POSTPARTUM CEREBRAL EMBOLISM IN A NORMAL PATIENT:
A CASE REPORT

Cerebral embolism in a normal postpartum patient is so rare that it deserves to be recorded. This case is of particular interest to anesthesiologists because the delivery was accomplished under spinal analgesia. Any neurologic complication that follows spinal anesthesia, especially in a normal parturient, tends to be attributed to the anesthesia unless proved otherwise. Anesthesiologists and obstetricians should, therefore, be reminded of two uncommon mechanisms which can cause cerebral embolism after the delivery of a normal patient.

A 23-year-old primigravida, at term, was admitted on February 17, 1949. Her history, prenatal course, physical and laboratory data were normal. After eighteen hours of labor the cervix was fully dilated, the occiput presented posteriorly and the vertex was level with the ischial spines. She was given 50 mg. of ephedrine sulfate subeutaneously and spinal analgesia was administered through a 26 gauge lumbar puncture needle in the fourth lumbar interspace. The anesthetic solution was 1 cc. of nupereaine (1 to 200), 1 cc. of glucose 10 per cent, and 1 cc. of ephedrine sulfate (50 mg.). Analgesia appeared promptly. There was no drop in blood pressure and no untoward symptoms during the remainder of labor and delivery. Ninety minutes later, with the fetal head below the spines, she was delivered of a normal male infant by Kielland forceps rotation and extraction. Pitocin and ergotrate (1 cc. of each) were given hypodermically and the placenta was expressed. The loss of blood was estimated at 250 cc. The patient left the delivery room in good condition.

The first and second postpartum days were uneventful. On the third day, while sitting on a bedpan following an enema, she cried out and was found flat in bed, lightly cyanotic, and unable to move her right upper and lower extremities. She could not express what she meant to say and speech was thick. The anesthetist examined the patient two hours after the onset of hemiplegia and found her in no apparent distress, except when she tried to talk and could not express herself adequately. Temperature, pulse and respiration were normal; the blood pressure was 110 mm. systolic and 68 mm. diastolic. There were no abnormal signs in the heart and lungs, no tenderness or edema of legs and no Homans’ sign. The right lower extremity had full range of motion, the right upper extremity was weak; she could grasp objects but could not easily relax her grip. The right side of the face was flattened and was pulled to the left when she tried to pucker her lips. She could wrinkle her brow and close the right eye firmly; the tongue protruded in the midline; the pupils were equal, central and reacted to light and accommodation; ocular movements were normal; Kernig, Brudzinski, Babinski, and Hoffman tests were negative; ankle, knee, biceps and abdominal reflexes were equal and active. Our diagnosis was cerebral embolus to the left middle cerebral artery, source undetermined. Four hours later a neurologist confirmed our findings. In addition, he noted astereognosis on both sides; the patient was unable to feel objects with the right hand and was unable to release her right hand grasp quickly. The nasal halves of both optic disks were blurred. Vibratory sense was absent in the right upper half of the body. His diagnosis was diffuse embolization involving the left half of the cerebral cortex with edema of the brain. The emboli, he believed, arose from a latent bacterial endocarditis.

The next morning, a lumbar puncture was performed and clear spinal fluid obtained under 200 mm. of pressure, containing 4 polymorphonuclear leukocytes and 4 lymphocytes per cubic millimeter. Urine was negative; hemoglobin 64 per cent, erythrocytes numbered 3,200,000 and leukocytes 14,200 with normal differential, bleeding and clotting times were normal; a blood culture remained negative for four days.

The postpartum course continued afebrile and improvement was noted daily. The last residua were numbness of the right upper extremity and some difficulty in speech. The patient was allowed out of
bed on the thirteenth and discharged on the sixteenth postpartum day. Six weeks later recovery was complete, except for numbness of the right index finger.

**DISCUSSION**

The sudden onset of cerebral signs forty-eight hours after a spinal anesthesia makes it very unlikely that the anesthesia was the cause. There was no period of arterial hypotension to favor thrombosis, as in Watter’s case (1). There was no episode of arterial hypertension due to vasopressor drugs to produce cerebral hemorrhage as in the case reported by Greene and Barcham (2). There was no postspinal hypotension of cerebrospinal fluid to predispose to subdural hematoma, subarachnoid hemorrhage or multiple hemorrhagic foci in the cerebrum (3). The use of a 26 gauge spinal needle prevented hypotension of cerebrospinal fluid and the diagnostic tap confirmed its absence.

The common sources of emboli to the brain are as follows: (1) bacterial endocarditis of the aortic or mitral valves; (2) myocardial infarction with thrombosis of the mural endocardium in the left auricle or ventricle; (3) auricular fibrillation or mitral stenosis with thrombosis in the left auricle; (4) atheroendarteritic ulcer with thrombosis in the aorta or carotid arteries; (5) pulmonary venous thrombosis in pneumonia, tuberculosiis, abscess or neoplasm, and (6) air entering the pulmonary venous circulation, for example, during pulmonary operations or artificial pneumothorax. All of these possibilities were eliminated by the absence of any findings which might support even the suspicion of these causes. The neurologist strongly favored the possibility of a latent subacute bacterial endocarditis. This was never confirmed by blood culture or physical finding and was finally dismissed by the continued recovery and good health of the patient.

Only after eliminating the most frequent causes of cerebral embolism may we consider the last two possibilities, paradoxical embolism and transuterine air embolism. For a case as rare as ours, however, a rare cause must be considered.

Paradoxical embolism was defined by Cohnheim in 1877 when he found an embolus in a middle cerebral artery which had originated in thrombosed leg veins and had by-passed the lungs, floating from the right auricle through a widely patent foramen ovale into the left auricle. This mechanism should be thought of in every case of unexpected cerebral symptoms or signs, especially in postpartum or postoperative patients who are prone to form venous thrombi. Anatomical patency of the foramen ovale has been found in 35 per cent of 1100 consecutive autopsies (4); in 29 per cent the opening admitted a probe but in 6 per cent it was wide enough to admit a pencil. Functional patency of a foramen ovale is usually associated with congenital heart disease, which may not be clinically evident. A functionally patent foramen may occur also in a normal heart without physical signs. More of the foramina becomes functionally patent when the right auricular pressure rises, as in pulmonary embolism, or left auricular pressure falls, as in shock. Then the membranous flap on the left side of the interauricular septum no longer closes the foramen ovale during auricular systole. Thus in Wittig’s series (5) 50 per cent of the cases of paradoxical emboli were preceded by pulmonary embolism.

In a large series of cases of cerebral emboli 4 per cent were of the paradoxical variety. Hemmings (6) in a study of sudden postpartum death, found that 14.5 per cent of all cases were due to embolism; of these, 8 per cent were cerebral, 6 per cent coronary and 8 per cent pulmonary emboli. Paradoxical cerebral emboli have appeared during and many days after operation (7, 8, 9).

In our case paradoxical embolism is a real possibility because venous thrombosis was a likely consequence of this difficult delivery and a possible source of embolization when the patient strained while using the bedpan on the third postpartum day. Against this idea are the following facts: (a) no signs of venous thrombosis could be found; (b) there were no conditions such as shock or pulmonary embolism to favor the functional patency of a foramen ovale, and (c) the cerebral signs regressed more rapidly and completely
than would be expected were the embolus to have originated in a venous thrombosis.

The remaining possible mechanism is air emboli entering the circulation through the placental wound in the uterus and passing through the lungs or the foramen ovale into the arterial circulation. Transuterine air embolism is a well established cause of postpartum and postabortal death (10). In the vast majority of these cases the air was forced into the vagina or uterus by syringe or insufflation. But it is not well known that air under positive pressure may be trapped in the vagina and uterus as a result of changes in posture postpartum (10). Four deaths and one transient case of cerebral air embolism have been reported following the assumption of the knee-chest position on the seventh to eleventh day postpartum; in 2 cases the patient had performed the exercise without symptoms on the day prior to the day when the posture led to embolism (11-14). Our patient probably trapped air in her genital tract and forced it into the venous sinuses of the placental site while straining on the bedpan on the third postpartum day.

Air emboli arising in the uterine venous circulation usually produce pulmonary signs or shock. The lung is generally regarded as an effective barrier to the further passage of air into the left side of the heart. While this is usually true it is not always so (11). Villaret et al. photographed air in the cerebral vessels after injection of a peripheral vein (15). Cormack (16) found air in the cerebral vessels in one of 3 patients who died suddenly, without apparent cause, five hours after delivery. Garcia et al. (17) reported cases of cerebral air embolism following abortion. Deadman (18) presented two instances of post-abortal air embolism with postmortem evidence of air in the cerebral vessels. Rangell’s case (11) of cerebral air embolism quite closely resembled our own experience. His patient was a young woman who developed convulsions and a transient organic psychosis immediately after assuming the knee-chest position on the seventh postpartum day; she recovered completely.

Air emboli may be transported to the brain from the peripheral venous circula-

lation through a patent foramen ovale, as already discussed. A momentary period of shock and pulmonary arteriolar obstruction might allow the left auricular pressure to fall and the right auricular pressure to rise sufficiently to allow the paradoxical transmission of air emboli arriving in the right auricle at that moment. Cyanosis was observed in our patient at the time of onset of her cerebral signs. The cyanosis might have been caused, however, by respiratory depression not specifically related to any type of cerebral embolism. Regardless of the source of origin, only 1 cc. of air in the systemic circulation can cause cerebral signs (19).

The diagnosis of cerebral air embolism is difficult to prove in a patient who has survived. Certain characteristic signs such as pallor of the tongue and air bubbles in the retinal vessels were not thought of by the physician who saw her immediately after the onset of the cerebral signs. Skin petechiae were never found. But the diagnosis in our case is most probably that of cerebral air embolism because the onset was sudden, following straining on a bedpan; the signs were diffuse and the areas affected were multiple; the recovery was rapid and complete, and all other possible causes were much less likely or completely inapplicable.

The treatment of cerebral air embolism consists of halting the act which permits air to enter the circulation, placing the patient in Trendelenburg position to decrease the extent and duration of air blockade in the cerebral vessels and the administration of 100 per cent oxygen by mask to hasten the absorption of nitrogen from the air bubbles and so minimize permanent damage. There is reason to believe that the cerebral signs are as much due to spasm of cerebral vessels as to air blockade (20). An anesthetic block of the stellate ganglion on the same side as the embolus might, therefore, be indicated to overcome vascular spasm (21, 22). The convalescence of our patient might have been speeded by the application of these specific measures.

**Summary and Conclusion**

A normal young woman suddenly developed organic signs of cerebral embolism on
the third day following vaginal delivery under spinal analgesia and recovered spontaneously. The differential diagnosis is discussed; the conclusion is that probably air was trapped in the genital tract and was forced, by the act of straining, into the vessels of the placental site, then transported to the brain through the lungs or a patent foramen ovale.

Paradoxical embolism and transpulmonary transmission of air emboli are discussed in detail.

REFERENCES


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