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Noncardiac Surgery Brain Injury: Etiologic Factors and Prevention

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David A. Stump, PhD, D. M. Colonna, D. Kilgus, D. M. Moody, D. Deal

Departments of Anesthesiology and Radiology, Wake Forest University School of Medicine, Winston-Salem, North Carolina, USA

During total joint arthroplasty, showers of bony spicules, marrow fat, and clot are carried by venous blood to the lungs [Capan 1993], creating conditions not unlike those present in patients who have suffered traumatic long bone fractures. There is recent evidence that, like the fat embolism syndrome (FES), which often has a component of neurologic dysfunction, total joint arthroplasty and femoral nailing are associated with intraoperative brain embolization as determined by transcranial Doppler ultrasonography [Colonna 1999, Sulek 1999, Edmonds 2000] and magnetic resonance brain imaging [Fisher 1995]. Although there are good data demonstrating that intraoperative brain embolization occurs during total joint arthroplasties, the makeup and, even more importantly, the clinical significance of these emboli remain speculative.

Brain microemboli resulting from cardiac surgery occur by the millions and may cause focal ischemia resulting in significant neurologic dysfunction. Our studies suggest that the major source of these microemboli is lipid droplets of the patient's fat that drip into the blood in the surgical field. This lipid-laden blood is aspirated and then returned to the patient via the cardiopulmonary bypass (CPB) apparatus. Our investigations have focused on the causes (microemboli), consequences (brain damage), and strategies for elimination of brain lipid microemboli resulting from salvaged blood collected during surgery.

We recognize many similarities in the kind of brain injury seen in orthopedic and cardiac surgery. For example, recent data suggest that brain embolization occurs in 40% to 60% of patients during total joint arthroplasty [Colonna 1999, Sulek 1999, Edmonds 2000], though the structural nature of the emboli and the degree to which the brain is embolized are unknown.

We believe that brain embolism during total joint arthroplasties has clinical importance, because of our suspicion that it may underlie postoperative delirium (or confusional states and/or cognitive dysfunction). This condition is common in the elderly, occurring in 10% to 15% of general surgery patients, and in as many as 44% to 55% in particular subgroups of orthopedic surgery patients [Fisher 1995, Lipowski 1998].

Patients undergoing CPB may develop systemic inflammation response syndrome (SIRS) and will possibly suffer from postsurgical cognitive deficits (transient, 061%; permanent, 023%). The foreign surfaces of the CPB extracorporeal circuit cause blood contact activation, which exacerbates the endothelium irritation caused by emboli (gaseous, lipid, and particulate), delivered from the pump to the patient.

Typically the shed blood during both cardiac and orthopedic surgery is aspirated from the surgical field. The aspirated blood will include lipid components released from subcutaneous fat from the surgical incision (further increased by the use of electrocautery) and lipid-rich ooze from cut bone surfaces (sternum, hip acetabular socket, femur, tibia). The negative force of the aspiration suction can damage the blood cells as well. We have associated the return of shed blood during CPB with increased levels of cerebral lipid microemboli and have found that processing the shed blood with a cell-saver device before reinfusion significantly reduces cerebral lipid microemboli.

Total hip or knee replacement orthopedic surgery requires the insertion of joint prosthesis components into bone (cemented or uncemented). The prosthesis implantation compresses the medullary canal, forcing lipid emboli into vascular spaces and resulting in systemic embolization. The elevated temperature caused by the exothermic reaction of the cement hardening as the components are implanted also may cause further release of lipid emboli. Systemic lipid embolization causes endothelium irritation and neutrophil activation and initiates the production of proinflammatory mediators. This deleterious cascade may lead to FES, causing a sudden drop in blood pressure, impaired lung function, heartbeat irregularities, sudden heart failure, or any combination of these life-threatening complications.

We have demonstrated in a canine model of vertebroplasty that the glue transits the vertebral body and forms a cast of the venous system. In addition, we found a remarkable amount of the glue hardened in the lungs but not the brain. The appearance of small lipid droplets in the brain of the dogs suggested that the lungs act as a "strainer" and not a filter for lipid microemboli, resulting in smaller lipid droplets than those documented coming from the CPB circuits but still numerous. In a recent case report we documented millions of lipid microemboli in the brain and other organs of a woman who died shortly after the injection of methyl methacrylate cement in the absence of a patent foramen ovale [Colonna 2002].

The identification of fat emboli within the brains of patients undergoing orthopedic procedures raises many important questions: What is the true incidence of systemic (brain) fat embolization in orthopaedic surgery patients? What is the prognostic importance of lipid grain embolization? What patient subgroups are most susceptible to cognitive dysfunction after

Address correspondence and reprint requests to: David A. Stump, PhD, Departments of Anesthesiology and Radiology, Wake Forest University School of Medicine, Winston-Salem, North Carolina, USA 27157.

brain embolization during orthopaedic surgery? Specifically, are patients with cardiac septal defects at greater risk for brain embolization during orthopedic surgery? Are there differences in brain embolization rates between patients who have cemented versus uncemented prostheses? Further study is warranted.

REFERENCES

Capan LM, Miller SM, Patel KP. 1993. Fat embolism. Anesthesiol Clin North Am 11:25-54.

Colonna DM, Kilgus DJ, Brown W, Challa V, Stump DA, Moody DM. 2002. Acute brain fat embolization occurring after total hip arthropasty in the absence of a patent foramen ovale. Anesthesiology 96(4):1027-9.

Colonna DM, Stump DA, Kilgus DJ, Manuel JC, Hilbawi HR. 1999. Total hip arthroplasty produces intraoperative brain embolization and neuropsychologic dysfunction up to 6 weeks postoperatively. Anesthesiology 91:A79.

Edmonds CR, Barbut D, Hager D, Sharrock NE. 2000. Intraoperative cerebral arterial embolization during total hip arthroplasty. Anesthesiology 93:315-8.

Fisher BW, Flowerdew G. 1995. A simple model for predicting postoperative delirium in older patients undergoing elective orthopedic surgery. J Am Geriatr Soc 43:175-8.

Lipowski ZJ. 1998. Delirium in the elderly patient. N Engl J Med 320:578-82.

Sulek CA, Davies LK, Enneking FK, Gearen PA, Lobato EB. 1999. Cerebral micro-embolism diagnosed by transcranial Doppler during total knee arthroplasty: correlation with transesophageal echocardiography. Anesthesiology 91:672-6.