Research letter

Benztropine for acute muscle spasm in the emergency department

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Many patients arrive in the emergency department with acute lumbar and cervical muscle spasm (focal dystonia) that occurs either spontaneously or secondary to nonimpact axial injuries (i.e., lifting and twisting). Current treatment options, such as opioids, NSAIDs and anxiolytics,[1] provide either minimal or no relief or precipitate undesirable side effects. My hypothesis was that benztropine, an anticholinergic medication, would reverse the state of muscle spasm and thus provide good pain relief for these patients.

Entry criteria for this study were age between 16 and 50 years, no prior lumbar or cervical problems such as arthritis or herniated discs, no previous injury in the affected area and onset of muscle spasm symptoms within the 24 hours preceding presentation. I treated 8 patients during 1999 who met these criteria: 5 had acute cervical torticollis, and 3 had paralumbar muscle spasm. All of the patients were asked to rate their pain using a visual analogue scale (0 to 10) on presentation and at discharge.[2] Their range of motion — flexion, extension and bilateral rotation — was determined also on presentation and at discharge.

Most of the patients had nonimpact axial injuries, either from lifting or twisting or from hyperextension or hyperflexion related to motor vehicle accidents or sports-related

Table 1: Characteristics of 8 patients with acute focal dystonia given benztropine intramuscularly

Case	Age, yr	Sex	Presenting problem	Estimated duration of symptoms before presentation, h	Pain at presentation*	ROM at presentation	Benztropine dose, mg IM	Time from treatment to pain relief or discharge, min	Pain at discharge*	ROM at discharge
1	16	M	Torticollis from playing football	6	9/10	None	4	40	2/10	Flexion: 60° Extension: 30° Lateral: 80°
2	25	F	Torticollis from MVA	8	8/10	Flexion: 10° Extension: 10° Lateral: none	4	50	1/10	Normal
3	43	F	Spontaneous torticollis	12	8/10	Virtually none	4	65	3/10	Flexion: 45° Extension: 30° Lateral: 45°
4	28	M	Spontaneous torticollis	10	9/10	None	4	55	2/10	Flexion: 60° Extension: 30° Lateral: 60°
5	23	F	Spontaneous torticollis	4	10/10	None	2	20	5/10	Flexion: 45° Extension: 30° Lateral: 60°
6	44	F	Paralumbar spasm from aerobic exercise	4	7/10	Flexion: 20° Extension: 10° Lateral: 20°	2	60	0/10	Normal
7	33	F	Paralumbar spasm from MVA	5	8/10	Flexion: 10° Extension: 10° Lateral: none	4	55	3/10	Flexion: 60° Extension: 45° Lateral: 45°
8†	42	M	Paralumbar spasm from lifting	12	10/10	Virtually none	4‡	80	5/10	Flexion: 30° Extension: 30° Lateral: 20°

Note: ROM = range of motion, IM = intramuscularly, MVA = motor vehicle accident.
*Patients used a 10-point visual analogue scale to rate their pain.
†This case was a transfer of care.
‡Morphine (two 10-mg doses subcutaneously) was given 3–4 hours before the benztropine treatment but provided no pain relief.

injuries. The others had pain and spasm occurring spontaneously. All experienced intense pain with profound diminution or complete loss of range of motion.

All but 2 of the patients were given 4 mg of benztropine intramuscularly; the other 2 were women who, because of their size, required only 2 mg.[3]

Every patient experienced substantial relief and improved range of motion (Table 1). One patient was a 42-year-old man whose condition did not respond to treatment with an opioid, administered 3 to 4 hours before the benztropine therapy. The only adverse reaction was mild sedation, in 3 patients; this effect may have been due in part to the opioid given before the benztropine in 1 case. None of the patients returned to the hospital during the 72 hours following discharge.

The pathophysiological features of cervical dystonia and paralumbar spasm are complex and unclear. The literature suggests that the sensory input to the spinal cord at the level of injury is altered and secondarily affects the higher brain stem and basal ganglia and thalamus. The basal ganglia transmit these changes to the motor complex, which alters motor neuron activity to the muscle at the same level. The acetylcholine in the basal ganglia is among multiple neurotransmitters (e.g., gamma-aminobutyric acid and dopamine) that affect this process or circuit. Therefore, anticholinergics are thought to work centrally, as opposed to peripherally at the myoneural junction.[4–8]

There have been several prospective controlled studies of anticholinergic drugs for the treatment of dystonia. In one study 4 of 8 patients with a chronic neurological disorder (e.g., idiopathic spasmodic torticollis, writer's cramp and cranial dystonia) had marked relief after receiving benztropine intravenously; however, the sample was very small, and the relief was often transitory.[9] In another study central anticholinergic drugs, primarily trihexyphenidyl, were significantly beneficial in 61% of 23 children and 38% of 52 adults with dystonia of various causes.[10] In a study involving children and adults with severe dystonia, benzhexol was effective at various doses.[11] In a single-blind study involving 13 adults with torticollis given 2 mg of benztropine intravenously, symptoms were alleviated in 6 and worsened in 1.[12] Not all of the cases of dystonia in these studies were necessarily post-traumatic, and none was treated immediately after symptom onset.

Anticholinergic drugs, such as benztropine, may be effective in the treatment of acute cervical torticollis and acute paralumbar spasm in the emergency department setting. A randomized trial is needed to corroborate the findings in this case series.

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