J Anesth (1997) 11:208–212

Journal of
Anesthesia

Effects of vagal nerves or vagosympathetic trunks stimulation on the hemodynamics during spinal anesthesia in cats

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Abstract

Purpose. To clarify the sudden onset of profound bradycardia or hypotension during spinal anesthesia, we stimulated vagal nerves (VN) or vagosympathetic trunks (VST) to examine the effects on the autonomic nervous system during spinal anesthesia with different degrees of cardiac sympathetic nerve block.

Methods. Cats were anesthetized and mechanically ventilated. The left stellate ganglion was exposed to record cardiac sympathetic nerve activity (CSNA). Systolic and diastolic blood pressures (BP), heart rate (HR), and CSNA were measured before and after intrathecal injections of 0.2, 0.5 and 1.0 ml of 1% lidocaine. After each intrathecal injection of lidocaine, bilateral VST ($n = 5$, group A) or VN ($n = 5$, group B) were stimulated and measurements were repeated.

Results. After 1.0 ml intrathecal injection of 1% lidocaine, CSNA was blocked completely, and BP and HR were decreased. In group A, BP were unchanged following VST stimulation while in group B, BP decreased approximately 30% by VN stimulation from the pre-stimulation levels after 0.2, 0.5, and 1.0 ml injection of 1% lidocaine, respectively. HR decreased further, approximately 35% in group A and 50% in group B, by each stimulation from the prestimulation levels after 0.2, 0.5, and 1.0 ml injection of 1% lidocaine.

Conclusion. These results suggest that hypotension and bradycardia during a high level of spinal anesthesia are due to the block of CSNA, and vagal reflex may produce profound hypotension and bradycardia especially in high spinal anesthesia.

Key words: Spinal, Lidocaine, Cardiac sympathetic nerve activity, Vagal nerve stimulation, Vagosympathetic trunk stimulation

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Received for publication on July 22, 1996; accepted on March 24, 1997

Introduction

Caplan and colleagues [1] reported 14 episodes of sudden cardiac arrest in healthy patients during spinal anesthesia. They speculated that unappreciated respiratory insufficiency might have been the important factor in approximately 50% of their cases. However, the sudden onset of profound bradycardia or asystole during spinal anesthesia without hypoxia has been reported [2,3]. This bradycardia has been attributed to the reductions of cardiac sympathetic stimulation and venous return to the heart [4,5]. Another possible mechanism for profound bradycardia or asystole would be preexisting autonomic imbalance or dysfunction. Spinal anesthesia changes sympathetic nerve activity at different degrees according to dose by intrathecal administration. Vagal stimulation generally produces a depressor response; however, the effect of sympathetic stimulation on the heart is excitatory [6–8].

We consider vagal nerve or vagosympathetic trunk stimulation as useful to know the autonomic influence, with or without sympathetic effects, during spinal anesthesia. Therefore, to clarify the sudden onset of profound bradycardia or hypotension during spinal anesthesia, we stimulated vagal nerves or vagosympathetic trunks to produce autonomic imbalance during spinal anesthesia with different degrees of cardiac sympathetic nerve block.

Materials and methods

With permission from the Animal Care Committee, cats (2.5–3.5 kg) were anesthetized with intraperitoneal sodium pentobarbital 40 mg·kg^{-1}. The femoral vein was cannulated for i.v. infusion of lactated Ringer's solution 3 ml·kg^{-1}·h^{-1} and drug administration. The femoral artery was cannulated for blood sampling and monitoring blood pressure. After endotracheal intubation, they

were paralyzed with intermittent injections of pancuronium bromide 0.2 mg·kg^{-1}, and mechanically ventilated with a respirator (SN-480-5, Shinano, Tokyo, Japan). The respiratory rate and tidal volume were adjusted to keep $PaCO_2$ at 37–40 mmHg using a capnometer (Capnomac, Datex, Helsinki, Finland). The rectal temperature was maintained between 35°C and 37°C with a heating pad.

Laminectomy was performed at L6 level. An intrathecal catheter was inserted through the dura for injections of local anesthetics and covered with an adhesive agent for the prevention of cerebrospinal fluid leakage. The left stellate ganglion was exposed by resecting the first and second ribs overlying it. The inferior cardiac nerve was identified, freed from surrounding connective tissue, and placed on a silver hook electrode to record cardiac sympathetic nerve activity (CSNA). A signal from the recording electrode was passed through an amplifier (AVB-21, Nihon-Kohden, Tokyo, Japan) and displayed on an oscilloscope (VC-11, Nihon-Kohden). In addition, CSNA was rectified and integrated (EI-601-G, Nihon-Kohden) every 5 s. CSNA was calculated by subtracting the height of integrated waves of the baseline noises, which were recorded after the cats were sacrificed by overdosing of sodium pentobarbital, from the height of integrated waves as nerve activities. The phrenic nerve was exposed and nerve activities were recorded using silver hook electrodes to confirm that the lidocaine did not spread to the upper cervical area. These nerves and electrodes were immersed in paraffin solution. Stimulation electrodes were placed on the bilateral vagosympathetic trunks in five cats (group A) to stimulate both sympathetic and parasympathetic nerves, while five cats (group B) had stimulation electrodes placed on the bilateral vagal nerves exposed in the neck. Stimulation pulses (duration 0.3 ms) were generated by a stimulator (SEN-3201, Nihon-Kohden) at a rate of 30 Hz. The stimulation intensity was maintained at 50 V. CSNA, systolic and diastolic arterial blood pressures and heart rate were measured in a basal-anesthetic state as control values. The effects of vagosympathetc trunks or vagal nerves stimulation to these parameters were measured. A single dose of 0.2 ml of 1% hyperbaric (1.040) lidocaine with glucose was injected into the subarachnoid space through the catheter. Fifteen minutes after initial injection of lidocaine, all measurements were repeated. After that, the measurements of the effects of vagosympathetc trunks or vagal nerves stimulation to these parameters were repeated. Twenty minutes after initial injection, a supplementary dose of 0.5 ml lidocaine was injected intrathecally through the catheter and measurements were repeated 15 min after this injection. The effects of stimulation were also measured. Furthermore, 20 min after the second injection, 1.0 ml of lidocaine was injected intrathecally and all measurements were repeated. Systolic and diastolic arterial blood pressures were measured at the point of its maximal changes during vagal stimulation for 30 s. Heart rate was determined from the electrocardiograms with a heart rate counter and recorded for 10 s between 5 and 15 s after the start of stimulation. All data were measured with a thermal-recorder (Omniace RT2108, San-ei, Tokyo, Japan) and stored in a data-recorder (RD-120TE, TEAC, Tokyo, Japan).

All values are expressed as mean ± SD. Within each group, the results were analyzed by one-way analysis of variance and post-hoc Scheffe's procedure as a multiple comparison procedure. The differences between the two groups were analyzed by unpaired t-test. Stat View (Abacus, Berkeley, CA) was used for these analyses. $P < 0.05$ was considered statistically significant.

Results

The results in groups A and B are shown in Fig. 1. Systolic and diastolic blood pressures and heart rate decreased gradually following the increases in injection dose, and then decreased significantly after injection of 1.0 ml of 1% lidocaine in the two groups. There were no differences between the two groups in systolic and diastolic blood pressures and heart rate.

Vagosympathetic trunk stimulation in group A produced a significant increase in blood pressures from 137/100 to 178/128 mmHg, while in vagal stimulation in group B, it produced a significant decrease in blood pressures from 141/100 to 106/75 mmHg before intrathecal injection of 1% lidocaine. In group A, systolic and diastolic blood pressures were unchanged following vagosympathetic trunks stimulation after 0.2-, 0.5- and 1.0-ml injections of 1% lidocaine. In group B, systolic and diastolic blood pressures decreased by vagal stimulation from the prestimulation levels by 30%, 36%, and 25% in sytolic and 39%, 45%, and 35% in diastolic blood pressures after 0.2-, 0.5-, and 1.0-ml injection of 1% lidocaine, respectively. Heart rate decreased from 188 to 140 beats·min^{-1} in group A, and from 208 to 114 beats·min^{-1} in group B with each stimulation before intrathecal injection. After 0.2-, 0.5-, and 1.0-ml injection of 1% lidocaine, heart rate decreased suddenly with each stimulation from the prestimulation levels by 27%, 34%, and 37% in group A, and 52%, 52%, and 51% in group B, respectively. No differences in the reduction of heart rate were found between the two groups.

Phrenic nerve activity was not blocked by intrathecal injection of lidocaine in this study. However, vagal nerve or vagosympathetic trunk stimulations suppressed the phrenic nerve activity (Fig. 2).

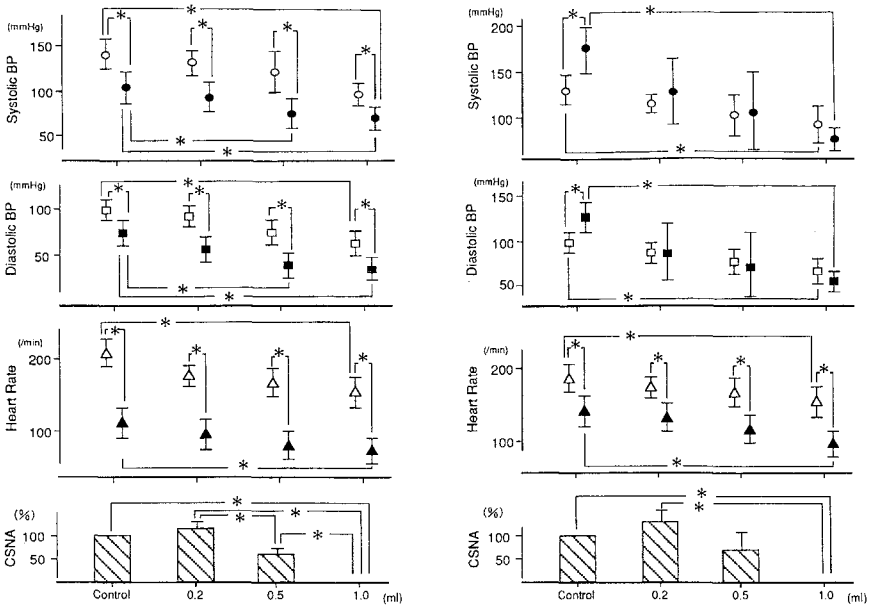

Fig. 1. Changes in systolic and diastolic blood pressures (*BP*), heart rate, and cardiac sympathetic nerve activity (*CSNA*) before (*open circles*, *squares*, *triangles*) and after (*solid circles*, *squares*, *triangles*) vagal nerve stimulation (*left*) or vagosympathetic trunk (*right*) stimulation with 0.2, 0.5, and 1.0 ml intrathecal injection of 1% lidocaine. Data are presented as means ± SD. $^*P < 0.05$

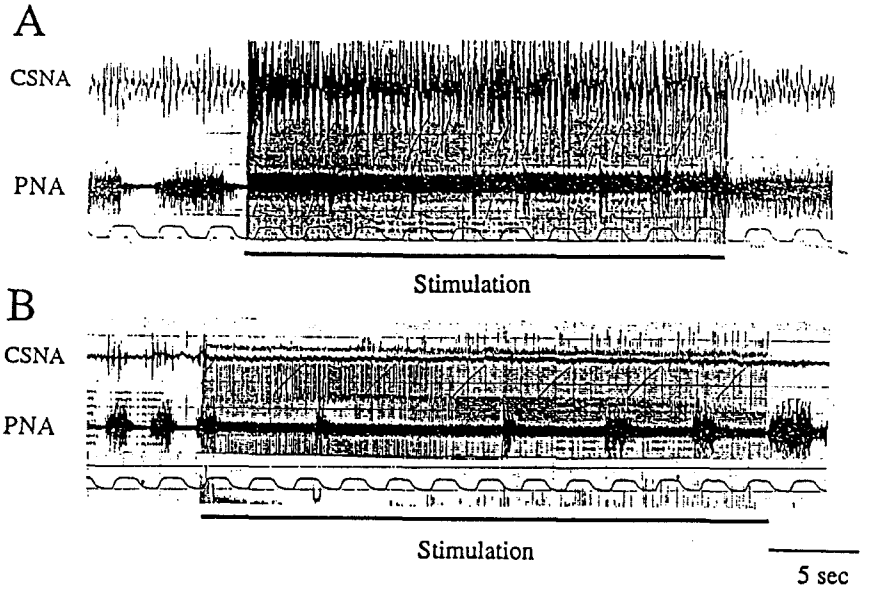

Fig. 2. Changes in cardiac sympathetic nerve activity (*CSNA*) and phrenic nerve activity (*PNA*) as a result of vagosympathetic trunk stimulation (**A**) and vagal nerve stimulation (**B**) before spinal anesthesia. Vagosympathetic trunk stimulation increased CSNA but vagal nerve stimulation suppressed it. Phrenic nerve activity was suppressed in the two groups

Discussion

Intrathecal injection of 1.0 ml 1% lidocaine, on the disappearance of cardiac sympathetic nerve activity (T1–T4), decreased systolic and diastolic blood pressures and heart rate. Though vagosympathetic trunk stimulation prevented the decrease of blood pressure, vagal nerve stimulation further decreased the blood pressure and heart rate compared to prestimulation values during spinal anesthesia. This means that vagal reflex produced sudden severe hypotension and bradycardia, especially in high spinal anesthesia.

Spinal anesthesia itself decreases sympathetic nerve activity from caudal to rostral and reduces the venous return [4,5]. Baroreceptor reflexes have a major role in the regulation of sympathetic discharge to the heart to keep up the level of blood pressure [9]. With 0.2 ml of 1% lidocaine, CSNA increased due to the decrease in blood pressure because of compensatory sympathetic nerve stimulation of the non-blocked region. As the block extends higher, leading to vasodilation of a greater area, the ability to compensate with vasoconstriction decreases. In clinical study, the level of sympathetic nerve block was considered according to the level

of sensation. Greene [10] reported that sympathetic nerve block was two segments higher than that of sensory analgesia level, while Chamberlain and Chamberlain [11] reported that the level of sympathetic block is six segments higher than the sensory block. In cases with a higher-level block of pain sensation in normotensive patients, considered complete block of sympathetic nerve resulted in a mean arterial pressure decrease of 30% or 40% [12,13] during spinal anesthesia. Though we could not measure the sensation level, we recorded CSNA directly and compared it with systolic and diastolic blood pressures and heart rate. In our animal study, there was a 31% or 34% decrease in systolic blood pressure by complete suppression of CSNA. It is difficult to predict the degree of hypotension because many factors contributing to hypotension include decreasing vascular resistance and cardiac output. However, the denervation of sympathetic nerve was considered to decrease the systolic and diastolic blood pressures by more than 30% of the control preinjection values.

In addition to the block of the sympathetic nerve, vagal stimulation further decreased the blood pressure approximately 30% compared to prestimulation values, and up to about 50% compared to the control preinjection values after 1.0 ml injection of 1% lidocaine, while vagosympathetic stimulation kept the systolic and diastolic blood pressures at the control preinjection values. Direct cardiac sympathetic nerve stimulation, regardless of spinal block, was considered to prevent further decrease of the systolic and diastolic blood pressures.

As well as changes of systolic and diastolic blood pressures, heart rate decreased similarly as a result of vagal nerve or vagosympathetic trunk stimulation with any injection dose of 1% lidocaine. Alterations in heart rate are brought about by simultaneous reciprocal changes in sympathetic and parasympathetic influences on the heart [14,15]. However, with high levels of vagal activity, sympathetic activity had a negligible effect on the heart rate [15]. Our study also shows that the vagosympathetic trunk stimulation tended to decrease the heart rate less than vagal nerve stimulation; the changes of heart rate did not significantly differ in the presence or absence of sympathetic activity from the prestimulation levels.

Sudden onset of profound bradycardia or asystole during spinal anesthesia without hypoxia has been speculated by Bainbridge reflex [2] and/or Bezold–Jarisch reflex [3]. Stimulation of cervical vagal afferents affects neurons in the rostroventral medulla and produces intensity-dependent changes in blood pressure [6]. Stimulation of cardiopulmonary vagal efferent C-fiber elicits bradycardia or asystole [16]. These changes agree with Shorten and Furness [17] who reported that bladder distention and vagal overactivity during spinal anesthesia have been shown to produce hypotension. The decision as to what degree of hypotension and bradycardia require treatment can be made only for individual patients, but there are few who cannot tolerate a decrease of 25%–30% quite safely [18]. Both blood pressure and heart rate decreases of up to 50% compared to control value, like vagal nerve stimulation in our study, need rapid treatment to prevent organ impairment.

We recorded phrenic nerve activity which originates from the cervical nerve (C5–6) to confirm that the lidocaine did not spread to the upper cervical area. This record also showed that phrenic nerve activity was suppressed by vagal stimulation, which means respiratory depression. In our experiments, cats had no trouble with oxygenation because of mechanical ventilation. In spontaneous ventilation, sudden onset of hypotension and bradycardia and/or hypoxia due to vagal excitation may trigger profound hypotension and bradycardia, especially in high spinal anesthesia.

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