

# Complications of Spinal Anesthesia

HATIM HYDERALLY, M.D.

## Abstract

Serious neurological complications after spinal anesthesia are rare, but do occur. The most common are postdural puncture headache and hypotension. Hypotension after spinal anesthesia is a physiological consequence of sympathetic blockade. The diagnoses and management of these sequelae are discussed.

**Key Words:** Regional anesthesia, complications, spinal anesthesia, postdural puncture headache, transient radicular irritation, cauda equina syndrome, neurologic deficits.

---

PROFESSOR AUGUST BIER performed the first surgical operation using spinal anesthesia at the Royal Surgical Hospital of the University of Kiel, Germany on August 16, 1898, heralding the advent of major regional anesthesia using neuraxial blockade (1). In 1927, spinal anesthesia was performed by Gaston Labat at The Mount Sinai Hospital (2). Since then, of course, it has been well incorporated into the practice of anesthesiology.

The complications of spinal anesthesia may be categorized as:

- spinal – postdural puncture headache
- cardiovascular
- neurologic

## Postdural Puncture Headache

Postdural puncture headache is the most common complication of spinal anesthesia. It occurs most frequently in young adults including obstetric patients, with an incidence rate of 14%, compared to 7% in individuals older than 70 years.

The use of smaller needles with pencil-point tips has markedly reduced the incidence of postdural puncture headaches. These intense headaches occur when cerebrospinal fluid (CSF) escapes through the dural puncture site, resulting in intracranial tension on meningeal vessels and nerves. Prompt treatment is essential and consists of bed rest, adequate hydration (orally or intravenously), and the use of analgesics. If conservative therapy fails, an epidural blood patch with 10–15 mL of autolo-

gous blood injected at the site of the “meningeal” tear may be necessary to minimize further leakage of CSF. This modality is very effective in relieving symptoms despite the fears of infection and neurological sequelae (3).

## Cardiovascular Complications

The incidence of hypotension following spinal anesthesia is 10–40%. The hypotension is related to the extent of sympathetic blockade, which is responsible for a decrease in systemic arteriolar and venous tone. Cardiac output may fall as a result of the decreased venous return (4).

Significant hypotension should be treated with appropriate administration of intravenous fluids and judicious use of vasoactive drugs such as ephedrine or phenylephrine.

Cardiac arrest has been reported in healthy patients during administration of a spinal anesthetic (4). The cardiac arrest occurs suddenly, often preceded by severe bradycardia in an otherwise hemodynamically stable patient. In these cases, hypotension or hypoxemia is not a precipitating cause. It is postulated that the etiology of the bradycardia and asystole might be a reflex mechanism such as the Bezold-Jarisch reflex (4).

## Neurologic Complications

The incidence of severe neurologic deficits following spinal anesthesia is low. In a prospective study of 40,640 cases of spinal anesthesia, the authors reported an incidence rate of serious neurological deficits of 0.5 per 10,000 (confidence intervals 0.2–1.1 per 10,000) (5). When they do occur, they are of great concern to both the patient and the practitioner. Despite the low incidence, many pa-

---

Address correspondence to Hatim Hyderally, M.D., Associate Clinical Professor of Anesthesiology, Department of Anesthesiology, Box 1010, Mount Sinai School of Medicine, One East 100th Street, New York, NY 10029-6574.

tients reject spinal anesthesia because they fear this complication.

The most benign neurologic complication is aseptic meningitis. This syndrome usually presents within 24 hours of spinal anesthesia and is characterized by fever, nuchal rigidity and photophobia. Microscopic examination of CSF is characterized by polymorphonuclear leukocytosis; bacterial CSF cultures are negative. Aseptic meningitis requires only symptomatic treatment and usually resolves within a few days.

Cauda equina syndrome presents after regression of the neuraxial blockade. This syndrome may be permanent, or it may regress slowly over weeks or months (6). It is characterized by a sensory deficit in the perineal area, urinary and fecal incontinence, and varying degrees of motor deficit in the lower extremities.

The most serious neurologic complication is adhesive arachnoiditis. This reaction usually occurs several weeks or even months after spinal anesthesia. The syndrome is characterized by a gradual progression of sensory deficits and motor weakness in the lower limbs. There is a proliferative reaction of the meninges and vasoconstriction of the spinal cord vasculature.

Spinal cord ischemia and infarction may occur after prolonged periods of arterial hypotension (6). The use of epinephrine in anesthetic solutions may reduce blood flow to the spinal cord (6). Traumatic injury to the spinal cord and nerve roots is a rare cause of neurologic deficits. In the past, syringes and ampules of local anesthetic medications were sterilized by immersion in phenolic disinfectant solu-

tions. This chemical contamination has been implicated as a cause of neurologic complications following spinal anesthesia.

Transient neurologic symptoms consisting of severe radicular back pain after neuraxial blockade regression were reported in 1960 following spinal anesthesia with 5% lidocaine. There were no sensory or motor deficits, and the symptoms resolved spontaneously within a few days. The etiology of this radicular pain was unclear. The incidence was greater with the use of lidocaine compared with bupivacaine and was more common in patients having surgery with the hips or knees in the flexed position (7).

### References

1. Van Zundert A, Goerig M. August Bier 1861–1949. *Reg Anesth Pain Med* 2000; 25:26–33.
2. Bacon DR. Regional anesthesia and chronic pain therapy: A history. In: Brown DL, editor. *Regional anesthesia and analgesia*. Philadelphia: WB Saunders; 1996. Chap. 2. pp.14–15.
3. Bridenbaugh PO, Greene NM. Spinal (subarachnoid) neural blockade. In: Cousins MJ, Bridenbaugh PO, editors. *Neural blockade*. 2nd ed. Philadelphia: JB Lippincott; 1998. Chap. 7. pp. 213–251.
4. Mackey DC. Physiological effects of regional block. In: Brown DL, editor. *Regional anesthesia and analgesia*. 1st ed. Philadelphia: WB Saunders; 1996. Chap. 22. pp. 397–422.
5. Auroy Y, Narchi P, Messiah A, et al. Serious complications related to regional anesthesia: Results of a prospective survey in France. *Anesthesiology* 1997; 87:479–486.
6. Kane RE. Neurologic deficits following epidural or spinal anesthesia. *Anesth Analg* 1981; 60:150–161.
7. Horlocker TT, Wedel DJ. Neurological complications of spinal and epidural anesthesia. *Reg Anesth Pain Med* 2000; 25:83–98.