EFFECTS OF SUFENTANIL ON MEDIAN NERVE SOMATOSENSORY EVOKED POTENTIALS†

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SUMMARY

We have studied the effects of a single i.v. dose of sufentanil 5 \( \mu \text{g kg}^{-1} \) in combination with pancuronium on the median nerve short latency somatosensory evoked potentials (SSEP) in 15 unpremedicated patients undergoing thoracic or lumbar spinal surgery. The latency and amplitude of the SSEP response over the second cervical vertebra (SC) and sensory cortex (P17, N20, P25), heart rate and arterial pressure were recorded for 30 min after the injection of sufentanil. A significant increase in mean latency occurred for N20 (\( P < 0.003 \)) and P25 (\( P < 0.002 \)) within 2 min, but the absolute increase in latency was small. The mean amplitudes of all peaks decreased to 60% (SC), 70% (P17), 60% (N20) (\( P < 0.012 \)) and 45% (P25) of the baseline value within 7 min. The results suggest that the major change in median nerve SSEP produced by this dose of sufentanil is a reduction in amplitude, and that major changes in latency after sufentanil and pancuronium are probably caused by other influences.

KEY WORDS


Evoked potential monitoring is an accepted technique for objective measurement of function in the sensory spinal tracts and has been used to monitor spinal integrity during surgery. Many factors influence the ability to monitor the somatosensory evoked potentials (SSEP), including anaesthetic agents, physiological variables, technical problems and surgery. Veilleux, Daube and Cucchiara reported reductions in ulnar nerve and tibial nerve cortical evoked potential amplitudes shortly after induction of anaesthesia in 12% of their patients [1]. Drug-induced changes in SSEP must be determined to permit their discrimination from adverse effects of patient positioning for surgery.

As inhalation agents appear to affect monitoring ability more than opioids [2, 3], an opioid-based anaesthetic technique is often used. Sufentanil, an opioid agonist selective for the mu receptor [4, 5], has a high therapeutic index [6] and is used widely in a dose of 5 \( \mu \text{g kg}^{-1} \) [7–9].

The present study was undertaken to determine the effects of a single bolus of sufentanil 5 \( \mu \text{g kg}^{-1} \) in combination with pancuronium on the short latency median nerve SSEP in humans.

PATIENTS AND METHODS

Fifteen ASA physical status I and II patients undergoing thoracic or lumbar spinal surgery with SSEP monitoring gave written consent to participate in the study, which was approved by the institutional review boards. Patients with neurological abnormalities in the median nerve pathway or intracranial pathology were excluded.

Patients came to the operating room without premedication. An i.v. catheter was inserted and the overnight fluid deficit replaced with 5% glucose in lactated Ringer’s solution. Routine monitoring included precordial stethoscope, spirometer, continuous electrocardiogram,
automated oscillotonometry, temperature and capnograph.

After skin abrasion, disc recording electrodes with electrode paste were affixed with collodion at Fz (reference), the inion (SC), and 2 cm posterior to C3 and C4 (International 10-20 System). A silver-silver chloride ground electrode was placed on the shoulder. All electrode impedances were less than 3000 Ω and electrode pairs matched within 1000 Ω. Median nerve SSEP were obtained using stimulation at the wrist with a bipolar bar electrode and electrode gel. Constant current stimulation used 300-μs square wave impulses at 5.7–8.7 Hz, 1 mA greater than that sufficient to produce a motor response.

For each averaged waveform, 250 artefact-free responses were acquired using a Pathfinder II or CA-1000/DC-2000 signal averager (Nicolet Biomedical Instruments, Madison, Wisconsin), with bandpass filter settings of 5–250 Hz (60 Hz notch filter was not used) and an analysis window of 50 ms. All acquired data were stored on magnetic discs for later analysis.

After two satisfactory baseline recordings were obtained, pancuronium 0.02 mg kg⁻¹ was given i.v., followed by sufentanil 5 μg kg⁻¹ over 30 s. The SSEP, arterial pressure (AP) and heart rate (HR) were recorded at 1-min intervals for 9 min, and then every 3 min until 30 min after injection. Additional pancuronium (to a total of 0.12 mg kg⁻¹) was given as required. Ventilation was assisted manually as necessary using a facemask with 40% oxygen in air to maintain end-tidal Pco₂ at 4.7–6 kPa. All patients were normothermic at the end of the study.

Latency and amplitude values were calculated after the study by recall of stored averages. Post-stimulus latency of the major cervical negative wave (SC) (approximately 15 ms) and the latencies of the primary cortical response waves (P17, 17 ms; N20, 20 ms; and P25, 25 ms) were recorded. Conduction times were calculated as the time intervals between various peaks. Amplitude was measured as the voltage from the peak of interest to the following peak of opposite polarity (e.g., cortical N20 amplitude was measured from N20 to P25).

SSEP latencies, conduction times, amplitudes and physiological variables (AP and HR) were compared using Friedman two-way analysis of variance. Differences were confirmed by Wilcoxon matched pairs signed ranks test. Statistical significance was assumed when P < 0.05.

RESULTS

We studied nine women and six men: mean age 35 yr (range 24–55 yr), height 169 cm (range 152–185 cm) and weight 73 kg (range 55–115 kg).

There was no difficulty obtaining the post-stimulus latencies immediately after the sufentanil bolus in any patient. The median nerve SSEP obtained from one patient are shown in figure 1, and mean (SD) values for group data acquired during the entire study period are presented in table I. A statistically significant increase in latency occurred in the cortical N20 (P < 0.003) and the cortical P25 (P < 0.002). Wilcoxon analysis demonstrated a significant difference between time 0 and later times, confirming that the increase occurred immediately after injection (1–2 min). The absolute latency increase was, however, small. Changes in the latencies of the cervical and cortical P17 were also small and not statistically significant.

Analysis of the interwave latencies (conduction times) revealed significant differences between SC and cortical N20 (P < 0.002) and between SC and cortical P25 (P < 0.003). Again, the major changes
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TABLE I. SSEP and cardiovascular data (mean (SD)) at selected times after administration of sufentanil and pancuronium. SC = SSEP response over second cervical vertebra; P17, N20 and P25 = SSEP response over sensory cortex. HR = Heart rate; SAP and DAP = systolic and diastolic arterial pressures.

<table>
<thead>
<tr>
<th>Latency</th>
<th>Baseline</th>
<th>1 min</th>
<th>2 min</th>
<th>6 min</th>
<th>12 min</th>
<th>30 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>SC</td>
<td>14.9(1.1)</td>
<td>14.8(1.2)</td>
<td>14.6(0.9)</td>
<td>14.9(1.1)</td>
<td>15.0(1.3)</td>
<td>15.0(1.3)</td>
</tr>
<tr>
<td>P17</td>
<td>17.0(1.0)</td>
<td>17.1(1.2)</td>
<td>17.3(1.3)</td>
<td>17.3(1.2)</td>
<td>17.3(1.2)</td>
<td>17.4(1.4)</td>
</tr>
<tr>
<td>N20</td>
<td>20.2(1.3)</td>
<td>21.1(1.8)*</td>
<td>21.3(1.8)*</td>
<td>21.4(1.8)*</td>
<td>21.4(1.4)*</td>
<td>21.3(1.4)*</td>
</tr>
<tr>
<td>P25</td>
<td>25.2(2.5)</td>
<td>26.2(3.0)*</td>
<td>26.7(2.9)*</td>
<td>26.7(2.9)*</td>
<td>26.6(2.7)*</td>
<td>26.6(2.8)*</td>
</tr>
</tbody>
</table>

| Central conduction time | SC-N20 | 5.3(1.3) | 6.2(1.4)* | 6.1(1.4)* | 6.4(1.3)* | 6.4(1.1)* | 6.2(1.2)* |
| Amplitude           | SC      | 2.7(1.6) | 1.6(0.8)  | 1.4(1.0)  | 1.5(0.8)  | 1.8(1.6)  | 1.5(1.0)  |
|                    | P17     | 1.6(1.2) | 1.0(0.8)  | 1.1(0.9)  | 1.0(0.9)  | 1.2(0.8)  | 1.1(1.0)  |
|                    | N20     | 3.6(2.5) | 2.2(1.7)* | 2.1(1.6)* | 2.3(1.5)* | 2.1(1.4)* | 2.3(1.9)* |
|                    | P25     | 4.4(2.7) | 2.3(1.8)  | 2.3(1.8)  | 2.1(2.3)  | 1.9(1.5)  | 2.2(1.8)  |

<table>
<thead>
<tr>
<th>Physiological variables</th>
<th>HR (beat min⁻¹)</th>
<th>SAP (mm Hg)</th>
<th>DAP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>84(14)</td>
<td>135(16)</td>
<td>76(14)</td>
</tr>
<tr>
<td>1 min</td>
<td>99(23)*</td>
<td>134(19)</td>
<td>78(12)</td>
</tr>
<tr>
<td>2 min</td>
<td>98(19)*</td>
<td>137(18)</td>
<td>70(31)</td>
</tr>
<tr>
<td>6 min</td>
<td>89(2)</td>
<td>123(19)*</td>
<td>80(14)</td>
</tr>
<tr>
<td>12 min</td>
<td>98(25)*</td>
<td>132(20)</td>
<td>79(14)</td>
</tr>
<tr>
<td>30 min</td>
<td>91(35)*</td>
<td>134(17)</td>
<td>73(13)</td>
</tr>
</tbody>
</table>

occurred between the baseline and later times, but were small.

Baseline amplitudes varied between patients and were in the ranges 0.21–6.96 μV (SC), 0.14–4.64 μV (P17), 0.71–11.3 μV (N20) and 0.92–8.78 μV (P25). The amplitudes of all peaks consistently decreased rapidly (within the first 1 min) and reached a stable value by 6–7 min. The final values for the amplitudes were approximately 60% (SC), 70% (P17), 60% (N20) and 45% (P25) of the baseline value. The change in N20 amplitude was statistically significant (P < 0.012).

AP and HR changed little during the study. A transient reduction in systolic arterial pressure with maximal change at 6 min was statistically (P < 0.0001), but not clinically, significant. A sustained increase in HR occurred which also was statistically (P < 0.032), but not clinically, significant.

DISCUSSION

The anaesthetic technique used during evoked potential monitoring should be chosen to allow adequate SSEP signal acquisition. Often, several drugs are used during induction of anaesthesia, each contributing an effect on the SSEP. These effects and their time course should be noted after induction, to permit detection of changes resulting from positioning, temperature, ischaemia or surgery.

The effects of sufentanil on SSEP have not been described previously. Several studies have examined the effect of fentanyl on evoked potentials. McPherson, Sell and Traystman noted an increase in N20 and P23 latencies and a decrease in amplitude after a bolus dose of fentanyl 25 μg kg⁻¹ [10], but they did not specify the time of measurement. These changes are similar to those found with sufentanil in the present study. Loughman and colleagues measured the median nerve SSEP after a bolus dose of fentanyl 200 μg and noted a downward trend in amplitude [11]. After high-dose fentanyl (53.2 μg kg⁻¹), Schubert and co-workers noted an increase in the cortical N20 latency and a decrease in cortical amplitude. However, their patients received diazepam and morphine premedication before the study [12]. Although these studies with fentanyl did not examine closely the early time course after injection, the minimal changes in latency with decreases in amplitude were similar to the changes seen with sufentanil in this study. This similarity in action has been seen in EEG studies [13–15].

Pharmacokinetic studies suggest that sufentanil should have a rapid onset of action in the central nervous system with a peak effect at about 5 min [8]. Sufentanil has a high tissue affinity with rapid, extensive uptake into tissues which may be related to its lipophilic nature. Cooke and Scott used the EEG as a continuous quantitative measure of sufentanil activity and showed that a 125-μg bolus caused a 50% reduction of the
on 27 July 2018 by guest

This study was supported in part by a grant from Janssen.

ACKNOWLEDGEMENT

REFERENCES

13. Cooke JE, Scott JC. Fentanyl vs. sufentanil: The same onset time. Anesthesiology 1987; 67: 3A.