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Lidocaine-induced Spinal Block Can Relieve Central Poststroke Pain: Role of the Block in Chronic Pain Diagnosis

PETER A. CRISOLOGO, M.D.,* BLAKE NEAL, M.D.,* RON BROWN, M.D.,*
JUDY MCDANAL, M.D.,† IGOR KISSIN, M.D., PH.D.‡

The use of differential spinal block for diagnostic purposes in chronic pain syndromes was suggested by Arrowood and Sarnoff.[1] The main objective of their suggestion was to determine "whether the pain is of local origin or a projection from the sensory cortex." One of the most important conclusions based on the results of the block is as follows: if there is no pain relief despite complete sensory loss over the affected area, the pain is central or psychogenic in origin.[2] We present here observations that contradict this conclusion.

REPORT OF THREE CASES

With the diagnosis of central poststroke pain, we used the approach based on the studies by Leijon *et al.*[3] and Boivie *et al.*[4] All three patients had the following major diagnostic criteria: appearance of pain after the stroke, hemidistributed pain, abnormal temperature sensibility, and presence of dysesthesia. No patient had evidence of peripheral neuropathy or radiculopathy.

Intrathecal injection of lidocaine was performed with the patient in the sitting position. Through a suitable interspace (L2–3 or L3–4) under aseptic conditions, the subarachnoid space was entered with a 22-G needle, with the needle bevel parallel to the direction of the dural fibers. A 28-G catheter (Durr-Fillauer Medical, Inc.) was placed for subarachnoid injections. First, normal saline (2 ml) was injected; next, 2 ml 0.5% lidocaine in 7.5% dextrose was administered in 20 min; and, finally, 2 ml 2% lidocaine was injected (also in 7.5% dextrose and 20 min after the previous injection). The effect of the injections on pain intensity was assessed with the use of a visual analog scale.[5] The scale consisted of a 100-mm horizontal line, at the left end of which was "no pain at all," and at the right, "as severe as pain could be"). Pain intensity was determined separately over the arm and the leg before injections and 20 min after each injection. Pain intensity also was assessed, 1, 2, and 5 h after the last injection.

Case 1. The patient was a 70-yr-old man with the complaint of a constant burning pain over the left side of his body. He also had some unpleasant sensations on the right side of his body. The left-sided pain appeared 3 yr previously, a few weeks after a thalamic infarct. Six months after the first stroke, a second stroke, on the opposite side, was followed by the development of mild burning sensation on the right side. During a 3-yr period, various pain treatments, including implantation of a deep brain stimulator, were tried, but with no significant success. Neurologic examination showed no major impairment. Deep tendon reflexes were decreased but symmetrical. Sensations to pin prick and cotton wool were decreased over the left side of the body, and allodynia was present over the left arm. Sensibility to temperature was decreased on the left side. The patient had tingling dysesthesia on both sides of the body. The diagnosis was postthalamic stroke pain.

Intrathecal injections of saline and then lidocaine 0.5% (2 ml) did not cause any pain relief despite an increase in the skin temperature by 3° C and loss of the feeling of sharpness in the pin prick test after lidocaine. The injection of lidocaine 2% (2 ml) resulted in a complete sensory block (up to the T6 level) as well as motor block of the lower extremities and simultaneous complete pain relief in the left leg (100-mm visual analog scale levels were 35 before and 0 mm after the block). The intensity of pain in the left arm (35 mm on the visual analog scale) did not change. Five hours after the injection, the pain in the leg returned to the preinjection level.

Case 2. The patient was a 60-yr-old man with the complaint of aching pain in the left extremities. The patient had sustained two right cerebral hemispheric infarcts; the first had occurred 5 yr earlier and the second 3 yr earlier. He had been well before the second stroke, which left him with hemiplegia and pain in the arm and the leg. The hemiplegia gradually became less severe, but the pain worsened. Neurologic examination showed that the patient was alert and oriented and had normal speech. He had a left homonymous hemianopia and a left seventh cranial nerve motor deficit. Mild left hemiplegia was most pronounced in the arm; the leg had only distal weakness. The patient was able to ambulate with the use of a cane. His deep tendon reflexes were 3+ on the left extremities and 2+ on the right extremities. Babinski's test was positive on the left. Sensory examination revealed decreased sensibility to temperature and pin prick in the left leg and arm. Dysesthesia could be produced in the left foot. The diagnosis was central pain following right hemispheric cortical infarct.

Intrathecal injection of saline or lidocaine 0.5% did not change the pain intensity. An additional injection of lidocaine 2% resulted in complete pain relief from the initial level of 60 mm on the 100-mm visual analog scale. The patient completely lost the feeling of sharpness in the pin prick test and partially lost the feeling of touch. Motor block was very pronounced: the patient was unable to flex the knee, but some flexion of the foot was still possible. The patient did not feel pain for 1 h, after which the intensity of pain gradually increased and returned in 5 h to its initial level.

Case 3. The patient was a 78-yr-old man with the complaint of constant aching pain in his left leg and arm. The pain had appeared 6 yr earlier, several weeks after a stroke. For the past 3 yr the pain had increased gradually. The patient was alert and oriented but had mild word-finding difficulty. There was flattening of the left facial fold. The strength of the extremities on the left side was decreased and the tone of the left arm was increased. Deep tendon reflexes were increased on

* Resident in Anesthesiology.
† Associate Professor of Anesthesiology.
‡ Professor of Anesthesiology.

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Address reprint requests to Dr. Kissin: Department of Anesthesiology, University of Alabama School of Medicine, University of Alabama at Birmingham, UAB Station, Birmingham, Alabama 35294.

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the left side. The patient was able to ambulate safely with a walker. Temperature sensibility and sensitivity to pin prick were decreased on the left side. Tactile stimulation evoked dysesthesia in the left extremities. The diagnosis was postthalamic stroke pain.

Intrathecal injections of saline, lidocaine 0.5%, and lidocaine 2% did not provide any pain relief. The pain intensity in the leg remained at the preinjection level (80 mm on the 100-mm visual analog scale) despite complete sensory loss (up to the T8 level) and complete motor block.

Discussion

The occurrence of the pain after a stroke in our patients does not constitute the only evidence of its central origin. The pain distribution, abnormal temperature sensibility, and presence of dysesthesia, which constitute three other major diagnostic criteria for the central poststroke pain,[3,4] also were present. In addition, none of the patients had any evidence of peripheral neuropathy or radiculopathy. These observations together led us to believe that in each of three cases the pain was of central origin.

Complete pain relief in two of the three patients diagnosed with central poststroke pain contradicts the notion that spinal block would not relieve central pain. In one of the patients (case 3), sensory block did not provide any pain relief. This patient's pain was similar to that of the two others. However, his pain has been present for a longer duration than that of the others (6 *vs.* 3 yr, respectively) and was of greater intensity (80 *vs.* 35 and 60 mm in cases 1 and 2, respectively, on the visual analog scale). Additionally, he had a moderately developed paresis, at a time when the first patient did not have any paresis and the second patient at the time of the blockade demonstrated only slight paresis. These differences are not sufficient to explain the difference in outcomes after the blockade.

It has been reported that intravenous administration of lidocaine 3 $mg \cdot kg^{-1}$ can provide complete pain relief in various chronic pain syndromes, including central pain (thalamic and phantom pain syndromes).[6] Therefore, the action of lidocaine after its absorption from the site of injection should always be considered as a possible mechanism of action. However, in our cases the total dose of lidocaine was much lower than 3 $mg \cdot kg^{-1}$, and more importantly, despite complete pain relief in the leg, there was no change of the pain intensity in the arm.

Ours is not the first observation that spinal block can relieve central pain. Most curiously, Arrowood and Sarnoff,[1] who introduced spinal block to determine whether pain is of local origin, observed phantom pain in two of seven patients in their series. In both cases, spinal block provided complete relief of phantom pain, which presently is considered to be central pain.[7] In 1960, Kibler and Nathan[8] presented a series of cases with spinal cord or spinal root lesions in which local anesthetic blockade of various peripheral nerves produced pain relief. They suggested that the pain was due to impulses arriving from the periphery and altered by the central lesion. We previously reported a series of cases in which relief of sciatic radicular pain was induced by sciatic nerve blocks distal to the lesion.[9]

Relief of the central pain by spinal block can be explained on the basis of recent experimental advances in the pathophysiology of pain. It has been reported that nerve injury can alter the neurons' receptive field properties and change sensory processing.[10] These changes can result in altered perception of peripheral stimuli, including the feeling of pain in response to innocuous stimuli.[11] A patient with a thalamic injury may have an area in the neuraxis that processes peripheral sensory input in a way that contributes to the pain. The spinal blockade of the normal peripheral input to the damaged central neuraxis could eliminate the mechanism responsible for pain maintenance.

We have presented three successive cases of central poststroke pain when complete sensory block was induced by subarachnoid injection of lidocaine. In two of the three patients, the central pain was completely relieved. We believe that the value of spinal block in the diagnosis of central pain is questionable.

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