

# Systemic lidocaine and human somatosensory-evoked potentials during sufentanil-isoflurane anaesthesia

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*The effect of systemically administered lidocaine on somatosensory evoked potentials (SSEPs) during general anaesthesia has not been widely reported. Knowledge of the influence of anaesthetic agents on evoked potentials assists in interpreting evoked potential waveforms. Accordingly, we studied the behaviour of cortical and subcortical (recorded at the second cervical vertebra) SSEPs after administration of intravenous lidocaine ( $3 \text{ mg} \cdot \text{kg}^{-1}$  bolus followed by infusion at  $4 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{hr}^{-1}$ ) during a sufentanil-based anaesthetic regimen in 16 patients undergoing abdominal or orthopaedic surgery. When compared to awake baseline recordings, the sufentanil-nitrous oxide, low-dose isoflurane anaesthetic depressed  $N_1$  amplitude by approximately 40% and prolonged latency by 10%. Fifteen minutes after establishment of this anaesthetic, the amplitude and latency of  $N_1$  were  $1.13 \pm 0.56 \mu\text{V}$  and  $19.81 \pm 1.63 \text{ msec}$ , respectively. Within five minutes of adding lidocaine, amplitude decreased further to  $0.84 \pm 0.39 \mu\text{V}$  ( $P = 0.001$ ), while latency was extended to  $20.44 \pm 1.48 \text{ msec}$  ( $P = 0.01$ ). Lidocaine did not affect cervical amplitude and prolonged latency only minimally. Despite the observed effects on amplitude and latency, SSEP waveforms were preserved and interpretable. Plasma lidocaine levels obtained at 5, 20, and 40 minutes after lidocaine were  $5.17 \pm 1.33$ ,  $3.76 \pm 1.14$ , and  $3.66 \pm 0.9 \mu\text{g} \cdot \text{dL}^{-1}$ , respectively. Our*

*results indicate that systemically administered lidocaine at therapeutic plasma levels acts synergistically with a sufentanil-based anaesthetic to depress the amplitude and prolong the latency of SSEPs.*

*Il existe peu de publications sur l'effet de la lidocaïne administrée par voie systémique sur les potentiels évoqués somatosensitifs (SSEP) pendant l'anesthésie générale. La connaissance des effets des agents anesthésiques sur les potentiels évoqués aide à l'interprétation des ondes de potentiels évoqués. Nous avons étudié l'effet de l'administration de lidocaïne par voie intraveineuse (bolus de  $3 \text{ mg} \cdot \text{kg}^{-1}$  suivi d'une perfusion de  $4 \text{ mg} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ ) sur les SSEP corticaux et sous-corticaux (enregistré au niveau de la deuxième vertèbre cervicale) chez 16 patients anesthésiés avec une technique à base de sufentanil pendant une chirurgie abdominale ou orthopédique. Comparativement aux enregistrements de base en état d'éveil, l'anesthésie à l'aide de sufentanil-prot oxyde d'azote et faible dose d'isoflurane déprimait l'amplitude  $N_1$  d'environ 40% et prolongeait la latence de 10%. Après 15 minutes d'anesthésie, l'amplitude et la latence de  $N_1$  étaient de  $1,13 \pm 0,56 \mu\text{V}$  et  $19,81 \pm 1,63 \text{ msec}$  respectivement. Cinq minutes après l'addition de lidocaïne, l'amplitude a diminué à  $0,84 \pm 0,39 \mu\text{V}$  ( $P < 0,001$ ) tandis que la latence a augmenté à  $20,44 \pm 1,48 \text{ msec}$  ( $P < 0,01$ ). La lidocaïne n'a pas affecté l'amplitude cervical et a très peu prolongé la latence. Malgré les effets observés sur l'amplitude et la latence, les ondes SSEP étaient préservées et interprétables. Les niveaux plasmatiques de lidocaïne à 5, 20 et 40 minutes après l'injection étaient de  $5,17 \pm 1,33$ ,  $3,76 \pm 1,14$  et  $3,66 \pm 0,9 \mu\text{g} \cdot \text{dL}^{-1}$  respectivement. Nos résultats démontrent que la lidocaïne administrée par voie systémique à des niveaux plasmatiques thérapeutiques agit en synergie avec une technique anesthésique à base de sufentanil en provoquant une dépression de l'amplitude et une prolongation de la latence des SSEP.*

## Key words

ANAESTHETICS, INTRAVENOUS: sufentanil;  
ANAESTHETICS, LOCAL: lidocaine;  
MEASUREMENT TECHNIQUES: electroencephalography;  
MONITORING: evoked potentials.

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Somatosensory evoked potentials (SSEPs) are frequently used to indicate central nervous system integrity during certain neurosurgical and orthopaedic procedures. However, anaesthetics affect SSEP amplitude and latency,

particularly in cortical recordings.<sup>1-3</sup> Knowing the effect of anaesthetics and other therapeutic agents that may be administered perioperatively helps with the interpretation of evoked potential waveforms and in the management of anaesthesia for patients who require such electrophysiologic monitoring.

Lidocaine is administered intravenously either as a component of the anaesthetic or in the management of intraoperative dysrhythmias. However, the influence of systemic lidocaine on SSEPs during general anaesthesia has not been widely reported. This is of interest to the anaesthetist who cares for patients requiring SSEP monitoring during procedures that are associated with the risk of injury to spinal or brain function. We therefore investigated the effect of intravenously administered lidocaine on median nerve SSEPs during a background anaesthetic technique commonly used for neurosurgical patients.

### Methods

With institutional approval and written informed consent, we studied 16 patients (ages 22 to 46 yr) undergoing elective abdominal or orthopaedic surgery. All patients received premedication with triazolam (0.25 to 0.50 mg *po*) and morphine (0.1 to 0.15 mg · kg<sup>-1</sup> *im*) 60 to 90 min before induction. Anaesthesia was induced with thiopentone (4 to 5 mg · kg<sup>-1</sup>) and sufentanil (0.5 to 1.0 µg · kg<sup>-1</sup>) and maintained with sufentanil (by continuous infusion at a rate of 0.25 to 0.5 µg · kg<sup>-1</sup> · hr<sup>-1</sup>), nitrous oxide (50% inspired) and isoflurane (0.5% inspired). Muscle relaxation was provided by non-depolarizing relaxants. After a postinduction equilibration period of 15 minutes, lidocaine (3 mg · kg<sup>-1</sup>) was infused over two minutes, followed by a constant infusion at 4 mg · kg<sup>-1</sup> · hr<sup>-1</sup>.

Using a Cadwell 5200A (Cadwell Laboratories, Inc., Kennewick, WA, USA) electrophysiological monitoring system, SSEPs were recorded immediately before induction, at six and one minutes before lidocaine administration, and at five- and ten-minute intervals thereafter. Stimuli consisting of 150 µsec constant current pulses were applied to the skin overlying the median nerve at the wrist. Stimulus intensity was adjusted to motor threshold plus 50%. Signals were recorded simultaneously from surface gold-cup electrodes (affixed with collodion) at the level of the second cervical vertebra and the area of the scalp overlying the contralateral cortex (C3' or C4', International 10-20 System). The electrical signals were recorded over a time base of 50 msec, with band pass filters of 30 and 500 Hz. The Cadwell preamplifier setting was 10 µV per division. Electrode impedance was adjusted to less than 3 kOhms. Three hundred signals were averaged into one evoked potential waveform. For each channel, an 8-bit analogue-to-digital converter digitized

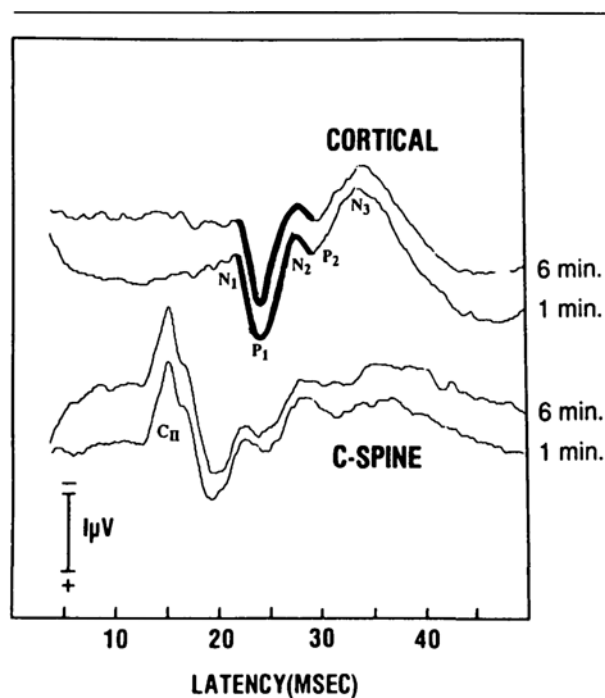


FIGURE 1a Example of scalp ("cortical") and cervically ("C-spine") recorded SSEP waveforms obtained during the sufentanil-isoflurane anaesthetic, one and six minutes prior to the systemic administration of lidocaine.

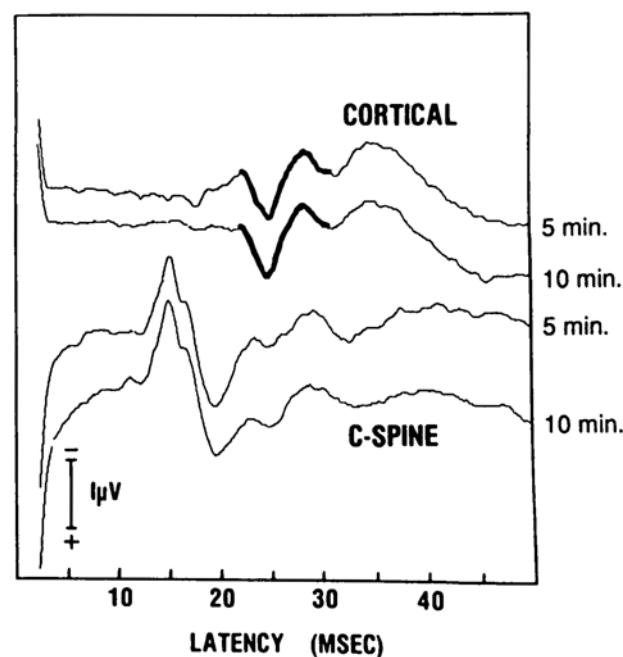


FIGURE 1b SSEP waveforms five and ten minutes after lidocaine 3 mg · kg<sup>-1</sup> *iv*. Note the contraction in N<sub>1</sub> waveform amplitude with relative sparing of cervical amplitude.

TABLE I Cardiorespiratory data and anaesthetic levels

	Pre-lido	5 min	10 min	20 min	30 min	40 min
MAP (mmHg)	73 ± 12	76 ± 9	75 ± 10	71 ± 11	79 ± 13	79 ± 16
HR (beats · min <sup>-1</sup> )	66 ± 11	66 ± 11	65 ± 12	64 ± 11	62 ± 10	66 ± 9
T (°C)	35.9 ± 0.2	35.8 ± 0.3	35.8 ± 0.3	35.7 ± 0.4	35.7 ± 0.3	35.6 ± 0.3
PETCO <sub>2</sub> (mmHg)	30.1 ± 2.6	29.9 ± 3.0	30.8 ± 2.4	30.7 ± 2.4	30.9 ± 2.4	31.0 ± 3.0
FET <sub>ISO</sub> (%)	0.32 ± 0.03	0.34 ± 0.03	0.34 ± 0.03	0.34 ± 0.03	0.35 ± 0.03	0.34 ± 0.03
Plasma lidocaine (µg · dl <sup>-1</sup> )	–	5.17 ± 1.33	–	3.76 ± 1.14	–	3.66 ± 0.46

waveforms at 18 kHz. All evoked potential waveforms were stored on magnetic disc for later analysis. The pertinent waveform generated from the cervical recording electrode consisted of a negativity at approximately 14 msec (C<sub>II</sub>s). In tracings recorded from the cortical electrode, negativities at approximately 20, 24, and 33 msec (N<sub>1</sub>, N<sub>2</sub>, and N<sub>3</sub>) followed by positive deflections at approximately 22 and 27 msec (P<sub>1</sub> and P<sub>2</sub>) were analyzed (Figures 1a, 1b). Peak-to-trough amplitude values and central conduction time (CCT), that is, the C<sub>II</sub>s-N<sub>1</sub> interlatency difference, were also measured.

With each SSEP measurement, mean arterial blood pressure (MAP), heart rate (HR), oesophageal temperature (T), end-tidal CO<sub>2</sub> (PETCO<sub>2</sub>), and end-tidal isoflurane (FET<sub>ISO</sub>) were also recorded. Venous plasma lidocaine levels were obtained 5, 20, and 40 min after lidocaine was begun. The infusion was maintained for at least 40 min; however, some anaesthetists elected to continue intravenous lidocaine anaesthesia throughout the case. Lidocaine levels were measured by fluorescence polarization immunoassay. Heated humidification and warming blankets were used to maintain body temperature. Cooling of the stimulated extremity was avoided by using the opposite arm for intravenous fluid administration. Mechanical ventilation was adjusted to maintain a stable level of PETCO<sub>2</sub>.

Statistical analyses included the paired t test or Wilcoxon signed rank test, where appropriate, to evaluate the stability of the pre-lidocaine postinduction measurements and to detect differences over time when compared with the pre-lidocaine baseline data. Pre-lidocaine baseline data were compared with five follow-up times in pairwise fashion. Paired t tests were used instead of repeated measures analysis of variance, because of occasional missing data points. Since the latter were distributed over four patients at different observation points, the use of ANOVA would have resulted in deletion of a disproportionate amount of data. To help account for multiple comparisons, the Bonferroni method of adjustment (0.05/# pairwise comparisons) was used to arrive at a stricter criterion for statistical significance ( $P = 0.01$ ).

TABLE II Baseline SSEP data prior to induction (A) and prior to administration of lidocaine (B, C)

Amplitude (µV)	A	B	C*
C <sub>II</sub> s	3.25 ± 1.06	2.95 ± 1.12	3.0 ± 1.04
N <sub>1</sub> -P <sub>1</sub>	2.0 ± 0.67†	1.16 ± 0.58	1.09 ± 0.55
P <sub>1</sub> -N <sub>2</sub>	1.16 ± 0.69	0.81 ± 0.68	0.78 ± 0.61
N <sub>2</sub> -P <sub>2</sub>	0.85 ± 0.70	0.64 ± 0.48	0.56 ± 0.45
P <sub>2</sub> -N <sub>3</sub>	1.91 ± 0.98	1.41 ± 0.74	1.31 ± 0.72
Latency (msec)			
C <sub>II</sub> s	13.16 ± 1.07	13.40 ± 1.09	13.40 ± 1.12
N <sub>1</sub>	18.85 ± 1.01†	19.76 ± 1.57	19.85 ± 1.70
P <sub>1</sub>	21.45 ± 1.14†	22.07 ± 1.39	22.18 ± 1.33
N <sub>2</sub>	24.10 ± 1.73	24.27 ± 1.39	24.30 ± 1.35
P <sub>2</sub>	26.23 ± 2.12	27.07 ± 1.80	27.15 ± 2.07
N <sub>3</sub>	30.91 ± 2.16†	33.50 ± 2.21	33.47 ± 2.39
CCT (N <sub>1</sub> -C <sub>II</sub> )	5.69 ± 0.47†	6.36 ± 1.0	6.45 ± 1.17

\* $P > 0.05$  vs B (ns).

† $P < 0.01$  vs average of B and C.

A = pre-induction; B = post-induction, six min pre-lidocaine; C = post-induction, one min pre-lidocaine.

## Results

Although HR and MAP decreased during induction of anaesthesia, no further changes were observed after the administration of lidocaine. Similarly, T, PETCO<sub>2</sub> and FET<sub>ISO</sub> were unchanged during the study period (see Table I).

Baseline SSEP results recorded before and after induction of anaesthesia are shown in Table II. The sufentanil-based background anaesthetic resulted in a reduction in N<sub>1</sub>-P<sub>1</sub> amplitude of approximately 40% with a small (<10%) increase in latency (see Table II). In no case was the N<sub>1</sub> waveform obliterated. However, in four patients, the later peaks P<sub>1</sub>, N<sub>2</sub> and P<sub>2</sub> were not identifiable. Somatosensory evoked potential measurements obtained six and one minutes before administration of lidocaine did not differ. Therefore, they were averaged and used as a baseline value for post-lidocaine data.

Tables III and IV show the effect of lidocaine on SSEP latency and amplitude. The addition of lidocaine to the

TABLE III Effect of lidocaine *iv* on SSEP latency

	Pre-lido†	5 min	10 min	20 min	30 min	40 min
C <sub>II</sub> S	13.40 ± 1.10	13.50 ± 1.17	13.58 ± 1.17	13.59 ± 1.22	13.67 ± 1.17*	13.72 ± 1.18*
N <sub>1</sub>	19.81 ± 1.63	20.44 ± 1.48*	20.38 ± 1.49*	20.35 ± 1.56*	20.25 ± 1.79	20.37 ± 1.60
P <sub>1</sub>	22.12 ± 1.28	23.0 ± 1.65*	22.8 ± 1.57*	22.89 ± 1.54*	22.78 ± 1.38*	22.88 ± 1.59*
N <sub>2</sub>	24.28 ± 1.37	24.85 ± 1.61*	24.91 ± 1.51*	24.96 ± 1.53*	25.11 ± 1.51*	25.43 ± 1.40*
P <sub>2</sub>	27.11 ± 1.96	27.90 ± 1.46*	27.86 ± 1.32*	27.97 ± 1.45*	28.02 ± 1.58*	28.06 ± 1.56*
N <sub>3</sub>	33.49 ± 2.23	34.66 ± 2.25*	35.16 ± 2.6*	35.45 ± 2.82*	34.76 ± 2.31*	35.36 ± 2.77*
CCT	6.41 ± 1.07	6.94 ± 0.70*	6.8 ± 0.68*	6.76 ± 0.86	6.58 ± 1.12	6.65 ± 0.87

†Average of two pre-lidocaine data points.

\**P* < 0.01 vs pre-lidocaine average.

TABLE IV Effect of lidocaine *iv* on SSEP amplitude

	Pre-lido†	5 min	10 min	20 min	30 min	40 min
C <sub>II</sub> S	2.97 ± 1.06	3.11 ± 1.04	3.02 ± 1.02	2.88 ± 0.94	2.75 ± 0.92	2.75 ± 0.92
N <sub>1</sub> -P <sub>1</sub>	1.13 ± 0.56	0.84 ± 0.39*	0.79 ± 0.45*	0.75 ± 0.32*	0.90 ± 0.30	0.77 ± 0.30*
P <sub>1</sub> -N <sub>2</sub>	0.80 ± 0.64	0.51 ± 0.36*	0.53 ± 0.39	0.49 ± 0.31*	0.46 ± 0.31*	0.45 ± 0.29
N <sub>2</sub> -P <sub>2</sub>	0.60 ± 0.47	0.82 ± 0.44	0.67 ± 0.34	0.58 ± 0.37	0.46 ± 0.32	0.47 ± 0.33
P <sub>2</sub> -N <sub>3</sub>	1.36 ± 0.73	1.54 ± 0.74	1.28 ± 0.65	1.17 ± 0.66	1.15 ± 0.63	1.04 ± 0.45*

†Average of two pre-lidocaine data points.

\**P* < 0.01 vs pre-lidocaine average.

sufentanil-isoflurane anaesthetic resulted in a small but statistically significant prolongation of all latencies measured, as well as in a prolongation of CCT (Figure 2). Lidocaine also affected N<sub>1</sub>-P<sub>1</sub> and P<sub>1</sub>-P<sub>2</sub> amplitude, diminishing it significantly by 25–30% (Figure 3a). The amplitude at later waveform peaks (N<sub>2</sub>-P<sub>2</sub>, P<sub>2</sub>-N<sub>3</sub>) was largely unchanged although a trend towards a transient enhancement at five minutes was seen (Figure 3b). No waveform peaks were lost as a result of lidocaine. The SSEP effects of the lidocaine bolus plus infusion regimen were clearly evident five minutes after drug administration and persisted for at least 40 minutes.

### Discussion

Our data indicate that intravenous lidocaine depresses cortical SSEP amplitude by 25% to 30% and prolongs latency by approximately 5% when administered during an anaesthetic regimen frequently used for patients requiring neurophysiological monitoring. The dosing regimen employed in the study resulted in plasma lidocaine concentrations considered therapeutic for arrhythmia control.

During anaesthesia, lidocaine may be administered systemically for a variety of reasons. In humans, lidocaine reduces MAC by approximately 30% at plasma concentrations comparable to those attained in the present study.<sup>4</sup> It also has been reported to suppress the cough reflex and may blunt the haemodynamic and intracranial pressure responses to airway manipulation.<sup>5,6</sup> The agent has therefore been administered by continuous infusion to augment

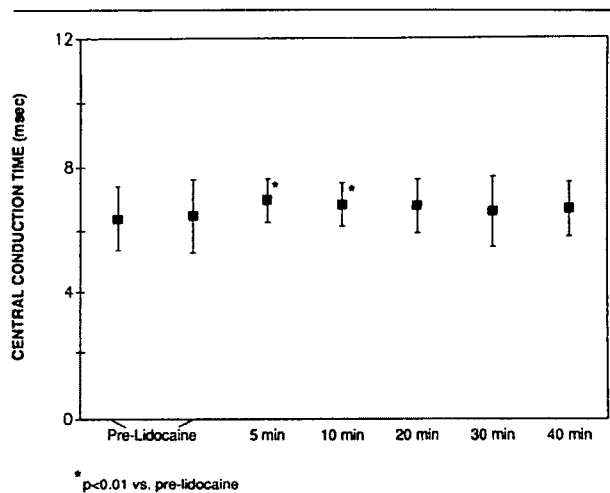


FIGURE 2 Despite achieving statistical significance, the prolongation of central conduction time after lidocaine was relatively minor (<10%). Note the stability of the pre-lidocaine measurements.

depth of anaesthesia, and by bolus to guard against stimulation from airway manoeuvres.<sup>7-9</sup> Lidocaine infusions have also been employed in the treatment of acute postoperative pain<sup>9,10</sup> and chronic pain syndromes.<sup>11,12</sup>

The influence of systemically administered local anaesthetics on evoked potentials has been investigated primarily in the context of the auditory-evoked response. Both systemically administered lidocaine<sup>13</sup> and its primary amine congener tocainide<sup>14</sup> have been found to influence

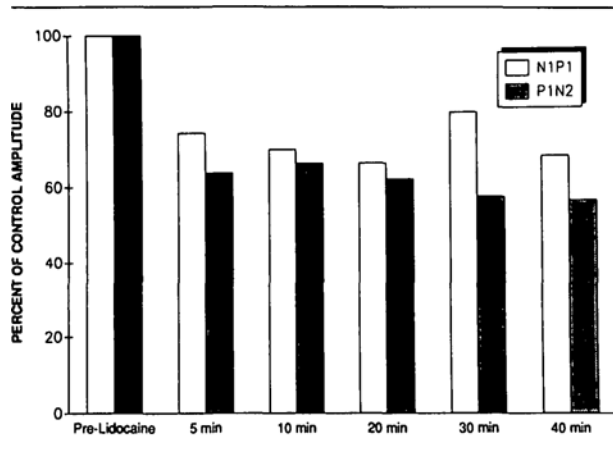


FIGURE 3a Amplitude effect of lidocaine:  $N_1$ - $P_1$  and  $P_1$ - $N_2$  amplitude is moderately depressed from pre-lidocaine, post-induction baseline.

the brainstem auditory response (BAER). At very low doses, the effect is minimal.<sup>15</sup> At continuous infusion rates commonly employed during arrhythmia control, lidocaine affected the more central components of the human BAER (wave V) by increasing latency and reducing amplitude. At much higher doses ( $20 \text{ mg} \cdot \text{kg}^{-1}$ ) all components of the cat BAER were prolonged,<sup>16</sup> and in guinea pigs the BAER response disappeared entirely at  $30 \text{ mg} \cdot \text{kg}^{-1}$ .<sup>17</sup>

Somatic-evoked sensory nerve activity at the spinal cord level is subject to the influence of systemically administered lidocaine and tetracaine. In spinal rats, lidocaine at doses greater than  $1 \text{ mg} \cdot \text{kg}^{-1}$  substantially reduced the amplitude of evoked polysynaptic reflexes.<sup>18</sup> Although also affecting A-beta-, A-delta- and C-fibre evoked activity, lidocaine and tetracaine preferentially depressed C-fibre-evoked activity.<sup>18,19</sup> Similarly, Dohi *et al.*,<sup>20</sup> found that intravenously administered lidocaine suppressed noxious-evoked activity in dorsal horn neurons of decerebrate cats. At the spinal level, systemic lidocaine may enhance monosynaptic reflexes,<sup>21</sup> possibly through depression of local inhibitory neuronal influences. Cortical neurons, however, appear to be subject to a direct inhibitory action of the drug at doses encountered clinically.<sup>1,22,23</sup>

Depression of multisynaptic pathways at the spinal and supraspinal levels is therefore associated with the systemic administration of lidocaine. The results of the present investigation were consistent with this notion. The major depressant effect of lidocaine on SSEP latency in the present study occurred at the level of  $N_1$  and  $N_2$  waveforms, but the latency of the cervical potential ( $C_{II}$ s) was also prolonged to a lesser degree (Table III). The transient enhancement of later cortical waveform components ( $P_2$ - $N_3$ ) is unexplained but may represent release of inhibitory activity.

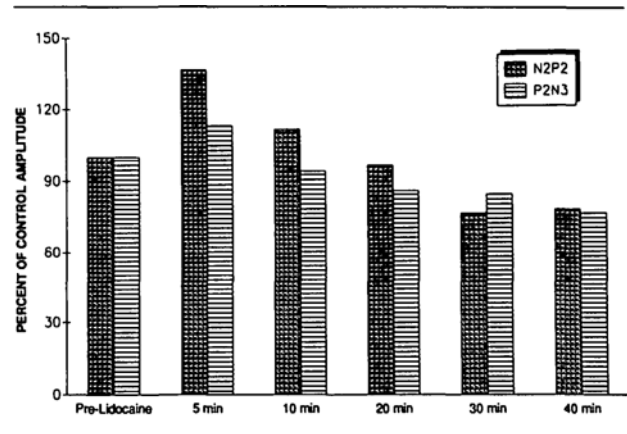


FIGURE 3b The amplitude of later ( $N_2$ - $P_2$ ,  $P_2$ - $N_3$ ) waveform components is variably affected by lidocaine.

The influence of systemic lidocaine on intraoperatively recorded SSEPs has only recently been recognized.<sup>24,25</sup> In a study of intravenous lidocaine for cerebral protection during focal ischaemia, Gelb *et al.*<sup>26</sup> reported that lidocaine  $5 \text{ mg} \cdot \text{kg}^{-1}$  resulted in a 40% reduction of feline cortical SSEP amplitude prior to middle cerebral artery occlusion. Mackie *et al.*<sup>27</sup> reported a lack of changes in both the SSEP and BAER after lidocaine was administered systemically to four healthy awake human volunteers at a dose sufficient to achieve blood levels of 2 to  $5 \mu\text{g} \cdot \text{dl}^{-1}$ . Their small sample size may well have prevented the detection of a significant effect, as a trend toward prolonged SSEP latency was evident in the data. The unanaesthetized state of their subjects could also have contributed. It is conceivable that lidocaine acts synergistically with general anaesthetics to depress SSEP waveforms.

Our data may be criticized because of the possibility that factors other than the administration of lidocaine could have accounted for the SSEP changes observed. However, we believe that this is unlikely for several reasons. First, such potential confounding variables as temperature, carbon dioxide tension, or inhaled anaesthetic concentration were rigidly controlled during the entire recording period. Second, the two pre-lidocaine baseline recordings were similar, suggesting that the 15-min equilibration period had, indeed, resulted in a stable anaesthetic state. Furthermore, the changes observed were generally maximal at the five- or ten-minute recordings and progressed no further with time. This stability, together with the stable baseline recordings, argues against the observed changes having occurred merely as a result of the passage of time.

The present findings should be of interest to the anaesthetist and neurophysiologist involved in the care of patients who are to have SSEPs monitored intraoperatively. When an anaesthetic similar to the one in the

present study is employed, the systemic administration of lidocaine may reduce cortical amplitude to a point where the cumulative change could give the appearance of neural injury. It would, therefore, be advisable to curtail the administration of *iv* lidocaine during critical SSEP monitoring periods. Similarly, if baseline SSEP amplitude is already low, the addition of lidocaine to the anaesthetic regimen is not desirable because of the potential for further amplitude depression. If lidocaine is needed for arrhythmia control, it may be necessary to discontinue nitrous oxide to counteract its SSEP-depressant effect. Nitrous oxide reduces SSEP amplitude by up to 50%.<sup>28</sup>

Kasaba *et al.*<sup>24</sup> observed prolongation of median nerve SSEPs after epidural lidocaine anaesthesia and postulated that this effect resulted from the central action of systemically absorbed local anaesthetic. These investigators later showed similar SSEP latency prolongation with intravenously administered lidocaine at plasma lidocaine levels comparable to those achieved during epidural anaesthesia.<sup>25</sup> Our findings support their conclusion that SSEP changes during epidural anaesthesia may in part be the result of systemic absorption. In light of these observations, previous data reporting the influence of epidural local anaesthesia on SSEPs should be re-evaluated, since the systemic effect of local anaesthetic may have accounted for some of the changes observed. Epidural administration of lidocaine can result in plasma levels closely approximating those observed in the present study.<sup>24</sup> Depression of cortically<sup>19,30</sup> and spinally<sup>30</sup> recorded amplitude and prolongation of latency after administration of local anaesthetic thus may, in part, have been occasioned by systemically absorbed drug.

In summary, we observed a reduction in SSEP amplitude and a slightly prolonged latency when patients anaesthetized with a sufentanil-nitrous oxide-isoflurane regimen were given intravenous lidocaine to achieve mean plasma levels of 3.7 to 5.2  $\mu\text{g} \cdot \text{dl}^{-1}$ . This information can be of use in the intraoperative management of patients requiring SSEP monitoring when lidocaine is administered intravenously either as an adjunct to anaesthesia or as an antiarrhythmic agent.

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