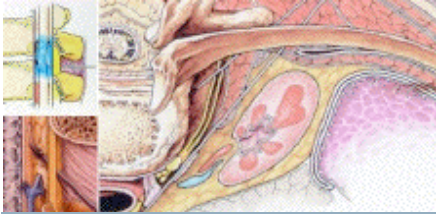


AnesthesiaDotCalm



News You Can Use

Insights Into Clinical Practice

Cardiac Arrest Under Spinal Anesthesia

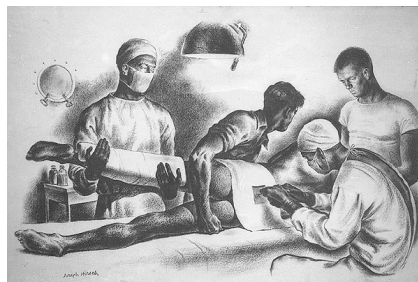
Physiology of Heart Rate

Neural and, to a lesser extent, humoral mechanisms control heart rate. Baroreceptors, chemoreceptors and both divisions of the autonomic nervous system are the major components of the neural control. Sympathetic innervation stimulates the heart (cardioacceleration by enhanced SA node automaticity, faster AV and intraventricular conduction, increased contractility). Parasympathetic innervation inhibits the heart. These influences exist in dynamic equilibrium. Inhibition of preganglionic sympathetic nerve fibres by subarachnoid local anesthetic blockade above T5 disturbs the balance. The cardiovascular effects of spinal anesthesia manifest predominantly as hypotension and bradycardia

REFERENCES:

THYS D. ADVANCES IN CARDIOVASCULAR PHYSIOLOGY. IN: KAPLAN J (ED). CARDIAC ANESTHESIA. TORONTO: WB SAUNDERS; 1999: 217-40

GREENE N, BRULL S. PHYSIOLOGY OF SPINAL ANESTHESIA, 4TH ED. BALTIMORE: WILLIAMS & WILKINS, 1992



Investigators have recently found that in a group of 254 healthy women undergoing cesarean section under spinal anesthesia in which peak sensory levels ranged from T7 to C7 using a fixed dose of subarachnoid bupivacaine and morphine, the incidence of sinus arrhythmia was 30.3%; the incidence of premature beats was 27.2% and the incidence of bradyarrhythmias was 13.8%. More specifically, sinus bradycardia with a heart rate of less than 50 bpm seems to occur anywhere from 2% to 13% of the time. It seems that bradycardia as a result of spinal anesthesia is an ominous sign. A prospective study of 40,640 spinal anesthetics found an incidence of cardiac arrest of 6.4 per 10,000 patients and all of these patients had bradycardia just before the arrest. Similarly a closed claims analysis of complications during spinal anesthesia conducted approximately 10 years earlier also implicated bradycardia as the event leading to cardiac arrest.

The underlying mechanism of severe bradycardia and asystole during

spinal anesthesia remains a matter of debate. It has been proposed that the bradycardia from high spinal anesthesia stems from unopposed normal parasympathetic tone and/or is a physiological response to decreased venous return that may lead to increased parasympathetic tone (a reverse of the Bainbridge reflex). There is also evidence that the forceful myocardial contractions that normally accompany a rapid decrease in left ventricular afterload activate mechanoreceptors in the left ventricle leading to bradycardia (Bezold-Jarisch reflex). Whatever the mechanism behind bradycardia, it has been suggested that the onset of a first degree heart block during spinal anesthesia may be a warning sign of impending complete heart block or asystole.

Since bradycardia can occur as a consequence of spinal anesthesia, it behooves anesthesia practitioners to be proactive in dealing with this premonitory sign. The immediate availability of atropine is a wise precaution and prophylactic anticholinergic administration should be considered for patients at increased risk of bradycardia (high sensory block required, slow pre-anesthetic heart rate, young patients, a prolonged P-R interval) In fact, the use of prophylactic glycopyrrolate (200 µg) in patients undergoing Cesarean section under spinal anesthesia have resulted in greater cardiovascular stability and less intraoperative

nausea and vomiting compared with placebo.

REFERENCES:

SHEN CL, HO YY, HUNG YC, CHEN PL. ARRHYTHMIAS DURING SPINAL ANESTHESIA FOR CESAREAN SECTION. CAN J ANESTH 2000; 47: 393-7

THYS D. ADVANCES IN CARDIOVASCULAR PHYSIOLOGY. IN: KAPLAN J (ED). CARDIAC ANESTHESIA. TORONTO: WB SAUNDERS; 1999: 217-40

GREENE N, BRULL S. PHYSIOLOGY OF SPINAL ANESTHESIA, 4TH ED. BALTIMORE: WILLIAMS & WILKINS, 1992

JUHANI TP, HANNELE H. COMPLICATIONS DURING SPINAL ANESTHESIA FOR CESAREAN DELIVERY: A CLINICAL REPORT OF ONE YEAR'S EXPERIENCE. REG ANESTH 1993; 18: 128-31

CARPENTER RL, CAPLAN RA, BROWN DL, STEPHENSON C, WU R. INCIDENCE AND RISK FACTORS FOR SIDE EFFECTS OF SPINAL ANESTHESIA. ANESTHESIOLOGY 1992; 76: 906-16

AUROY Y, NARCHI P, MESSIAH A, LITT L, ROUVIER B, SAMI K. SERIOUS COMPLICATIONS RELATED TO REGIONAL ANESTHESIA: RESULTS OF A PROSPECTIVE SURVEY IN FRANCE. ANESTHESIOLOGY 1997; 87: 479-86.

CAPLAN RA, WARD RJ, POSNER K, CHENEY FW. UNEXPECTED CARDIAC ARREST DURING SPINAL ANESTHESIA: A CLOSED CLAIMS ANALYSIS OF PREDISPOSING FACTORS. ANESTHESIOLOGY 1988; 68: 5-11.

BARON JF, DECAUX-JACOLOT A, EDOUARD A, BERDEAUX A, SAMI K. INFLUENCE OF VENOUS RETURN ON BAROREFLEX CONTROL OF HEART RATE DURING LUMBAR EPIDURAL ANESTHESIA IN HUMANS. ANESTHESIOLOGY 1986;64: 188-93.

SANDERS JS, FERGUSON DW. PROFOUND SYMPATHOINHIBITION COMPLICATING HYPOVOLEMIA IN HUMANS. ANN INTERN MED 1989; 111: 439-41.

JORDI, E; MARSCH, STEPHAN C. U. ; STREBEL, S. THIRD DEGREE HEART BLOCK AND ASYSTOLE ASSOCIATED WITH SPINAL ANESTHESIA. ANESTHESIOLOGY: VOLUME 89(1), JULY 1998 PP 257-260

CHESTER WL. SPINAL ANESTHESIA, COMPLETE HEART BLOCK, AND THE PRECORDIAL CHEST THUMP: AN UNUSUAL COMPLICATION AND A UNIQUE RESUSCITATION. ANESTHESIOLOGY 1988; 69: 600-2.

LIU S, PAUL GE, CARPENTER RL, STEPHENSON C, WU R. PROLONGED PR INTERVAL IS A RISK FACTOR FOR BRADYCARDIA DURING SPINAL ANESTHESIA. REG ANESTH 1995; 20: 41-4.

TARKKILA P, ISOLA J. A REGRESSION MODEL FOR IDENTIFYING PATIENTS AT HIGH RISK OF HYPOTENSION, BRADYCARDIA AND NAUSEA DURING SPINAL ANESTHESIA. ACTA ANAESTHESIOL SCAND 1992; 36: 554-8.

URE D, JAMES KS, MCNEILL M, BOOTH JV. GLYCOPYRROLATE REDUCES NAUSEA DURING SPINAL ANAESTHESIA FOR CAESAREAN SECTION WITHOUT AFFECTING NEONATAL OUTCOME. BR J ANAESTH 1999; 82: 277-9