The use of anticoagulation therapy in neurosurgery remains a problematic issue on a day-to-day basis. Postoperative immobility caused by neurological deficits, incisional pain, and the need for mechanical ventilation can predispose the patient to DVT, which may require intervention (that is, Level 1 or equivalent anticoagulation therapy). In addition, the cardiac stress produced by the general anesthesia required for major intracranial or spinal surgery can cause myocardial ischemia and/or infarction, thus necessitating a perioperative Level 1 (or higher) anticoagulation protocol. The neurosurgeon is thus left to weigh the risks of postoperative hematoma formation against the benefits of protecting against myocardial dysfunction or PE.

Although the issue of perioperative anticoagulation therapy for intracranial surgery has been extensively debated and studied, the use of anticoagulating agents after spinal surgery is less well delineated. We have conducted a review of the literature and we offer practical guidelines for the use of perioperative Level 1 anticoagulation therapy after spinal surgery.

Abbreviations used in this paper: DVT = deep venous thrombosis; PE = pulmonary embolism.
RESULTS

There were no prospective randomized studies regarding the use of Level 1 anticoagulation therapy after spinal surgery. The incidence of thromboembolic complications (DVT and PE) following spinal surgery is estimated to be 0 to 56.25%.4,6,7,13,16

There was only one article1 whose authors directly addressed the issue of perioperative therapeutic heparinization after spinal surgery and the complications associated with this therapy. This article contained the results of a retrospective, nonscientific survey of 22 members of the Scoliosis Research Society, encompassing more than 2400 cases. The survey was therefore classified as Level 3 evidence. Respondents to the survey were asked to identify from memory patients in whom clinically detected PE had developed after thoracic, lumbar, or combined surgery and who required Level 1 anticoagulation therapy.

In the survey, nine patients were identified who received heparinization according to a Level 1 protocol after development of PE. Of these nine, one suffered a postoperative spinal epidural hematoma requiring evacuation, whereas six (67%) of the nine patients experienced a major complication. The authors concluded that because of the high rate of major complications associated with postoperative heparinization, inferior vena cava filters were the method of choice for preventing fatal PE. In another case report with Level 3 evidence, its authors also advocated the use of inferior vena cava filters instead of Level 1 heparinization for the prevention of fatal PE.15

There were six studies involving identification of a postoperative spinal epidural hematoma as either a report of a single case or as one case that was part of a retrospective series of spinal surgeries.5,8,10,14,15,17 In one of the six single case reports, the patient in question received postoperative Level 1 anticoagulation therapy for a clinically detected DVT.17 There was a single publication in which two postoperative epidural hematomas that appeared after cervical surgery were reported in a series of 384 patients; neither hematoma was associated with postoperative heparinization.9

There was a single publication in which clinical outcomes were said to be related to the timing of evacuation of spinal epidural hematomas. In that study, Lawton, et al.12 identified 12 patients in whom epidural hematomas developed after spinal surgery; none of the patients was treated with postoperative anticoagulation therapy. The aforementioned study encompasses a 14-year review of all spinal epidural hematomas identified at a single institution, but the total number of surgeries was not stated. The evidence in this study was classified as Level 3.

Three other notable studies were identified. Catre4 conducted a systematic literature review and metaanalysis in 1997 to determine the true rate of thromboembolic complications after spinal surgery. Because this was a literature review, the article was classified as Level 3 evidence. Catre concluded that the data were insufficient to determine the rate of DVT and PE after spinal fusion. He also concluded that there were insufficient data to determine the efficacy of prophylactic doses of anticoagulant drugs for use after spinal surgery. Nevertheless, he failed to address the topic of Level 1 heparinization therapy after spinal surgery.

Kou, et al.,13 conducted a case-controlled study of risk factors for postoperative spinal epidural hematoma after lumbar laminectomy, which involved more than 400 cases at a single institution over a 10-year period. This work met the criteria for a Level 2 evidence study. Using a logistic regression model, they identified preoperative coagulopathy as an independent risk factor for postoperative epidural hematoma. Nevertheless, after reviewing the 12 identified cases of postoperative spinal epidural hematoma, they did not identify postoperative heparinization or PE as risk factors, although the exact incidence of these events in the entire study population was not mentioned.

Uribe, et al.,18 published a retrospective, case-controlled analysis of 4018 patients treated by six spine surgeons. These authors identified seven patients who presented with delayed postoperative epidural hematoma after spinal surgery. Their study met the criteria for Level 2 evidence. They concluded that previous surgery at the level of the operation significantly predicted the risk of delayed postoperative epidural hematoma. Postoperative Level 1 anticoagulation therapy was not involved in any of the cases.

A total of 40 cases of postoperative spinal epidural hematoma was reported; two of these (2.5%) were associated with postoperative Level 1 anticoagulation therapy. No articles were identified in which the use of intravenous Level 1 heparin was mentioned for treatment of myocardial infarction or ischemia in the perioperative period after spinal surgery.

A summary of recommendations and the level of evidence for postoperative Level 1 anticoagulation therapy is presented in Table 1. No studies were identified that met the criteria for Level 1 evidence. The most relevant published study, which was conducted by Cain, et al.,4 only met the criteria for Level 3 evidence. Some of the obvious weaknesses of that study were the lack of case controls and the fact that the respondents to the survey conducted no systematic chart review. Only nine clinically detectable pulmonary emboli were identified in more than 2400 patients who had undergone spinal fusion. Nonetheless, there was at least anecdotal evidence for a high (67%) rate of major complications after the perioperative administration of a Level 1 heparin protocol, including wound and epidural hematomas. Furthermore, the survey encompassed the experience of more than 2400 patients and 22 experienced surgeons. Of special interest in this series was the fact that in the six patients who suffered major complications after Level 1 heparinization, none of them received heparin before postoperative Day 4. It is also noteworthy that in a single case report, development of spinal epidural hematoma occurred 15 days postoperatively after Level 1 heparinization.17

The fact that so many of the patients in the aforementioned study1 suffered complications on a relatively delayed basis (> 4 days postoperatively) contrasts with findings in the recent neurosurgical literature regarding postoperative administration of Level 1 heparin in patients undergoing craniotomy. Specifically, in recent studies authors have suggested that Level 1 heparinization administered more than 48 hours after craniotomy carries an acceptable statistical risk in terms of the perioperative development of an intracranial hematoma.14 It seems pos-
sible that the extensive bone removal involved in spinal fusion and/or decompression leaves a wider potential bone surface area for rebleeding than does craniotomy. This could predispose patients undergoing spinal surgery to delayed postoperative hematoma.

**CONCLUSIONS**

After careful review of the literature, we have concluded that there is no reasonable peer-reviewed standard of evidence on which to base the use of a Level 1 heparin protocol (or its equivalent) in patients undergoing spinal surgery in whom PE arises postoperatively. On a practical level, however, we believe that the use of a vena cava filter alone may be the most judicious choice postoperatively for patients who have undergone spinal surgery. As Cain, et al., have previously discussed, the 1999 study by Becker, et al., of 2019 patients with PE has demonstrated successful prevention of repeated PE in 98.3% of patients treated with vena cava filters alone. Given the high success rate of treatment with inferior vena cava filters, the exposure of patients undergoing spinal surgery to the potentially high risk associated with postoperative heparinization may be unwarranted.

There is a need for at least a retrospective case-controlled study of the use of inferior vena cava filters compared with Level 1 anticoagulation protocols postoperatively in patients undergoing spinal surgery. To our knowledge, no such study exists, and it may be a reasonable option for quantifying the risk posed by heparinization compared with inferior vena cava filters in this group of patients.

The use of Level 1 heparin therapy in patients who experience myocardial ischemia or infarction is another matter entirely. Obviously, the alternate treatment for such cases does not include an inferior vena cava filter. In cases of myocardial insufficiency or ischemia in which there is no alternative treatment other than anticoagulation therapy, close neurological monitoring (that is, frequent neurological examinations) in conjunction with heparinization therapy seems prudent, because early (< 12 hours after onset of symptoms) evacuation of postoperative spinal hematomas can improve outcome.12

**References**


Address reprint requests to: Bryan Barnes, M.D., Wake Forest University, Department of Neurosurgery, Medical Center Boulevard, Winston-Salem, North Carolina 27157-1029. email: bbarnes@wfubmc.edu.