Anesthesia-related periodic involuntary movement in an obstetrical patient for cesarean section under epidural anesthesia: A case report

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Anesthesia-related periodic involuntary movements in a patient with regional anesthesia or etomidate anesthesia can be alarming.

This case report describes anesthesia-related periodic involuntary movement in a patient undergoing cesarean section with epidural anesthesia. The anesthesia-related periodic involuntary movement occurred postoperatively in the recovery room. The discussion includes a brief review of spinal cord anatomy and physiology, as well as a review of the literature describing similar events.

Key words: Myoclonus, nocturnal myoclonus, periodic movement in sleep.

Case summary
A repeat cesarean section was scheduled for a gravida V, para I patient. Epidural anesthesia was easily performed at L3-4 using a bicarbonated solution of 2% lidocaine with epinephrine 1:200,000, 20 mL. A T-4 level was obtained and the surgery proceeded uneventfully. In the recovery room, the patient was asked to move her toes. The patient could move both feet although she had no sensation and stated that she was not aware of the movement.

As the block wore off, both legs started moving involuntarily, and soon even her upper body seemed out of control, with her arms and head moving also. The patient remained calm throughout the experience but did find it distressing since she felt she could not control her movements.

She received 2 mg of midazolam titrated over 5 minutes and the symptoms ceased. There were no sequelae.

As early as 1685, Willis described "restless legs syndrome": "Wherefore to some, on being abed, they betake themselves to sleep, presently in the arms and legs, leaping and contractions of the tendons, and so great a restlessness and tossing of their members ensue that the diseased are no more able to sleep than if they were in the place of great torture."1

Review of the anatomy and physiology of the spinal cord
Each half of the spinal cord is divided into three longitudinal sections that run the length of the cord: the dorsal, lateral, and ventral columns. Within each of these divisions are distinct localized bands of nerve fibers, called tract. A fiber tract is a bundle of fibers which have the same origin, termination, and function. The tracts may be ascending, descending, or associative.

Ascending tracts bring sensory information to the central nervous system. Incoming information may include pain, temperature, crude and fine touch, conscious proprioception, vibration, itching, and tickling.

Nerve fibers from various parts of the brain to the motor neurons of the brainstem and spinal cord
are called descending tracts. The lateral and ventral corticospinal tracts represent the voluntary motor pathway in the spinal cord.

 Associative tracts are short ascending or descending tracts. Those associative tracts that connect between a few segments of the spinal cord are called intersegmental tracts.

 The spinal cord tracts are named to denote the origin and termination of the fibers. Origin designates the location of the cell bodies of the tract. Termination refers to the point at which the axon forming the tract ends. One may determine whether a tract is an ascending sensory or a descending motor tract by analyzing the name. For example, the rubrospinal tract is a descending motor tract with its cell bodies in the red nucleus of the midbrain and its axon termination in the spinal cord.

  ■ Descending pathways: The two major systems of motor pathways are classified as pyramidal and extrapyramidal. Pyramidal tracts are those whose fibers come together in the medulla to form the pyramid, hence the name. The pyramidal tracts consist of the lateral and ventral corticospinal tracts.

  ■ Voluntary motor pathway: The lateral and ventral corticospinal tracts are the major voluntary motor tracts of the spinal cord. The function of these tracts is controlling skilled movement of the extremities. Lesions of the corticospinal tracts produce Babinski’s sign and loss of performance of skilled voluntary movement.

  ■ Extrapyramidal system and basal ganglia: The extrapyramidal system includes all motor fibers not passing through the pyramids. It is composed of the basal ganglia and their circuits, cortical areas that project to the basal ganglia, cerebellar areas that project to the basal ganglia, parts of the reticular formation with connection to the basal ganglia, and the cerebral cortex and thalamic nuclei that connect the basal ganglia and the reticular formation.

 The primary function of the extrapyramidal system is to provide coarse control for voluntary muscles where the lateral and ventral corticospinal tracts are the major voluntary motor tracts, the extrapyramidal tracts are mostly involuntary. The system works as a unit providing for integration on three levels: cortical, striatal, and tegmental. The major effect is inhibition. Posture and performance of well-coordinated movements result from an integration of information received from both the cerebral cortex and extrapyramidal systems. The cortex initiates movement, and the extrapyramidal system provides the facilitation or inhibition needed for production of purposeful, coordinated, controlled movements. Disruption of the extrapyramidal influence results in abnormal uncontrolled movements.

 **Case reports of periodic involuntary movement with epidural, spinal, or etomidate anesthesia**

 Watanabe et al described alternating periodic movements of the lower legs in a elderly man during spinal anesthesia and during sleep (nocturnal myoclonus). The authors stated, “At the present time there is no clear understanding of the mechanism of sleep-related myoclonus, nor of the unusual response that we observed during spinal anesthesia. Physiologic changes commonly seen both during sleep and at this stage of spinal anesthesia are believed to be involved in inducing this type of movement. These changes include inhibition of an inhibitory pathway from the cerebral cortex. During sleep, the extrapyramidal tract is depressed, whereas spinal anesthesia blocks more intensively the inhibitory neurons in the postero-lateral column. This results in isolation of spinal interneurons that, being free from inhibition, become excited. In our patient, myoclonic movement initiated on the nondependent side of the lateral position on two separate occasions. This also suggests an uneven distribution of local anesthetic drug in the spinal cord.”

 Another article by Watanabe et al describes similar periodic leg movements occurring in an elderly man on two occasions, once during epidural anesthesia for a surgical procedure, then again during a spinal anesthetic performed with permission to elicit the periodic leg movements.

 In the same article, Watanabe describes two cases of anesthesia-related periodic leg movement occurring in relation to the level of anesthesia. In one case it occurred when the sensory level regressed to T-11. In the other case it occurred when the sensory level regressed to L-2. He describes another case in which there was a spinal cord lesion at this level, eliciting mild signs of corticospinal tract dysfunction.

 Watanabe describes the Babinski response during wakefulness as a reliable index of pyramidal tract disease. It is a spinal polysynaptic reflex that is normally inhibited by the descending impulses in the spinal tract. A study by Smith proposes a relationship between anesthesia-related periodic leg movement and the Babinski-like response due to changes in the pyramidal tracts during nonrapid eye movement (NREM) sleep.

 The action of local anesthetics and intravenous etomidate on the spinal cord and/or supraspinal system may lead to cortical disinhibition and pyramidal tract inhibition. The action of re-
ceptor occupation of opiate drugs may cause myoclonus. The changes of aging in the spinal cord may predispose the occurrence of anesthesia-related periodic leg movement. He quotes a study by Ancoli-Israel suggesting that the reduction of blood flow in the legs under regional anesthesia may trigger the periodic leg movement.¹

Then Watanabe asks, why does anesthesia-related periodic leg movement occur even without a history of sleep-related periodic leg movement? And, why isn't anesthesia-related periodic leg movement seen in other cases of regional anesthesia with these predisposing factors? He answers that any leg movement under regional anesthesia of spinal or epidural anesthesia is considered inadequate anesthesia and the progression to general anesthesia is used. Also, general anesthesia is used in conjunction with midthoracic epidural anesthesia. Therefore, some events of anesthesia-related periodic leg movement will go unnoticed in the busy clinical situation.²

Barauh states, “Spinal myoclonus, also called focal or segmental myoclonus, is due to a tumor, infection, injury, or degenerative process of the spinal cord.”³ He describes a case of a 45-year-old man who had a left midthigh amputation after a motor vehicle accident. One year later he had occasional episodes of jerky movements of the amputation stump. On examination he did exhibit brisk reflexes in the right lower extremity. It was concluded that he had a lesion of the spinal cord between T-11 and L-2 on the left side of the cord. The lesion probably came from his previous trauma. He responded to treatment with clonazepam.⁴

Laughlin et al present a case report of prolonged myoclonus after etomidate anesthesia. The patient was an elderly male undergoing an L4-5 discectomy and laminectomy. For anesthesia, he received etomidate, fentanyl, succinylcholine, and pancuronium maintained with an etomidate infusion.

“When arrival in the recovery room, 30 minutes after termination of the etomidate infusion, the patient remained unresponsive to command but began having purposeless twitches in the face and all four extremities, the latter soon progressing to myoclonic movements requiring four point restraints used for the patient’s protection.” His laboratory studies and temperature were normal. “The myoclonic movements continued, but after 1 hour, the patient exhibited some purposeful movement and could converse minimally but did not obey commands.”

“Seventy-five minutes after arrival in the recovery room myoclonic movements began to decline in intensity and for the first time the patient could follow simple commands such as moving his toes and hands. The myoclonic activity ceased 2.5 hours after discontinuation of the etomidate infusion.”

“When interviewed 10 hours after discharge from the recovery room the patient had no recall of post-operative events. Physical examination, including a neurologic examination, was unremarkable.”⁶

“Myoclonus has been reported to occur during induction of anesthesia with etomidate. In the awake state, the involuntary movements of myoclonus, which are produced in the extrapyramidal motor system, are normally prevented by inhibition from the cortex. During anesthesia, etomidate is believed to suppress these inhibiting cortical influences, thereby allowing the myoclonus. This myoclonus can be suppressed by treatment with diazepam or fentanyl, the main sites of actions of which are in subcortical structures including the extrapyramidal motor system.”⁶

“When etomidate is used only for the induction of anesthesia, it is assumed that rapid redistribution and enough metabolism of etomidate takes place before the end of the operation that anesthesia of the cortex by etomidate is no longer present so that any myoclonus can be inhibited by the cortex. When etomidate is administered as a continuous infusion, other drugs given concurrently may inhibit the extrapyramidal system to prevent myoclonus. If etomidate is the primary anesthetic, it is postulated that myoclonus could occur when awakening from anesthesia if the extrapyramidal system emerged more quickly than the cortex that inhibits it. The time sequence of myoclonus and arousal observed in our patient supports this premise. On termination of the etomidate infusion no myoclonus was observed, indicating anesthesia of both the extrapyramidal system and the cortex. With time, the myoclonus appeared indicating emergence of the extrapyramidal system but residual anesthesia of the cortex. When the patient was fully awake, the myoclonus disappeared.”⁶

Causal effects

The following is a summary of possible causal effects of anesthesia-related periodic involuntary movement or myoclonus:

1. People with a history of periodic leg movements during NREM sleep may have a higher incidence of involuntary movement.

2. Certain levels of spinal anesthesia may free normal inhibitory pathways and cause involuntary movement.

3. The level of spinal anesthesia in regard to anterior-posterior distribution may free some inhibitory tracts.
4. Nocturnal myoclonus is more prevalent in the elderly population.
5. Age-related blood flow changes in the spinal cord may be a factor in involuntary movement.
6. The regional anesthetic level between T-11 to L-2 seems to be the level for the occurrence of involuntary movement.
7. Spinal cord lesions may cause myoclonus.
8. Etomidate anesthesia may cause myoclonus.

Discussion

In the operating room setting with an adequate regional anesthetic, if the patient moves, the sequence is to proceed to general anesthesia. Because of this sequence, some events of anesthesia-related periodic involuntary movement may be unnoticed.

In the recovery room when anesthesia-related periodic involuntary movement occurs in unconscious patients, they may be treated with diazepam or midazolam and soft restraints for their protection. Conscious patients may be treated with diazepam or midazolam and reassurance that the condition is a self-limiting phase of their recovery and without sequelae.

Our patient, who experienced the episode of anesthesia-related periodic involuntary movement in the recovery room, had movements in all four extremities but did not perceive the movements to be alarming. She was treated with midazolam, the involuntary movement was suppressed, and by the time that the midazolam had worn off, so had the epidural anesthetic. The next day she was functioning normally and taking care of her baby.

REFERENCES


AUTHORS

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