Cerebral Air Embolism Complicating Therapeutic Pneumoperitoneum

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The following case study is presented to illustrate the concept that recovery from the invariably disastrous effects of arterial air embolism, accidentally incurred during the therapeutic administration of pneumoperitoneum, may be possible if certain recognized and specific maneuvers are practiced correctly. This particular subject is of extreme importance because since the institution of pneumoperitoneum in the treatment of tuberculosis, 19 fatal accidents have occurred due to embolic complications.12,13 Three occurred in the Veterans Administration Hospital, Oteen, North Carolina, since 1942, during approximately 150,000 refills given to about 2,400 patients.

The following case represents an embolic accident which fortunately was followed by recovery.

L. J., age 32, known to be suffering from far advanced pulmonary tuberculosis since August 1950, was admitted on October 5, 1950 and two days later was started on chemotherapy consisting of 0.5 gram streptomycin intramuscularly once daily concurrently with four grams of para-aminosalicylic acid, by mouth, three times daily. On November 11, 1950, pneumoperitoneum was induced because, following subsidence of the initial toxic symptoms, it was felt that pulmonary collapse would also be necessary in treatment of the bilaterally extensive exudative and cavitary disease. After one year's continuous combined drug and collapse therapy, an excellent result, with bilateral clearing and cavitary closure, was obtained. Pneumoperitoneum was induced by a standardized procedure using the Zavod apparatus connected by sterile rubber tubing with a glass syringe and number 19 gauge short bevel needle measuring five inches in length. After sterile preparation and infiltration of an area to the left of the umbilicus with one per cent novocaine, 450 cubic centimeters of air were slowly introduced. The intra-abdominal pressure at the end of the injection was equivalent to a positive pressure of two centimeters of water, as measured on the manometer. Refills were repeated at weekly intervals, the amount being gradually increased to 800 cubic centimeters. Novocaine was not used after an adequate air space was obtained. The usual initial positive pressures fluctuated between five and six centimeters of water, the final positive pressures between six and eight centimeters. Fluoroscopy was utilized before each refill to determine the degree of diaphragmatic elevation and to make certain that no complication had developed. These observations demonstrated a fairly stable sub-diaphragmatic air chamber. Until September 20, 1951, at which time the embolic accident occurred, the patient had received 44 refills without incident.

On the day of the accident, the needle was inserted in the usual manner and, after having made certain that a free oscillation of the manometer existed, a reading was taken and the notation made that it approximated a positive pressure of eight centimeters of water. The needle was then quickly withdrawn, after 700 cubic centimeters of air were injected. At the end of the procedure however, a final positive pressure of 18 centimeters of water was noted. He rose slowly from the table, walked a short distance and then suddenly collapsed. An apneic phase with severe cyanosis intervened and he became rigid, following generalized tonic

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muscular contractures. The nearest person to the victim turned him on his right side, but this was immediately corrected and he was placed in the left lateral recumbent position. It will be shown later that this position was of virtually no value because stethoscopic auscultation of the heart in this particular instance did not disclose the typical churning mill-wheel murmur diagnostic of the cardio-venous form of air embolism. In quick succession, oxygen by intra-nasal catheter and an injection of 1/150 grain of atropine sulfate were administered. The latter agent was used to relieve respiratory embarrassment because by paralyzing the parasympathetic nerves, it would abolish bronchospasm and therefore dilate the pulmonary bronchi. After several minutes, respiratory movements gradually resumed. As consciousness slowly returned, it was noted that the left arm and leg were completely motionless. He was immediately returned to the operating table and deflation of the pneumoperitoneum was begun. After withdrawal of 1,050 cubic centimeters of air and return of the intraperitoneal pressure to a positive reading of eight centimeters of water, he was able to respond to direct questioning, which now disclosed a disturbance of vision described as a blurring sensation. He had normal blood pressure of 100/70, and frequent subsequent check-ups did not reveal any evidence of the existence of hypotension. Intravenous plasma was later administered on a purely empirical basis. It was given with the hope that it might alleviate the suspected anoxic injury to the brain. The left hemiplegia partially resolved in two hours and the extremities completely returned to normal in four hours. Sedation was used to relieve the severe headache which subsequently developed. This and the blurring vision vanished in 48 hours and to date no permanent disabling residuals have been observed.

Prior to Durant, Long and Oppenheimer's experimental studies on air embolism, the management of such accidents in humans consisted principally of resuscitative measures involving the use of artificial respiration, oxygen, stimulants and the Trendelenburg position. In other words, the treatment in most cases did not differ materially from that customarily employed in ordinary shock. The application of such measures were sometimes but not always successful in the hands of Banyal, Fremmel, Trimble and Wardrip, Warring and Thomas and Rilance and Warring. In 1947, Durant et al published an illuminating treatise on the subject of air embolism in which they distinguished two forms. They described the symptoms and pathogenesis and proposed a method of treatment for each entity. Briefly, they described the pulmonary or venous and the cerebral or arterial form. The site of entry of the air bubble in the former is a systemic vein; in the latter, a pulmonary venous channel. The course of the embolus in the former instance is through the right heart into the pulmonary circulation where it is excreted, of the latter through the left ventricle into a systemic artery, e.g. vertebral or coronary. A large volume of air is necessary to prove fatal in the venous form; a small dose may prove fatal in the arterial form. The cause of death in the former is the formation of an air trap in the right ventricular outflow tract with subsequent cardiac arrest; in the latter the cause is attributed to a coronary occlusion or cerebral damage. The clinical signs are a precordial mill-wheel murmur in the former and, in the latter, the well known signs of a coronary occlusion or of a cerebral accident. In the treatment of the venous form, because it relieves the obstruction by displacing the air trap in the right ventricle, the most favorable position is the left lateral; for the
latter, with one reservation, it is the head down position. The exception to the rule is that the head down position will prevent embolism only if this position is present when the accident occurs; it will not clear the cerebral signs or affect the coronary obstruction if the position is assumed after the accident occurs.

Hollander\(^{11}\) appears to have been one of the first to successfully apply the recommendations suggested by Durant et al. As he reported, the left lateral position produced immediate recovery in one patient who developed a venous air embolism following a pneumoperitoneum refill. In another, developing a cerebral air embolism under similar circumstances, the head down position assumed before the refill was started, aided recovery. He was less fortunate in the management of a third patient because of unavoidable delay in the application of the correct position. The accident occurred after the patient rose from the table and death followed in a matter of minutes. At autopsy, a perforated vein in the abdominal muscle tissues and frothy blood in the right auricle, ventricle and pulmonary artery were found. The author affirmed that the left lateral position should be maintained until obstruction of the pulmonary conus due to the air embolus is completely relieved even if this requires several hours, and that stimulants and oxygen are of little value unless this precaution is observed.

Some workers\(^{4}\) believe that air embolism complicating a pneumoperitoneum refill almost always occurs following direct entry of air into a blood vessel. Granted that this is so, it is difficult to conceive how the accidental injection of air into a vein of the abdominal wall could reach the brain without passing through the right heart unless one postulates the existence of anomalous vascular channels. It is known that the inferior epigastric vessels coursing through the abdominal wall communicate at their source with the external iliacs and anastomose above the umbilicus with the superior epigastric branches of the internal mammary which first empty into the subclavian and eventually into the right heart. So, unless there is evidence to support the presence of an interauricular or ventricular septal defect, air cannot reach a cerebral artery if injected into a peripheral venous channel. No clinical evidence of such a defect was uncovered in the case under discussion.

To effectively combat the complications resulting from this type of air embolism, it is necessary not only to be reasonably prompt in the application of the correct maneuver after the accident occurs but also to have a thorough knowledge of the precautions that should be observed in administering therapeutic pneumoperitoneum. Particularly extreme caution should be exercised at the moment of induction because it is at this time that accidents are most prone to occur. As the needle punctures the peritoneum, one should be certain that it reposes in a free space. Any signs of resistance to the flow of air such as is usually indicated by the absence of manometric oscillations, should warn the operator that the needle may be in muscular or connective tissues and that it should be immediately adjusted. If blood appears in the syringe, showing obviously
that some vessel has been punctured, the needle should be immediately withdrawn before air is injected and another site selected. It is good policy, moreover, that, if a certain area of skin has been found to permit facility of introduction of the needle without risk of injuring a blood vessel, the same site be used again if refills are continued. Fluoroscopy should serve primarily as a guide for the determination of the presence or absence of diaphragmatic elevation and should not influence the operator regarding the quantity of air to be injected. The quantity should depend rather on the intraperitoneal pressure and, if it is found that it is unusually high without correspondingly adequate elevation of the diaphragm, serious consideration should be given to abandoning the pneumoperitoneum. A prophylactic measure that enjoys the support of some operators is the use of the Trendelenburg position in all cases before the refill is given. The reason advanced for this is that the resultant flow of the subdiaphragmatic air mass away from the brain is believed to hinder invasion of a damaged blood vessel. The para-umbilical injection route is preferred to the infra or intra-costal approach because of the likelihood of puncturing the lung or liver with the latter technique. Such trauma to these organs inevitably produces an embolus if air is introduced with the puncture. Particular attention should be paid to the patient's complaints of dizziness and nausea which, despite the inclination to minimize them, are oftentimes reasonably good indicators of impending disaster. It is advisable also to regulate the flow of air into the peritoneal cavity by injecting, for example, small quantities of 50 cubic centimeters with frequent pressure readings. These precautions, if carefully observed, should eliminate those complications secondary to improper technique but unfortunately will not prevent the rare complication which is due to no fault of the operator. If the accident occurs on the operating table, a meticulous examination will almost consistently give some indication as to the type of procedure that might be of assistance. In the presence of a typical precordial mill-wheel murmur, the left lateral position is the only procedure that will succeed, keeping in mind, however, that it should not be discontinued until the air obstruction is completely relieved and until there is some certainty that the traumatized blood vessel has had a chance to heal. On the other hand, no reliable method of treatment has as yet been devised to manage arterial embolic accidents after the patient has apparently safely completed his pneumoperitoneum refill. Although no claim can be made at present concerning the effectiveness of the decompression technique in these cases, it is nevertheless believed to be worth a trial for the following reason. Where the head down position merely shifts and immobilizes the intraperitoneal air mass, abdominal deflation, on the other hand, helps to remove this potential hazard, thereby offering greater assurance that further embolization will not occur.

**SUMMARY**

One case with recovery from cerebral air embolism complicating therapeutic pneumoperitoneum is reported. Similar accidents reported in the
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literature are briefly discussed and precautions to be observed in administering pneumoperitoneum are reviewed. An additional safeguard represented by abdominal decompression is suggested for managing this complication.

RESUMEN

Se refiere un caso de embolia cerebral gaseosa complicando neumoperitoneo terapéutico, con recuperación. Se han relatado accidentes similares en la literatura los que se discuten así como las precauciones que deben observarse al administrar el neumoperitoneo. Se sugiere una medida adicional consistente en la descompresión abdominal para tratar esta complicación.

RESUME

L’auteur rapporte un cas d’embolles gazeuse cérébrale, compliquant un pneumopéritoine thérapeutique. Il discute rapidement les accidents de cet ordre jusqu’ici rapportés dans la littérature, et énumère les précautions que l’on doit observer lorsqu’on pratique un pneumopéritoine. Il conseille la décompression abdominale en tant que procédé de sécurité supplémentaire.

REFERENCES