Effects of Abdominal Surgery on Somatosensory Evoked Potentials during Nitrous Oxide-enflurane Anesthesia

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The effect of abdominal surgery on median nerve somatosensory evoked potentials (SEPs) was studied in 8 enflurane and nitorous oxide anesthesia (GOE) patients. We further compared the effect of epidural anesthesia. The first recording was done immediately prior to induction. Anesthesia was then induced with 5 mg·kg^{-1} i.v. of thiopental and maintained with 1.0% enflurane, 66% N_2O and 33% O_2. Before skin incision for abdominal surgery, the second recording was performed under GOE anesthesia and the third recording during surgery. Then 2% lidocaine was injected into the epidural space through a preinserted catheter, and after 15 min the fourth recording was obtained. The latencies of peaks N1, P2 and N2 and the amplitudes of N1-P2 and P2-N2 were measured. The latencies of N1, P2 and N2 increased and the amplitudes of N1-P2 and P2-N2 decreased significantly after the induction of anesthesia compared with the control values. During abdominal surgery the latencies of N1 and P2 decreased and the amplitudes of N1-P2 and P2-N2 increased. After epidural anesthesia, however, the latencies of N1 and P2 increased and the amplitudes of N1-P2 and P2-N2 decreased significantly and returned almost to the values recorded under preoperative GOE anesthesia. These phenomena indicated that the excitations produced by surgical stimulation in nerve ending might have been transmitted to the central nervous system via spinal nerves and blocked by epidural anesthesia. (Key words: somatosensory evoked potentials, abdominal surgery, epidural anesthesia, enflurane)

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Somatosensory evoked potentials (SEPs) are the electrophysiologic response of the nervous system to sensory stimulation. These reflect the functional integrity of specific sensory pathways and serve to some extent as more general inducers of function in adjacent structures[1]. Monitoring of these potentials is made difficult by anesthetic changes which may cause alterations in neural function. It is reported that volatile anesthetics such as halothane, enflurane and isoflurane prolong the latencies and decrease the amplitudes of SEPs[2–4].

Under general anesthesia, a surgical trauma evokes an endocrine response characterized by the increased production of cortisol and catecholamines[5–9]. This means that afferent inputs affect the central nervous system (CNS) through spinal cord pathways during operation. Input of a surgical stimulus may also change the latencies and amplitudes of SEPs under general anesthesia.

To determine the effect of surgery on

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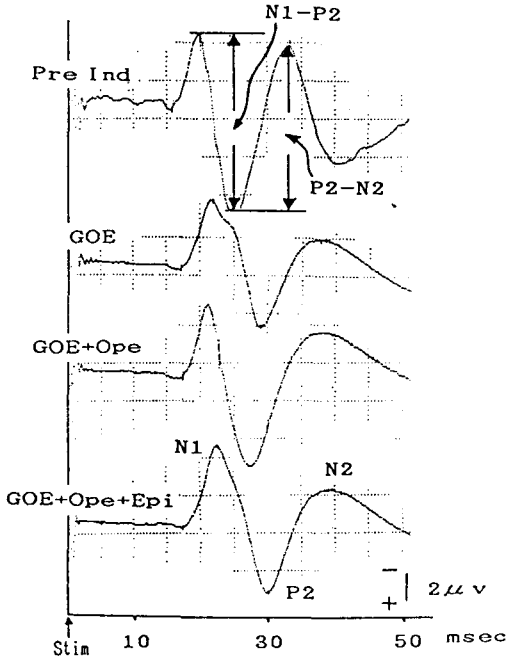

Fig. 1. A typical example is shown in the figure. Somatosensory evoked potentials were recorded before induction (Pre Ind), before skin incision for surgery (GOE), during operation (GOE + Ope), and after injection of lidocaine (2%; 10 ml) through preinserted (T10/11) epidural catheter (GOE + Ope + Epi).

CNS, we compared median nerve SEPs before and during operation. We further compared the effect of epidural anesthesia.

Methods

Eight ASA Class I and II patients with no neurological problems scheduled for elective surgery (mainly gastrectomy or cholecystectomy) were selected for the study. Informed consent was obtained from each of the patients at the time of their preoperative visit. All the patients were premedicated with intramuscular atropine (0.5 mg) and hydroxyzine (50 mg) 60 min before the scheduled operation. SEPs were measured using a signal averager (Neuropac II, Nihon Kouden). A transcutaneous stimulator was used to locate the median nerve at the wrist. The stimulus intensity was supramaximal of the motor thresholds. Five hundred and twelve constant current stimuli of 200 microsecond duration were delivered at a rate of 2 Hz. The time base of 50 msec following stimulation was analysed. Input filtering was set to a bandwidth of 20–3000 Hz. Following the 10–20 international system, a recording electrode was placed at C3' in reference to FPz. An ear clip served as ground. The electrode impedance was maintained at less than 20 Kohms. Before induction a catheter was introduced to the extradural space via the T9-T10 or T10-T11 interspace. The waveforms of SEPs were recorded immediately prior to induction of anesthesia. Anesthesia was then induced with 5 $mg \cdot kg^{-1}$ i.v. of thiopental and maintained with 1.0% enflurane, 66% N_2O and 33% O_2. Before skin incision for abdominal surgery, the second recording was performed under GOE anesthesia at approximately 20 min following induction. The third recording was obtained when an abdominal retraction was placed (about 10 min from the start of operation). The fourth recording was obtained 15 min after injection of 2% lidocaine (7 $\sim$ 10 ml; 3 $mg \cdot kg^{-1}$) in the epidural space through the preinserted catheter. The latencies of peaks N1, P2 and N2 were measured in msec from the time of stimulation. In addition, the peak-to-peak amplitudes of N1-P2 and P2-N2 were measured in microvolts and were recorded at each sampling period. The changes in recorded values before induction, after induction, during surgery, and after epidural injection were assessed by student' t test for paired data. $P < 0.05$ was considered significant.

Results

An example of SEPs recording is shown in the figure. The change in latencies are shown in table 1. The latencies of N1, P2 and N2 increased significantly after induction of anesthesia compared with the control values (prior to induction). The latencies of N1 and P2 decreased significantly during abdominal surgery. After the epidural anesthesia, the latencies of N1 and P2 increased significantly to levels similar to the values before operation and during GOE anesthesia. The latency of N2 was not changed

Table 1. Changes in N1, P2 and N2 latencies

	Pre Ind	GOE	GOE+Ope	GOE+Ope+Epi
N1 (msec)	19.5 ± 2.1	21.1 ± 1.9*	20.8 ± 2.0*#	21.8 ± 1.9*§
P2 (msec)	24.9 ± 2.1	28.3 ± 3.0*	26.6 ± 2.5*#	28.3 ± 2.8*§
N2 (msec)	33.1 ± 1.5	37.6 ± 2.2*	38.3 ± 2.4*	39.9 ± 2.1*§

*Significantly increased ($P < 0.05$) compared with control (Pre Ind)
#Significantly decreased ($P < 0.05$) compared with GOE
§ Significantly increased ($P < 0.05$) compared with GOE+Ope

Table 2. Changes in N1-P2 and P2-N2 amplitudes

	Pre Ind	GOE	GOE+Ope	GOE+Ope+Epi
N1-P2 (μv)	7.4 ± 3.3	5.0 ± 2.9*	6.2 ± 3.4*#	5.3 ± 3.5*§
P2-N2 (μv)	6.5 ± 3.5	3.6 ± 2.2*	5.6 ± 2.7#	3.9 ± 2.7*§

*Significantly decreased ($P < 0.05$) compared with control (Pre Ind)
#Significantly increased ($P < 0.05$) compared with GOE
§ Significantly decreased ($P < 0.05$) compared with GOE+Ope

Table 3. Hemodynamic data

	Pre Ind	GOE	GOE+Ope	GOE+Ope+Epi
SBP (mmHg)	129 ± 14	99 ± 10*	149 ± 16#	95 ± 6*§
DBP (mmHg)	79 ± 10	59 ± 6*	90 ± 9#	56 ± 7*§
Pulse (bpm)	84 ± 12	72 ± 12*	79 ± 17	75 ± 16*

*Significantly decreased ($P < 0.05$) compared with control (Pre Ind)
#Significantly increased ($P < 0.05$) compared with GOE
§ Significantly decreased ($P < 0.05$) compared with GOE+Ope

by the surgical stimuli but increased after epidural anesthesia. The amplitudes of N1-P2 and P2-N2 increased significantly by surgery and returned to the preoperative values after epidural anesthesia. (table 2) The blood pressure decreased significantly after induction of anesthesia and increased after surgery, while it decreased again after epidural anesthesia. No significant changes in heart rate occured at any stages after GOE anesthesia..(table 3)

Discussion

Our results demonstrate that the abdominal surgical stimulus, under GOE anesthesia, decreases the N1 and P2 latencies and increases the N1-P2 and P2-N2 amplitudes on the median nerve somatosensory evoked potential. After epidural anesthesia, the latencies and the amplitudes return to levels similar to the presurgical values. This means that the surgical stimulus affects the central nervous system through spinal cord pathways and is surpressed by epidural anesthesia during operation. The effects of surgery on CNS were studied by the endocrine responses characterized by the increased production of cortisol and catecholamines[5–9]. Increases in the plasma levels of cathecholamines, cortisol, and other stress hormones represent the sum of the intensity of stimuli in stress situation such as trauma and surgical interventions, thus suggesting that they can be regarded as important components of

stress response. These hormones were also suppressed by epidural block[5,6]. As the endocrine response, we confirmed the effect of surgical stimulus on CNS and the effect of epidural anesthesia on surgery by the measurement of SEPs.

The intensity of surgical stimuli may be related to the changes in SEPs. We compared the changes in the latencies and amplitudes of SEPs before skin incision and after retraction of the abdominal wall which provoked an intense noxious stimulation and was an usual abdominal surgical simulus. The depth of anesthesia also influences the changes in SEPs. Roizen et al.[10] determined MAC-BAR (anesthetic concentration that blocks the adrenergic response) in surgical patients, and noted that these doses of anesthetics were all considerably higher than the original MAC[11]. Very high doses of opiates administrated systemically can block cortisol and glucose responses to surgery[12]. In most patients, the adrenergic response could be diminished or completely eliminated, resulting in a relatively stress-free state of anesthesia. MAC-BAR of enflurane is 1.6 times (1.6 MAC) as much as ordinary MAC of this agent. To observe the adrenergic response by the effect of surgical stimuli, we anesthetised the patients using about 1.2 MAC with enflurane (0.6 MAC) and nitrous oxide (0.6 MAC). With this concentration, the blood pressure decreased to about 100 mmHg in preoperative GOE anesthesia and increased to about 150 mmHg during abdominal surgery. These changes in blood pressure were within a clinical range and were considered justifiable for comparison of the SEPs before and after surgery. The changes in the latencies and the amplitudes of SEPs seemed to parallel with the changes in blood pressure; however, it is not considered that these changes of SEPs depend only on such blood pressure levels. Bunegin et al.[13] reported that the mean arterial pressure of 55 mmHg results in significant reduction of SEPs. A number of variables are known to alter SEPs waveforms. Volatile anesthetics such as halothane, enflurane and isoflurane prolong the latencies and decrease the amplitude of SEPs[2-4]. Besides anesthetics and blood pressure[13], the amplitude and latency of the waveform may also change with body temperature, Pa_{CO_2} and other drugs. During this study we maintained the body temperature and Pa_{CO_2} within normal values. In this study, anesthesia was induced with thiamylal and maintained with enflurane in nitrous oxide and oxygen. Shimoji and co-workers[14] have shown that thiamylal (5 $mg \cdot kg^{-1}$) has a significant effect on both scalp and spinal evoked responses. However, in their study SEPs returned to the control values at 8–10 min after the administration of thiamylal. In our study the minimum interval between administration of thamylal and recording of SEPs was set at least 20 min.

Input from the median nerve ascends in the dorsal columns which terminates in the cuneate nucleus. The fibers that leave the dorsal column nuclei ascend through the contralateral lower brain stem terminate in the thalamus. The thalamus plays a role in transforming information that reaches the cerebral cortex. N1 is considered to originate from the primary sensory cortex, and P2 and N2 represent more complex and multi-synaptic response that may have more than one generator. Tsuji et al.[15] reported that N1 is generated as a single horizontal dipole in the central fissure, whereas P2 could reflect multiple generators in pre- and postrolandic regions. The N1, P2 and N2 component may be derived by the dynamic interaction of afferent volleys, synaptic discharge, and changes in current flow at various boundaries of the volume conductors. An abdominal stimulus ascends through anterolateral spinothalamic tract pathways into the thalamus and reaches the cerebral cortex. An abdominal stimulus also projects to the brain stem. And the dorsal columun-medial leminiscal system and the anterolateral system converge at the thalamus (ventral posterior lateral nucleus). And many neurons in a posterior nuclear group project to the cortical sensory region[16]. So the changes in the N1, P2 and N2 latencies and the N1-P2 and P2-N2 amplitudes of SEPs may represent the summation of the exciting abdominal stim-

ulus delivered, when the behavior of SEPs is interpret. Though the latencies of N1 and P2 were decreased, the latency of N2 was not changed by the surgical stimulus. The generation source of the N2 component of SEPs is not clear. N2 may generate multisynaptic responses more than the N1 and P2 component. GOE anesthesia suppresses the synaptic transmission while a surgical stimulus activates sensory pathways and affects adjacent structures. When the number of synaptic transmission is increased, the surgical stimulus to the synaps is masked by the suppressive effects of GOE anesthesia. The latencies of SEPs may change the combination of anesthetic synaptic suppression and the activation by surgical stimulation. After epidural anesthesia, the latency of N2 was increased compared to that by GOE anesthesia. We previously reported that the N1, P2 and N2 peak latencies of SEPs was increased after epidural anesthesia compared with the control values[17]. This suggests that we need to consider not only the block of its neural noxious inputs but also the systemic effect of local anesthetics.

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