Effect of Near-Vacuum Exposures on Pulmonary Circulation in Dogs[1] (34593)

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Investigations concerning near-vacuum exposures, in support of current high altitude and space flights, are essential to help evaluate the risk and recovery potentials after encounters to reduced pressures. During and immediately after World War II, studies to pressures equivalent to about 72,000 ft (30 Torr) ascertained that general circulation ceased within less than 16 sec (1–4). More recently, it has been reported that vascular (5–8) and biochemical (9) changes measured at pressures approaching a vacuum are generally of greater magnitude than those measured at 30 Torr, and decompressions to 2 Torr within 1 sec result in circulatory arrest within a few seconds, as evaluated by blood-pressure gradients (5, 7). The present study is designed to investigate the direction and distribution of pulmonary blood flow during such an exposure, where ordinary techniques and methods of measurement are either difficult or unreliable owing to artifacts associated with pressures that are less than that of water vapor.

Method. Eight adult mongrel dogs, weighing about 20 kg each, were anesthetized with pentobarbital sodium. A catheter, placed in the jugular vein, penetrated the wall of the altitude chamber and connected with a three-way stopcock outside the chamber. The catheter was filled with bubble-free saline at room temperature. The location of the catheter tip in the blood vessel was estimated by monitoring the pulmonary arterial pressure with a previously calibrated P-23 series Statham Pressure Transducer and a Honeywell Visicorder (Model 1108). The catheter tip location was later verified by careful dissection.

Each animal breathed 100% oxygen through a tracheal cannula and a demand oxygen regulator, anad was slowly decompressed to 180 Torr. After 5 min, 0.1 cc of degassed corn oil was injected into the vascular cannula to serve as a visible marker for movement of fluid. One microcurie of ^{125}I, contained within 0.1 cc of 5% aqueous albumin solution, was introduced part way into the catheter, followed by 0.5 cc of degassed saline. The chamber was then rapidly decompressed within 1 sec to 2 Torr. After 20 sec, at which time previous work has shown that the venous and arterial pressures are about equal (7), the ^{125}I was injected into the blood vessel with additional saline. Each animal remained at 2 Torr for 10 min, at which time death had already occurred, and was then slowly recompressed within 5 min to ground level pressure. The body was refrigerated over night to prevent tissue decomposition while the blood coagulated, and the major blood vessels were ligated to prevent movement of blood during sampling. The samples were weighed, and scintillation counts were made with a Tracerlab Versa-Matic II Spectrometer. A 0.025-μCi water standard served as a reference.

Results. Whenever the catheter was passed into the pulmonary artery and carried forward by movement of blood, it generally entered the diaphragmatic lobe of the left lung as shown in Table I. A high level of the isotope was always detected in the immediate vicinity of the injection site. In four ani-

[1] The research reported in this paper was conducted by personnel of the Physiology Divison, USAF School of Aerospace Medicine, Aerospace Medical Divison, AFSC, United States Air Force, Brooks AFB, Texas. Further reproduction is authorized to satisfy the needs of the U. S. Government.

TABLE I. Detection of Albumin ^{125}I in Animals Exposed to a Near-Vacuum Pressure of 2 Torr.

Dog no.	Injection site	Location of highest isotope level	Counts/minute/gram of tissue				
			Left auricle	Left ventricle	Right auricle	Right ventricle	Pleural space
1	Pulmonary artery; diaphragmatic lobe, left lung	Diaphragmatic lobe, left lung (5600 cpm/g)	15	0	10	15	0
2	Pulmonary artery; diaphragmatic lobe, left lung	Diaphragmatic lobe, left lung; hemorrhagic tissue; pleural space (180–795 cpm/g)	17	15	8	40	795
3	Pulmonary artery; diaphragmatic lobe, left lung	Diaphragmatic lobe, left lung; hemorrhagic tissue (229–689 cpm/g)	13	73	0	10	40
4	Left pulmonary artery	Left lung; all lobes (128–350 cpm/g)	15	16	3	11	62
5	Left pulmonary artery	Left lung; hemorrhagic tissue (850–1160 cpm/g)	297	198	24	50	0
6	Common pulmonary artery	Evenly distributed; both lungs; heart (230–845 cpm/g)	403	845	27	34	33
7	Common pulmonary artery	Pleural space of both lungs; blood from non-hemorrhagic tissue (388–1376 cpm/g)	592	261	66	23	210
8	Superior vena cava; 1 inch from heart	Both lungs; pulmonary veins; pleural space (1449–4213 cpm/g)	146	225	218	255	530

mals, the isotope was identified in a nonhemorrhagic, colorless fluid in the pleural space. The isotope was readily detected in hemorrhagic regions of the lungs, especially in the proximity of the injection site. In all of the animals in which the isotope had been injected into a pulmonary artery, only an insignificantly low level of the isotope was detected in either the right ventricle or auricle (see Table I). In three of the seven animals, a low level of ^{125}I was detected in the left auricle and ventricle. No significant amount of the isotope was located in any of the coronary arteries or in the aorta.

Discussion. These results show that at the time of the injection of the isotope, 20 sec after the rapid decompression, blood flow had essentially ceased within the pulmonary circulatory system, with only a limited amount of blood movement occurring during the remainder of the near-vacuum exposure. A small amount of blood movement after this time appears to be associated with pressure differentials other than those caused directly by living processes. The absence or low level of ^{125}I in blood from the right heart in seven of eight animals shows that blood flow from the lungs back to the right heart is limited or did not occur either during the exposure or the recompression. The rapid rise in pressure on the venous side of the heart, the immediate outgassing of dissolved gases and water vapor from the pulmonary capillary bed, and action of the semilunar valve, were apparently effective factors in preventing blood from being forced back into the right ventricle.

Varying amounts (from 10 to 100 %) of the lung surface were found to be hemorrhagic, with high levels of ^{125}I apparently moving

into the damaged tissue during the latter part of the exposure or during the recompression. The effect of the recompression process itself upon tissue damage is not known, but the slow rate used in this study would not likely be an important consideration.

It can be concluded that a small, perhaps insignficiant, amount of pulmonary flow occurs after about 20 sec of near-vacuum exposure or during the recompression in the animals tested. This movement of blood appears to be independent of a functional heart. Also, both physical processes at this low pressure and anatomical factors assist in preventing either a normal or even a reversed blood flow.

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Received Oct. 9, 1969. P.S.E.B.M., 1970, Vol. 133.