LOCAL ANESTHESIA AND PAIN IV

A693

Title: REFLEX SYMPATHETIC DYSTROPHY IN CHILDREN: FOLLOW-UP OF 70 PATIENTS
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Introduction
Reflex sympathetic dystrophy (RSD), once considered only a disease of adults, is being recognized with increasing frequency in children and adolescents. We report on the characteristics and 1-8 year follow-up of 70 patients who developed RSD prior to the age of 18.

Materials and Methods
We examined the course of the first 72 of the 156 children with RSD seen since development of our pain treatment service. The diagnosis of RSD was made clinically by a formal scale requiring both neuropathic pain descriptors and physical signs of sympathetic hyperactivity. Patients were treated using a multidisciplinary approach: all patients received active physical therapy (PT), a trial of TENS, and cognitive/behavioral pain management techniques (CBPMT); patients with persistent limb dysfunction despite PT, TENS and CBPMT received sympathetic blocks (SB), usually via continuous catheter techniques. Tricyclic antidepressants (TCA) were used adjunctively. Follow-up was performed at clinic visits and by interview by the investigators or a nurse clinician. Pain was measured using a visual analog scale (VAS). Limb function was measured on a 0-10 point scale (0 = no function; 10 = unrestricted use). Data were analyzed using chi square, Wilcoxon rank-sum, and Spearman correlation.

Results
70 of the 72 patients participated in follow-up interviews at an average of 33 months (range 12-96 months) following initiation of treatment. Patients were predominantly female (M: F = 11:59) with an average age of 12.7 years (range 5 - 17) at the time of injury. Lower extremities were involved more frequently than upper (61:9). Pain scores and function scores improved with therapy (Figure, p<0.001). 69 of 70 patients had returned to school or work. Only 31 of the 60 patients participating in sports prior to their injury returned to sports. SB produced the greatest reduction in pain and improvement in function, although among the 33 patients who did not require SB, 27 showed reduction in pain scores by more than 70% with PT, CBPMT, TENS and TCA alone. Age, gender, and time from injury to diagnosis and initiation of therapy did not predict outcome (p>0.05).

Discussion
RSD in children and adolescents produces significant pain and dysfunction. Anxiety, depression and altered family dynamics are common. Goals of therapy should be improvement of function, reduction of pain, return to school and normalization of family dynamics. Treatment regimens should incorporate active PT, CBPMT, and selective use of SB. SB should not be undertaken in isolation.

References

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Title: EPIDURAL TRIAMCINOLONE CAUSES PROLONGED AND SEvere SUPPRESSION OF THE PITUITARY-ADRENAL AXIS.
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Suppression of cortisol after epidural administration of 160 mg of methylprednisolone lasts for at least 3 weeks in man (1). Suppression of the hypothalamic-pituitary-adrenal axis (HPA) lasts for up to 5 weeks in dogs given 2 mg/kg of epidural triamcinolone (2). The degree or duration of suppression of the HPA axis in humans given 80 mg of triamcinolone weekly for 3 weeks, a common regimen in treating lower back pain, has not been reported. We studied the HPA axis in such patients using cosyntropin testing and a new supersensitive two-site immunoradiometric assay (IRMA) for ACTH.

We studied 12 (6F, 6M) subjects (age 43±5 yr) who received 3 epidural injections (ESI) of 80 mg of triamcinolone in 1% lidocaine (7 cc) for low back pain (with informed consent and approval of the Human Research Committee at St. Luke’s Medical Center). Half of the patients received intravenous midazolam (M) before ESI. Cortisol (RIA) and ACTH (IRMA) were measured before and at 15, 30, and 60 min after ESI each week for three weeks. Cosyntropin (250 μg) stimulation tests were performed 1 month after the last injection and, if abnormal, were repeated 2 months later.

Before the first ESI, ACTH and cortisol were not different between groups nor were they affected by M per se (Figure). Plasma ACTH dramatically decreased after ESI in the M group and less so in the no M group. One week later, ACTH was still significantly depressed although higher in the no M group. Subsequent steroid injection further suppressed ACTH in both groups. These changes in ACTH were mirrored by plasma cortisol levels which, by the third treatment, were very low. One third of the patients had significantly decreased ACTH levels and subnormal cortisol responses (20 μg/dL) to cortisone 4-5 weeks after the last injection. All patients had recovered at 3 months after the last injection.

Weekly ESI for three weeks caused suppression of the HPA axis for up to 5 weeks after the last injection. Midazolam premedication accentuated this effect. Exogenous steroid coverage during the vulnerable period for patients undergoing major stress should be considered.

References

Graphs showing cortisol and ACTH levels before and after ESI with and without M.