Paraplegia Associated With Intraaortic Balloon Pump Counterpulsation

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SUMMARY Two patients developed paraplegia associated with the use of the intraaortic balloon pump. In one patient, transient spinal ischemic episodes ceased after removal of the intraaortic balloon and the second patient apparently sustained spinal cord infarction. Possible etiologic mechanisms include vascular occlusion due to balloon position, arterial spasm, thromboembolic phenomena, hypotension, hypoxia and arterial dissection.

SINCE THE FIRST TRANSFEMORAL INSERTION of the intraaortic balloon pump in 1968, numerous complications have been related to the balloon catheter: direct vascular injury with dissection or perforation, ischemic changes in the extremity used for the insertion and local changes owing to surgical insertion (groin infection, sepsis, hematoma and false aneurysm). Several neurologic complications related to intraaortic balloon pump insertion have also been reported. The development of paraparesis or paraplegia after intraaortic balloon pump insertion is described in two patients in this report.

Case Presentations

A 46-year-old man developed an acute myocardial infarction followed by ventricular fibrillation treated with electrical cardioversion. On a dopamine drip, his blood pressure was 105/78 mm Hg, pulse 150/minute, respiration 26/minute. Bilateral rales and an S4 gallop were present. The neurologic examination was normal. EKG revealed a recent inferolateral-posterior myocardial infarction and supraventricular tachycardia. Repeat cardioversion was performed and sinus rhythm established. CPK determinations were markedly elevated with a large MB fraction. On the fifth hospital day, because of persistent left ventricular failure and recurrent angina, a 35 ml capacity intraaortic balloon was inserted (D6-L3) via the right femoral artery. Cardiac catheterization revealed a markedly elevated left ventricular and diastolic pressure, occlusion of a dominant left circumflex artery and severe disease of the left anterior descending artery. With intraaortic balloon counterpulsation, there was immediate improvement in the patient’s clinical and hemodynamic status. He received heparin at 1000 units/hour and the following day, with the partial thromboplastin time (PTT) still in the control range, a bolus of 4000 units was given and the infusion increased to 1500 units/hour. Twenty-four hours after the balloon pump insertion, he developed transient numbness of and inability to move both lower extremities. Examination revealed weakness against resistance of both lower extremities including weakness of flexors of the hips.

SUMMARY Two patients developed paraplegia associated with the use of the intraaortic balloon pump. In one patient, transient spinal ischemic episodes ceased after removal of the intraaortic balloon and the second patient apparently sustained spinal cord infarction. Possible etiologic mechanisms include vascular occlusion due to balloon position, arterial spasm, thromboembolic phenomena, hypotension, hypoxia and arterial dissection.

There was hypoaesthesia below L1 level bilaterally and proprioception in the toes was impaired. The deep tendon reflexes were present and plantar reflexes were equivocal. During the episode, there was no clinical evidence of circulatory compromise to the lower extremities. No hypotension or cardiac arrhythmias were noted. After 45 minutes, full neurologic function returned. Heparin was increased to 3000 units/hour with achievement of PTT in the therapeutic range. The next day, an episode of numbness and heaviness of the lower extremities occurred. Neurologic examination revealed bilateral lower extremity weakness and hypoaesthesia below the inguinal area bilaterally. The episode resolved after a few minutes. Again, there was no evidence of decreased peripheral circulation. The patient’s hemodynamic status remained stable. One week after IABP insertion with the PTT 1½ times control on heparin 2000 units/hour, an episode of paresthesia and weakness occurred involving the right lower extremity. This resolved after 30 minutes. A similar episode occurred a few hours later. Anticoagulation was continued and weaning of the IABP was initiated. On the 18th hospital day, 13 days after it was inserted, the balloon pump was removed. Following a turbulent course including unstable angina, extension of myocardial infarction and dysrhythmia, the patient made gradual improvement and after six weeks, started progressive ambulation. His cardiac status was well compensated on Digoxin, diuretic and nitrate therapy. There was no recurrence of neurologic dysfunction during one year follow-up.

Patient #2

A 66-year-old, white female suffered an acute inferior myocardial infarction. The early post-infarction course was complicated by hypotension and congestive heart failure. A murmur of mitral regurgitation was noted on the second hospital day. Her clinical status improved after treatment with Dobutamine, Digoxin and Lasix. She experienced ventricular ectopy which was satisfactorily controlled with Lidocaine. Her peak CPK level was 5000 units. Twelve days after myocardial infarction, hypotension recurred and the patient was transferred to Westchester County Medical Center. Her blood pressure was 80/60 mm Hg, pulse 110/minute, respirations 35/minute and the temperature was 98.7°F. Bilateral rales were present over the lower 2/3 of the lung fields. Cardiac examination revealed an S3 gallop and a grade IV/VI holosystolic...
murmur at the apex radiating to the axilla. The patient was confused but had no focal neurologic deficit.

A 40 ml intraaortic balloon pump was inserted percutaneously via the left femoral artery and advanced to the level of the mid-descending aorta (upper end of balloon was at level of D7–8 intervertebral space and lower end opposite L4 vertebral body) from which point it could not be further advanced. A 5000 unit bolus of Heparin was given. The patient improved hemodynamically. Cardiac catheterization revealed severe proximal triple vessel coronary artery disease, severe mitral regurgitation, and severe left ventricular dysfunction.

One hour after cardiac catheterization, approximately 14 hours after the IABP insertion, she experienced sudden onset of paraplegia. She was disoriented and showed flaccid paraplegia, areflexia in the lower extremities, anesthesia below D10 level and position loss in the toes bilaterally. The femoral arterial pulsations were good and there was no evidence of vascular compromise of the lower extremities.

Because of the neurologic deficit, the IABP was removed. There was no clot seen on the catheter or balloon. There was no recovery of neurologic function.

After a few hours, the patient suddenly developed ventricular fibrillation. Resuscitation efforts were unsuccessful. Permission for autopsy was denied.

Discussion

Neurological complications associated with intraaortic balloon pump use include foot drop, unilateral lower extremity weakness, paraplegia, neuralgia over distribution of the medial cutaneous nerve of the thigh, and left hemiparesis (after ascending aortic balloon insertion).

Patient #1 developed transient paraparesis on two occasions (on days one and two of intraaortic balloon pumping) and two subsequent episodes of right lower extremity weakness and paresthesia (day 7). The fact that he never had any such symptoms prior to insertion or following removal of the intraaortic balloon pump suggests that there was a causal relationship. In patient #2 also, paraplegia was temporally related to IABP insertion. In these patients, during the neurologic episodes, there was no evidence of circulatory impairment to the lower extremities and cardiac arrhythmia, hypoxia, and hypotension were not noted. In both patients, spinal cord ischemia seems the most likely etiology of the neurologic manifestations.

In order to consider a possible ischemic mechanism, the blood supply of the spinal cord is briefly reviewed. The concept of descending and ascending blood flow currents in the anterior spinal artery has been controversial. According to one view, the blood flow current in the anterior spinal artery is from above downward. However, angiographic observation support the theory that the blood flow from two adjacent radicular arteries converge in the anterior spinal artery leading to "watershed" areas.

In the cervical area, the blood in the posterior spinal arteries flows from the posterior inferior cerebellar arteries downward. In the lower cord, the direction of flow in the posterior spinal arteries is upward. At the conus, the anterior spinal artery anastomoses via the "rami cruciantes" (Adamkiewicz) with the posterior spinal arteries. Thus, the descending blood current in the inferior part of the anterior spinal artery changes direction, reaching dorsally to the lower posterior spinal arteries, where the direction of flow is upward.

Thus, existence of anastomotic arterial channels would make the blood supply to the spinal cord a dynamic process with multiple areas of diminished bulk (watershed). Circumflex vessels ring the cord, uniting the anterior and posterior spinal arteries while anterior sulcal arteries enter the anterior fissure and turn left or right to supply gray matter and some of the white matter in the anterior two-thirds. Such vessels are poorly developed in the thoracic region, thus, making the thoracic cord the area most prone to developing ischemia.

The blood flow might be considerably altered and compensatory flow reversals may occur in pathological and even in some physiological situations. With obstructive vascular disease involving the spinal cord arteries or major extraspinal arteries (e.g. vertebral arteries or the aorta) a "steal phenomenon" may occur due to reversal of blood flow. It is conceivable that occlusion of an important radicular vessel may produce ischemia and even infarction particularly in the watershed zones several segments above or below the level of the obstructive lesion. The largest radicular artery exclusive of the vertebrals to reach the cord is the anterior radicular artery of the lumbar area, the arteria radicularis magna. This vessel occurs most frequently at the second lumbar segment but may be found anywhere between the eighth thoracic and the fourth lumbar segments.

Otomo, Van Buskirk and Workman have shown that the blood flow in the cervical region is in a caudal direction, while below this region, the flow is cephalic. Thus, the blood supply to the thoracic lumbar and sacral regions of the cord is heavily dependent on the arteria radicularis magna of the lumbar area. Insufficiency in this vessel could easily lead to loss of function in the lower cord.

The blood supply of the distal spinal cord can sometimes depend upon branches of the aorta below its bifurcation. Several small medullary arteries accompany the roots of the cauda equina and terminate in the anastomotic ring on the conus medullaris. These distal medullary arteries by themselves can supply the entire intrinsic vasculature of the distal spinal cord. Several mechanisms may account for compromise of spinal cord perfusion.

Severe hypoxia and drop in perfusion pressure in shock or cardiac arrest may cause spinal cord ischemia. It has been shown experimentally that hypotension will precipitate paraplegia after ligation of certain radicular vessels in dogs. In 11 patients with spinal stroke, Silver et al noted a high incidence of myocardial infarction with either ventricular arrhythmia or...
profund shock. The pathogenesis of the cord lesion in these cases was thought to be ischemia secondary to a drop in the perfusion pressure.

Thromboembolic episodes involving the cord are well documented but rare. Emboli consisting of fragments of atheromatous aortic plaques have been described. It is also possible that emboli originating in the heart may play a role. Ratlinov and Jimenez-Pabor reported intermittent ischemia of the cord in a patient with aortic occlusion and Rudar, Urbanke and Radonic described the sudden appearance of a paraplegia almost certainly attributable to blockage of the abdominal aorta by an embolus. Cook stressed the importance of aortic occlusion as a cause of acute or chronic paraplegia, even when the peripheral circulation appears normal.

Thromboembolic phenomena specifically related to the intraaortic balloon pump must also be considered. Schneider and Eckner performed scanning electron microscopic studies of the calf aorta following three days of IAB pumping with anticoagulation and demonstrated extensive deformation and patchy losses of the luminal endothelium adjacent to the balloon with mural thrombus firmly attached to the exposed subendothelium. In addition, renal embolism and infarctions, presumably from these mural thrombi, were noted. Among the 15 calves, each pumped with a dual chambered balloon, five developed bilateral hind quarter paralysis believed to result from balloon pumping episodes.

In addition to direct mechanical aortic injury, abnormal stimulation of the clotting system may be implicated in intraaortic balloon complications. Schneider in calf experiments found minimal physical changes in circulatory platelet structure in animals on an IABP. However, numerous irregularly shaped platelet aggregates were attached to a fibrin network on the IAB surface and catheter after either several days of discontinuous pumping or 7 days of continuous assist, despite systemic anticoagulation. This was particularly apparent in areas of diminished blood flow such as along that portion of the IAB catheter which was in the lower abdominal aorta and iliofemoral system. Schoen and associates conducted scanning electron microscopic studies of balloon surfaces that had been counterpulsed both experimentally (calves) and clinically. These investigators noted similar thrombotic patches approximately 2 mm in diameter. The extent of these patches appeared related to the duration of implantation. Embolic phenomena as a result of balloon induced thrombosis appear to be rare in humans. Scheidt and coauthors reported death associated with balloon thrombus attributable to renal emboli. This patient had not been anticoagulated. Dunkman and coauthors did not observe any clinical evidence of peripheral embolization but noted the presence of two small emboli, one testicular and one renal, at postmortem examination.

The influence of IAB pumping on flow in intercostal arteries is not known. Occlusion of or damage to the mouths of intercostal vessels especially those adjacent to the diaphragm on the left side near the origin of the arteria magna, by IABP counterpulsation, might be expected to cause spinal cord ischemia. Likewise, local mechanical trauma might induce vascular spasm, possibly compromising intercostal blood flow. Dissecting aneurysms from IAB catheter insertion may occur and may occlude intercostal arteries.

Repetitive transient spinal cord ischemic episodes in patient #1 suggest a reversible mechanism such as arterial compression or spasm. The fact that the neurologic episodes ceased entirely after removal of the balloon catheter further supports this contention. The level of sensory neurologic deficit (below L1) could have been due to compression of the arteria radicularis magna of Adamkiewicz which arises from lower thoracic or upper lumbar roots, and often show anatomic variation in individuals. However, a thromboembolic etiology cannot be ruled out on clinical grounds. In patient #2, apparently spinal cord infarction occurred. The anatomic location of the intraaortic balloon (D7–8 to L4) correlated fairly well with the topographic diagnosis in this patient (anesthesia below D10 level). Resistance was met during balloon catheter insertion such that it could only be advanced to the mid-descending thoracic aorta. These facts suggest the possibility of aortic dissection as the mechanism of spinal cord injury in this patient.

The neurologic complications described here further underscore the potential hazards of the intraaortic balloon pump. The patient’s circulatory and neurologic status must be continuously monitored. Prompt detection of neurologic dysfunction and appropriate intervention might prevent irreversible spinal cord injury.

References
Nerve Terminal Damage in Cerebral Ischemia: Greater Susceptibility of Catecholamine Nerve Terminals Relative to Serotonin Nerve Terminals

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SUMMARY The energy-dependent uptake of (3H)-dopamine (DA), (3H)-norepinephrine (NE) and (3H)-serotonin (5-HT) was measured in synaptosomes isolated from either the whole cerebral hemispheres or the striata of gerbils after cerebral ischemia. Ischemic stroke was induced in the Mongolian gerbil by left common carotid ligation. Uptake values in the affected hemisphere (expressed as a percent of the corresponding control hemisphere) were 32.6% for DA, 35.1% for NE, and 52.0% for 5-HT, 16 hours after stroke. The differential reduction in uptake of the catecholamines relative to 5-HT was significant (p < 0.005). This differential persisted when measures were made on isolated striata from the ischemic and control hemispheres. In the latter measurements, uptake of DA was 20.7% of control and uptake of 5-HT was 44.7% of control. Uptake of both DA and NE were significantly reduced in animals exhibiting milder circling behavior, while uptake of 5-HT was not. There was no significant reduction in uptake in animals subjected to left common carotid ligation not exhibiting signs of stroke. These studies indicate a selective sensitivity of catecholamine nerve terminals to damage in ischemic stroke.

PRIOR STUDIES IN THIS LABORATORY examined the active uptake of dopamine, GABA and glutamate into synaptosomes that were prepared from gerbils at 16 hours after unilateral carotid ligation. The high-affinity uptake of neurotransmitters by synaptosomes is an energy-dependent process that requires the integrity of neuronal membrane function. No changes in uptake were noted for up to 8 hours after carotid ligation, even though animals exhibited signs of stroke. However, by 16 hours, the uptake of dopamine (DA) into synaptosomes prepared from the ischemic hemisphere was reduced to 15.2% of the unaffected hemisphere. GABA uptake was reduced to 28.0% and glutamate uptake to 47.5%. The greater effect of ischemia on the uptake of dopamine relative to glutamate was significant (p < 0.001). Potassium-stimulated release of radiolabelled transmitter from the synaptosomal preparation, following labeling of the tissue via uptake, was not affected. This indicated that the residual uptake of transmitter was into normally functioning synaptosomes and that the reduction in uptake was most probably due to loss of nerve terminals.

Marked reductions in the levels of the catecholamines DA and norepinephrine (NE) have been observed in the ischemic hemispheres of gerbils exhibiting stroke after unilateral carotid ligation. Layne et al and Meyer et al have proposed that increased release of NE and 5-HT occurs during ischemia, and accounts for the decreased levels in the brain. However, this would not explain why 5-HT levels are not reduced to the same extent as dopamine and NE in cerebral ischemia. Therefore, we studied the high-affinity active uptake of DA, NE and 5-HT into synaptosomes prepared from gerbils 16 hours after unilateral carotid ligation, in order to determine if there was a differential susceptibility of these neuronal populations to the effects of ischemia.

Materials and Methods

Male gerbils weighing 50 to 70 gm were anesthetized with approximately 35 mg pentobarbital/kg (i.p.), with the dosage titrated to the stage of surgical anesthesia for each animal. Animals could breathe spontaneously without a respirator. The left common
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