MASSIVE VENOUS AIR EMBOLISM

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In a surgical crisis, rapid blood transfusion may be needed, and this is sometimes effected by raising the pressure of air within the transfusion bottle with a Higginson's syringe, or with compressed air or oxygen. This dangerous practice can cause massive venous air embolism. These accidents may be rare, but for obvious reasons they are seldom published. Dornette[2] (1956) describes one such death in a series of 108 fatalities complicating 63,105 anaesthetics. We have heard of 2 cases in other Johannesburg hospitals during the past decade. We here report the case of a patient who survived this disaster. This may be a unique event; it is described to outline the steps taken to resuscitate the heart as well as to emphasize once more the danger of administering blood under positive air pressure. During the emergency the distribution of air within the heart and coronary vessels was noted and later, in an attempt to

Fig. 1: Cine-angiogram of canine heart in the left lateral position. This frame precedes the sudden injection of 100 ml. of air into the inferior vena cava. The inferior vena cava, aorta and heart chambers are opaque.

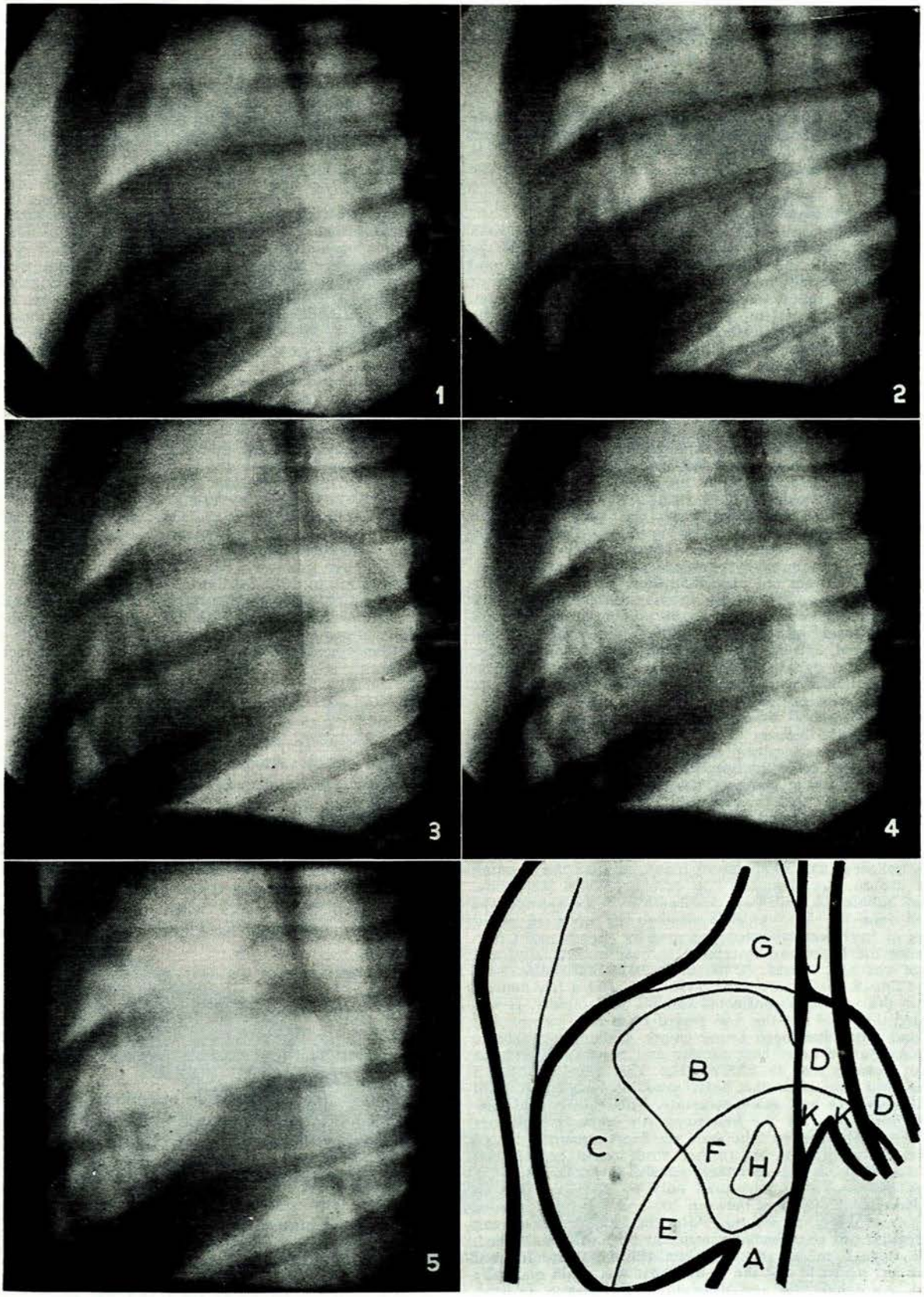
Fig. 2: Frame taken ½ second after the injection of air. The inferior vena cava can no longer be seen, the right atrium is translucent, and the right ventricle contains some air but is not distended. The left ventricle is opaque.

Fig. 3: At 10 seconds. The right ventricle, right atrium and pulmonary artery are filled with air and are distending. The coronary sinus has filled. The ventricular septum, previously convex forwards, is becoming concave. The left heart chambers and aorta remain opaque.

Fig. 4: At 30 seconds. The right heart chambers and pulmonary artery have distended. The coronary sinus is seen.

Fig. 5: At 100 seconds. Heart fibrillated at about this time. Gross distension of the right heart chambers. The left heart and the aorta are opaque and contracted.

Key to Figs. 1-5: Composite tracing of contours of chambers of the heart. A=inferior vena cava. B=right atrium. C=right ventricle. D=pulmonary artery. E=left ventricle. F=left atrium. G=aorta. H=coronary sinus. J=trachea. K=bronchi.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clarify our observations and to explain the mechanism of circulatory failure, a series of animal experiments were conducted.

CASE REPORT

A Bantu female, aged 35 years, was admitted to Rietfontein Hospital on 15 December 1961, with a lung abscess in the left upper lobe. Despite prolonged conservative treatment the abscess failed to resolve and it was decided to remove the affected lobe.

Operation (9 August 1962)

The left chest was entered through the bed of the sixth rib. The pleura was densely adherent and mobilization of the lung was difficult and haemorrhagic. After dissection and ligation of the vessels there was a negative blood balance of some 600 ml. A request was made to speed up the transfusion, which was being given into an arm vein. This was done by pumping air into the blood bottle with a Higginson's syringe. The attention of the person administering the blood was diverted to other demands and, when next inspected, the bottle was empty and air occupied the full length of the transfusion set. It was not possible to guess what amount of air had entered the body. The tubing was immediately clamped and the head of the patient was lowered steeply. The lung was retracted to expose the heart. The pericardium was widely opened. The heart was distended and beating weakly but there was no effective pressure in the aorta. The right atrium, and the right ventricular outflow tract and pulmonary artery, were grossly dilated and tense. Froth was seen through the stretched right atrial wall and churning of blood was audible. The left atrium and ventricle were conspicuously small and, from the rapid examination possible under the circumstances, it seemed that air was not present in these chambers. Bubbles were oscillating to-and-fro in the anterior descending coronary vein and some air was thought to be present in the adjacent artery. The heart was dislocated from the pericardium and inspected behind, and the coronary sinus, the circumflex veins, and the left and right posterior coronary veins, were seen to be tensely distended with froth, and bubbles had reached the apical venous radicles.

For fear of cerebral air embolism, the heart was not immediately massaged. Because of previous successful experience with coronary air embolism, during open-heart surgery, the anterior coronary artery and vein near the apex of the heart were pricked with a number-24 hypodermic needle. Froth escaped vigorously and continuously from the venous prick. The right heart remained distended and so an incision was made in the right atrial appendage, and a gush of frothy blood spurted to a height of about 20 cm. The tension was rapidly relieved, and when liquid blood escaped the opening in the atrium was clamped. The coronary veins were now clear of bubbles. A wide-bore needle on a 20-ml. syringe was inserted into the left ventricle through its apex. A small amount of froth was aspirated, followed by pure blood. Up to this point the heart had continued to beat ineffectually, and massage was now started. 10 ml. of 1/10,000 adrenaline were injected into the left ventricular cavity and after a few manual squeezes the ventricles contracted forcibly and rapidly. It was estimated that 2 - 3 minutes had passed from the moment that the blood bottle had been found empty. Under the influence of adrenaline the heart beat rapidly and powerfully and the systolic pressure rose to 220 mm.Hg. This continued for 5 minutes, but thereafter the heart gradually weakened until manual assistance was again necessary. This precipitated ventricular fibrillation for the first time. An immediate attempt at electrical defibrillation failed and the heart remained dilated and weakly fibrillating. No further froth could be felt and none was seen in the coronary vessels. Over the next 15 minutes massage was interrupted only by four attempts at defibrillation with voltages between 100 and 150 for durations varying from 0·1 to 0·5 seconds. After the sixth shock, a weak and irregular but coordinated ventricular beat was established. At this stage 6 ml. of 10% calcium chloride were injected into the left ventricle and the heart's beat was again manually assisted. An immediate and dramatic improvement resulted. Gradually the irregularity lessened, and 20 minutes after ventricular fibrillation had begun a reasonably normal beat was restored.

The removal of the upper lobe was then completed. The systolic blood pressure had risen to 180 mm.Hg, and bleeding was profuse from all the raw areas of the pleura. This was eventually controlled by electrocoagulation and packing and the blood volume was maintained by transfusion. In all, 9 pints of blood were given. 1½ hours after the air embolism had occurred the operation was completed.

Postoperative Progress

The patient's pupils, which had dilated at the time of the air embolism, slowly contracted, and at the completion of the operation spontaneous respiration returned and voluntary movements were made. Fearing cerebral embolism, we started preparations for mechanical respiration with an Engström unit and a tracheostomy was performed. Mechanical respiration did not prove necessary and the patient, breathing normally, and fully conscious, was returned to the ward. She responded to commands and was able to move her limbs freely and without signs of weakness.

During the following 4 days there was some difficulty in maintaining an adequate blood pressure. In the first 24 hours, 30 mg. of 'methedrine' and 100 mg. of 'solucortef' were given intravenously, followed by 45 mg. of 'meticorten' orally. This latter was decreased daily by 5 mg. 'Levophed' in a drip was given in controlled doses during the first few hours and was then replaced by 'hypertensin', 2·5 mg. in 500 ml. of normal saline, and was followed by 20 mg. of 1% 'aramine' in 500 ml. of normal saline. By the second day the intravenous administration of pressor agents was unnecessary, but 10 mg. of aramine were administered intramuscularly at first 3-hourly and later only when necessary. Because of the massive blood transfusions, 10 ml. of 10% calcium gluconate were given 8-hourly during the first postoperative day. Broad-spectrum antibiotics were administered for a week. Serial electrocardiograms showed flattening of the T waves, which sometimes is normal in the Bantu, and did not alter in the 3 weeks that recordings were taken.

COMMENTS

Because of the need for action during the emergency, our observations were cursory and we were later unsure of our impressions. Animal experiments were, therefore, undertaken, the nature of which were determined by the following facts and impressions:

1. Cerebral air embolism did not occur. This excluded the presence of a patent foramen ovale. However, our first reaction had been to lower the head of the patient steeply and air may have bypassed the carotids. Moreover, some froth entered the syringe when the left ventricle was aspirated but this might have been due to a faulty needle connection.

2. The right side of the heart was grossly distended and tense and the pressure was high. This was shown by the gush of frothy blood when the right atrium was opened.

3. The aortic pressure was minimal when the heart was first exposed. The left ventricle was flaccid in contrast to the tense right ventricle.

4. Air was present in large quantities in the coronary veins, but little, if any, was seen in the arteries.

The impression gained from reconstructing the episode was that blood and air was churned by the beating heart. The expansion of the frothy mixture distended the ventricle and atrium and produced acute tricuspid incompetence. This happened immediately, and no blood or froth could be driven through the lungs, so that the circulation ceased. Air was forced into the coronary sinus and coronary veins because of the high right atrial pressure. The coronary

arteries may have filled in a retrograde manner *via* the heart capillaries. If air was present in the left ventricle it may have entered through the thebesian veins.

This impression accords with the concepts of others. Rangell[6] discussed the opinions of various authors who agree that frothy blood obstructs the outflow tract of the right ventricle and blocks the pulmonary capillaries, causing asphyxia. Martland[5] and Durant, Long and Oppenheimer[3] hold that the airlock in the right ventricle, either in the form of a large bubble or a foaming mixture, overdistends the right heart and interferes with ventricular contraction. Whether air can cross the capillary bed from pulmonary artery to pulmonary vein is controversial. Rangell[6] maintains that in certain cases air will pass to the left side through a patent foramen ovale. This must be a grave danger, for Martland[5] has shown, by postmortem study, that in 25% of people the foramen ovale opens when the right atrial pressure rises above that of the left. Kent and Blades[4] believe that air emboli are held up at the pulmonary capillary bed in dogs, but Birch[1] holds that air passes through the pulmonary capillaries in dogs, but not in man.

ANIMAL EXPERIMENTS

Fifteen mongrel dogs weighing from 12 to 35 kg. were used. The dogs were anaesthetized with intravenous 5% pentobarbital and, if the chest was opened, were ventilated mechanically with a 50% mixture of nitrous oxide and oxygen. Arterial, venous or intracardiac pressures were recorded on an Elema unit. Lead 2 of the ECG was used throughout.

The Distribution of Emboli following Massive Intravenous Injections of Air

Four dogs had sudden injections of 200 ml. of air into the femoral vein. In one, the course of the air was followed by cine-radiography with a 9″ Phillips image intensifier (Figs. 1 - 5). In the others, right thoracotomies were performed and the hearts inspected. A colour cine-film was made of one of these experiments. Central venous pressures and peripheral arterial pressures were recorded. The following observations were made:

After a rapid massive intravenous injection of air, blood immediately churns within the right atrium and ventricle. The central venous pressure immediately rises and the arterial pressure falls (Fig. 6). The right atrium and ventricle continue to beat for some minutes but the arterial pressure drops progressively. The right side of the heart remains tensely distended and froth can be seen and felt in the venae cavae, right atrium, right ventricle and pulmonary artery. No bubbles appear in the pulmonary veins or left heart chambers. The small contracted left ventricle remains in sharp contrast with the dilated right ventricle. After a varying interval, air bubbles appear within the coronary veins, firstly in the posterior veins, and they clearly enter through the coronary sinus. These bubbles are large and take some time to reach the smaller venous radicles. They oscillate back and forth synchronously with the heart beat, with sudden spurts towards smaller radicles when large bubbles fragment or when smaller bubbles enter. Only in one dog did a few bubbles appear in a coronary artery

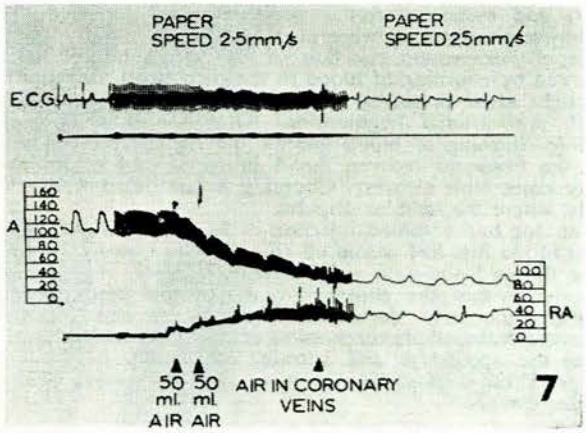

Fig. 7: Right atrial and aortic pressures recorded after the injection of 100 ml. of air into a peripheral vein with the heart exposed. Air appeared in the coronary veins when the atrial pressure had risen to between 40 and 50 mm.Hg and the arterial pressure had dropped to a similar level. The right atrial pressure curve shows a well-marked systolic peak denoting tricuspid incompetence. A=aorta. RA=right atrium.

but their origin was obscure. The right heart remains distended, its beat gradually weakens as it becomes cyanosed, and it eventually fibrillates. This happened between 90 seconds and 3 minutes after injection in all cases.

Air enters the coronary vessels when the right atrial pressure reaches 30 - 40 mm.Hg (Figs. 7 and 8). In most experiments the arterial pressure had dropped profoundly by the time air appeared within the coronary veins. The rise in the central venous pressure appears to be partly due to the expansion of its contents owing to frothing of blood, and partly to the development of acute tricuspid incompetence, so that the right ventricular pressure is transmitted to the right atrium. In all right atrial tracings taken at fast speeds, ventricular systolic pressure waves were seen (Figs. 7 and 11). At necropsy air is distributed in all the visceral veins excepting the portal vein. Bubbles are present in every organ except the spleen, the stomach and the

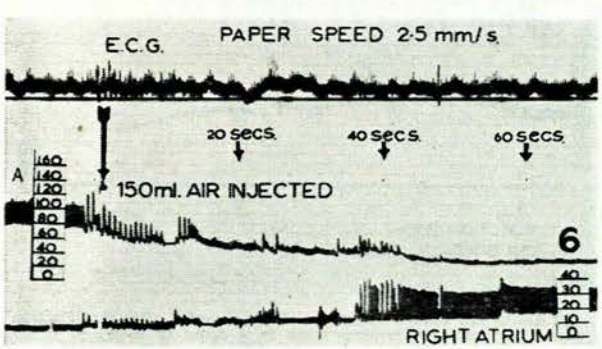

Fig. 6: ECG and arterial and right atrial pressure records of an experiment in which 150 ml. of air were injected rapidly into the femoral vein. The heart had not been surgically exposed, but tricuspid incompetence probably developed at about 35 seconds, when the right atrial pressure rose suddenly. Ventricular fibrillation occurred after 3 minutes. A=arterial.

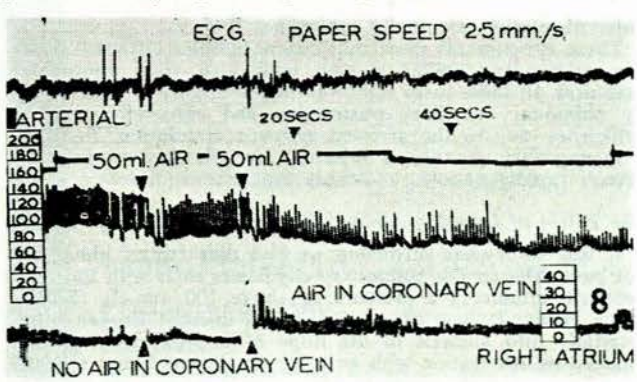

Fig. 8: Arterial and right atrial pressures recorded during intermittent injections of 50 ml. of air into a peripheral vein. After the first injection the arterial pressure drops but recovers rapidly. The venous pressure is unaffected. The second injection was made before full recovery from the first and immediately the venous pressure rose, the arterial pressure dropped, and air appeared within the coronary veins. Following right atrial decompression and cardiac massage this heart was resuscitated.

small and large bowel. The liver is always involved and obviously acts as a filter preventing the entry of air into the portal vein. Apart from a few bubbles in one dog's coronary arteries, no air was found in the systemic arteries, left side of the heart, or pulmonary veins. The valves in the peripheral veins prevent retrograde filling, so that bubbles are scanty in the muscles of the limbs.

The Effect of Small, Graduated Intravenous Injections of Air

Two dogs had small measured quantities of air injected into the femoral veins. Their hearts were directly observed. Right atrial and systemic arterial pressures were recorded. The following observations were made:

Rapid intravenous injection of up to 50 ml. of air is followed by churning of blood in the right heart, elevation of the right atrial pressures, distension of the right side of the heart, and arterial hypotension. After 15 - 30 seconds the audible churning of blood lessens, the right heart contracts, and the pressures recover. Small injections can be repeated many times with recovery. Churning is last heard within the lungs, where the bubbles disperse.

One dog had a second injection of 50 ml. of air before the effect of the first had passed off (Fig. 8). The systemic pressure again dropped, the right atrial pressure suddenly rose, and air appeared within the coronary veins. In this case tricuspid incompetence only developed after the second injection. Recovery followed decompression of the right atrium by incising the appendage and allowing a quantity of froth to escape. These dogs survived without apparent neurological or cardiac damage.

The Effect of Injections of Air Directly into the Pulmonary Arteries

In the previous experiments air occupied the whole right side of the heart, distending the ventricle and atrium, and obstructing the pulmonary arteries. So as to determine the part played by peripheral pulmonary arterial obstruction, 3 dogs were used in which varying quantities of air were injected directly into the main pulmonary artery. Right ventricular and peripheral arterial pressures were recorded. The following observations were made:

After an injection of 50 ml. of air into the main pulmonary artery, the right ventricular pressure immediately rises and the systemic arterial pressure drops precipitously. Blood churns within the lungs. The right ventricle distends but continues to beat vigorously to produce very high pressures. No air appears in the left side of the heart or within the coronary vessels. Churning of the blood in the lungs ceases within about 30 seconds when the right ventricular pressure drops and the systemic arterial pressure rises and overshoots its pre-injection level. When 100 ml. of air is injected, acute right ventricular hypertension occurs, pulmonary valvular incompetence follows, and air appears within the right ventricle and atrium. Ventricular fibrillation eventually supervenes (Fig. 9).

These experiments show that coarse bubbles do not traverse the pulmonary capillaries even with high right ventricular pressures. In these cases right ventricular failure is precipitated by pulmonary capillary obstruction and coronary-artery insufficiency due to the arrested systemic circulation. Provided a manageable quantity is injected, air will disperse into the alveoli rapidly enough to enable the heart to recover.

The Effect of Injecting Air and Oxygen into a Systemic Artery

It was somewhat surprising to find that frothy blood did not pass through the pulmonary capillaries even with the right ventricle producing a pressure of above 100 mm.Hg (Fig 9). Some years ago one of us (P.M.) had injected oxygen intra-arterially into animals in the hope of improving anoxaemia induced by respiration with an inert gas. It was found that the introduction of more than 100 ml. of oxygen per minute resulted in gas embolization to the right side of the heart, and eventually to universal gas embolism. The impression was then gained that bubbles of oxygen passed readily through capillaries. It was decided to repeat these experiments with both air and oxygen injected into the abdominal aorta.

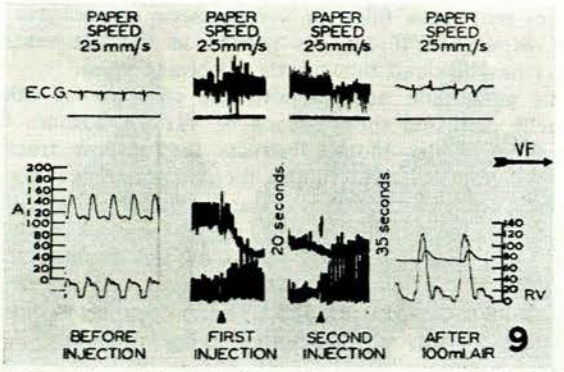

Fig 9: Systemic arterial and right ventricular pressures recorded during 2 injections of 50 ml. of air into the pulmonary artery at 30-second intervals. After the first injection the right ventricular pressure rose to about 80/0 mm.Hg, and the systemic pressure dropped to 60/45. This was due to pulmonary capillary and arteriolar obstruction. The heart was recovering (drop in pulmonary and rise in systemic pressure) when the second injection was made. The right ventricular pressure now rose to 120/0. This time the heart did not recover. No air appeared in the left heart chambers. A=arterial. RV=right ventricle. The arrow indicates the onset of ventricular fibrillation (VF) in 70 seconds.

Observations with air. In one experiment, injections of 200 ml. of air in 4 doses of 50 ml. at 10-second intervals were made into the aorta. After the first injection the systemic pressure immediately rose. This was due to the increased peripheral resistance caused by blockage of the arterioles and capillaries. No further pressure rise followed subsequent injections, but the systemic pressure gradually dropped and the heart fibrillated within 100 seconds (Fig. 10). At no stage was the central venous pressure affected. When the heart and lungs were opened under water, fine bubbles of air escaped from the left ventricle and pulmonary veins as well as from the right side of the heart and the systemic veins. It is possible that air refluxed into the left side of the heart when the left ventricle failed. Bubbles would, however, have had to pass the barrier of both aortic and mitral valves to fill the left atrium, and this would seem unlikely in view of the known barrier effect of the venous valves and in the absence of left ventricular dilatation. To resolve this query, 100 ml. of air were injected into the aorta below the renal arteries under X-ray screen control. Air was seen to fill the aorta beyond the point of injection, and the peripheral arterial tree was immediately outlined. Almost simultaneously air was seen in the right heart. Systemic hypertension was followed by failure of the heart, hypotension, and ventricular fibrillation. As

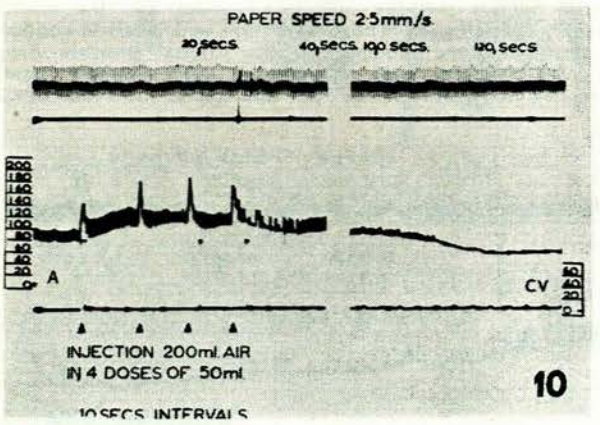

Fig. 10: Aortic and central venous pressures recorded after the injection of 200 ml. of air into the terminal aorta in 4 doses at 10-second intervals. The arterial pressure peaks reflect the pressures of the injection. The arterial pressure rose maximally after the first injection due to peripheral arteriolar obstruction and further injections had no effect. The heart fibrillated within 3 minutes. Fine micro-bubbles were dispersed throughout the veins and arteries of the body. No frothing occurred and consequently no rise in the venous pressure resulted. A=aorta. CV=central vein.

hypotension progressed, air was obviously present in the proximal aorta. It was not possible to decide by screening whether the left ventricle and atrium contained air. Necropsy revealed fine bubbles of air throughout the body occupying both the right and left sides of the heart and the pulmonary artery and vein. The bubbles were identical in the left and the right heart chambers. The impression gained from this experiment was that the pulmonary venous bubbles had entered from the pulmonary artery and not from reflux from the aorta. The air bubbles are different from those resulting from intravenous injections. They are very fine and best seen with a magnifying glass, whereas those resulting from venous injection are large and coarse and associated with frothing of the blood. After an arterial injection frothing is minimal, and the fine air bubbles appear evenly suspended in the blood.

Observations with oxygen. Three separate experiments were performed, where 150 ml. of oxygen were injected into the inferior vena cava, pulmonary artery and aorta respectively. The findings were identical with those following similar air injections. After the venous injection the blood frothed within the right side of the heart, tricuspid incompetence was produced and, with the development of high venous pressures, coarse bubbles appeared within the coronary and other veins but not in the pulmonary or portal veins (Fig. 11). After a pulmonary arterial injection of 150 ml. of oxygen, right

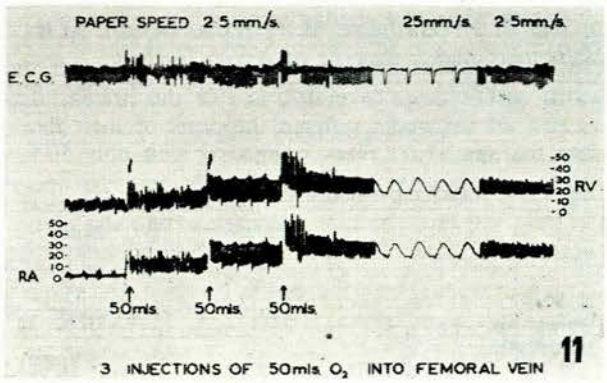

Fig. 11: Right ventricular and right atrial pressure tracings taken during the injection of 150 ml. of oxygen into the femoral vein. The right atrial pressure tracing follows the right ventricular curve from the time of the first injection and indicates the development of tricuspid valve incompetence. Massive injections of oxygen behave in the same way as injections of air. RA=right atrium. RV=right ventricle.

ventricular hypertension followed and the heart eventually fibrillated. Acute pulmonary and tricuspid valve incompetence occurred and bubbles of oxygen were present in the right ventricle and in the veins of the body. After an intra-arterial injection fine bubbles were dispersed within the whole vascular tree both arterial and venous. Little frothing occurred and the right ventricular and right atrial pressure did not rise.

DISCUSSION

A massive intravenous injection of air obstructs the pulmonary capillaries and, with the froth resulting from the churning of blood by the heart's action, distends the right ventricle and atrium. This causes incompetence of the tricuspid and pulmonary valves and, when the right atrial pressure rises above 30 mm.Hg, bubbles are forced retrogradely into the veins of the body. The bubbles are coarse and do not traverse capillaries, so that air does not appear within the pulmonary or portal veins. The lung and liver are effective barriers to the passage of bubbles. The coronary veins fill *via* the coronary sinus, and some bubbles may enter the left ventricle through the thebesian veins. Peripheral venous air emboli cannot cause irreversible damage and, provided a significant quantity of air can be evacuated and the circulation restored, the remaining bubbles can be driven to the lungs to be quite rapidly absorbed. In 4 of the dogs, determined resuscitative attempts were made and the animals survived. The presence of a patent foramen ovale alters the outlook, for air then enters the left heart to be driven to the cerebral vessels, where as little as 10 ml. may cause profound cerebral damage. A patent foramen ovale must be exceptional in dogs, but is present in 25% of humans.[5]

With the chest open at the time of air embolization, prompt and accurate resuscitative measures are possible. Theoretically it should be possible to resuscitate a case of accidental venous air embolism even with a closed chest. In the absence of a septal defect, only the heart is immediately endangered. We would suggest that the external jugular vein in the neck be immediately opened by incision and external cardiac massage instituted. Digital compression on the cerebral side of the vein should control haemorrhage while froth escapes from the central end. No valves are present between the external jugular vein and the heart, and an incision should adequately decompress the right side of the heart if assisted by external cardiac massage. There is no danger of forcing air into the systemic circulation unless a septal communication exists, in which case all would be lost whatever was attempted. Provided the right heart can be adequately decompressed, the lungs should absorb air bubbles delivered to it. Blood transfusion and external or internal defibrillation should be used if required.

The lungs are effective filters of air when the bubbles are large. It is a constant finding that, after a massive quantity of air or oxygen is injected into the aorta, bubbles will pass through the systemic and pulmonary capillaries. These form a fine suspension within the blood and little frothing occurs. Universal embolization and death are inevitable. This indicates that at least a proportion of the capillaries of bone, muscle, skin and vital organs are of larger calibre than those of lung. The pulmonary capillaries were shown by Schulze[7] in 1871 to have an extremely fine mesh. It is possible that bubbles first forced through the systemic capillaries by the high systemic pressure are reduced sufficiently in size to be forced through the lung capillaries. It cannot only be a question of air being squeezed through capillaries by the greater systemic pressure, for after direct pulmonary arterial injection the right ventricular pressure may rise to 120 mm.Hg without bubbles appearing in the pulmonary veins.

SUMMARY AND CONCLUSIONS

The accidental human case reported here together with the experimental results shows that, in the absence of a cardiac septal defect, air or oxygen injected into the systemic veins in a coarse stream does not traverse the lung capillaries. Coronary air emboli are confined to the veins, though it is theoretically possible that a small quantity of air could enter the left ventricle through the thebesian veins. Death is due to right heart failure that has directly resulted from obstruction to the pulmonary arteries and the air lock in the right ventricle and atrium. Acute pulmonary and tricuspid valve incompetence is produced, so that the contraction of the right ventricle forces air retrogradely into the systemic veins throughout the body.

No air enters the portal vein because the liver is interposed between it and the heart.

It is possible to resuscitate the heart by decompressing the right atrium and allowing most of the froth to escape. Thereafter, standard resuscitative procedures should succeed in establishing a normal heart beat, provided intervention is prompt. The article reports successful resuscitation on a human, duplicated on 4 occasions with dogs.

Air introduced intra-arterially is inherently more dangerous, even when injected beyond the vital organs. In dogs a massive intra-arterial injection of air is followed by the appearance within the veins of fine bubbles that do not froth, and these pass readily through the lungs and enter the left heart. It is thought that the systemic capillaries are larger than those of lung, and that the systemic pressure is able to force the bubbles through these capillaries, and in so doing alters the physical properties and reduces the size of the bubbles sufficiently to allow them to be forced in turn through the pulmonary capillaries by the right ventricle. This does not happen when coarse bubbles of air are introduced directly into the pulmonary artery.

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