



FACTFINDERS FOR PATIENT SAFETY

FALLS AND INTERVENTIONAL PAIN PROCEDURES

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MYTH: Patients are at no greater risk of falling after an uncomplicated interventional spine or joint procedure than before the procedure.

FACT: Although the incidence of falls following interventional spine and joint procedures has not been established, several procedure-related factors may contribute to fall risk, including sedation, transient sensory or motor blockade from local anesthetic exposure, and vasovagal reactions.

Sedation-related Instability and Falls

Procedural sedation and analgesia may be administered during interventional spine procedures to improve procedural tolerance. Ideally, this is achieved with minimal rather than moderate or deep sedation for most spinal procedures [1].

Benzodiazepines are frequently utilized for minimal procedural sedation. Commonly prescribed benzodiazepines for procedural anxiolysis include lorazepam, alprazolam, and diazepam. After oral administration, peak concentration is reached at 1–2 hours for alprazolam, 1–6 hours for lorazepam, and 1–2 hours for diazepam [2]. Half-lives for alprazolam, lorazepam, and diazepam are 6–27 hours, 10–20 hours, and 10–80 hours, respectively [2]. For moderate sedation, intravenous (IV) midazolam is commonly utilized for its ability to penetrate the blood-brain barrier quickly and result in both amnesia and anxiolysis [3]. Onset of action occurs in 1–2 minutes, with peak effect in 3–5 minutes and a duration of action of 30–60 minutes [4]. Benzodiazepines are often used in conjunction with analgesics such as IV fentanyl, a synthetic opioid. IV fentanyl takes effect within 2–3 minutes and typically lasts 30–60 minutes [3]. Propofol achieves deep sedation within 40 seconds and lasts 6 minutes [5].

These medications are central nervous system-acting agents and are considered fall-risk-increasing drugs [6]. Older individuals may demonstrate increased sensitivity to sedatives, including their adverse effects such as falls [7–9]. Additionally, reduced metabolic clearance of benzodiazepines in the elderly may prolong their clinical effects [3]. Prolonged effects may also occur in patients with hepatic impairment due to reduced drug metabolism and in patients with renal impairment when active metabolites accumulate, as may occur with certain benzodiazepines (e.g., diazepam) [2,3]. Similarly, patients older than 55 may be more sensitive to propofol, and lower doses are recommended [5].

Evidence supports the association between benzodiazepine use and increased fall risk. In a multivariate analysis of 7,908 hospitalized patients, very short and short half-life benzodiazepine use was significantly associated with falls, with an odds ratio of 1.8–1.9, respectively [10]. In a case-control study of 1,222 patients 65 years of age or older with hip fracture and 4,888 matched controls, all benzodiazepine doses greater than or equal to 3 mg/day in diazepam equivalents significantly increased the risk of hip fracture by 50% [11]. Furthermore, a meta-analysis that examined the association of medication and fall risk included 22 original studies and a total of 79,081 patients over age 60 and demonstrated a significantly increased risk of falls with benzodiazepines, with an odds ratio of 1.57 [12]. Concomitant use of other centrally acting medications may potentiate cognitive side effects and increase the risk of post-procedural falls. Although these studies were performed in non-procedural settings, it is important to recognize that benzodiazepines utilized for procedural sedation could lead to increased fall risk through similar mechanisms. Strategies to mitigate this risk have also been examined in procedural settings; in an outpatient endoscopy suite, pre-procedural fall-risk education and planned post-sedation assistance were associated with fewer falls [13].

Transient Nerve Block and Local Anesthetics

Transient gait dysfunction and falls following spine or joint injections may result from sensory blockade, motor blockade, or both. Sensory blockade may impair proprioception and balance, particularly when the sensory distribution is broader than anticipated or when local anesthetic spreads to adjacent, non-targeted nerves. Motor blockade may produce weakness, gait instability, and falls. The likelihood and extent of sensory and motor blockade depend on the pharmacologic properties of the local anesthetic, injectate volume and concentration, needle location, and the anatomy and distribution of the targeted and adjacent nerves.

Lidocaine has a more rapid onset but shorter duration of action compared with bupivacaine and ropivacaine, which have slower onsets and longer durations of action [14,15]. Local anesthetics differ in their sensory and motor effects, with lipid solubility playing a key role in determining potency, depth of nerve penetration, and sensory-motor selectivity. More lipophilic local anesthetics, such as bupivacaine, are more likely to produce clinically significant motor blockade than ropivacaine due to enhanced penetration into large myelinated fibers, whereas ropivacaine generally provides similar sensory blockade with less motor blockade [14–17].

Although data from regional anesthesia describe the onset and duration of motor effects of various local anesthetics used in regional blockade [14,15,17], these findings may not directly predict the timing or severity of motor impairment following small-volume injections near spinal nerve roots. Motor weakness has been reported infrequently following spinal injections (~1%) and is typically identified by patient report or immediate assessment [18]. When local anesthetic is injected near a neural structure, consideration should therefore be given to post-procedure observation and assessment of motor function before ambulation, recognizing that the optimal duration of observation and its effect on fall risk have not been established. Patients should be instructed to bear weight slowly following the procedure, with staff immediately available to assist with ambulation and a wheelchair readily available when needed.

Dural puncture is a known risk when performing epidural steroid injections, with reported incidences of 0.2% and 0.04% for interlaminar and transforaminal approaches, respectively [19]. Dose-response studies of intrathecal local anesthetics show that motor block can occur at relatively low doses, with an ED50 (median effective dose, defined as the dose at which 50% of patients exhibit any measurable reduction in motor function) of approximately 13.7 mg for lidocaine (about 0.7 mL of 2% lidocaine) and 3.4 mg for bupivacaine (about 0.7 mL

of 0.5% bupivacaine), making bupivacaine about four times more potent per milligram than lidocaine [20]. These amounts are consistent with volumes physicians may inject during epidural steroid injections (1.37 mL of 1% lidocaine, 1.36 mL of 0.25% bupivacaine, and 0.68 mL of 0.5% bupivacaine). With inadvertent intrathecal injection, these amounts may be sufficient to produce motor effects in some patients [20].

Furthermore, because local anesthetics may impair sensory and proprioceptive function without producing clinically apparent motor weakness, gait instability can occur even with preserved motor strength. In addition, local anesthetic injection during cervical medial branch procedures may induce ataxia through disruption of cervical proprioceptive signaling [21], thereby increasing the risk of falls. Patients with pre-existing neurologic disease may be particularly susceptible to local anesthetic effects, as compromised neural tissue may show increased sensitivity to sodium-channel blockade and further impaired neural conduction [22,23]. Consequently, sensory or motor blockade may be more pronounced or prolonged in patients with underlying neurologic dysfunction, potentially increasing the risk of post-procedural gait instability and falls.

Sensory or motor block may also occur during non-epidural injections as a complication of unintended spread of local anesthetic to adjacent neural structures. For example, imaging studies show that injectate from sacroiliac joint injections may extend to the posterior S1 neuroforamen in approximately 15% of cases, even when as little as 1 mL of contrast is injected; however, none of the patients in the retrospective review complained of motor block, even when 3 mL of 0.25% bupivacaine was injected [24]. Contrast extravasation beyond the sacroiliac joint capsule has been demonstrated along the dorsal sacral foramina, the L5 epidural sheath, and the lumbosacral plexus [25]. Additionally, extra-articular contrast extravasation has been described during hip arthrography [26], providing a theoretical mechanism by which intra-articular local anesthetic could extend beyond the joint capsule and contribute to transient femoral nerve blockade. However, these studies did not report motor blockade or falls [24-26], and the clinical significance of this unintended injectate spread with respect to fall risk remains uncertain.

Vasovagal Reaction and Fall Risk

A vasovagal reaction (VVR) can be triggered by pain, psychological distress, or another noxious stimulus. VVR can cause a sudden drop in heart rate, blood pressure, or vascular tone. Patients may experience symptoms such as nausea, diaphoresis, weakness, visual disturbances, and, in severe cases, vasovagal syncope. Syncope-related injury is not uncommon, and syncope occurring while standing has been independently associated with increased injury risk [27,28]. Although these data [27,28] are not specific to interventional spine or joint procedures, they underscore the importance of recognizing VVRs following these procedures.

Several patient factors can contribute to the risk of VVR. A study of more than 8,000 patients showed that patient factors such as male sex, age (<65 years old), and lower preprocedural pain scores (<5/10) were associated with an increased likelihood of VVR [29,30]. A history of previous VVR increases the likelihood of a VVR during subsequent procedures [31]. Another study comparing cervical epidural steroid injections to lumbar epidural steroid injections reported a higher likelihood of VVR reaction in the cervical epidural steroid injection group [32]. Additionally, it is suggested that pre-procedure anxiety could be a potential contributing factor to VVR risk [33]. Although direct evidence is lacking, minimizing unnecessary patient discomfort and time on the procedure table is a reasonable strategy to reduce procedural anxiety, a recognized trigger of VVRs.

For interventional spine and joint procedures that do not require sedation, there is no universal protocol to limit VVRs and VVR-related falls. At a minimum, patients who experience VVRs should be monitored and not allowed to stand or ambulate independently until symptoms completely resolve.

Conclusion/Recommendations

- The incidence of falls following interventional spine and joint procedures remains unknown. Potential contributors include sedation-related impairment, transient sensory or motor blockade due to local anesthetic exposure, and VVRs.
 - When sedation is utilized, clinicians should recognize that residual sedative effects may contribute to post-procedural gait instability, particularly in older patients and those with hepatic or renal impairment.
 - When local anesthetic is administered near neural structures, patients should be assessed for clinically significant sensory or motor impairment before ambulating. Patients should be instructed to bear weight gradually, with staff nearby and a wheelchair readily available if needed.
 - Patients who experience a VVR should not be discharged until symptoms have completely resolved.
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Use of Artificial Intelligence: Generative artificial intelligence was used to assist with literature organization, manuscript drafting, editing, formatting, and reference management. All scientific content, interpretations, citations, and conclusions were independently reviewed and verified by the authors against the source materials. The authors take full responsibility for the content of this manuscript.

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