AJNR

# Spinal myoclonus induced by contrast material: an alternative explanation.

S N Heyman, Z Greenfeld, M Behar, P Mogle and M Brezis

*AJNR Am J Neuroradiol* 1989, 10 (3) 653
http://www.ajnr.org/content/10/3/653.citation

## Spinal Myoclonus Induced by Contrast Material: An Alternative Explanation

Tonic and clonic muscle spasm of the lower extremities and trunk is a well-known complication of myelography [1, 2] and accidental subdural injection of contrast material [3]. Occasionally, myoclonus also appears after aortography. It is postulated that some of the contrast medium reaches the spinal column through intercostal, lumbar, and lumbosacral segmental arteries. The risk is higher in distal aortic occlusion [4] and when the procedure takes place with the patient supine [5]. Spinal myoclonus associated with aortography may result from a direct toxic excitatory effect of the contrast agent [6], as happens during myelography, or it may be due to ischemic myelopathy, as sometimes occurs in spinal cord angiography during placement of the catheter, even before the injection of contrast material [7].

Recently, we introduced a new in-vivo model of contrast nephropathy [8], in which rats were given Angio-Conray (80% sodium iothalamate) at a dosage of 6 ml/kg through a polyethylene catheter placed in the femoral artery with the tip of the catheter directed proximally. The femoral artery was ligated on the catheter, so all the contrast material was injected against flow. During the injection, all animals developed myoclonus of the hindlimbs, trunk, and tail that was more prominent in the cannulated leg, which concomitantly became cyanotic.

The injection of contrast material was slow (an average amount of 1.5 ml injected over 90–120 sec). Fluoroscopy and films indicated that it did not reach above the aortic bifurcation. Spinal vessels were not filled.

Injection of contrast material was done the same way in rats with epidural anesthesia to evaluate the origin of the myoclonus. Despite full epidural anesthesia obtained by administering lignocain through a long, indwelling epidural catheter, the injection of contrast material resulted in the same myoclonus.

We conclude that the myoclonus produced by the intraarterial femoral injection of sodium iothalamate in the rat does not originate at the spinal level but at a lower level such as peripheral nerve, muscle, or the neuromuscular junction. This phenomenon may result from a direct excitatory effect of the contrast material or from transient ischemia of the lower extremities due to vasospasm and RBC microaggregation caused by the hyperosmolar material. The administration of iopamidol (Omnipaque) and iohexol (Hexabrix) caused minimal spasm only.

In summary, our observations suggest that the term *spinal myoclonus* may be a misnomer, because angiography-induced myoclonus may not result from spinal irritation or ischemia but from a lower neuromuscular toxic reaction.

S. N. Heyman
Z. Greenfeld
M. Behar
P. Mogle
M. Brezis
*Hadassah University Hospital, Mt. Scopus*
*Jerusalem, Israel*

REFERENCES

1. Praestholm J, Lester J. Complications of myelography with Conray meglumin. *Acta Radiol [Diagn] (Stockh)* **1977**;13:860–864
2. Bohutova J, Vojir R, Kolar J, Grepl J. Some unusual complications of myelography and lumbosacral radiculography. *Diagn Imaging Clin Med* **1979**;48:320–325
3. Feingold A, Elam JO, Dobben GD. Severe muscle spasms after visualization of a subarachnoid catheter. *JAMA* **1970**;212:879
4. Tornell G. Spinal cord tolerance to roentgen contrast media particularly during aortography with temporary occlusion of the aorta. *Acta Radiol [Diagn] (Stockh)* **1969**;8:257–283
5. Margolis G. Pathogenesis of contrast media injury: insights provided by neurotoxicity studies. *Invest Radiol* **1970**;5:392–406
6. Junck L, Marshall WH. Neurotoxicity of radiological contrast agents. *Ann Neurol* **1983**;13:469–484
7. Djindjian R. *Angiography of the spinal cord*. Paris: Masson & Cie, **1970**:52–56
8. Heyman SN, Brezis M, Reubinoff CA, et al. Acute renal failure with selective medullary injury in the rat. *J Clin Invest* **1988**;82:401–412