

gastrectomy, $n = 4$; pancreatectomy, $n = 4$; or lower abdominal procedure, $n = 5$). Catheter tip location was tested with a solution (3 ml) of local anesthetic and epinephrine 1/200,000. General anesthesia was induced with alfentanil, propofol, and pancuronium and maintained with isoflurane.

At least 1 h after test-dose injection and before surgical stimulation, a dose of 8 $\mu\text{g/kg}$ clonidine was injected epidurally.

The site of catheter puncture, allocation of the patients, hemodynamic data, duration of operative analgesia are summarized in table 1.

In this limited clinical study, the hemodynamic data support the hypothesis of increased cardiovascular depression with upper thoracic injection of epidural clonidine. Although no patient suffered undesirable long term consequences from this dosage regimen and despite the excellent quality of surgical analgesia obtained, we elected to interrupt this clinical trial because of the magnitude of the hypotensive episodes encountered.

MARC DE KOCK, M.D.

Staff Anesthesiologist

Department of Anesthesiology

Université Catholique de Louvain

Cliniques Universitaires Saint Luc

Avenue Hippocrate 10 UCL 10.1821

1200 Bruxelles, Belgique

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Halothane Inhibits Residual Fast Sodium Channels in Human Atrial Muscle

To the Editor:—In isolated human atrial tissue, both isoflurane and halothane may depress electromechanical activity through a reduction of Ca^{2+} influx across the cell membrane.¹ However, halothane also reduces the fast Na^+ current in voltage-clamped rat myocardial cells.² In the present experiments, we used both conventional and ion-selective microelectrode techniques to investigate in human atrial tissue whether halothane reduces intracellular sodium activity (a_{Na}^i), as it would be expected from a decreased Na^+ influx. We studied partially depolarized atrial specimens obtained from two patients (61- and 64-yr-old men) during corrective cardiac surgery for coronary artery disease. Informed consent was obtained before surgery.

Trabeculae from these specimens were perfused in a tissue bath with oxygenated (97% O_2 , 3% CO_2) Tyrode solution at 37° C and driven electrically at 1 Hz, as described in detail previously.¹ Figure 1 shows a recording in normal $[\text{K}]_o$ (4 mM) Tyrode solution. In figure 1A (control), the action potentials show a reduced maximum diastolic potential (MDP; -78 mV) and a plateau beginning near -20 mV. The maximal rate of rise (\dot{V}_{max}) was about 75 V/s, and a_{Na}^i was normal (7.38 mM). In figure 1B, exposure to 0.5 vol% of halothane within 10 min decreased \dot{V}_{max} and the upstroke amplitude by 23 and 10%, respectively. Halothane also slowed phase-3 repolarization, reduced MDP to -73 mV, and reduced a_{Na}^i from 7.38 to 5.56 mM (-24.7%).

In the other atrial preparation, perfused in 8 mM $[\text{K}]_o$ Tyrode solution containing 0.3 μM epinephrine, MDP was -67 mV and \dot{V}_{max} was about 105 V/s. Halothane (0.5 vol%) decreased \dot{V}_{max} and the amplitude of upstroke by -72 and -32%, respectively, and reduced a_{Na}^i from 3.50 to 2.64 mM (-24.6%) within 6 min. Both preparations recovered from halothane effects within 14 min of washout.

In a previous study in six well polarized human atrial preparations (average MDP = -85 mV),¹ halothane at concentrations up to 0.75 vol% did not change significantly the upstroke. In this connection, it should be noted that 1 μM tetrodotoxin (which selectively blocks the fast Na^+ channel) barely affects \dot{V}_{max} (as for halothane in well-polarized tissue), although it depresses the plateau and reduces the action potential

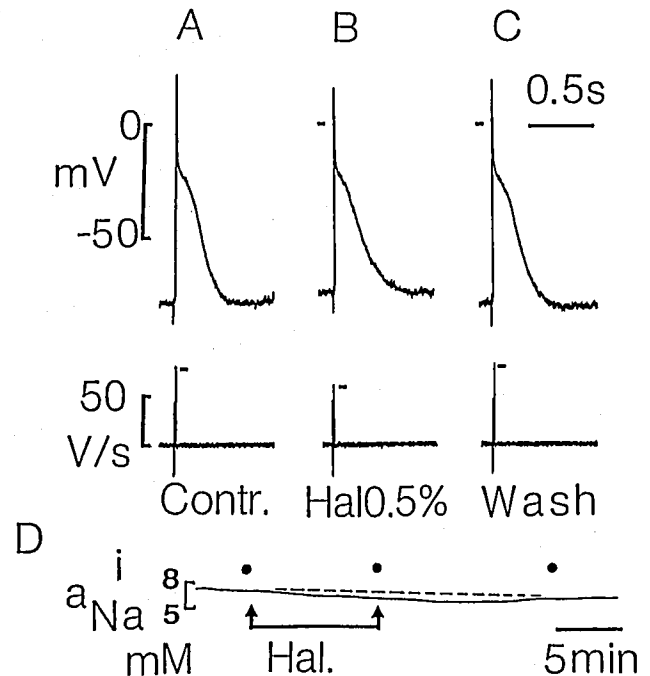


FIG. 1. Halothane decreases intracellular Na^+ activity. (a_{Na}^i) Top: The action potential (upper traces) and first derivatives (lower traces) before (A), during the 10th min of exposure to halothane (B) and after 14 min of recovery (C). In the top panel, the small horizontal bars indicate either the zero potential (upper traces) or the \dot{V}_{max} (lower traces). D: A continuous slow speed chart record of a_{Na}^i . The dots above the trace in D indicate the time at which the fast speed traces shown in A-C were recorded.

duration of dog Purkinje fibers³ as well as of human atrial tissue.¹ The underlying mechanism is a block by tetrodotoxin of the steady state Na^+ current.⁴ Because in the present experiments the fibers were partially depolarized and therefore the residual available fast Na^+ channels were fewer, halothane may have decreased the fast Na^+ current, as suggested by the decrease in \dot{V}_{max} and a_{Na}^1 . The reduction of \dot{V}_{max} and of the upstroke amplitude was greater in the fiber depolarized in 8 mM $[\text{K}]_0$ than in the fiber perfused in normal $[\text{K}]_0$ (fig. 1). Since Ca^{2+} current may contribute to the upstroke, especially in the presence of β -adrenergic agonists, the depression of Ca^{2+} current by halothane might contribute to the observed effect. Still, the present demonstration that halothane decreases a_{Na}^1 in partially depolarized fibers suggests that halothane might decrease the fast Na^+ current more when the cells are depolarized (and therefore with fewer available Na^+ channels⁵). A diminished fast Na^+ current might contribute to electrical uncoupling in cardiac cells treated with halothane.⁶ Thus, the present findings may be significant in relation to the arrhythmogenic actions of halothane in patients with ischemic heart disease.

An alternative explanation is that halothane decreases a_{Na}^1 by reducing Ca^{2+} influx and a_{Ca}^1 ; the consequent increase in the transmembrane Ca^{2+} electrochemical gradient in turn would decrease a_{Na}^1 through the $\text{Na}^+-\text{Ca}^{2+}$ exchange. The determination of the mechanism of a_{Na}^1 decrease requires further experimentation. However, the present results indicate that a decrease in a_{Na}^1 in human cardiac tissues is to be added to the effects of halothane and should contribute to the decrease in contractile force.

YUAN-HWA CHOU, M.D., M.S.
Research Fellow

JENG WEI, M.D., M.S.D.
*Associate Professor and Chairman
Department of Surgery*

CHENG-I LIN, B.D.S., PH.D.
Professor of Pharmacology

*National Defense Medical Center, Taipei
Taiwan, Republic of China*

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Failed Spinal Anesthesia with the Sprotte Needle

To the Editor:—Recent reports^{1,2} have compared Quincke point spinal needles to the 24-G Sprotte (pencil-point) needle with respect to the incidence of post-dural puncture headache in various patient subgroups. These reports and a review of the literature comparing various spinal needles have not mentioned failure of the technique as a complication of needle design.

Since the introduction of the Sprotte needle to our hospital, a perceived increase in the failure rate of spinal anesthetics using the Sprotte needle was suggested by anesthesia staff and residents. We therefore undertook a retrospective analysis of the anesthetic charts from September, 1990 to March, 1991 to address this specific issue. This period was chosen to eliminate the "learning period" of new residents in the program and, it was hoped, to account for lack of experience with regional anesthesia techniques. A "failed technique" was defined as the lack of acceptable anesthesia for the proposed surgical procedure, following the injection of local anesthetic after free-flow cerebrospinal fluid (CSF) was identified with any spinal needle.

The failure rate of spinal anesthesia using the Sprotte needle (22- and 24-G) was compared to the failure rate with all other spinal needles (22-, 25-, and 27-G Quincke point needles) used during the same time period. Of 394 spinal anesthetics performed at our institution there were 20 (5.1%) failures. Charts with incomplete data totaled 49 of 394

(12%) and were excluded from analysis. Sprotte needles were used in 87 cases, with 9 (10.3%) failures, as compared to 11 of 258 (4.3%) with all other needle types. Within the failure group of 20 there were 4 unknown needles used that were included in the non-Sprotte group. There was a significant difference ($P = 0.02$) (chi-squared analysis) between the groups. The type of local anesthetic used included lidocaine (6), bupivacaine (2), and tetracaine (1) in the Sprotte group and lidocaine (3), bupivacaine (4), tetracaine (1), and 3 unreported local anesthetics in the non-Sprotte group. Free-flow CSF was documented on 16 of 20 anesthetic records. Although difficult to evaluate, the experience of the anesthetists in the two groups was considered similar.

Possible explanations for this difference may include inexperience with the new needle type, experience of the operator, or the local anesthetic used. A more plausible reason could relate to the design of the needle, specifically the dimensions and placement of the sideport, allowing for free flow of CSF and deposition of local anesthetic solution into both the CSF and the epidural space, resulting in an inadequate spinal block. The length from the needle tip to the opening of the sideport is 1.2 mm (22- and 24-G). The length of the sideport opening is 1.75 mm on the 24-G and 2.0 mm on the 22-G Sprotte needle. From cadaveric studies³ and our own unpublished observations of fresh cadaveric dural specimens, dural thickness can vary from 0.5 to almost