CARDIOPULMONARY EFFECTS OF INTRAVENOUS GAS EMBOLISM; WITH SPECIAL REFERENCE TO FATE OF INTRAVASCULAR GAS Bubbles

Yohtaro Oyama, M.D.* and Merrill P. Spencer, M.D.**

To determine the graded cardiopulmonary effects and fate of pulmonary gas embolism, 11 sheep (with chronically implanted Doppler ultrasonic flow probes on pulmonary and brachiocephalic arteries) were subjected to experimental intravenous injection of nitrogen, oxygen and carbon dioxide. Three different rates of injection of each gas (0.03, 0.09 and 0.15 ml/kg/min) were used for 30 minutes.

Doppler ultrasonic flow probes were found to be very sensitive to the injected intravascular gas and produced characteristic “chirps.” The electromagnetic blood flowmeter was insensitive to the passage of these gases. Cardiopulmonary responses include elevation of pulmonary arterial pressure, diminished cardiac output and arterial hypoxemia. Nitrogen produced the greatest response and carbon dioxide the least, correlating with differences in solubility and diffusion rates. These responses developed with a one-half time of 15–20 minutes, with recovery, following cessation of injection, at the same rate. No statistically significant changes occurred in heart rate, systemic arterial pressure, pH or Pco₂. Passage of intravenous gas through the pulmonary vasculature to the systemic circulation occurred only at the 0.15 ml/kg/min dosage rate. The opening of the intrapulmonary arteriovenous shunts was primarily responsible for the decrease in arterial oxygen tension and passage of gas through the lung.

Gross pulmonary gas embolism occurs in many medical and surgical situations such as right ventriculotomy, craniotomy, angiography, retroperitoneal insufflation and other procedures involving venous catheterization and intravenous infusion. Recently¹⁻³ the use of Doppler ultrasonic technique in decompression sickness and open heart surgery has disclosed an unsuspected microaerembolism.

The principal cardiopulmonary disturbances shown by others in experimental pulmonary gas embolism include pulmonary arterial hypertension and systemic arterial desaturation. Systemic arterial hypotension and bradycardia have also been reported in acute massive gas embolization. In most gas embolism studies, air was injected as a large single bolus into the vein of an anesthetized animal⁴ Wycoff and Cann⁵ however, infused air continuously into the vein and showed that a rate of injection of 0.69 ml of air/kg/min produced heart failure, a decided rise of the systemic venous pressure, and death.

Key Words: Doppler Ultrasonic Flowmeter Electromagnetic Flowmeter Ventilation-Perfusion Relationship Arterial P0₂ Reduction Dissolution and Dissipation Intravascular Gas Pulmonary Hypertension Bubble Signals Intrapulmonary Arteriovenous Shunt Aeroembolism

(Received for publication, May 17, 1971)

* Clinical Cardiovascular Research Unit, Tonan Hospital, Sapporo
** Institute of Cardiovascular Physiology, Virginia Mason Research Center, Seattle, Washington, U.S.A.

Japanese Circulation Journal Vol. 35, December 1971 1541
For the prevention, detection and management of pulmonary gas embolism, it is important to understand the cardiopulmonary effects and fate of venous gas bubbles. The use of carbon dioxide in diagnostic procedures requires more information on dosage response and the potential practicality of intravenous oxygenation of blood requires quantitation of the maximal tolerable dosage. Also, the effects of graded dosages of nitrogen is important in the understanding of decompression sickness. In addition, it is important to know when and to what extent the gas bubbles are transported through the pulmonary vascular network.

The occurrence of systemic gas embolization following experimental intravenous gas injection or rapid decompression has been observed microscopically and macroscopically. However, the questions of whether or not and how the decompression bubbles can be transported across the pulmonary vasculature have not been conclusively resolved. If intravenous gas bubbles do travel through the pulmonary circulation, it has not been well documented when they will first appear in the systemic circulation.

The present investigation was made to evaluate the comparative cardiopulmonary responses and tolerance to continuous intravenous injection of nitrogen, oxygen and carbon dioxide at different dosage rates and to determine more precisely the conditions necessary for passage of gas bubbles through the pulmonary vascular network. Of additional importance was the definition of the types of signals that gross intravenous gas may produce in the Doppler ultrasonic and electromagnetic flowmeters.

**Materials and Methods**

Eleven healthy sheep between 29.5 and 35.9 kg were subjected to intravenous gas injection in a total of 32 experiments. One to 2 weeks prior to the first experiment on each animal, Doppler perivascular ultrasonic flow transducers were surgically implanted on the pulmonary and brachiocephalic arteries and catheters were also inserted through the femoral artery and vein to the aorta and vena cava.

On the day of the experiment, percutaneous right heart catheterization was performed through the right jugular vein, and the animals were subjected to the intravenous gas injection under a conscious condition, without any pharmacologic agent. One hundred per cent nitrogen, oxygen or carbon dioxide was injected into the vena cava at 3 different rates (0.03 ml/kg/min, 0.09 ml/kg/min and 0.15 ml/kg/min). These rates will be referred to as “0.03 rate,” “0.09 rate” and “0.15 rate” respectively. The indicated gas volumes were introduced with a syringe each minute for 30 minutes.

Before, during and for 30 minutes following embolization, the electrocardiogram, systemic arterial pressure, right ventricular pressure and Doppler flow signals from both the pulmonary and brachiocephalic arteries were monitored on a strip chart recorder and tape recorded simul-

\[ \Delta f = f_T \left( \frac{V_b - V_b \cos \theta}{V_b - V_b \cos \varphi} - 1 \right) \]

- **T**: TRANSMITTER CRYSTAL
- **R**: RECEIVER CRYSTAL
- **B**: BLOOD BUBBLE
- **θ**: ANGLE BETWEEN DIRECTION OF BUBBLE AND SOUND FROM T
- **ϕ**: ANGLE BETWEEN DIRECTION OF BUBBLE AND RETRANSMITTED SOUND FROM B
- **f_T**: TRANSMITTER FREQUENCY
- **V_b**: VELOCITY OF SOUND IN BLOOD
- **V_b**: VELOCITY OF BUBBLE (in the direction the crystals face)

Fig.1. Geometry and equation of the perivascular cuff type Doppler ultrasonic detector.

*Japanese Circulation Journal Vol. 35, December 1971*
Cardiopulmonary Effects of Intravenous Gas Embolism

Fig. 2. Left panel: The initial effect of intravascular nitrogen bubbles superimposed on the Doppler spectrum of pulmonary arterial blood flow. A dramatic increase in amplitude of each frequency band occurs 3 heart cycles after injection of gas into the inferior vena cava. Right panel: Typical bubble "chirping" signals detected with heartbeats 13 seconds after injection.

taneously. In 3 nitrogen experiments, changes in pulmonary blood flow was measured with the square wave electromagnetic flowmeter. Once every 5 minutes arterial blood samples were withdrawn and analyzed for \( P_{O_2} \), \( P_{CO_2} \) and pH. In terminal experiments, 5 animals were sacrificed by injecting large quantities of air.

The geometry of the perivascular cuff type Doppler ultrasonic detector used in this study and theoretical equation for bubble detection are illustrated in Fig. 1. The cuff contained 2 piezo-electric crystals, one for transmitting 10 mHz ultrasound and the other for receiving the Doppler shifted signal, mounted so as to face diagonally into the vascular lumen at an angle of about 60 degrees. The moving acoustical interface between gas and liquid produces a Doppler shifted frequency. The signal has considerable magnitude with relation to that produced by the normal cellular elements in blood and appears as audible "chirps" on the Doppler spectrum.

RESULTS

When the gas was ejected from the right ventricle into the pulmonary artery, Doppler signals of pulmonary blood flow were masked by high amplitude signals which sound to the ear like chirps, whistles and snaps (Fig. 2). Though the gases were injected in small boluses at one-minute intervals, their trapping and delay in the right heart produced continuous embolism with each heart beat.

During the 30 minute embolization period, there always occurred a progressive elevation of the right ventricular pressure and reduction of the arterial \( P_{O_2} \) (Fig. 3). Both the magnitude and initial rate of change of the response was dependent on the type of gas introduced as well as the
TABLE I  MAXIMAL CHANGES OF SRVP AND $P_{aO_2}$ DURING GAS INJECTION (EXPRESS AS PERCENTAGE OF CONTROL VALUE)

<table>
<thead>
<tr>
<th></th>
<th>SRVP</th>
<th>Arterial $P_{aO_2}$</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$N_2$</td>
<td>$O_2$</td>
<td>$CO_2$</td>
</tr>
<tr>
<td>0.03 ml/kg/min</td>
<td>130.5 (3)</td>
<td>121.7 (3)</td>
<td>100.0 (3)</td>
</tr>
<tr>
<td>0.09 ml/kg/min</td>
<td>171.5 (3)</td>
<td>155.7 (3)</td>
<td>100.0 (3)</td>
</tr>
<tr>
<td>0.15 ml/kg/min</td>
<td>196.4 (5)</td>
<td>165.6 (5)</td>
<td>113.1 (4)</td>
</tr>
</tbody>
</table>

Number of experiments in ( )

TABLE II  VALUES OF CARDIO-PULMONARY VARIABLES IN CARBON DIOXIDE EXPERIMENTS (FIVE EXPERIMENTS, WITH INJECTION RATE OF 0.15 ml/kg/min) ± FIGURES REPRESENT STANDARD DEVIATIONS

<table>
<thead>
<tr>
<th>Time</th>
<th>$P_{aO_2}$ (mmHg)</th>
<th>$P_{aco_2}$ (mmHg)</th>
<th>pH</th>
<th>SRVP (mmHg)</th>
<th>MSAP (mmHg)</th>
<th>HR (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>78.8 ± 2.6</td>
<td>36.2 ± 1.5</td>
<td>7.458 ± 0.026</td>
<td>25.3 ± 1.9</td>
<td>93.8 ± 4.5</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>74.5 ± 3.4</td>
<td>35.7 ± 2.1</td>
<td>7.456 ± 0.026</td>
<td>28.3 ± 2.6</td>
<td>93.1 ± 4.5</td>
<td>103.9 ± 6.3</td>
</tr>
<tr>
<td>30</td>
<td>69.9 ± 1.4</td>
<td>37.1 ± 1.3</td>
<td>7.456 ± 0.040</td>
<td>28.6 ± 2.3</td>
<td>92.5 ± 9.1</td>
<td>104.3 ± 6.9</td>
</tr>
<tr>
<td>End Injection</td>
<td>45</td>
<td>71.8 ± 2.7</td>
<td>37.8 ± 2.9</td>
<td>7.458 ± 0.027</td>
<td>25.9 ± 1.9</td>
<td>93.1 ± 6.2</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>75.9 ± 3.6</td>
<td>37.1 ± 2.0</td>
<td>7.454 ± 0.017</td>
<td>26.0 ± 1.0</td>
<td>95.0 ± 7.1</td>
</tr>
</tbody>
</table>

SRVP: Systolic right ventricular pressure.
MSAP: Mean systemic arterial pressure.
HR: Heart rate.

rate of injection. Table I shows the maximal changes of the systolic right ventricular pressure (SRVP) and arterial $P_{aO_2}$ ($P_{aO_2}$). Nitrogen caused the greatest response, while carbon dioxide had the least effect. As shown in Fig.3 and Tables II, III and IV, both hemodynamic and blood gas changes began with the first intravenous gas introduction, but the rate of change in SRVP and $P_{aO_2}$ during injection approached a plateau at the end of the 30 minute injection period. The maximal changes were not sustained beyond the cessation of gas injection, but were immediately reversed, returning toward the control with a one-half time of 5 to 10 minutes for the large nitrogen dosage. Arterial $P_{aO_2}$ did not change significantly in any of these experiments, but pH decreased slightly and the heart rate increased slightly (Tables II, III and IV). Systemic arterial pressure either remained at its control value or showed a slight decrease.

Fig.4 illustrates the pulmonary blood flow recorded simultaneously by both the ultrasonic Doppler zero crossing method and the electromagnetic method. Because of the large deformation of the ultrasonic flow curve during gas embolization, we were unable to compute the blood flow from the Doppler signal at that time. On the other hand, bubbles in the blood stream did not affect the flow contour obtained by the electromagnetic flow probe. This finding is contrary to our previous reports on decompression bubbles1,2. In order to gain information on quantitative cardiac output, we conducted 3 nitrogen experiments with the electromagnetic flow probe substituted for the Doppler cuff on the pulmonary artery. Nitrogen was injected at the 0.15 rate and we found the pulmonary blood flow to be progressively reduced during gas injection, reaching to 79.1 per cent of the control value. Pulmonary vascular resistance, computed from mean BA pressure and pulmonary flow, increased by approximately 248.3 per cent.

None of the 3 gases produced embolic signals on the brachiocephalic artery (BCA) if the injec-

*Japanese Circulation Journal  Vol. 35, December 1971*
TABLE III  VALUES OF CARDIO-PULMONARY VARIABLES IN OXYGEN EXPERIMENTS  
(FIVE EXPERIMENTS, WITH INJECTION RATE OF 0.15 ml/kg/min)

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>PaO₂ (mmHg)</th>
<th>PacO₂ (mmHg)</th>
<th>pH</th>
<th>SRVP (mmHg)</th>
<th>MSAP (mmHg)</th>
<th>HR (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>77.6 ± 3.6</td>
<td>36.7 ± 2.5</td>
<td>7.461 ± 0.031</td>
<td>22.3 ± 1.7</td>
<td>93.0 ± 13.7</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>64.1 ± 4.4</td>
<td>36.9 ± 2.7</td>
<td>7.454 ± 0.032</td>
<td>31.3 ± 2.1</td>
<td>91.5 ± 8.0</td>
<td>114.0 ± 7.1</td>
</tr>
<tr>
<td>30</td>
<td>57.8 ± 3.4</td>
<td>37.4 ± 2.9</td>
<td>7.456 ± 0.035</td>
<td>36.9 ± 3.8</td>
<td>94.0 ± 14.7</td>
<td>118.9 ± 10.7</td>
</tr>
<tr>
<td><strong>End Injection</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>65.3 ± 3.1</td>
<td>37.9 ± 2.1</td>
<td>7.464 ± 0.015</td>
<td>28.8 ± 5.6</td>
<td>86.5 ± 17.0</td>
<td>112.2 ± 6.2</td>
</tr>
<tr>
<td>60</td>
<td>70.9 ± 3.6</td>
<td>36.4 ± 2.3</td>
<td>7.465 ± 0.032</td>
<td>25.8 ± 3.0</td>
<td>92.5 ± 14.7</td>
<td>109.1 ± 8.9</td>
</tr>
</tbody>
</table>

TABLE IV  VALUES OF CARDIO-PULMONARY VARIABLES IN NITROGEN EXPERIMENTS  
(FIVE EXPERIMENTS, WITH INJECTION RATE OF 0.15 ml/kg/min)

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>PaO₂ (mmHg)</th>
<th>PacO₂ (mmHg)</th>
<th>pH</th>
<th>SRVP (mmHg)</th>
<th>MSAP (mmHg)</th>
<th>HR (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>81.0 ± 4.2</td>
<td>37.0 ± 1.8</td>
<td>7.460 ± 0.023</td>
<td>23.3 ± 1.1</td>
<td>82.0 ± 8.6</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>60.1 ± 1.4</td>
<td>37.7 ± 1.5</td>
<td>7.459 ± 0.027</td>
<td>39.4 ± 4.2</td>
<td>83.0 ± 9.3</td>
<td>126.4 ± 18.3</td>
</tr>
<tr>
<td>30</td>
<td>56.1 ± 5.4</td>
<td>38.2 ± 1.2</td>
<td>7.453 ± 0.011</td>
<td>45.8 ± 3.3</td>
<td>85.5 ± 10.8</td>
<td>141.8 ± 7.2</td>
</tr>
<tr>
<td><strong>End Injection</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>45</td>
<td>73.2 ± 6.5</td>
<td>37.6 ± 1.6</td>
<td>7.465 ± 0.061</td>
<td>28.4 ± 3.4</td>
<td>79.0 ± 12.8</td>
<td>113.6 ± 11.3</td>
</tr>
<tr>
<td>60</td>
<td>75.8 ± 5.1</td>
<td>36.9 ± 1.2</td>
<td>7.462 ± 0.014</td>
<td>26.8 ± 1.3</td>
<td>84.0 ± 8.6</td>
<td>112.8 ± 13.8</td>
</tr>
</tbody>
</table>

Fig.4. Simultaneous recording of electrocardiogram (top), pulmonary arterial blood flow obtained by Doppler ultrasonic zero crossing method (middle) and square-wave electromagnetic method (bottom). The Doppler flow signal is deformed by intravascular gas bubbles, while they have no effect on the electromagnetic flow signal.

Japanese Circulation Journal  Vol. 35, December 1971
tion rate was less than the 0.15 rate. When gas was injected at the 0.15 rate, 1 out of 5 oxygen experiments, and 3 out of 5 nitrogen experiments, produced distinct Doppler bubble signals on this major systemic artery, indicating the passage of intravenous gas bubbles through the pulmonary vasculature. In these 4 cases of arterial embolization, the minimal systolic right ventricular pressure at the time of the first gas bubble's appearance into the systemic circulation was 34.4 mmHg with the oxygen experiment. In the 3 nitrogen experiments, SRVPs at the time of first systemic embolization were 38.8, 42.2 and 45.0 mmHg. Fig.5 illustrates the passage of bubble signals from the pulmonary to the brachiocephalic artery. In the terminal experiments, in which large quantities of air were injected, signals of bubble showers were observed in the systemic artery.

Post-mortem examination of the heart and large vessels excluded the existence of obvious right-to-left shunts in all animals.

**DISCUSSION**

**The Doppler Ultrasonic Method for Bubble Detection**

Many devices for the detection of intravascular microbubbles have been used, including a glass air trap, the stethoscope and the microscope. The use of an ultrasonic Doppler flowmeter was first used in this laboratory in decompression experiments. We have also proven the method to be of great value in detecting intravascular bubbles during open heart surgery and showed that one can use many types of transcutaneous and surgically implantable Doppler flow transducers as well as catheter detectors. Further refinement of these detectors is needed and wider application in clinical monitoring is urgent. Also urgently needed are techniques of determining the numbers and sizes of bubbles as well as for computing the volume of gas passing through a given vascular channel.

Flowing intravascular gas bubbles produce characteristic signals on the Doppler spectrum superimposing the basic flow signal with such a large amplitude and frequency distribution that it was impossible to compute blood flow accurately. The Doppler shift frequency, due to the movement of intravascular bubbles, is a function of the velocity of bubbles in the blood stream but its amplitude can be affected by the degree of density discontinuity, the geometry of the bubble and the distance between bubble and sensor. In vitro generated gas bubbles passing through a cuff type detector at constant velocity has been shown by us to produce not a constant frequency but a frequency varying according to the bubble position relative to the transmitter and receiver crystals. The resultant signal sounds to the ear like a chirp. We have further found that clicks rather than chirps are produced when the sonic crystals are oriented directly facing the axis of the vessel. The greatest signal and purest Doppler shifted tone might be expected from a crystal orientation facing directly up or downstream, such as might be provided by an "end-looking" catheter tip detector.

Cardiopulmonary Effects of Intravenous Gas Embolism

Arterial Oxygen Tension Reduction

Explanations in the literature of the mechanism of arterial desaturation during pulmonary thromboembolism include 1) anatomical pulmonary changes such as edema, atelectasis and subsequent diffusion impairment, 2) changes in the ventilation-perfusion ratio including increased velocity through the non-embolized lung capillaries, and 3) opening intrapulmonary arteriovenous shunts.\(^{10-12}\)

Pulmonary anatomical changes may develop to some extent during gas embolization and may also participate in the establishment of arterial hypoxemia. In our experiments, however, the rapidity of recovery following cessation of gas injection argues against this as the predominant mechanism.

Matching between intrapulmonary circulation and ventilation must be deteriorated when partial or disseminated perfusion blockage occurs in the lung by gas embolization.\(^{13}\) It is likely that non-perfused alveoli are still ventilated and many ventilated alveoli are over-perfused. Though some compensation may occur, like an intrapulmonary ventilation shift to correct this unevenness of ventilation perfusion relationship,\(^{14}\) relative over-perfusion of the lung area with normal or hypoventilation may participate in the causation of arterial oxygen tension reduction. Levy et al.\(^{16}\) ruled out regional hypoventilation by normal nitrogen washout. They also observed non-beneficial effects of oxygen breathing on hypoxemia secondary to autologous pulmonary thromboembolism and excluded the role of ventilation-perfusion abnormality as Steinitz et al.\(^{15}\) did. They concluded right-to-left shunt as the mechanism. Similar results were obtained from denitrogenation experiments.\(^*\) Arterial PO\(_2\) was reduced 103 mmHg from its control level by a calculated 5 per cent right-to-left shunt increase.

Hyman recently presented valid evidence for opening of potential intrapulmonary arteriovenous shunts by using non-occlusive distension on the main pulmonary arterial trunks.\(^{16}\) The opening of anatomical shunts, whose size is considered to be at least 420 micra in diameter,\(^{11}\) seems likely when PA pressure increases. These shunts might not only cause the arterial PO\(_2\) reduction but also provide the pathway for the passage of gas bubbles through the pulmonary vasculature in our present study.

From these considerations, increase of venous admixture, probably due to opening of intrapulmonary arteriovenous shunts, should be concluded as the most important factor for the arterial hypoxemia by pulmonary gas embolism.

Pulmonary Hypertension

In pulmonary arterial embolism, it is believed that the pressure rise is caused by mechanical blockage of vasculature, while active vasocostriction may superimpose on this mechanism in miliary (arteriolar or capillary) embolism.\(^{11,17}\)

In our experience, mechanical blockage of the pulmonary vascular bed by gas embolism is no doubt the primary cause of pulmonary hypertension. Size-changeable abilities of intravascular gas bubbles, which are quite different from the emboli like blood clots, plant seeds or solid particles, should however be considered in the evaluation of the pressure responses caused by pulmonary gas embolism. When gas bubbles reduce their size to be small enough to produce miliary embolism, active pulmonary vasoconstriction might be expected. Additional participation of humorally mediated vasoconstrictive substance like serotonin\(^{18,19}\) pulmo-pulmonary reflex\(^{16}\) and concomitant arterial oxygen tension and pH decrease\(^{20}\) can also be speculated.

Fate of Intravenous Gas

Gases administered intravenously may be absorbed into the blood or may be dissipated directly in the gas state through the alveolar membrane into the expired air. Nitrogen, as expected from its lower solubility in blood and tissues, is dissipated more slowly than oxygen and carbon dioxide. The stability of bubbles impacted into the pulmonary vascular bed is undoubtedly affected by hemodynamic forces breaking them and forcing them into the smaller vascular channels. Fractionation will in turn increase the gas-tissue-interface, promoting dissipation into the blood and alveoli. Mandelbaum and King's failure to find nitrogen in the expired gas when air was injected into the animal breathing oxygen is probably inconclusive because of low sensitivity of the nitrogen meter.\(^2\) Whatever the eventual fate, it is apparent from the “on”

\(^*\) As a part of intravenous oxygenation experiments, 3 sheep were denitrogenated by respiration within a box containing 100 per cent oxygen for 6 hours prior to the introduction of oxygen embolization at the 0.15 rate. This disclosed that systolic pulmonary arterial pressure rose to 148.5 per cent of control level while arterial oxygen tension diminished from 559 mmHg to 455 mmHg. The calculated shunt ratio increase for these experiments was approximately 5 per cent.
and “off” time response of our experiments that dissipation is rapid and continuous during the injection so that for each given dosage rate a steady plateau of cardiopulmonary responses can be reached where the dissipation rate equals the dosage rate. Measurement of these plateau values may give a good indication of the embolization rate in aeroembolism conditions and a quantitation of the volume of emboli in solid or thrombotic embolization.

**Passage of Gas Bubbles through the Pulmonary Circulation**

Aeroembolism experiments of decompression sickness are typically represented by the work of Wagner who observed the appearance of gas bubbles in the pial vessels of cats following acute decompression. We feel, as did Lever et al., it is invalid to assume microscopic observation of only the peripheral circulation that peripheral bubbles pass through the lung. In many of these hyperbaric exposures and decompression, nucleation in the arterial blood may be expected. It seems to us equally invalid to conclude from experiments in which 0.5 ml of air was injected that gas cannot pass the pulmonary circulation.

Mandelbaum and King injected intravenously 0.5 ml/kg of air and at autopsy found gas bubbles in the pulmonary vein, clearly indicating their passage through the lung. Our experiments also confirm that gas emboli can, at dosage rates above 0.09 ml/kg/min transverse the pulmonary vasculature and indicate that this can occur at a systolic pulmonary arterial pressure as low as 34 mmHg.

When opened, intrapulmonary arteriovenous shunts are believed to have a considerably larger diameter than that of the normal capillary vessels. Even if the gas bubbles were small enough to pass through the pulmonary capillary bed, complete dissipation of intracapillary gas bubbles into the alveoli can be expected. We accept the evidence for the presence of intrapulmonary arteriovenous shunts in dogs and consider this to be the primary pathway of gas through the pulmonary vasculature in our sheep.

The minimal amounts of intravenous gas which results in systemic bubbles was between 100 and 150 ml in our sheep experiments. These quantities are not as great as others reported as the lethal dose. The absence of external signs in our animals at the time of the occurrence of gas embolism in the brachiocephalic artery correlates well with our finding during open heart surgery of considerable tolerance by the brain to aeroembolism. The goal in all clinical situations should be to prevent any systemic gas embolism and to seriously consider hyperbaric compression treatment in a suitable chamber if cerebral or coronary embolism occurs or is suspected.

**REFERENCES**


