

TITLE: THE EFFECTS OF INFRARENAL AORTIC OCCLUSION ON CSF PRESSURE

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Studies have shown that aortic clamping results in a significant increase (50-100%) in cerebral spinal fluid (CSF) pressure¹. This pressure increase will reduce spinal cord perfusion and may be partially responsible for the high incidence of post-operative neurologic complications seen after thoracic aortic surgery. Intra-theatal (IT) catheters and CSF drainage have been employed to reduce CSF pressure and increase spinal cord perfusion pressure with good results. Patients scheduled for abdominal aortic surgery with infrarenal aortic occlusion, whose anesthetic plan included catheter spinal anesthesia (CSA) were evaluated for changes in IT pressure.

After careful chart review and physical exam revealed no contraindications to regional anesthetics, the patients were advised of their anesthetic options. All agreed to CSA as part (7 of 8) or all of their (1 patient) anesthetic. Patients had cannulization of their IT space accomplished in the lateral decubitus position with either a 17 or 18 gauge Touhy needle. The needle was then connected to a 3-way stopcock to which pressure tubing (attached to a transducer) was also attached. The transducer was "zeroed" to the level of the needle and pressure readings recorded. An 18 gauge nylon catheter was then passed through the needle and the patient returned to the supine position. The stopcock apparatus was attached to the IT catheter and was used (open to the patient) only for a brief period during pressure recordings. The IT catheter was then used to establish the desired level of anesthesia with pressure readings taken at frequent intervals. The patients were seen daily until their discharge from the Intensive Care Unit and spinal narcotics were given as needed.

Initial mean, CSF pressure was 16.13 mm Hg (range 10-26 mm Hg). Only one patient showed a significant increase in CSF pressure with local anesthetic injection (1cc volume), 16 to 27mm HG which returned to baseline in less than 5 minutes. Six of the eight patients showed significant increases in CSF pressure immediately after aortic occlusion (mean 35.83, range 26-40) with two patients showing a slight increase. These changes persisted throughout the occlusion period (CSF pressure monitored every 30 minutes) and CSF pressure remained elevated 45 minutes in 3 patients and for 150 minutes in one patient. No patients had new neurologic defects noted post-operatively.

As other studies have shown elevations in CSF pressure with aortic occlusion, the rise seen in our patients was not unexpected. However, the magnitude and duration of pressure increases after the aortic cross-clamp was removed were surprising. This may imply that spinal cord autoregulation (intact to perfusion pressure of 50mm Hg)², may be compromised by episodes of hypotension after the cross-clamp has been removed. Further, epidural anesthesia, often included in the anesthetic regime for aortic surgery may itself elevate CSF pressure. A recent study noted that epidural injections and CSF pressure increases seen with them raised questions concerning the compliance of the epidural space. It was noted that "large" (15 ml) epidural injections placed the epidural space on the "high end" of the compliance curve, with the displacement of the CSF being the "main safety valve limiting extradural pressure".³

If the CSF cannot be displaced due to persistent pressure elevations then epidural injections may further increase CSF pressure and compromise spinal cord perfusion. These preliminary results suggest a subset of patients may experience persistent CSF pressure increase after aortic occlusion with possible anesthetic implications.

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Title: NALBUPHINE IN PATIENTS RECEIVING LOW DOSE INTRATHECAL MORPHINE: A PROSPECTIVE DOUBLE BLIND EVALUATION

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Nalbuphine has been shown to be effective in reversing side effects of intrathecal morphine in rats.¹ Its usefulness following low dose intrathecal morphine in humans has not been established. We assessed the effectiveness of IV nalbuphine in reducing side-effects after low dose intrathecal morphine.

Twenty-two unpremedicated, ASA I-II, adult females undergoing hysterectomy were studied following IRB approval and informed consent. All patients received morphine sulfate 0.25 mg intrathecally before induction, thiopental (3-5 mg/kg) for induction, and nitrous oxide, oxygen and isoflurane for maintenance. Following emergence, patients were given either IV nalbuphine 10 mg (n=11) or saline 1 ml (n=11) according to a blinded, randomized protocol. Each hour for 6 hrs. pain and pruritus were assessed by visual analogue scales. Episodes of nausea and vomiting were recorded. Satisfaction with anesthetic care was evaluated by questionnaire. Psychological tests² consisting of tapping (a mechanical counter pressed as rapidly as possible for two 30-sec periods) and symbol cancellation (target letters crossed out of

lines of letters for 2 min) were administered preoperatively and at 3 and 6 hrs. postoperatively. Tapping is a measure of motor coordination and speed. Symbol cancellation is a cognitive test with a perceptual motor component. Total taps and correct letters crossed out were counted. Unpaired t-test, repeated measures ANOVA and ANOVA supplemented by planned comparisons (for psychological test) were used for statistical analysis. $P < 0.05$ was accepted as significant. Data are Mean \pm SD.

There were no differences in demographics, pain, pruritus, nausea or patient satisfaction with anesthetic care between groups. Results of psychological tests are shown below:

	Nalbuphine-treated Group		
	Baseline	3hr	6hr
Taps	445 \pm 169	335 \pm 128*	350 \pm 136*
Letters	34 \pm 6	23 \pm 6*	28 \pm 7*
	Saline-treated Group		
	Baseline	3hr	6hr
Taps	355 \pm 107	304 \pm 107*	314 \pm 77*
Letters	31 \pm 12	26 \pm 9	28 \pm 10

*=Significant changes, baseline versus 3 and 6 hr

Nalbuphine did not mitigate the side-effects of low dose intrathecal morphine but, instead, caused increased sedation.

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