Concerning Neurologic Sequelae of Spinal Anesthesia

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The goal of this study was to determine the incidence and severity of neurologic complications after spinal anesthesia and to examine the factors contributing to these complications. Early and late effects of 10,098 spinal anesthetics were analyzed in 8,460 patients treated at the Hospital of the University of Pennsylvania between 1948 and 1951. The comparison groups were comprised of 1,000 patients undergoing similar procedures under general anesthesia and 75 patients who received spinal anesthesia after general anesthesia. Follow-up data on 8,987 spinal anesthetics were obtained 6 months after surgery via mailed questionnaire or by physical examination. Patients of both sexes, ranging in age from 10 to 89 yr, were included. Exclusion criteria for spinal anesthesia were reports of a previous unsatisfactory spinal anesthetic, neurologic disease, backache, frequent headaches, difficulty with the legs, or infections of the back. Lumbar puncture was performed with needles ranging from 16- to 24-gauge. Spinal anesthetic preparations used included tetracaine, procaine, dibucaine, piridocaine, and pyrrolocaine with or without epinephrine and dextrose. Patients were examined postoperatively to discover neurologic disease. Follow-up questions determined whether patients would choose a spinal anesthetic again, if they experienced any untoward effects, and their current condition. When possible, additional information was obtained from patients with suspicious symptoms. Only one case of incapacitating neurologic disease was observed in the 6-month follow-up examinations after the spinal anesthetic. The patient had an asymptomatic meningioma of the spinal cord. No patients developed cauda equina syndrome, transverse myelitis, or meningeval or epidural sepsis. The primary minor neurologic sequela was headache (9% in male patients and 15% in female patients). Transient minor sequelae included backache, pain and numbness in the extremity, and an occasional weakness in the leg. Neurologic complications are uncommon after spinal anesthesia with careful patient selection, meticulous technique, and use of safe concentrations of spinal anesthetic mixtures.

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ROBERT Dunning Dripps, Jr., M.D., then located at the Department of Anesthesiology, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania, mentor, then my confrere, surely would have been gratified to learn that his studies on neurologic complications of spinal anesthesia are once again under scrutiny, after nearly half a century. Actually, while still alive (he died in 1972), he would have known that his endeavors had already been cited both by Little in his monograph Classic Anesthesia Files and by Faulconer and Keys in their two-volume opus Foundations of Anesthesiology. My involvement with this work began soon after 1948, when Bob asked me to participate at the beginning of residency training at the University of Pennsylvania. A photograph is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org. Bob’s study was supported in part by a contract with the Office of the Surgeon General, U. S. Army, Washington, D.C., almost in parallel with the multi-institutional prospective study of the deaths associated with anesthesia and surgery performed by Beecher and Todd.

Historical Background

During the 1940s, concerns over the complications of spinal anesthesia reached a crescendo, whereas most anesthetists doubtlessly never knew or had forgotten the early sequelae that were noted almost immediately after its introduction by Drs. August Bier and August von Hildebrandt, Royal Surgical Clinic of Kiel, Kiel, Germany, in 1898. Notably, Bier reported a classic lumbar puncture headache, although he noted that “other agents related to cocaine might not cause these unpleasant side reactions, or that other additions to cocaine might abolish them altogether. Therefore, it did not seem justified to make further
experiments on human beings.” On November 2, 1899, Rudolph Matas, M.D., the pioneering vascular surgeon from the Department of Surgery, Tulane University, New Orleans, Louisiana, was the first to apply the subarachnoid method in the United States and probably the first to inject morphine into the cerebrospinal fluid with the hope of prolonging and intensifying the effect of cocaine.

Matas likewise referred to some disturbing consequences of the method: “As to the immediate accidents of the operation, there are as a rule none. Nevertheless, Gumprecht (Deutsche Med. Wochenschr XXVI, June 14, 1900) collected 17 cases from the clinics of Quincke, Förbringer, Lenhartz, Lichtisein, Wilms, Krönig, Bull, and his own, in which death has followed quickly after lumbar puncture for exploratory purposes” (probably related to the presence of a grave illness or to herniation of the hindbrain). Symptoms referable to injury of the cauda equina were referenced by Scard and Cardol, and Heumberg (January 19, 1900) reported a case in which an intradural and medullary hemorrhage followed injury to the veins that accompany the filum terminale.

Racovicceanu-Petesci, a surgeon who by 1900 had operated on 125 patients using the method, knew of two deaths that had occurred in Romania as a result of spinal cocaization. In three of his own patients, it had been necessary to resort to artificial respiration and stimulants. According to Dr. August Bier, “In reading detailed reports in Romaina Medica for 1900, one is struck by the frequency with which symptoms of collapse occur, necessitating the most energetic methods.” There were five deaths among theodor Tuffier’s patients that he thought could not be related to the anesthetic; however, the fifth patient died of asphyxia, and the autopsy showed mitral insufficiency and two fresh lung emboli (1899).

A. E. Barker in the United Kingdom recorded technical improvements in spinal anesthesia in his report of 1907. By that time, stovaine (amylocaine hydrochloride) had replaced cocaine as the anesthetic of choice. Adrenaline had also been added to anesthetic mixtures to prolong their effect. Positioning of patients and the addition of glucose to yield a hyperbaric mixture were found to be important elements in the spread and control of anesthetic levels, and the physical constitution of the solution (density) was deemed quite important.

The delayed effects included headache of varying degree, pressure sensations in the head, and difficulty with vision and sensitivity to light. Becker, in 1906, clearly described the development of sixth cranial nerve, or abducens, palsy in a patient given stovaine for anesthesia. Often there were meningitic signs, but because the cerebrospinal fluid was clear on diagnostic puncture, aseptic meningitis was thought to be the cause. Finally, motor disorders of the legs might appear. In relation to all of these disorders, the agents used were listed in order of decreasing numbers of complications, as follows: stovaine, tropocain, and novocain.

The Anesthesia Milieu in the 1940s

Toward the end of that decade, Gunnar Thorsen, a Swedish neurologist surgeon, published a monograph on neurologic complications after spinal anesthesia involving 2,493 cases. However, the immediate catalyst of Dripps’s study was a report by Kennedy et al., in which the authors averred that “paralysis below the waist (chronic adhesive arachnoiditis) is too high a price for the patient to pay for the fine relaxation afforded the surgeon.” In that era, anesthesiology was a rapidly developing specialty. Thus, in a way, Dripps was responding to a plea by William T. Salter, a pharmacologist at Yale University, New Haven, Connecticut, who in essence editorially proclaimed, “Without vision and research, the professions die.” Prophetically, within a decade or so, the field of anesthesiology would mount a multi-institutional study of the reported hepatotoxic properties of the newly introduced inhalant, Fluothane.

Appraisal

I do not propose to delve into the details of the article reviewed here, for the goals and outcome of the study are available to the reader. Surely, as investigations progressed, the technique of spinal anesthesia under such close scrutiny must have improved. For example, refer to table 4 in the original article for a list of anesthetics used over time. Almost simultaneously, manufacturers improved anesthetic equipment in the form of prepackaged, sterilized trays containing syringes, needles, and local anesthetics, as we have come to expect some 50 yr later. Finally, although the first report emanating from this extensive survey revealed no major neurologic sequelae, several classes of minor reactions could be classified and are discussed in the accompanying references.

References

6. Barker AE: Clinical experiences with spinal analgesia in 100 cases and some reflections on the procedure. BMJ 1907; 1:665

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