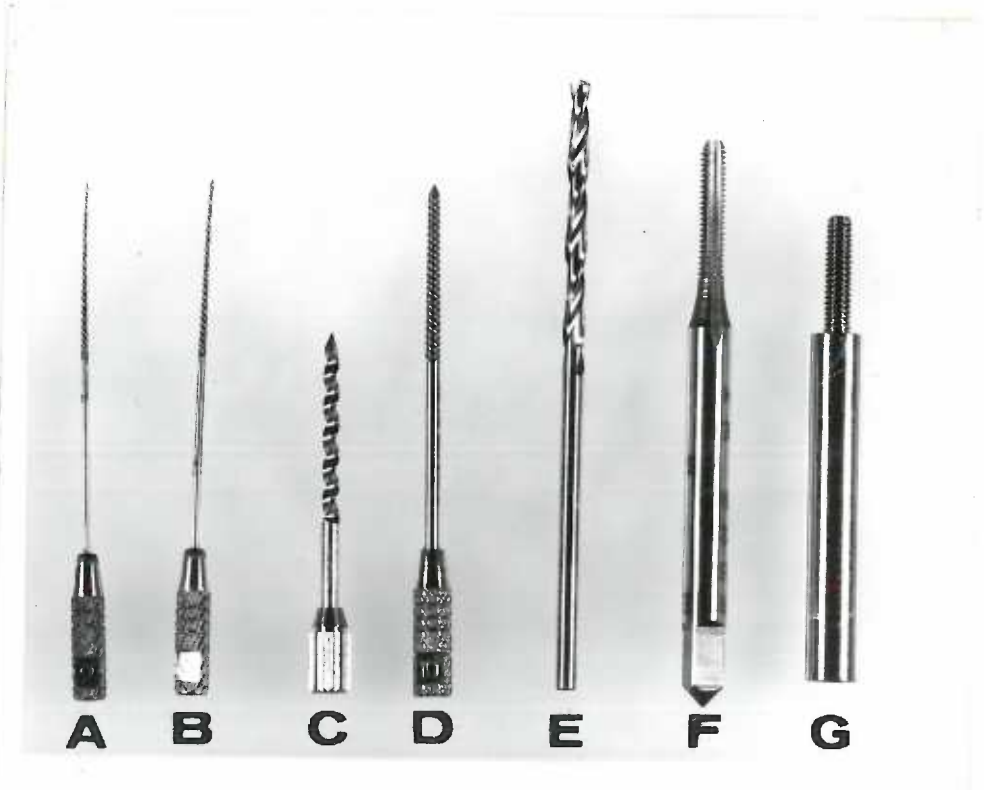

A B C D E F G

Figure 4. The figure represents a dog being seated in a specially prepared wooden chair during the experiment. The chair consisted of

A) The back rest
B) A horizontal adjustable platform
C) A base
D) Adjustable metal bar

Notice the air canula fitted into the tooth

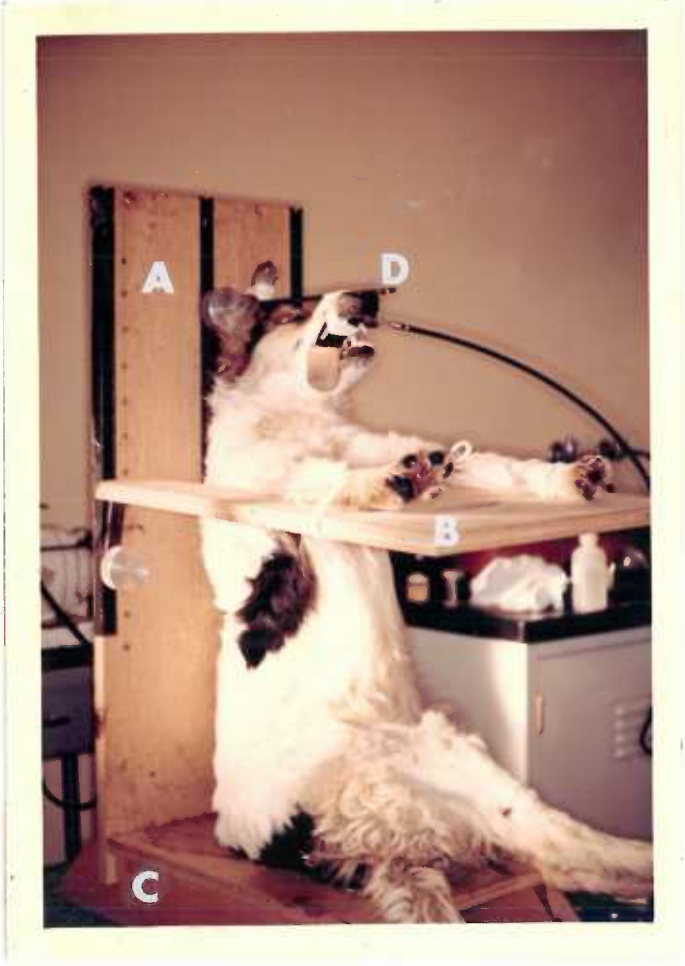

A
D
B
C

Figure 5. Dog number eight (Group 2) at the end of the experimental procedure. Notice the air canula inserted into the tooth. The arrows indicate moderate emphysema of the face and periorbital tissues.

Figure 6. A close up view of the left anterior segment of the upper jaw of dog number eight. The arrow indicates the left upper third incisor with canula inserted into the root canal. Notice the pallor of the buccal mucosa.

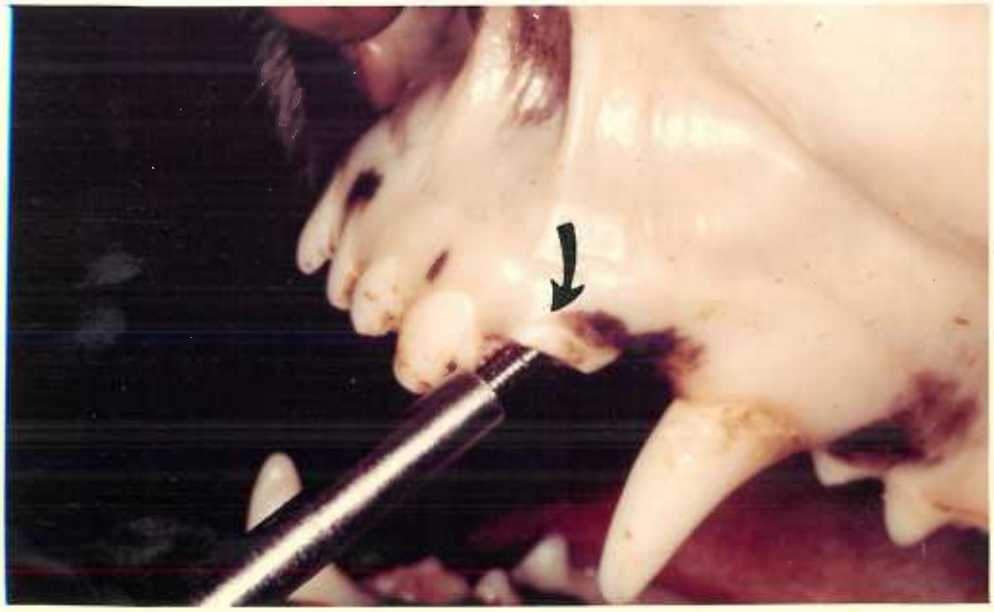
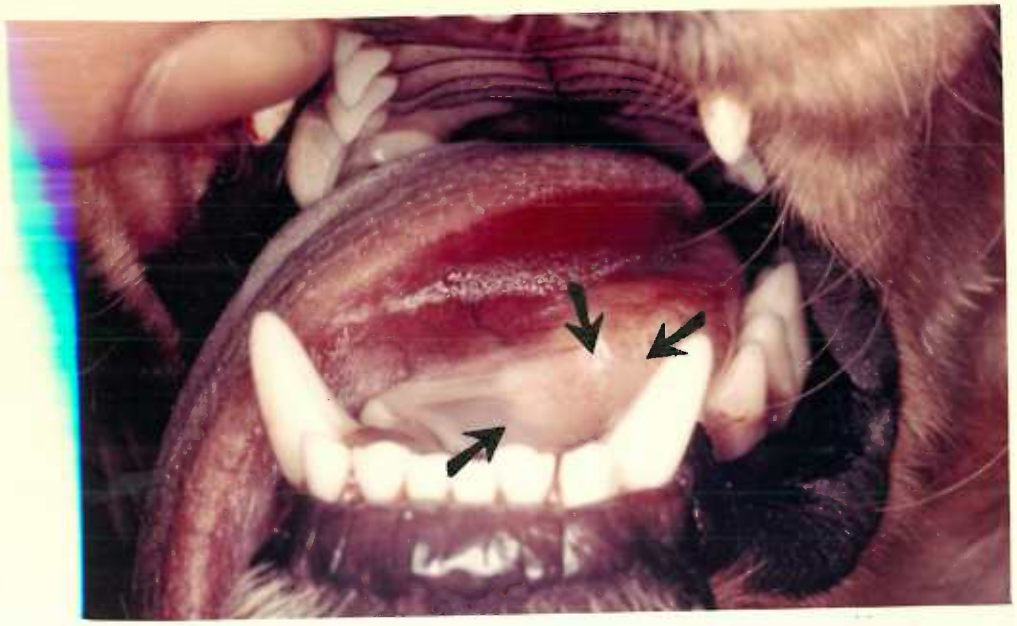


Figure 7. A close up anterior view of the oral cavity of dog number three (Group 1) after air administration. The arrows indicate the emphysematous bulging of the tongue and floor of the mouth.

Figure 8. A close up lateral view of the oral cavity of dog number three. The arrows indicate the same emphysematous area seen in figure 7.



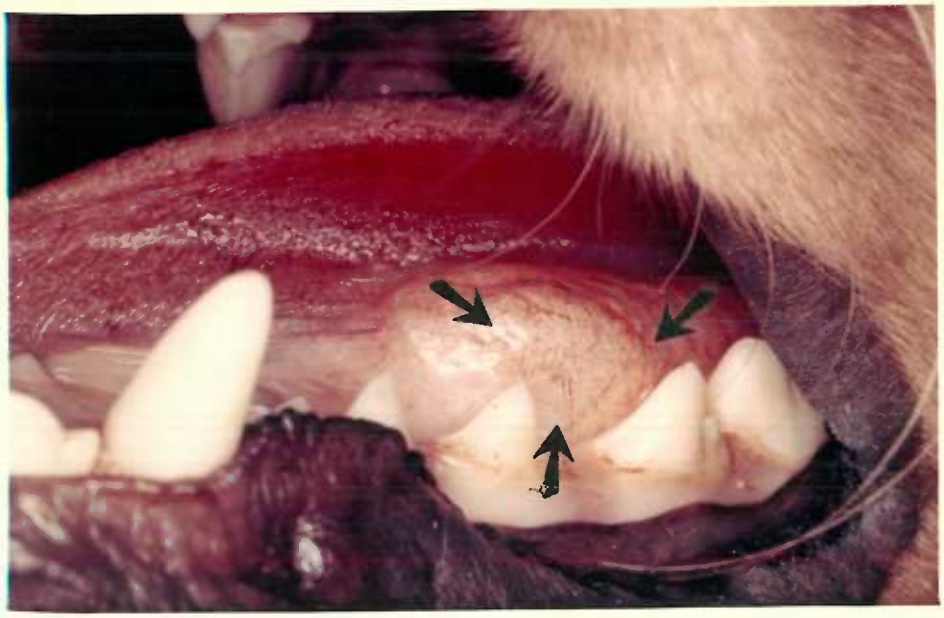


Figure 9. Anterior view of the emphysematous bulging of the left face and periorbital tissues of dog number twelve (Group 2).

Figure 10. The lateral view of the same emphysematous area seen in figure number 9.

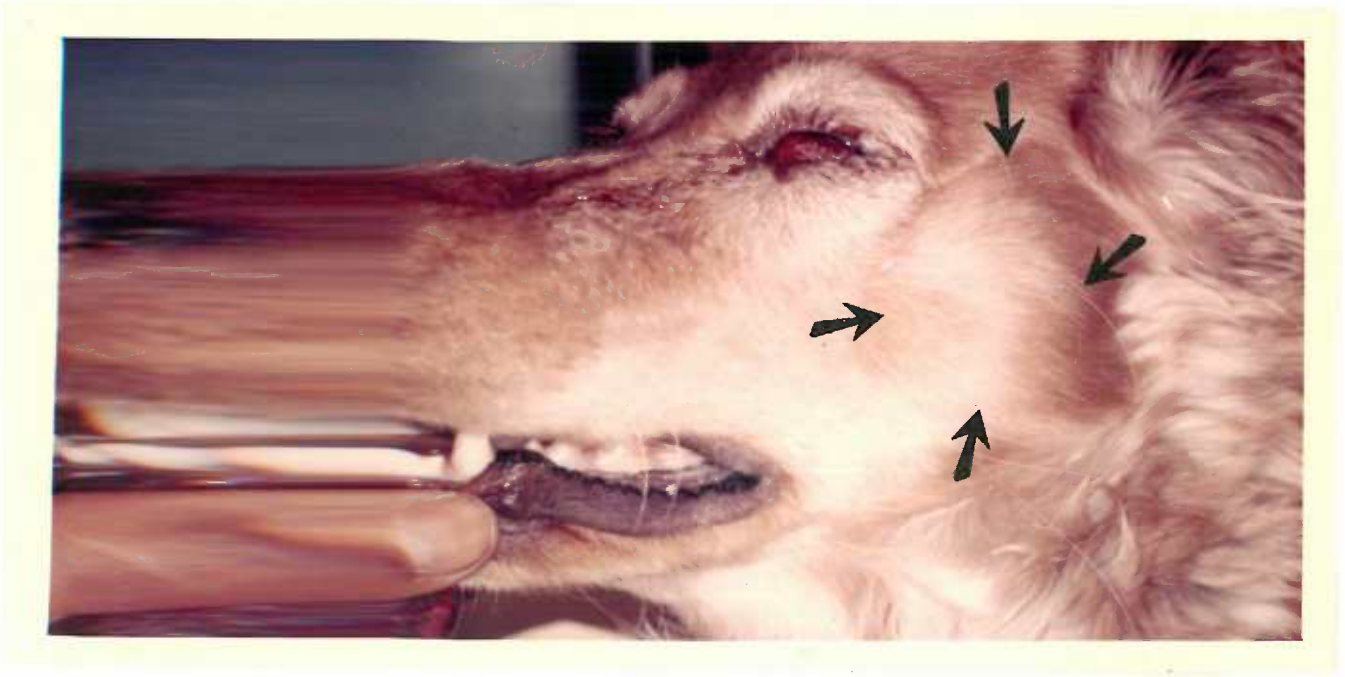


Figure 11. Typical radiograph of a young dog at the end of one of the procedures. Dotted lines outline the probable passage of introduced air from the root canal at A to the heart chambers. The air in the right ventricle of the heart reveals a radiolucent area B. An area of subcutaneous emphysema is noted in the floor of the mouth-C.

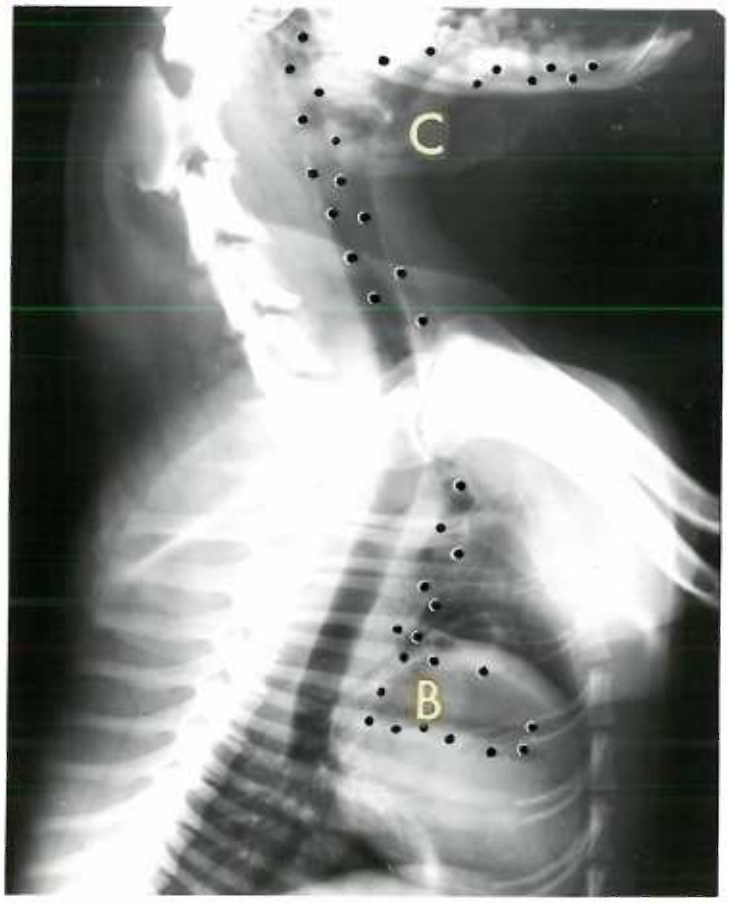

C
B

Figure 12. Typical finding of air emboli in coronary vessels of a young dog. A close-up view of the ventral surface of the heart revealing the presence of minute air bubbles in the left coronary vessels and their tributaries. This indicates a passage of air through the lungs from the venous to the arterial system.

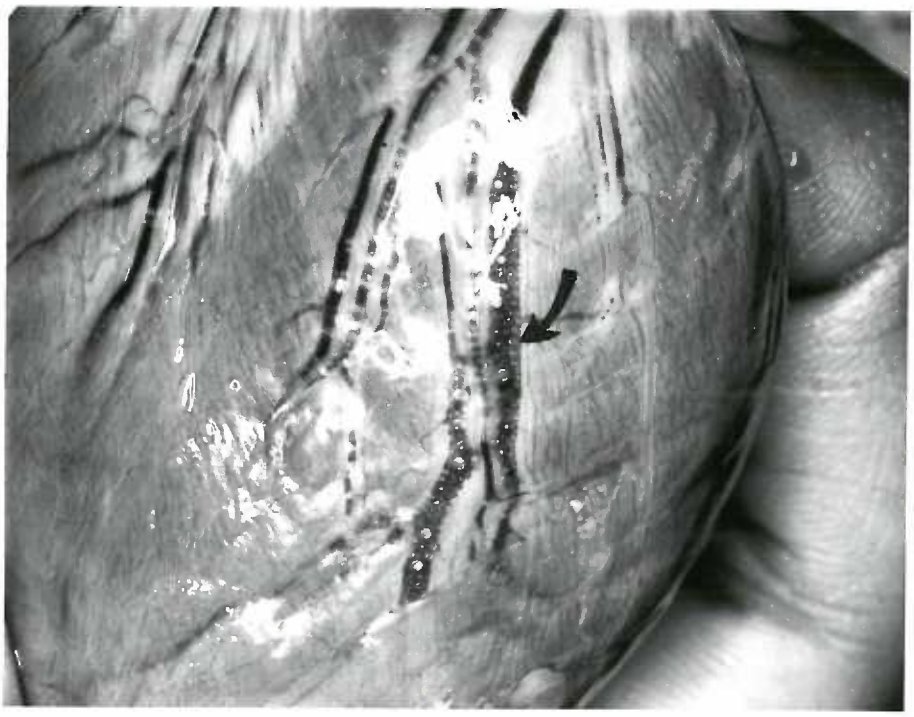


Figure 13. Typical air emboli in a young dog. Air emboli are seen in the proximal portion of the superior vena cava (A), and to the left of (A), and the thoracic (mammary) vein (B).

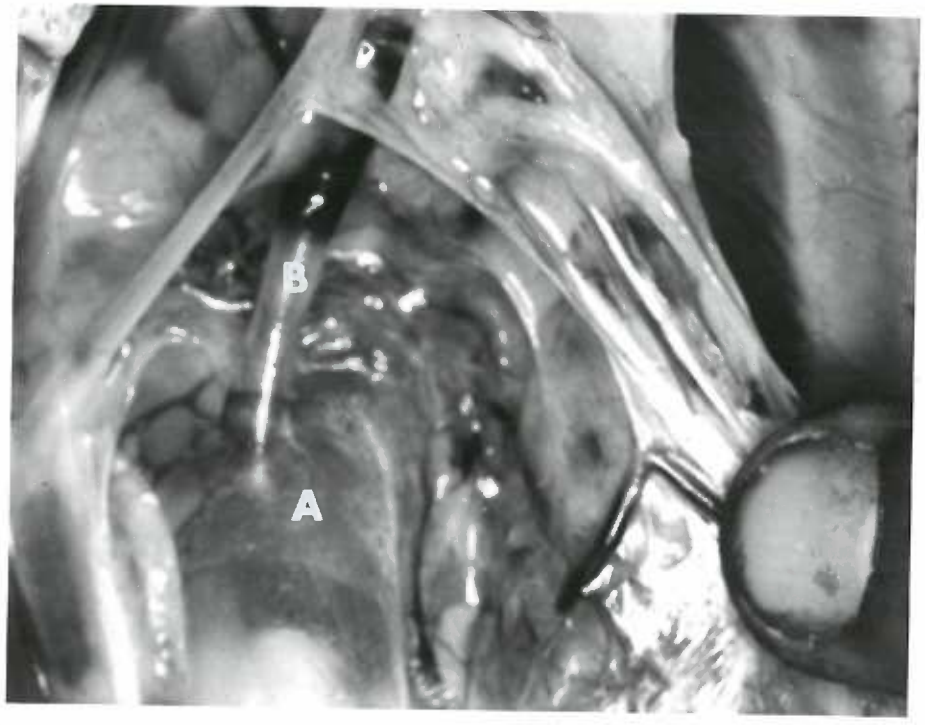

B
A

Figure 14. Typical air emboli in a young dog. Alternating bubbles of air and blood seen in a thoracic vein attached to the internal surface of the anterior chest wall. The surrounding fibroadipose tissue and intercostal muscles (A) are seen.

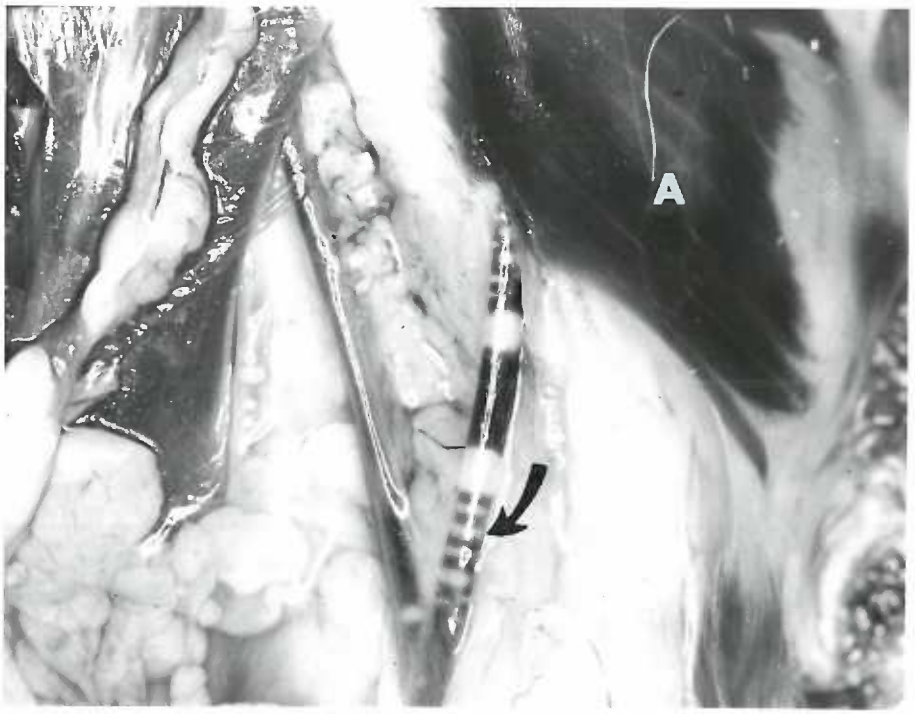
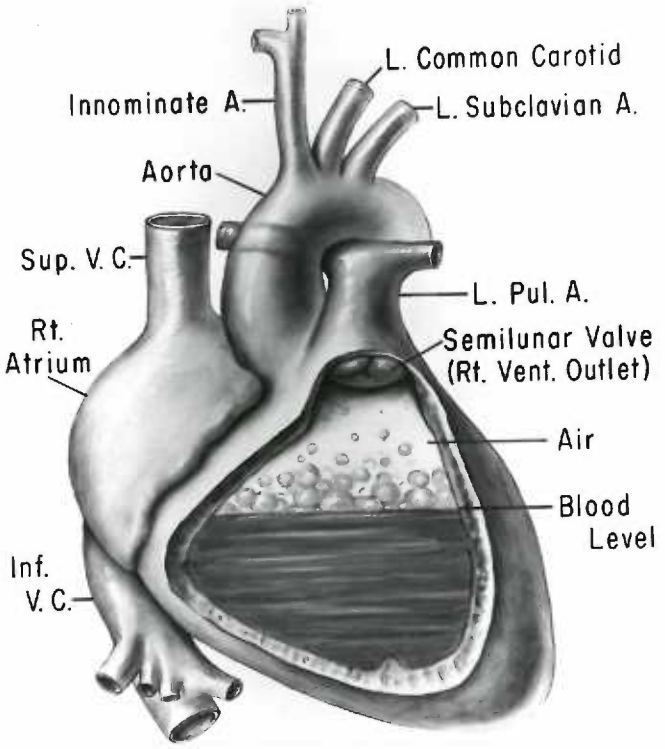

A

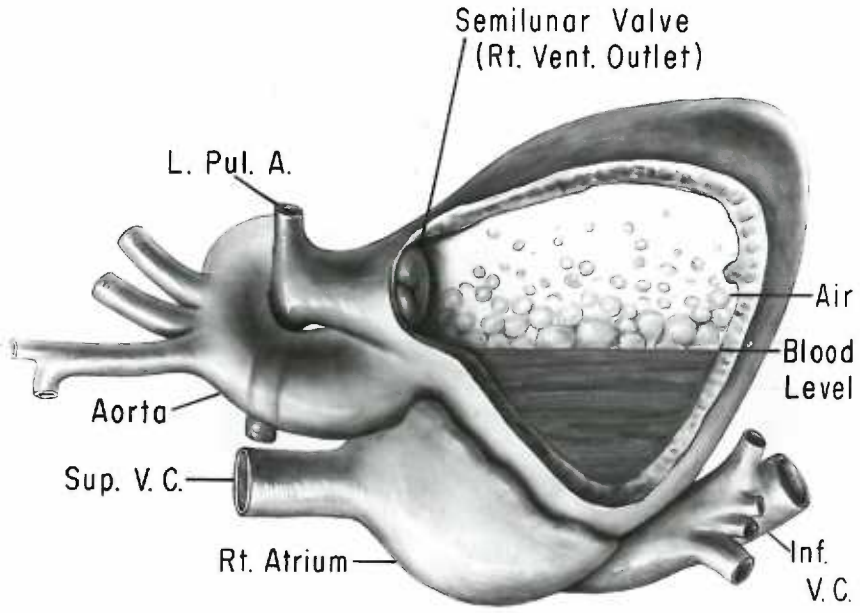
Figure 15. Orientation of the heart when the patient is seated. This drawing demonstrates the obstruction of blood flow from the right ventricle by the accumulated buoyant air present between the blood and the superiorly located semilunar valve. Serious circulatory block is likely to occur in this position.



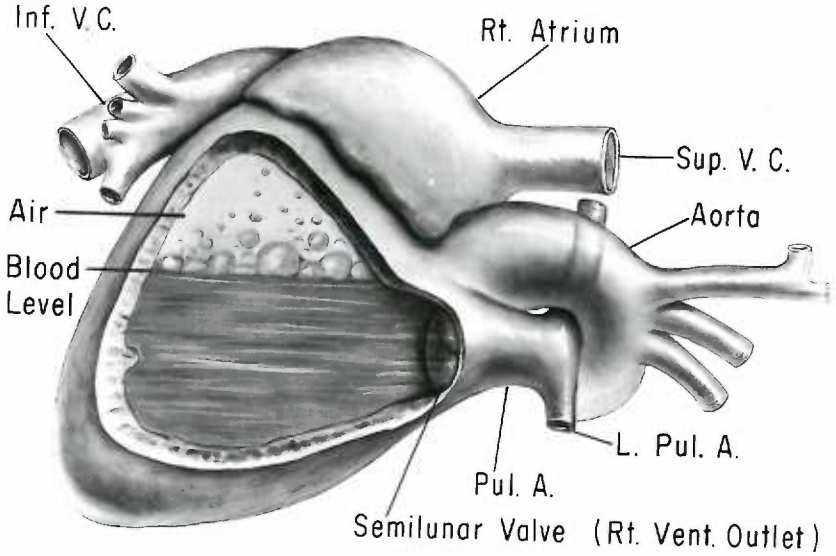
SITTING POSITION

Figure 16. Orientation of the heart when the patient is in the right lateral position. The semilunar valve is located superiorly in this position; therefore, obstruction of blood outflow due to the accumulated buoyant air may well occur.

Figure 17. Orientation of the heart when the patient is in the left lateral position. Since the semilunar valve is located inferiorly in this position, the probability of the superiorly located air blocking the outflow of blood is greatly lessened. Changing a patient from any other position to this one may prove to be lifesaving.



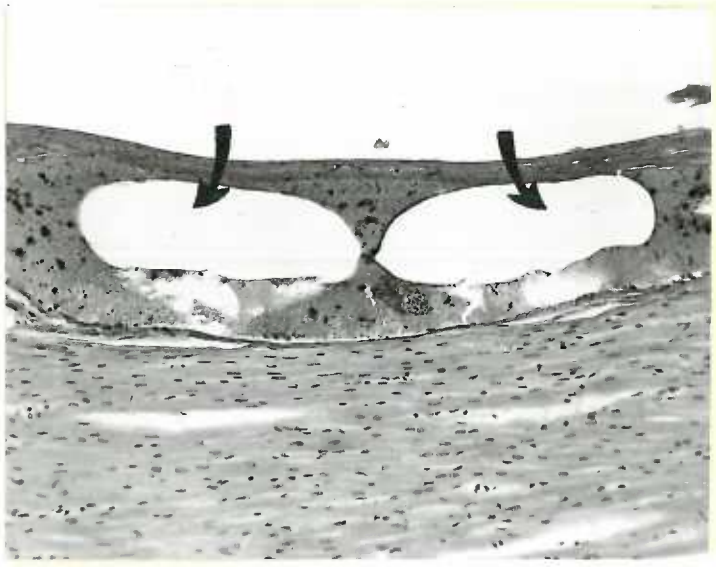
RIGHT LATERAL POSITION



LEFT LATERAL POSITION

Figure 18. Photomicrograph of heart tissue from one of the experimental animals. Notice large vacuolated areas in the lumen of a coronary vessel. H & E. 10x

Figure 19. Photomicrograph of heart tissue from a control animal revealing moderately large empty areas in the lumen of a deeply seated vessel. H & E. 10x



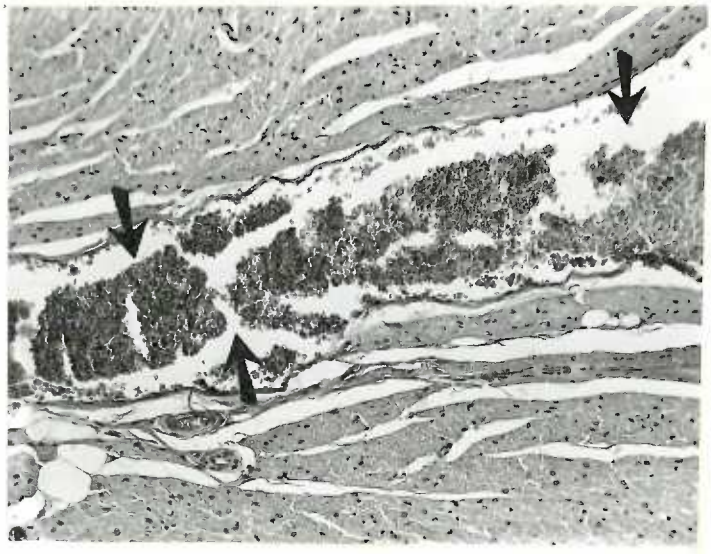
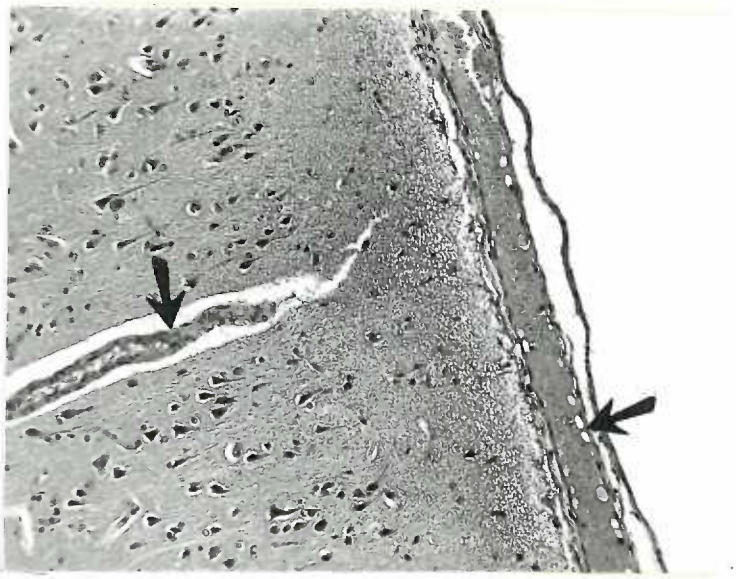


Figure 20. Photomicrograph of brain tissue from one of the experimental animals revealing the presence of minute round empty spaces in the lumen of the superficial vessel. Also, notice the similar areas in a deeply located vessel and separation of endothelial layer from the parenchyma. H & E. 10x

Figure 21. Photomicrograph of brain tissue from the control animal revealing larger empty areas in the lumen of a large superficial vessel. H & E 10x



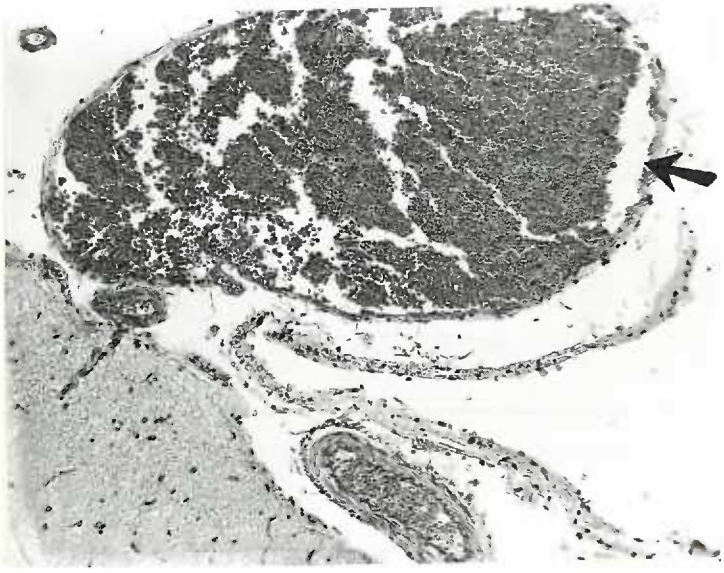


Figure 22. Photomicrograph of lung tissue from an experimental animal. Notice small, round empty areas resembling minute air bubbles, seen at the periphery of the lumen. Lung parenchyma also reveals emphysema and accumulation of edematous fluid. H & E. 10x

Figure 23. Photomicrograph of lung tissue from a control animal. Notice a large empty area in the lumen. (A). Minute, round vacuolated spaces are seen on the other side. These resemble closely to the ones seen in Figure 22. H & E. 10x