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VENOUS AIR EMBOLISM

A REVIEW OF THE LITERATURE

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Submitted in Partial fulfillment for the degree of Doctor of Medicine

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TABLE OF CONTENTS

- I. History
- II. Incidence
- III. Physiology and Pathology
 - A. History
 - B. Amount of air necessary to produce death
 - C. Heart
 - D. Lung
 - E. Type of gas
 - F. Arterial side
 - IV. Diagnosis
 - A. Mill wheel murmur
 - B. Cardiovascular system
 - C. Pulmonary system
 - D. ECG
 - E. X-ray
 - F. Autopsy
 - V. Treatment
 - A. General
 - B. Position
 - C. Respirations
 - D. Needle aspiration of air
 - E. Cardiac massage
 - F. Vasopressors
 - G. Neurological surgery

VI. Etiology

- A. Surgical
 - 1. Neck
 - 2. Chest
 - 3. Neurosurgery
 - 4. Uterine procedures
 - 5. Other procedures
- B. Diagnostic air injections
 - 1. Into the peritoneal cavity
 - 2. Into presacral cavity, urinary bladder and large joints
 - 3. Encephalography and angiocardiography
- C. Therapeutic air injections
- D. Obstetrical
- E. Accidental
- VII. Summary

I. HISTORY

Venous air emboli have been known to man for three to four centuries 56-579 approximately. Waepfer as early as the 17th century was said to have killed an ox of stupendous size by experimentally blowing air into its jugular vein.

In Europe in the early nineteenth century mangy and diseased horses were destroyed at times by injection of air. It was noted however that in view of the extreme apprehension shown by the animals before they died it could hardly be described as a humane method of killing.

The first recorded case of air embolism during a surgical operation occurred around the year 1800. Barlow, a surgeon in Blackburn, was removing a tumor of the neck. Suddenly there was a hissing sound and his patient expired instantly without either a grown or struggle. It was 30 years later when Barlow realised that this had been a case of air embolism.

Surgical interest in this complication wasn't really stimulated until Sansom in 1824 reported a case. At this time a tumor of the head and neck was being removed. A whistling sound was heard and the surgeon remarked that if he had not known that he was some distance away from the air tubes he would have supposed that he had divided them. Moments after he had said that his patient exclaimed, "I am dead," leaned against the chair and fell lifeless. He wrote that every possible means was employed to excite the action of the heart but without success. Autopsy revealed an open vein and air in the right auricle which was distended. By 1843 at least forty cases had been described in the literature.

II. INCIDENCE

It is impossible to ascertain the true incidence of death from air 46-634 emboli as this diagnosis is seldom made. Many believe the diagnosis would be greater if it were to be considered by pathologists in all sudden, unexplained death in which autopsy was performed.

There have been many attempts to ascertain the incidence of venous air emboli, but all admit the diagnosis to be made no where near the true 26-1353 incidence. One study of 108 operating room deaths showed venous air embolism to account for one of these deaths. However, one report in 2-375 1960 stated almost 20% of accidents in surgery and anesthesia appeared to involve air embolism.

Other studies have been done. A survey of 1,267 urologists showed 58 deaths and 64 severe nonfatal cases of gas embolism resulting from all 30-259 the retroperitoneal pneumography that they had done. Another study of 281 neurogurgical cases including craniotomies, posterior and middle fossa explorations, cervical laminectomies, explorations of the upper thoracic and cervical spine revealed six cases of air embolus and five of these were in the sitting position. Projected true incidence would probably be much higher.

-2-

III. PHYSIOLOGY AND PATHOLOGY

A. HISTORY

In the eighteenth century there were many theories put forth to 56-582 explain the cause of death in venous air embolism. The first one of these theories suggested death was due to resistance afforded by the air to the venous return to the heart. Shortly after this another theory stated it was due to direct depressive effect of air on the brain. Later it was thought to be that the heart became distended by the air and unable to contract. Finally Erichsen around 1844 demonstrated that air mixed with blood doubled or trebled the force required to drive it through capillaries. This led to the view that death was usually due to inadequate cerebral circulation caused by the inability of a foamy mixture of air and blood to pass through the pulmonary capillaries. This theory comes close to the theories which stand today.

B. AMOUNT OF AIR NECESSARY TO PRODUCE DEATH

The amount of air necessary to cause death in venous air embolism is still not completely decided. Fatal accidents in persons have happened 22-67 with as little as 50ml. The most generally agreed upon minimal 19-542,53-5 amount in the literature is approximately 100 ml. It must be emphasized that this can vary greatly. If a patent foramen ovale exists and air enters the left heart and coronary circulation as little as 5 ml 1-96 of air can be fatal whereas the venous side will tolerate much more. 30-262 In dog experiments a dose of 1,377 cc of air was tolerated, but this was over a period of 460 minutes before dying. This aspect of time will be discussed later.

In dog experiments air doses of as little as 0.3cc/kg caused increased

-3-

41-237 systolic and diastolic pressures in the pulmonary artery However doses of lcc per kilogram per minute of air can be tolerated for several 30-262 hours in some dogs. It must be pointed out that extrapolation from data from dog experiments to man does not always hold. 400 to 600 ml 22-67 of air could be tolerated if extrapolation from dogs were used. It is well known that 100 ml of air can be fatal to a man.

C. HEART

There are several different mechanisms by which air emboli of the venous channels affect the heart. Durant with his dog experiments while observing the heart injected large amounts of air. The typical millwheel murmur of the heart, which will be discussed later, was heard. Almost immediately following the injection of air. there was noted a sudden marked dilatation of the right auricle and ventricle associated with the rise in 21-273 venous pressure. With this dilatation of the right ventricle which was especially profound in the out flow tract region, there developed an area of easily observed ischemia just to the right of the lower reaches of the anterior descending branch of the left coronary artery. The most ventral portion of the right ventricular outflow tract was found by transillumination to contain a large air bubble. The right ventricle continued to dilate and contractions became more and more feeble until death supervened.

With the use of stereoscopic anglocardiography and cinefluorography Durant was able to demonstrate that the air causes a gas trap in the right 22-66 ventricle outflow tract. The air accumulates in the pulmonary arterioles, pulmonary arteries, right ventricle and right atrium thus producing right heart dilatation. While visualizing this dilatation one can see

-4-

beginning pallor of the heart muscle. This dilation causes muscular 19-542 ischemia and a decrease in coronary blood flow and death from arrhythmias.

Dasher has shown also that small amounts of air may cause fibrin 19-542 clots or thrombosis and obstruct cardiac blood flow.

The valvular action of the heart is also affected by this air which is backed up from the lung arteries and accumulates in the right heart thus causing dilatation. There are two phases of valvular action in the atrioventricular valves. The first phase appears to be muscular and involves an actual narrowing of the right at the base of the valve. The second phase is hydrodynamic and requires the presence of sufficient fluid within the ventricle to swing the valve leaflets into position for effective closure. If the ventricle is filled with air, the second phase does not occur, and the valve remains partially open. The ventricle develops no head of pressure to open the outflow valves. There is no propulsion of either fluid or air. The aortic and pulmonary valves remain closed. Thus the heart cannot build up pressure enough to move the air out or $\frac{46-637}{1000}$

The cause of the myocardial ischemia in the air embolism and resultant right ventricle dilatation can be explanned by the coronary blood flow 21-275 findings of Visscher. The flow of coronary blood depends on a pressure gradient measured by the differences in pressure between the aorta at one end of the coronary sinus and right ventricle at the other end. The amount of blood flow to the coronary sinus and right ventricle (thru the thebesian channels) is determined by the aorta-pulmonary artery pressure difference. The greater this pressure difference, the greater the thebesian flow and the less the coronary sinus flow. With a low aorta-

-5-

pulmonary artery pressure difference there will be a great falling off of the thebesian flow. The right ventricle is greatly dependent on coronarythebesian circuit for its nourishment. It is the right ventricle which is especially vulnerable to increased pressure in its outflow tract. This is in contrast to the left ventricle which can tolerate increased loads for long periods of time. When the right ventricle pressure is greatly increased as in a venous air embolism the aortic pressure is reduced and the thebesian flow decreases. These two combine to markedly reduce the pressure gradient and decrease blood flow to the right ventricle. Ischemia is the result.

Another point should be mentioned while discussing the heart. As early as the 1800's it was recognized that inspiratory phase of respiration produced negative venous pressure and thus drew air into open vein. This negative thoracic pressure is responsible for many of the accidents which occur. The air once in the veins finds its way to the heart.

D. LUNG

High pressures are needed to force air through the lungs to the 23-518 arterial side (above 60 mmHg) through pulmonary capillaries. Isolated air bubbles are stopped in pulmonary arterioles when they are of greater than 30 to 40 u in diameter. Air bubbles of 30 to 40 u or more in diameter behave like plastic solidsand become trapped in smaller pulmonary vessels 22-66 throughout the lungs. Gordon has shown arteriovenous vessels average 15 to 19 u in diameter so it seems unlikely that bubbles should be carried through except when marked rise in pulmonary pressure occurs. This rise 23-518 may only occur in right-side-down position.

When small amounts of air are injected into pulmonary arterial circulation the air produces obstruction and leads to pulmonary arterial

-6-

hypertension and to certain reflex phenomena. These reflexes are a systemic hypotension, bradycardia, and disturbances of respiratory rhythm. 23-517 These are akin to the pressoreceptor and von Bezold reflexes. With the small amounts recovery usually takes place by means of solution. utilization and pulmonary excretion of the air. When larger amounts of air enter the pulmonary vessels the amount is in excess of the eliminating capacity and accumulates. Reflexes occur and the difficulties under which right heart labors are enhanced by the existence of unfavorable gradient of coronary flow as the aortic blood pressure falls due to reflex and at the same time the pressure within the chambers to which coronary venous blood is returned increases. The drop of systemic pressure is proportional to 30-263 the amount of gas injected. 41-237

Mandelbaum injected air into dogs and then did autopies on them. The autopies revealed in 80% of the dogs that there were numerous small ecchymotic foci scattered throughout the subpleural areas of several lobes of the lungs. They ranged in size from 2-3 cm. Alveolar edema was present microscopically. Also pulmonary hemorrhage and infarction was found. The adjacent pulmonary capillaries were engorged with blood and alveolar collapse was frequent.

E. TYPE OF GAS

An important consideration in venous air emboli or more properly termed venous gas emboli is the type of gas injected. The most important gas characteristic other than volume is its solubility. Carbon dioxide is at least 20 times as soluble as air in serum at 38 degrees C. It is 22-67 this difference that has made it a safer negative contrast media. In dogs the LD_{50}^{\dagger} for air is 5.1 ± 0.5 cc per kilogram according to Graff. 30-264 For CO₂ the LD_{50} is 25 ± 1.7 per kilogram. Therefore air is five times more toxic than CO₂ in dogs. The LD₅₀ for CO₂ in a 50 kilogram

-7-

person would be around 1,200 to 1,300 cc. Nitrous Oxide has also been recommended for use due to its solubility.

In contrast to the soluble gases it is found that air, oxygen and 22-67 nitrogen are very insoluble. The fact that oxygen is in this category is not appreciated by many physicians as seen in many reports containing the statement, "We have used injections of oxygen in order to avoid the danger of air embolism." Oxygen was found to have approxemately the same fatality rate as air.

F. ARTERIAL SIDE

I'll just say a quick word about arterial air emboli because for example if a patent foramen ovale exists a venous air embolus can go to the arterial side. Death is most often due to impaction of an air bubble in the coronary artery which promptly leads to ventricular fibrillation of a kind resistant to treatment because of the air bubble, once arrested in a narrow vessel it is difficult to displace. Also cerebral embolism may occur.

-8-

IV. DIAGNOSIS

A. MILL-WHEEL MURMUR

To make the diagnosis of venous air embolism it first must be thought of. The signs and symptoms of the air embolus are quite characteristic. Probably the single most diagnostic finding is the presence of a Millwheel murmur heard by a stethoscope placed over the precordium. Investigators as early as the mid 1800's heard the mill-wheel cardiac murmur and 56-581 described it as a beating up the white of an egg. The mill-wheel murmur is a peculiar, gurgling, churming and rushing heart sound, which has been attributed to the churning of the frothing blood within the 11_2 heart chambers. Actually the murmur probably represents the right 26-1353 ventricle beating against the bubbles of air. 55-87

In the work done by Shivpuri and others, they have found that a sign appears earlier than the mill-wheel murmur which they call the drum sign. It was found that after injecting air in dogs changes in heart sounds appeared in 10-15 seconds while symptoms appeared in 40-50 seconds. In 10-15 seconds the heart sounds became metallic and resonant in character and the heart rate increased. In 20-25 seconds the sounds became drum like in character. It is felt this is pathognomonic of venous air embolism. After 25-30 seconds the sounds resemble a galloping horse on a wooden bridge. The sound can be simulated by stroking the diaphragm of the stethoscope with the finger nails. At 30-40 seconds the systolic churning murmur occurs. At 40-50 seconds the first symptoms of groaning or convulsions appear.

Just under a minute after the air has been injected the patient will experience abrupt onset of chest pain, dyspnea or unconsciousness. In many of the cases I have reviewed the awake patient will abruptly sit up

-9-

with a startled appearance then fall unconscious. At this time the mill-wheel murmur can be heard. The murmur has also been called the 22-70 bruit de Moulin.

B CARDIOVASCULAR SYSTEM

46-635

The cardiovascular system is affected. The venous pressure increases. Cyanosis becomes readily apparent. Retrograde circulatory failure ensuses then evidence of forward circulatory failure appears. The blood pressure drops. The pulse becomes rapid and thready. Syncope occurs due to cerebral ischemia. An important finding is that the heart continues to beat after respirations have stopped so many can recover if prompt treatment is instigated.

C. PULMONARY SYSTEM

The pulmonary reflexes have already been discussed but the respirations are affected. Shivpuri found only an increase in depth of respirations 55-87 but no change in rate. However, others have said the primary change 1-96, 21-273 is one of increase in depth, but sometimes an increase in rate. The rule generally then is hyperpnea with tachypnea occasionally present.

Other diagnostic helps are offered by several investigators. If the pulps of the fingers are placed over the jugular vein, bubbles of air can 7-284 be felt moving beneath. Nicholson feels the diagnosis of venous air embolism can be considered almost established if the patient makes a 46-638 rapid recovery when quickly placed on the left side down. (This will be discussed in the section on treatment.)

In the neurosurgical patient in the sitting position fatal air embolism is usually preceded by an initial minor warning embolism which causes a small fall in blood pressure, a rise in pulse-rate and some 1-96 increase in depth and sometimes rate of breathing. These signs may be interpreted as just insufficient anesthesia. But if the anesthetist compresses the jugular veins in the neck when such disturbances appear, he will produce enough local venous congestion in the operation area to demonstrate an open vein if one is present, and thus be able to diagnose air embolism before a major catastrophe has resulted. It is felt that routine application of this test reduces the mortality from air embolism 1-96during neurosurgery in the sitting position. Also in neurosurgical procedures Marshall recommends the use of an esophageal stethoscope for h2-255the best early detection of air embolus.

D. ELECTROCARDIOGRAM

The ECG findings in venous air embolism have been recorded by several 21-275 investigators. Durant found there is ST segment depressions in leads II and III without obliteration of the upright T waves. No change in the S-T segment in lead I is seen but an inverted QRS or deep S wave is 19-543 often seen. Distrubances in rhythm consisting of varying degrees of A-V block or development of nodal rhythm is often seen. The deviation of the S-T segments disappear rapidly if recovery occurs. If QRS changes and intraventricular block continue, ventricular fibrillation and death occur.

When the electrodes are placed directly on an animals heart the sequence of events are as such. Immediately sharp T wave inversion and in 10 seconds an elevation of the S-T segment appears and as it becomes more marked, the T wave inversion disappears. Then develops a more extreme 21-275 S-T segment deviation. It surpasses the R wave in height.

30-263

Graff found the ECG findings quite typical of acute cor pulmonale with increase in amplitude of p waves, deepening of S waves, and heart block. These were all transient if survival occurred.

E. X-RAY FINDINGS

A chest x-ray of a patient who has just experienced a venous air embolism would show a marked decrease in pulmonary artery roentgen density, 23-510 indicating presence of air in this vessel. It is quite evident this cannot be of any value in the immediate diagnosis since the equipment is usually not present and the time involved would be entirely too long. However, the chest roentgenogram can be of great value in the postmortem period in the diagnosis of venous air embolism especially if an autopsy 46-638 permit is not obtainable. Nicholson recommends to clarify the relationship of air embolism and sudden death to obtain a postmortem roentgenogram of the chest of all patients who die suddenly and unexpectedly. This is easy and requires no special permission. Roentgenogram would show air in the vena cava, right heart and pulmonary tree.

F. AUTOPSY

The autopsy can be very helpful not only in establishing the diagnosis but also in establishing the site of entrance, cause and the type of gas involved. The heart should be opened under water inorder to prove the presence of gas within it. This can be done by ligating the vessels and 11-2removing the heart and then opening it under water. In dogs who survived an embolus of air they were found at necrosy to contain no air in 23-511the heart or great vessels 20 to 30 minutes after the injection. In fatal cases there was air in the right heart, in the pulmonary artery and its branches, and in the vena cava. 39-248

The autopsy findings are usually a right heart that is dilated. The right chambers are filled with blood foam which can be aspirated. The site of entrance of air into the venous circulation can be established by injecting air under pressure. The frothy blood and air bubbles are also

-12-

found in the pulmonary trunk, inferior or superior vena cava and the coronary veins. In four autopsies done by Larson no air had passed to the left 39-248 side of the circulatory tree.

During autopsy it is not standard practice to measure the volume of air present or to demonstrate in it the presence of atmospheric oxygen by gas analysis. It may be preferable to make this scientific observation, 53-3which would distinguish air from putrefactive gases. It should be kept in mind that bubbles of air can be forced into the right heart during post mortem pulmonary collapse through damaged alveolar membranes into the capillaries and then to the heart. At the autopsy one should demonstrate air in the veins leading to the source such as the uterus if abortion is expected. -14-

V. TREATMENT

A. GENERAL

The keystone to successful treatment is early diagnosis. A high index of suspicion and a precordial stethoscope are mandatory in operations upon the head and neck, and in which the patient is in the reverse 26-1354 Trendelenburg position or pressure-driven instruments are to be used. After a venous air embolus has occurred if the patient survives fifteen 11-2 minutes the prognosis is good. If the survival is over the first hour, usually the coma and paralysis will clear. Studies by Nicholson have shown that large amounts of air injected into dogs who survived injection largely eliminated the air from the heart in fifteen minutes and completely to within thirty minutes in those who survived for that period of time. 21-27h

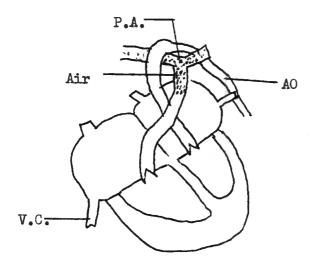
There are several factors which relate closely to mortality. These factors which the treatment of venous air embolism is based on are: 1. The amount of air injected. There is no dosage / weight relationship exactly because of the other factors.

2. The <u>speed</u> of injection. Animals can tolerate large quantities of air if injected slowly. As an example one dog took 1000 cc of air in 100 cc amounts at intervals of 5-10 minutes.

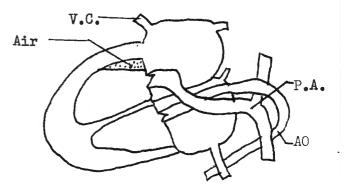
- 3. Position of the patient. This is very important. One can tolerate more air while on the left side than anyother position.
- 4. Effectiveness of the pulmonary excretory mechanism. Tachypnea acts as a protective mechanism and tends to prevent death.

B. POSITION

The choice of position during the treatment of venous air embolus probably means life or death for the patient. If the patient is turned on his right side he will surely die. The proper position is to turn the patient on his left side. Durant has shown experimentally that placing animals on their left side relieves the intra-cardiac obstruction of the pulmonary air embolism. The air that is trapped in the right ventricular outflow tract floats toward the apex of the right ventricle and the ECG returns to normal. The air in the right ventricle is then churned into small bubbles and passes to the pulmonary vessels to be excreted by the lungs. It is also advisable to place the head down thus avoiding fatal cerebral air embolism is a patent foramen to the arterial side exists. The following diagrams are to show what happens to the air trap when the patient is $\frac{16-611}{10}$



A. Air trapped in the pulmonary artery with patient on back



B. Shifting of air trap from pulmonary artery to right heart by turning patient on left side

To show how important position actually is in the recovery Durant 22-68 experimented with animals. Animals were given 7.5 ml/kg of air and while lying on their back had a mortality rate of 69.8%. Animals turned on their left side had a mortality rate of 39%. Animals lying on their right side had a 100% mortality rate.

-15-

In 93 cases collected from the literature by Gottlieb, 40 were untreated and 37 died, a mortality of 93%. 53 patients were treated all together. 27 were treated by left lateral decubitus position. The mortality of this group was one third that of the group where oxygen and vasopressors alone or open-chest cardiac massage were used.

Durant found while observing the heart that dilatation of the right ventricle could be reduced after air embolism by turning the patient on the left side. Heart contractions again would become strong and the animals recover.

By X-ray studies it was found after turning on the left lateral side 23-512 the air is slowly removed. The removal of gas from the right ventricle in the left lateral position, in defiance of the principle of air buoyancy, is evidently accomplished by maintenance of vigorous ventricular contractions and by the aspiration effect of deep inspiration.

Right lateral position by far is the worse. It is found that tachypnea does not occur when the animal is in this position. When on the left side the outflow tract assumes a position inferior to the body of the right ventricle. The air rises and is churned into froth and mixed with blood and is transported to the lungs where it is excreted as previously described.

C. RESPIRATIONS

It is necessary for the heart to be able to respond to a rapid increase in its pressure load while suffering from a diminution of coronary flow. The ventilatory function also must beable to respond with hyperventilation to accelerate excretion of gas from obstructing bubbles in the lungs. Artificially controlled respiration in experimental animals reduces the 22-69 chances of survival since it can't hyperventilate.

-16-

Hyperventilation acts as a protective mechanism and tends to prevent death. If on a respirator and respirations are timed hyperventilation 21-274cannot occur and death will result. If also the patient is on his right side this protective hyperventilation does not occur. Durant has shown that the minimal lethal dose of air is increased when the respirations are artificially maintained. This is not so when respiratory reflex 23-518disturbances are allowed free play. Also this is true when a surgically opened thorax exists, even though intratracheal artificial respiration is maintained. This undoubtedly is due to interference with the inspiratory aspiration effects of the normal chest movements.

Some investigators recommend positive pressure oxygen by means of an 55-87, 56-583 anesthetic circuit or its like. The pure oxygen by pressure helps by increasing partial pressure of oxygen in alveoli and ensures better oxygenation of whatever amount of blood is passing through lung capillaries. Secondly it reduces the partial pressure of nitrogen or other gasses in the alveoli thus making it possible for the gas from the air embolus in the lungs capillaries to diffuse out.

One author reported good results with rapid and marked intrapulmonary pressure changes produced through intensive pumping of the breathing bag 37-319 helped on air embolus recovery. They termed it pneumatic massage.

D. NEEDLE ASPIRATION OF THE AIR

If gas is great enough to fill the entire right heart, position change 22-68 is of little benefit. Only by aspiration of the gas can we then hope to restore circulation.

It is felt it is necessary to aspirate the air the aspirating needle should be inserted into the right ventricle rather than the right atrium. The aspiration of air from the atrium is not as effective a means of re-establishing blood flow, and the point of insertion of the needle in

-17-

the atrium is likely to be the site of bleeding which may lead to secondary 39-250 cardiac tamponade. The needle should be introduced below the xiphoid process or in the fifth interspace just to the right of the stermum. The needle should be directed upwards after entering the heart to allow the 39-248 floating air to enter.

Nicholson recommends that if things that have been discussed up to now fail then emergency thoracotomy within 2-3 minutes so that manual systole and displacement of the air by aspiration with a needle and syringe 46-641 should be done. However this generally is not necessary.

E. CARDIAC MASSAGE

The place of cardiac massage in venous air embolism has not been fully established. It is still quite controversial just how much importance should be placed on cardiac massage in the primary treatment. Larson reported that cardiac massage alone without aspiration of air was unsuccessful 39-250 in several surgical cases he reported on.

Gottlieb and Ericsson report of five cases of venous air embolism all of which were treated only with oxygen, vasopressors and closed-chest 29-778cardiac massage. All survived. They suggest this is an effective way of treatment and is good because it is quick. They also point out that sometimes it is impossible to immediately turn the patient on the left side. They feel the chief factor in successful treatment is the speed with which resuscitation measures are begun. They claim here lies 26-1353the advantage of closed-chest cardiac massage. It can be instituted in any position. They do state that whether closed-chest cardiac massage is better than other measures thay cannot say.

Shires attempted open cardiac massage just 30 seconds after the air embolus occurred. He found that it was not effective as long as the right ventricle had air in it. However, when the air was removed then effective

-18-

-19-54-1484

volume output was possible.

I still have several unanswered questions about how closed-chest cardiac massage can be successful. It appears to me that somehow the blockage of the right outflow tract must be corrected before heart contractions will be of any value. This can be done only by a change of position or aspiration. Since the work by Gottlieb is just recent perhaps the answers will be forthcoming in the future.

F. VASOPRESSORS

Many of the authors I have read recommend the use of vasopressors in conjunction with the other modes of treatment. In 93 cases in the literature vasopressors were used along with the other forms of treatment 26-1353and generally it was agreed that they are of value.

The use of adrenaline intravenously or intracardiac has been used 55-87 by many in the treatment.

G. NEUROLOGICAL SURGERY

A special word should be said about the treatment of venous air embolism of the neurological surgical patient in the sitting position. It is often difficult to place the person in the left lateral position after the h2-258 embolus has occurred. A suggestion by Marshall is the use of a leg less armchair and place it on the operating table. The cephalic end of the table is elevated to the vertical position and the chair is placed flush with this support. Surgical draping is done after strapping the patient firmly to the vertical portion of the operating table. To place the patient in the left lateral recumbent position it is necessary merely to rotate the entire table through 90 degrees. In this way the patient doesn't change position relative to the table nor is draping disturbed. The surgeon still has full view of the operating field. Larger amounts of air can be tolerated while in the vertical 1-96 position (sitting) than horizontal. This is because the distribution of air in the vascular system depends largely on the density difference between it and the blood. When animals are laid horizontal they promptly die. Usually people are laid down because of resulting hypotension. If there is a possibility of an air embolus they should be laid on their left side.

VI. ETIOLOGY

In this part I plan not only to discuss the etiology but also the recommendations of the physicians who experienced these venous air emboli if they offered any in their writings. These will be classified as to surgical, diagnostic air injections, therapeutic air injections, obstetrical and accidental. The various etiologies by which various venous air emboli have occurred will be in capital letters and any recommendations will follow.

A. Surgical

- 1. Neck: Many of the venous air emboli that occur do so during head and neck surgery. Of the eleven cases of venous air embolism recorded at Charity Hospital of Lowisiana for the period 1942-1964 six occurred 40-620 during head and neck operations and five were fatal. Recommend: The clamping of all veins before division to avoid aspiration of air during inspiration.
 - a. SCALENE LYMPH NODE BIOPSY: During dissection the right subclavian
 vein was wounded and air was sucked in through the opening.
 <u>Recommend</u>: Instruct all members of the junior house staff delegated
 this operation that complications can arise and knowledge of the
 regional anatomy and emergency treatment of the complication is
 9-308
 essential.
 - b. TRACHEOSTOMY: An 18 month old infant sat up and suddenly dropped ~dead a few hours after a successful tracheostomy for acute laryngeal obstruction. Autopsy revealed the cause of death to be an air embolism. The infant coughed just after the operation was completed and bleeding began. The infant must have dislodged a ligature from the vein. Bleeding was controlled with pressure only.

-21-

The infant must have taken a large inspiration before the cough and drew in air. <u>Recommend</u>: All venous hemorrhage especially from the lower end of the incision must be completely controlled. A non-slipping ligature should be used, such as silk. Tracheostomy bove the thyroid isthmus would be the safer standard procedure. 56-581

- c. SUICIDE ATTEMPTS BY CUTTING THE THROAT 56-581
- d. INTRODUCTION OF NEEDLE INTO THE NECK
- e. ROOT CANAL SURGERY IN DENTISTRY: Seven dogs were subjected to varying pressures of air after root canals had been cleaned and 48-402 widened. Four dogs died of air emboli.
- 2. Chest

a.

11-2 LOBECTOMY

11-2

- b. RADICAL BREAST DISSECTION
- c. CARDIAC SURCERY: Pulmonary air emboli during open heary surgery may result in significant increase in pulmonary hypertension at the critical time of bypass termination in patients with preoperative pulmonary wascular changes. <u>Recommend</u>: To prevent pulmonar, air embolism at operation it is recommended that clamping of the pulmonary artery be done before opening the right heart, and also prevention of forceful respiratory motions when the heart 4-445 is open. Since Anderson has taken precautions not to let air enter the pulmonary artery, 130 clinical cases have had marked reduction in postoperative pulmonary problems. Pulmonary air embolism besides increasing artery pressure, causes at least temporary decrease in compliance and decrease in pulmonary function.

3. Neurosurgery

- a. OPERATIONS INVOLVING DURAL SINUSES OR VEINS AROUND CERVICAL : 46-635 AND UPPER THORACIC VERTEBRAL COLUMN IN THE SITTING POSITION.
- b. POSTERIOR FOSSA EXPLORATION: In the sitting position posterior fossa and posterior cervical region has been found to be the 42-255 largest involved region of air embolism in neurosurgery. Air emboli result from air entering open veins as in one case where a self retaining retractor held a vein open. Venous air emboli have not been recorded with operations on the anterior or middle cranial fossa or cervical spine using the anterior 42-256 Because of the type of supporting structures approach. the dural sinuses are held open even after they have been severed. Whereas, veins in other parts of the body tend to collapse after 10-56, 42-258 The use of Recommend: 1. they have been cut. an endo-esophageal stethoscope for all neurological cases. 2. Special positioning arrangement so the patient can be quickly turned on the left lateral side. 3. Adequate depth of anesthesia must be maintained during surgery to prevent any straining or coughing which might cause deep inspiration and increase in negative venous pressure. A. Elevation of the feet and use of the G-suit to help keep venous pressure above atmospheric. 5. Assisted positive-pressure anesthesia during critical periods of surgery is helpful to prevent air embolism by preventing negative intrathoracic pressure during inspiration. 6. Flood open veins with saline to keep air from entering. 7. Use of bone wax to keep air from entering into the bone marrow. 8. Watch for venous bleeding and try to prevent it.

- a. POST PARTUM SURGICAL STERILIZATION: The post partum uterus provides easy access of air through the wide venous sinuses in the endometrium. With an abdomenal procedure for sterilization in the multiparous women air can be trapped in the uterine cavity and then forced into the uterine sinuses on change of position from Trendelenberg to flat after the operation is completed. This change can cause a drop in the position of the uterus by 44-274 15 cm. When a women has fat thighs they can trap the air.
- b. TRENDELENBURG POSITION IN THE POST PARTUM UTERUS
- c. TUBAL PLASTIC WORK WITH INSUFFLATION WITH AIR: In one case described a probe was placed through the stump of the follopian tube with difficulty and trauma to the uterine wall resulted during attempts to insert the polyethylene catheter. Venous sinuses were opened so that the repeated insufflation to demonstrate tubal patency introduced air directly into the circulation. <u>Recommend</u>: Insufflation with saline solution tinged with methyleme blue instead of air.
- 5. Other procedures

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- a. AMPUTATION THROUGH THE SHOULDER JOINT
- b. OTHER AMPUTATIONS AND VENESECTIONS
- B. Diagnostic air injection
 - 1. Into the peritoneal cavity
 - a. RUBINS TEST: Recommend: The use of carbon dioxide instead of either air or oxygen.
 - 2. Into presacral cavity, urinary bladder and large joints PRESACRAL AIR INSUFFLATION: Retroperitoneal pneumongraphy for the

delineation renal, adrenal and retroperitoneal lesions by introduction of gas insufflation can cause air emboli. In a survey of 1267 urologists there were reported 58 fatal reactions and 28-382
68 near fatal ones. <u>Recommend</u>: 1. Carbon dioxide only.
2. Contraindications to these procedures are: a. Acute infection in path of injection. b. Large renal or retroperitoneal tumors which frequently have large dilated veins on their surface.
c. Fascial plane malignancies in which adhesions or excessive vascular lesion could be dissected by entering air.

- b. OXYGEN INJECTION OF THE KNEE: Pneumoarthrosis for the diagnosis of loose fragments of patells. Upon aspiration at first no return 38-172 flow of blood was seen.
- c. URETHROCYSTOGRAPHY: Insertion of 150cc of air into the bladder. There was a congenital anterior urethral membrane found. Increasing the pressure was necessary to overcome the obstruction offered by the membrane. A vesicosigmoidal fistula was found and postulated that air may have been forced into veins of the fistula. 27-978 Recommend: Do not use air.

3. Encephalography and angiocardiography

a. PNEUMOENCEPHALOGRAPHY: The incidence at Ohio State of deaths from 1,196 procedures in a ten year period was much higher in children where four had been recorded. There were a total of nine deaths altogether. The complication of air embolism is most likely in children since faulty placement of the needle is more likely. Since the needle used is bevelled, it is possible to have a portion of the lumen within the subarachnoid space and another

-25-

portion in the extradural or intravascular space. In this way spinal fluid can still be obtained but air can be injected into the vessel. <u>Recommend</u>: 1. Short bevelled needles in children. 2. Extreme care in placement. 3. Listen to heart when injecting 34-337

- air.
- b. VENTRICULOGRAPHY: Air embolus occurred eight hours after ventriculography done by typical parieto-occipital manner.
 Why death occurred eight hours later was reasoned by: The lobe shaped laceration of the venous wall probably kept the vessel shut like a valve. A sudden increase in venous pressure, perhaps by coughing, opened this valve. The air contained in the ventricle was aspirated into the vein because of the negative venous pressure. <u>Recommend</u>: Ventriculography only done when complete blockage of CSF and an ascending encephalography will not work. 32-291
- C. Therapeutic Air Injections
 - 1. Examples:
 - a. MAXILLARY SINUS LAVACE: 58 cases of air embolism have been 47-1216 collected from maxiElary sinus lavage by Pang. Thirty from puncture through the inferior meatus, 14 from middle meatus and six from natural ostium. Anatomically this would hold true because of the large plexus of veins in the medial wall of the antrum in the region of the entrance of the trocar. Air is often injected first to see if the needle is in the sinus then after lavage to dry the sinus out. <u>Recommend</u>: Discontinue entirely the insufflation of air into the maxillary sinuses.

PNEUMOPERITONEUM AND PNEUMOTHORAX: Over 100 instances of air b. embolism following pneumoperitoneum have been reported in the 19+542 Although "collapse therapy" is almost literature alone. 50-810 Such a case abandoned it is still used occassionally. was after the removal of a right upper lobe for tuberculosis, there was a small residual space remaining and it was decided to induce pneumoperitoneum by means of transdiaphragmatic injection of air. The needle was inserted through the tendenous portion of the diaphragm. Aspiration revealed no blood. After injection of fifty cubic centimeters of air, a mill-wheel murmur was heard and the blood pressure dropped. A needle was inserted into the right heart and air was removed.

This embolus may have been due to introduction of air into the liver. In animals air injected into the substance of the liver found its way immediately into the inferior vena cava and right heart, suggesting the pathway of the air in the above case. <u>Recommend</u>: Use of a catheter passed through a small incision in the diaphragm to prevent introduction of air into the liver and resulting air embolism. Adhesions between the liver and diaphragm 33-158, 50-810 should be considered a contra-indication.

c. VAGENAL POWDER INSUFFLATION ESPECIALLY DURING PREGNANCY: Several cases were reviewed. In one such case the insufflator was used to dilate and smooth out the folds in the vagina so that powder could be sprayed all over the mucosa. Especially dangerous times are during engorgement of endometrium around the menstrual period, open veins after uterine curettage, and the soft

-27-

cervix of a pregnant uterus. All might favor air embolism. <u>Recommend</u>: A vaginal speculum should be in place anytime insufflators are used so that the vagina will be held open and no pressure can develop.

D. Obstetrical

Of all the veins in the body, the uterine sinuses are probably the most vulnerable to the entrance of air. This circumstance is what makes obstetrics and gynecology the specialties in which this 21-272complication is mot feared.

1. Examples:

- a. TRENDELENBURG POSITION: This position favors the entrance of 39-250 air into the pelvic veins.
- b. RUPTURED UTERUS: In one case there was found at autopsy a long transverse laceration at the level of the internal os and thought to be the route of entry of air. <u>Recommend</u>: As a prophylactic measure during expression of the separated placenta the patient legs should be maintained in abduction to secure patency of the introitus and so to prevent positive pressure of air into the 52-486 vagina if a second attempt at expression is necessary.
 Also recommend a wider appreciation of hazards of intrapartum 36-189 rupture of the lower uterine segment.
- c. PLACENTA PREVIA: This is occassionally involved with air ll-3 embolism at term, especially if a version is performed.
- d. CESAREAN SECTION: Immediately after incision into the uterus has been made is the time of highest incidence. ll-3
- e. DOUCHING: Just before the onset of menses.
- f. PREMATURE SEPARTATION OF THE PLACENTA:

-28-

43-168

- CRIMINAL ABORTION: The low lying placenta provides venous g. sinuses for the ready introduction of air into the circulation and one may see immediate or delayed air emboli. In one case there was an attempted abortion by the placement of a rubber catheter into the cervix and blowing as hard as possible. There was a sudden loud cry, gasp for air and the women became Delayed air embolism has been unresponsive and died. reported in abortions. After the abortion attempt contractions of the uterus, coughing, bearing down and other things may cause more extensive separation of the membranes and/or placenta from the interior of the uterus. This may take place sometime after the initial interference. The trapped air can then be forced into the venous sinuses. Several cases of fatal air emboli have been 53-6 reported two to three hours after the abortion try.
- h. KNEE_CHEST POSITION: This should be avoided in the postpartum exercises due to reported air emboli. One such patient who died had fragments of placenta left in the uterus eight days after 11-3 delivery.

E. Accidental

1. Accidental:examples:

- a. FRACTURES OF THE LONG BONES 46-634, 3-461
- b. CAISSON DISEASE
- c. SELF INDUCED: In men it is rare but a couple of examples are found with older men. Air emboli are not rare in women because of abortion attempts. A 69 year old male accidently killed himself by self-insufflation of the urethra with a Higginson syringe. The autopsy showed the death due to air embolism as a result of hemorrhages in the urethral mucosa as a result of

17-1197 distension with air. Another example took a tire pump and inserted the needle into his scrotum and started pumping. 46-634 STAB WOUNDS

d.

e. GUNSHOT WOUNDS: One such case happened when the bullet entered above the right claviele, progressed inferiorly and medially, striking the superior aspect of the right second rib. It went through the right lung and came to rest against the lateral aspect of the body of the ninth thoracic vertebra. It was concluded at autopsy that the bullet had partially severed the right subclavian vein and had created a pneumothorax. Large amounts of air from the pneumothorax were sucked into the circulatory system due to the low, intermittently negative pressure in the large thoracic veins.

To test this a soft rubber catheter was inserted into the wound and a thin suspension of barium was injected. Subsequent films demonstrated flow of barium into the right supraclavicular vein, superior vena cava, and the right chambers of the heart. 24-954 The heart was opened under water and it contained air.

f. HILEBOTOMY: Three different causes of death have been reported. In obtaining blood from a donor if a flask is used which has lost its vacuum the pressure of the air in the flask will increase as it is warmed by the blood. If the technician then decides to shirt to another vein because of the slow rate of flow, and removes the tourniquet before withdrawing the needle, air under considerable pressure will enter the vein. Recommend: Remove 22-67 the needle before removing tourniquet.

-30-

Another donor death happened while giving blood using a closed system to withdraw blood. The suction bulb was placed wrong and instead of megative pressure positive pressure was applied to the bottle. The result was death from air embolism. Recommend: 1. Centralized blood taking under the direction of a specific department. 2. Suction bulbs used should be so constructed that they can be used only for aspiration. 3. Strict 12-203 adherence to the technic which is proper.

Still another way pressure may build in the bottle besides heat is pressure generated by arm tourniquet, increased venous pressure, and gravity during the phlebotomy. One death occurred when there was no patent air vent in the container to permit the air to escape. In experiments venous pressure fifty times resting pressure is generated by touriquets. Using a dummy setup the dose of air possible was 100 ml. It takes three seconds to deliver this 51-425 air to the circulation. Recommend: 1. Clamp phlebotomy tube before releasing tourniquet. 2. Use of plastic bags. ADMINISTRATION OF BLOOD: Administration of blood or other fluids rapidly under pressure in a bottle by the injection of air into the bottle should not be done. Many deaths have occurred from 49-1477, 54-1481, 13-98 this technique. In most of the cases a sucking noise is described as being heard in the blood administering set just before the patient developed the signs and symptoms of an air embolism. Recommend: Discontinue the use of bottles for the storage of blood and to use only plastic bags where pressure by means of a cuff can hasten blood administration.

g.

-31-

It should be mentioned that the volume of oxygen and carbon dioxide held in physical solution in the blood is increased as the temperature is decreased. So when blood that has been equilebrated with oxygen is warmed as in the body, the formation of gas bubbles is possible when tension of the gas in the blood 20-110 exceeded the local hydrostatic pressure.

A young girl died from an air embolism because of a defect in 14-429 the tubing above a Martin pump used for pushing blood.

Air emboli have been reported from bag administration of blood. The filter used just below the bag of blood became partially occluded with coagulum. The blood level was low and thus the top of the filter was in the air and thus the air was taken into lh-29, 29+7h the tubes. There was a small defect above to let the air in.

Another point should be made in the placing of the clamp of tubing used in administering fluids. If the clamp regulating flow is placed above the level of the heart and partially occludes the tubing, a defect in the tubing between the clamp $\frac{1}{2}$ and the level of the heart will permit the entrance of air into the system. If the clamp is placed below the level of the heart a defect between the drip chamber and the clamp will produce a leaking of fluid 29-778rather than entrance of air.

-32-

Summary

The history of venous air embolism when it first became known to man goes back to four centuries ago. However, it wasn't until the middle of the 19th century that the importance of air emboli in surgical procedures first became known. Even today many physicians do not appreciate the significance of venous air emboli.

The true incidence of air emboli can only be guessed at since its actual diagnosis is only infrequently made. Estimates of incidence range up to almost 20% of accidents in surgery and anesthesia.

The physiology behind death from venous air embolism is based essentially on a block of the outflow tract of the right heart with air unable to pass the capillaries of the lung. This causes dilation and ischemia of the right heart. Also, so blood is being returned to the systemic circulation so cerebral ischemia results. These two points are the basic cause of death.

The diagnosis of venous air embolism can easily be made if it is thought of. The precordial mill-wheel murmur is almost always diagnostic of air embolism. Other findings are helpful such as cyanosis, blood pressure drop, venous pressure increase, hyperpnea, ECG changes and autopsy findings of a dilated right heart with air in it.

Probably the two most important things in treatment is first to have a preconceived plan of treatment and to practice prophylaxis and be aware of the dangers of some procedures. The patient who has suffered a venous air embolism should be immediately turned on his left lateral side. Administration of positive pressure oxygen should be started but no attempt should be made to artificially control respirations, unless breathing stops. The head should be lowered to prevent any possible

-33-

cerebral embolus in such cases where a patent foramen ovale might exist. At this time vasopressors can be considered. If the left lateral position does not improve immediately the state of the patient, it is probably best to try and aspirate air from the right heart after the source of the air entrance has been closed. After the air has been removed closed cardiac massage may be of value if it is needed. The keystone to successful treatment is early diagnosis and immediate treatment.

The etiologies of venous air emboli are discussed as to surgical, diagnostic air injections, therapeutic air injections, obstetrical, and accidental causes.

-34-

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