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# The Effect of Pitressin in the Treatment of Massive Air Embolism Following Cardiopulmonary Bypass: Case Report

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Although aortic air embolism is relatively uncommon in extra-corporeal circulation practice, it is so catastrophic when it occurs that further investigation into its etiology, pathophysiology, prophylaxis, and treatment is necessary. Moreover, as it was emphasized by other authors, its frequency may be much greater than has been recognized, since very few of these accidents are likely to be reported. In instances in which an autopsy is not done, death may be attributed erroneously to heart disease or some other condition.<sup>2</sup>

The site of entrance of air determines its severity and distribution within the vascular system, and it is therefore extremely important. In arterial embolism, the brain is more affected than the coronary arteries because the air gains entrance through the carotid arteries to the brain. A small amount of air can effectively block medium sized vessels and the consequent results can be seen in various stages when only few milliliters of air enters the cerebrovascular network. In the arterial form, the clinical picture is dependent upon ischemia of vital organs, especially the brain. If the head is higher than the level of the aortic arch, air will enter the cerebral circulation and cause various neurological manifestations such as aphasia, blindness, hemiplegia or monoplegia. The fundus oculi examination may show air bubbles in the vessels which is of significant diagnostic value in such cases.

## Case Report

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A twenty year old girl was admitted to the National Cardiovascular Centre for repair of an atrial septal defect (ASD). She was admitted ambulatory and showed no visual evidence of clinical illness.

The catheterization report revealed a small atrial septal defect (ASD) which did not require a patch

closure. She underwent intracardiac surgery for closure of ASD under cardiopulmonary bypass. During bypass there was an accident in which massive amounts of air were perfused. Approximately 150 milliliters of air was aspirated from the left radial artery. The perfusion accident occurred at the start of bypass when there was little priming fluid in the oxygenator reservoir. It was due to a delayed release of the venous clamp and 200 milliliters of air was estimated to have been infused. The arterial pump was stopped and the air was removed from the arterial line. Since circulatory arrest had not started, there was enough time to remove air from the line. The oxygenator used was Harvey Model No. H-1000\*. The operation went on and the ASD was closed. The operation took two hours, and the cardiopulmonary bypass took thirty minutes.

At the end of the operation, she never woke up from anaesthesia, nor did she respond to any stimuli. The left pupil was widely dilated, but all other vital signs were within normal limits. She showed signs of brain damage affecting mostly her left side. She was taken to the Intensive Care Unit where all postoperative management was carried out. After a few hours, she could move both limbs in spite of extensive brain edema, but the left side was rather weak. She remained semi-conscious for several days.

On the third day, daily injections of pitressin (vasopressin) were started in doses of 1/1000 units. After five days of injections, the brain edema subsided, the left pupil returned to normal, and both pupils reacted to light equally. One morning she was able to write her name and to respond to all questions, although, she could not talk properly. Three weeks later she was able

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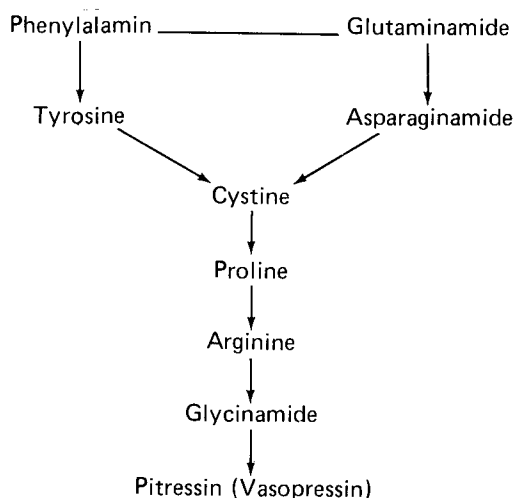


FIGURE 1. Diagram of structure of Pitressin (Vasopressin)

to talk and she was ambulatory. She was removed from the Intensive Care Unit (ICU) and sent to a general ward where she remained for a month. She was discharged from the hospital and continued to attend the out-patient cardiac clinic. After three months, she went back to school, and she was able to follow her lessons without any neurological impairment.

## Discussion

Pitressin is produced by nerve cells in the supraoptic and paraventricular nuclei of the hypothalamus. It appears then to be transported along the nerve fibers to the pituicytes of the posterior lobe of the pituitary. In the pituitary gland, they are bound to a specific group of proteins called neurophysin which contains three polypeptide binding sites per molecule. It can be discharged from this area by electrical stimulation or acetylcholine.

The control of pitressin release is mediated in part through osmoreceptors and increase in plasma osmolality resulting from water withdrawal or hydration leading to increased rates of hormone production. Pitressin (vasopressin) acts by increasing intracellular cyclic adenosin-3', 5'-monophosphate (cyclic AMP) in the cells of the collecting ducts. The cyclic AMP then acts in some undefined way to increase the permeability

of the renal epithelium thus increasing reabsorption of water.

There is evidence that many other hormones also exert their effect by increasing cyclic AMP in their target organs.<sup>8-10</sup>

This substance raises blood pressure by its vasoconstrictor effect on the peripheral blood vessels. Pitressin has been used in surgical shock as an adjuvant in elevating blood pressure.<sup>7</sup>

It may also be used occasionally in obstetrics in the case of delayed postpartum hemorrhage and a delivery for uterine inertia. Pitressin also exerts an antidiuretic effect as the so-called posterior pituitary antidiuretic hormone (ADH). The hormone affects the renal tubules and provides for the facultative reabsorption of water. It is of great interest that the above described treatment has not been used in the treatment of brain edema following massive air embolism.

The trial to use this drug proved quite effective. Since pharmacological action of this hormone extract seems to be irrelevant in the treatment of brain edema, it was very interesting for its action on this case. Pitressin is available in 20 units/ml, 0.5 and 1 ml ampules for subcutaneous or intramuscular injection.

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