TITLE: ROLE OF BRACHIAL PLEXUS BLOCK AFTER NEGATIVE RESPONSE FROM STALLATE GANGLION BLOCK FOR RSD

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Reflex sympathetic dystrophy (RSD) or causalgia minor is one of the most baffling pain syndromes. However, its treatment is one of the most rewarding if the syndrome is diagnosed properly. Pain relief following a stellate ganglion block (SGB) is diagnostic for RSD of the upper extremity (UE). However, because of variations in the sympathetic supply to UE, a negative response following SGB should be followed by brachial plexus block before a diagnostic conclusion is reached.[1]

In this study, we present 25 such patients from our routine diagnostic work-up. All patients with suspected clinical diagnoses of RSD of the UE required differential SGB as the initial diagnostic step. Those who failed to respond (after 3 SGB) received brachial plexus block (BPB). BPB techniques included axillary, interscalene, and subclavian perivascular. Mepivacaine 1.5% was the local anesthetic. All patients scored their pain using the visual analog scale at regular intervals. Sympathetic, sensory, and motor block were monitored according to Winnie's model for BPB.[2]

Of the 25 patients, 19 (76%) responded to BPB which was characteristic of sympathetic pain. 2 (8%) had somatic pain after repeated BPB, while 4 (16%) had no response or had "central" pain. Of the 19 patients with "sympathetic pain," 12 had long-term pain relief lasting over six months. 7 had short-term pain relief lasting 3 days to 3 weeks.

Because of the easily accessible location of the sympathetic chain in the neck, diagnosis and treatment of UE pain with a differential block is an anatomical rather than pharmacological venture. The use of a differential block has certain shortcomings which must be taken into consideration. Normally, the sympathetic fibers for the UE pass through stellate ganglion (SG) before entering the brachial plexus. However, in many patients, these sympathetic fibers may totally bypass the upper thoracic and SG and enter the brachial plexus directly (nerve of Kuntz[3]). In other patients, the sympathetic supply may come from as low as T_8. Horner's syndrome is indicative of the blockade of the fibers only to the superior cervical ganglion. Because of the above variability and inadequacy of Horner's syndrome, SGB alone is inadequate in many patients for sympathetic blockade of the UE. Moreover, if the patient has pre-existing Horner's syndrome (3 of our patients), SGB may not be totally reliable and BPB should not be the first choice if a differential blockade is indicated.

References:
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TITLE: FENTANYL PLUS LIDOCAINE VS. LIDOCAINE FOR BIER BLOCK

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Primary afferent nerve fibers have opioid receptors,[1] but their contribution to the analgesic activity of opioids is controversial.[2] Whether the addition of fentanyl to a local anesthetic solution intensifies a Bier block of the arm was, therefore, studied.

After approval from the Institutional Review Board, ten healthy male volunteers (29-45 yrs) were studied in a double-blind and cross-over fashion. Each subject received three different Bier blocks, the interval between blocks being at least 3 days, with: 1. 100 mg lidocaine in 42 ml saline (LIDO) (L), 100 ug fentanyl in 42 ml saline (FENT) (F) and 3. 100 ug fentanyl + 100 mg lidocaine in 42 ml saline (FENT + LIDO). No premediation was given and monitoring included EKG and automatic blood pressure recording.

Perception of sharp and blunt stimuli at six skin areas (corresponding to innervation of median, musculocutaneous, radial and ulnar nerves) and grip strength were recorded at 2 min intervals during the block (20 min) and recovery (20 min). Response to heat (55°C, 5 sec) applied to the palmar skin was assessed at 10 min intervals. ANOVA and Duncan's test were used for statistical analyses.

Results were nearly identical for LIDO and FENT + LIDO, except that with LIDO, grip strength exceeded control 20 min after cuff deflation (Table). Analgesia developed slower with FENT than with LIDO or FENT + LIDO ($P<0.05$). Grip testing in the FENT group induced transient cramping. During all blocks, there was hypersensitivity to heat, even after loss of sharp sensation. After cuff deflation, central nervous system symptoms (lightheadedness, sedation) occurred for a few minutes after LIDO and lasted much longer when fentanyl was used. The recovery of pinprick sensation was slowest in the FENT group ($P<0.05$ compared to LIDO).

	Sharp testing			Grip strength	
	Analg.* (min)	Anesth.* (min)	Recov.* (min)	Block 10 min (%)	Recov. 20 min (%)
LIDO	5.4±2.0	18.1±1.3	6.0±2.5	31±12	115±32
F + L	6.7±2.3	18.8±1.2	7.7±3.0	30±12	93±16
FENT	11.3±4.1	20.0±1.5	11.7±4.3	75±12	90±17

* average (± SD) of all six test spots.

In conclusion, 100 ug of fentanyl did not intensify the lidocaine Bier block. Peripheral opioid receptor binding of fentanyl, at this dosage range is clinically ineffective.

References
1. Science 210;76-78, 1980.
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