Case Report

Korean J Anesthesiol 2011 January 60(1): 52-53
DOI: 10.4097/kjae.2011.60.1.52

Periosteal nociceptors induced hypotension and bradycardia under spinal anesthesia
-A report of two cases-

Rakesh Garg, Pradeep Karunagaran, and Mridula Pawar

Department of Anaesthesiology and Intensive Care, Postgraduate Institute of Medical Education and Research, Dr. Ram Manohar Lohia Hospital, New Delhi, India

The sudden hemodynamic disturbance in the perioperative period can occur because of various surgical and anesthetic reasons but hemodynamic collapse due to noxious stimulus of periosteum stripping has not been described. We report two cases of severe hypotension and bradycardia during periosteum stripping in orthopedic surgery under subarachnoid block even though the block level was adequate. In our patients, hemodynamic collapse occurred specifically at a moment when surgeons manipulated periosteum and fall in blood pressure and heart rate was sudden in onset. The hemodynamic disturbance did not appear to be related to vagally mediated or due to blockade of sympathetic fibers but appeared to be related to periosteal nociceptors. (Korean J Anesthesiol 2011; 60: 52-53)

Key Words: Bradycardia, Hypotension, Nociceptors, Periosteum, Spinal anesthesia.

The sudden hemodynamic disturbance in the perioperative period can occur because of effect of anesthetic technique but sometimes also because of surgical procedure like neurosurgery, abdominal, laparoscopic, ophthalmic and facial surgery [1]. In most cases a vagally mediated reflex has been implicated, although experimental support for this is often lacking. Hemodynamic disturbances due to noxious stimulus of periosteum stripping has not been described. We report two cases of severe hypotension and bradycardia during periosteum stripping in orthopedic surgery under subarachnoid block even though the block level was adequate.

Case Reports

Case 1

A 28-year-old male, weighing 67 kg and height 180 cm with fracture tibia was scheduled for open reduction and internal fixation. Subarachnoid block was given with 3 ml of 0.5% bupivacaine (heavy) and a sensory level of T8 was achieved. Surgery was started and patient remained hemodynamically stable. Sixty minutes later, when surgeon was performing periosteal stripping, there was sudden hypotension (blood

Received: June 24, 2010. Revised: July 6, 2010. Accepted: July 12, 2010.
Corresponding author: Rakesh Garg, M.D., Department of Anaesthesiology and Intensive Care, Postgraduate Institute of Medical Education and Research, Dr. Ram Manohar Lohia Hospital, New Delhi, India. Tel: 91-11-981039495, Fax: 91-11-981039495, E-mail: drrgarg@hotmail.com

pressure decreased from 118/78 mmHg to 78/40 mmHg) and bradycardia (heart rate decreased from 84/min to 40/min). Intravenous atropine (0.3 mg repeated twice) was administered. Patient complained of discomfort in the lower limb at the surgical site. Intravenous fentanyl (25 μg) was administered. Hemodyanamic parameters became normal. Sensory level was T8. Patient remained stable thereafter.

Case 2

An 18-year-old male, weighing 78 kg and height 175 cm, was scheduled for open reduction and internal fixation of fracture tibia. Patient received combined spinal epidural block with 3 ml of 0.5% bupivacaine (heavy) intrathecally. Sensory block up to T6 level was achieved. After about 90 minutes of subarachnoid block, when surgeon manipulated the iliac bone for bone graft retrieval, suddenly there was fall in blood pressure and heart rate, which responded to intravenous atropine. Sensory block was still at the level of T12. Patient responded to intravenous fentanyl and remained stable throughout the surgery.

Discussion

In our patients, hemodynamic collapse occurred specifically at a moment when surgeons manipulated periosteum and fall in blood pressure and heart rate was sudden in onset. The effect of intrathecal bupivacaine leading to hypotension and bradycardia was ruled out as this event occurred after >60 minutes of subarachnoid block in both the patients. Also, prior to periosteum manipulation, hemodynamic parameters were stable. So it appears that the hemodynamic disturbance was probably not vagally mediated or due to blockade of sympathetic fibers.

The periosteum covers the outer surface of a bone and is richly innervated with sensory (pain and proprioception) nociceptor fibers thus making it very sensitive to manipulation. A nociceptor is a sensory receptor that reacts to potentially damaging stimuli by sending nerve signals to the spinal cord and brain. The cell bodies of these neurons are located in either the dorsal root ganglia or the trigeminal ganglia. The trigeminal ganglia are specialized nerves for the face, whereas the dorsal root ganglia associate with the rest of the body. Nociceptors have two different types of axons − Aδ and C fiber. Afferent nociceptor fibers travel to spinal cord where they synapse in the dorsal horn and use glutamate or substance P as the neurotransmitter. Substance P is an important element in pain perception. Substance P also has effects as a potent vasodilator which is dependent on nitric oxide release [2]. The activation of C fibres and thus release of substance P in our patients could be explained by the fact that some afferent fibres from the lower limb may traverse the sympathetic trunks for quite a distance i.e. the paraspinal pathways, and ultimate terminate in the spinal cord cephalad to the level of a spinal block [3]. So stimulus via C fibres can activate dorsal horn even when sensory level blockade is optimal in spinal anesthesia.

The presence of sudden bradycardia may be attributed to activation of Bezold-Jarisch reflex (BJR) due to sudden hypotension. BJR is elicited by activation of certain inhibitory reflexes, which have origin with cardiac sensory receptors and that leads to increases parasympathetic nervous system activity and inhibits sympathetic activity [4]. The receptors are the nonencapsulated terminals of the C-fiber afferents in the walls of the ventricles. These are activated in response to various stimuli like mechanical (pressure, inotropism, volume) and chemical (veratrum alkaloids, serotonin, adenosine triphosphate, serum, amidine derivatives, capsaicin and venoms from snakes, insects and marine animals) stimuli. In our cases sudden vasodilatation due to substance P leads to relative hypovolemia and thus elicited BJR and associated bradycardia.

We conclude that stripping or manipulation during orthopedic surgery under spinal anesthesia can lead to sudden hypotension and bradycardia. Any sudden hemodynamic collapse during periosteum manipulation should be managed not only be supportive therapy but also by providing adequate analgesia.

References

1. Doyle DJ, Mark PW. Reflex bradycardia during surgery. Can J Anaesth 1990; 37: 219-22.
2. Bossaller C, Reither K, Hehlert-Friedrich C, Auch-Schwelk W, Graf K, Gräfe M, et al. In vivo measurement of endothelium-dependent vasodilation with substance P in man. Herz 1992; 17: 284-90.
3. Dejong R, Cullen SC. Theoretical aspects of pain: bizarre pain phenomena during low spinal anesthesia. Anesthesiology 1963; 24: 628-35.
4. Campagna JA, Carter C. Clinical relevance of the Bezold-Jarish reflex. Anesthesiology 2003; 98: 1250-60.