Mid-latency auditory evoked potentials and circulatory response to loud sounds

D. Schwender, R. Haessler, S. Klasing, C. Madler, E. Pöppel and K. Peter

SUMMARY
We investigated in 60 patients scheduled for elective aorto-coronary bypass grafting if loud sounds by themselves can induce cardiovascular responses and if these could be related to mid-latency auditory evoked potentials (MLAEP). Anaesthesia was induced in group I (n = 20) with flunitrazepam–fentanyl 0.01 mg kg⁻¹ and maintained with flunitrazepam–fentanyl 1.2 mg h⁻¹. Patients in groups II (n = 20) and III (n = 20) received etomidate 0.25 mg kg⁻¹ and fentanyl 0.005 mg kg⁻¹ for induction and 0.6–1.2 vol% isoflurane and fentanyl 1.2 mg h⁻¹, or propofol 4–8 mg kg⁻¹ h⁻¹ and fentanyl 1.2 mg h⁻¹ for maintenance of general anaesthesia. After preparation of the sternum the operation was stopped for several minutes. Then, as a loud auditory stimulus, the sound of the running sternotomy saw was presented to the patients by putting the saw inverted on the sternum for several seconds. Heart rate (HR), arterial pressure (SAP), pulmonary capillary wedge pressure (PCWP), cardiac index, systemic vascular resistance and MLAEP were measured in the awake state, before and after presentation of the sound. Latencies of the peak V, Na, Pa, Nb and PI were measured. In group I there were statistically significant increases in HR (63.5–70.2 beat min⁻¹), SAP (123.9–146.5 mm Hg) and PCWP (9.2–11.7 mm Hg) after presentation of the sound. These haemodynamic changes were not observed in patients in groups II and III. In the awake state, AEP had high peak-to-peak amplitudes and a periodic waveform. During general anaesthesia brainstem auditory evoked potentials (BAEP) remained stable. In contrast, MLAEP in groups II and III showed a marked increase in latency and decrease in amplitude or were suppressed completely. In contrast, before and after the presentation of the sound, there was only a slight increase in latency or decrease in amplitude of the MLAEP in patients in group I. Latencies of the early cortical potentials Na and Pa correlated negatively with circulatory responses after presentation of the sound. Loud sounds by themselves may induce circulatory responses during general anaesthesia, when the electrophysiological conditions of primary cortical stimulus processing is preserved. (Br. J. Anaesth. 1994; 72: 307–314)

KEY WORDS

The transduction, transmission and processing of acoustic stimuli from the cochlea to the cortex can be monitored by recording auditory evoked potentials (AEP) [1]. Early components of the AEP are generated mainly in the peripheral auditory pathway and the brainstem (BAEP: brainstem auditory evoked potentials) and represent the process of stimulus transduction and transmission to the midbrain [1]. The mid-latency auditory evoked potentials (MLAEP) occur 10–100 ms after stimulus presentation and are generated by different overlapping areas of the primary auditory cortex [1–5]. They are the electrophysiological correlate of the primary cortical processing of the auditory stimuli. Late latency auditory evoked potentials (LLAEP) reflect neural activity of the association cortex, 100–1000 ms after stimulus onset. They are influenced strongly by processes of stimulus evaluation and cognitive analysis.

The early AEP, generated in the brainstem, remain nearly stable during anaesthesia [6–8]. The late cortical components show a great variation of latencies and amplitudes in the awake state [1, 9]. In contrast, mid-latency peaks of the AEP do not differ intra- and interindividually. Therefore, recordings of MLAEP offer the opportunity to monitor acoustic information processing in the primary auditory cortex during general anaesthesia.

Hyperdynamic cardiovascular responses, such as increases in heart rate, arterial pressure, pulmonary capillary wedge pressure and systemic vascular resistance may be observed during cardiac surgery, especially during sternotomy. These changes do not correlate with the dose of opioid [10–16]. Therefore, inadequate analgesia cannot explain this phenomenon satisfactorily. Furthermore, no correlation was found between circulatory changes and plasma concentrations of endocrine vasoactive substances liberated, for example, from mediastinal structures [13].

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During sternotomy, patients are exposed to the loud sound of the sternotomy saw. This raises the issue of whether or not loud sounds by themselves may contribute to the circulatory response during sternotomy. In this study, we investigated if loud sounds by themselves can induce cardiovascular responses and if these could be related to the primary cortical response to sounds (i.e. MLAEP).

**Patients and Methods**

With local Ethics Committee approval, informed consent was obtained from 60 patients scheduled for elective aorto-coronary bypass grafting. After oral premedication with a benzodiazepine (flunitrazepam 2 mg), 1 h before anaesthesia, patients were allocated randomly to one of three groups. Anaesthesia was induced in group I (n = 20) with flunitrazepam–fentanyl 0.01 mg kg\(^{-1}\) and in groups II (n = 20) and III (n = 20) with etomidate 0.25 mg kg\(^{-1}\) and fentanyl 0.005 mg kg\(^{-1}\). For maintenance of anaesthesia, all patients received high-dose opioid analgesia with fentanyl 1.2 mg h\(^{-1}\). Additionally, patients in group I received flunitrazepam 1.2 mg h\(^{-1}\), group II 0.6–1.2 vol % isoflurane and group III propofol 4–8 mg kg\(^{-1}\) h\(^{-1}\). If required clinically, additional bolus doses of fentanyl 0.5 mg were given. All patients received pancuronium 0.1 mg kg\(^{-1}\) for neuromuscular block. Episodes of hypertension in the period before bypass were treated with nitroglycerine. A three-lead electrocardiogram (ECG) and heart rate (HR), systolic and diastolic arterial pressures (SAP, DAP) via an 18-gauge cannula in the radial artery, central venous pressure (CVP) and pulmonary artery pressure (PAP) with a F7 pulmonary artery catheter via the internal jugular vein were measured continuously. Pulmonary capillary wedge pressure (PCWP) and cardiac output (CO) were measured intermittently. CO values were derived from the mean of triplicate measurements using the thermodilution method with an in-line injectate temperature probe. Systemic vascular resistance (SVR) and cardiac index (CI) were calculated.

After preparation of the sternum the operation was stopped for several minutes. Then, as a loud auditory stimulus, the sound of the sternotomy saw was presented to the patients by placing the inverted running saw on the sternum for several seconds.

The electrodiagnostic system Pathfinder I (Nicolet Instruments) was used for auditory stimulation, recording and analysis of evoked potentials. Rarefaction clicks of 0.1-ms duration and 70 dB above the normal hearing level were presented binaurally with a stimulation frequency of 9.3 Hz using acoustically shielded earphones (TDH 39). For recording, silver electrodes were positioned at Cz and A\(_{1}/A_{2}\) with Fpz as ground (according to the international 10–20 system). The impedance of all electrodes was maintained less than 0.5 kΩ. An epoch of 100 ms (bin width 0.2 ms) was bandpass filtered (1–1500 Hz) with an analogue Butterworth filter (roll-off 6 dB/octave) and averaged across 1000 stimulus presentations. The recording procedure was controlled visually on a monitor, and an automatic artefact detector rejected signals greater than 96% of full scale. To guarantee reliability of the signal and correct transmission and transduction of the auditory stimuli, evoked potentials without a brainstem response (peak V) were rejected also. Off-line data analysis was as follows: latencies of the peaks V, Na, Pa, Nb and P1 were measured. Measurements of HR, SAP, PCWP, CI, SVR and MLAEP were performed on three occasions: in the awake state; during operation under steady state conditions (preparation of the sternum) before presentation of the sound; and after operation was stopped and the sound was presented.

**Statistical Analysis**

Results are presented as mean (SD). For the haemodynamic data and the peak latencies V, Na, Pa, the Wilcoxon test was used within groups and the Kruskal–Wallis test between groups. Correlation of haemodynamic data and changes of AEP latencies were calculated as Spearman’s rank correlation. \(\alpha < 5\%\) (\(P < 0.05\)) was considered statistically significant. Statistical analysis included Bonferroni’s correction (\(\alpha\) adjustment).

**Results**

The three groups were comparable in patient characteristics and cardiovascular data before operation. The total amount of fentanyl given until the presentation of the sound was different between the groups. Patients in group I received more fentanyl (0.02 mg kg\(^{-1}\)) than patients in groups II and III (0.01 mg kg\(^{-1}\)). These differences between groups I and II and groups I and III were statistically significant (\(P < 0.001\)). Furthermore, 10 patients in group I, no patient in group II and one patient in group III required antihypertensive medication (nitroglycerine 0.01 (0.006) mg/kg body weight) after skin incision and before presentation of the sound.

Mean (SD) haemodynamic data for the three groups are shown in Table I for the following situations: wake; before presentation of the sound during operation under steady state conditions (preparation of the sternum); and after presentation of the sound. There were no differences in haemodynamic baseline values of the awake patients between the three groups. During general anaesthesia, and under steady state conditions, SAP, DAP, HR, PCWP and CI remained stable or were reduced slightly and SVR increased slightly compared with the awake state. The sound of the running sternotomy saw caused a significant increase in HR from 63.5 to 76.5 (\(P < 0.001\)) SAP from 123.9 to 146.5 mm Hg (\(P < 0.004\)) and PCWP from 9.2 to 11.7 mm Hg (\(P < 0.01\)) in patients in group I. DAP, CI and SVR also showed slight increases in mean values which did not reach statistical significance. In contrast, no statistically significant increases in HR, SAP, DAP, PCWP, SVR and CI were observed in groups II and III after presentation of the sound. These changes in haemodynamic variables after presentation of the sound were significantly different between groups I and II and groups I and III (\(P < 0.01\)).
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Table 1. Mean (sd) haemodynamic data (heart rate (HR), systolic (SAP) and diastolic (DAP) arterial pressures, pulmonary capillary wedge pressure (PCWP), cardiac index (CI) and systemic vascular resistance (SVR)) for the three groups in the following situations: awake; before presentation of the sound during operation under steady state conditions (preparation of the sternum); and after presentation of the sound. *P < 0.05 compared with before presentation of the sound

<table>
<thead>
<tr>
<th></th>
<th>Flunitrazepam—fentanyl group</th>
<th>Isoflurane—fentanyl group</th>
<th>Propofol—fentanyl group</th>
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<tr>
<td><strong>HR (beat min⁻¹)</strong></td>
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<tr>
<td>Awake</td>
<td>70.61 (16.26)</td>
<td>67.95 (19.58)</td>
<td>66.10 (13.25)</td>
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<tr>
<td>Before sound</td>
<td>63.48 (13.56)</td>
<td>65.52 (19.55)</td>
<td>61.60 (9.91)</td>
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<tr>
<td>After sound</td>
<td>70.24 (14.25)*</td>
<td>67.19 (27.67)</td>
<td>64.90 (11.41)</td>
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<td><strong>SAP (mm Hg)</strong></td>
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<tr>
<td>Awake</td>
<td>147.57 (21.86)</td>
<td>139.00 (14.88)</td>
<td>138.40 (17.63)</td>
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<tr>
<td>Before sound</td>
<td>123.95 (17.45)</td>
<td>128.76 (24.90)</td>
<td>132.30 (17.07)</td>
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<tr>
<td>After sound</td>
<td>146.52 (20.32)*</td>
<td>128.67 (24.50)</td>
<td>134.50 (15.99)</td>
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<td><strong>DAP (mm Hg)</strong></td>
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<tr>
<td>Awake</td>
<td>75.48 (9.75)</td>
<td>75.50 (12.52)</td>
<td>71.90 (10.37)</td>
</tr>
<tr>
<td>Before sound</td>
<td>70.35 (13.28)</td>
<td>70.10 (9.57)</td>
<td>76.70 (11.73)</td>
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<tr>
<td>After sound</td>
<td>75.19 (10.89)</td>
<td>69.76 (9.15)</td>
<td>73.90 (12.04)</td>
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<td><strong>PCWP (mm Hg)</strong></td>
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<tr>
<td>Awake</td>
<td>11.20 (4.47)</td>
<td>9.89 (4.84)</td>
<td>9.84 (3.88)</td>
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<tr>
<td>Before sound</td>
<td>9.22 (3.77)</td>
<td>7.21 (3.94)</td>
<td>7.75 (2.90)</td>
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<tr>
<td>After sound</td>
<td>11.17 (7.30)*</td>
<td>8.53 (3.52)</td>
<td>9.45 (4.05)</td>
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<td><strong>CI (litre min⁻¹ m⁻²)</strong></td>
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<tr>
<td>Awake</td>
<td>2.81 (0.74)</td>
<td>2.61 (0.64)</td>
<td>2.29 (0.45)</td>
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<tr>
<td>Before sound</td>
<td>2.27 (0.57)</td>
<td>2.07 (0.40)</td>
<td>2.16 (0.55)</td>
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<tr>
<td>After sound</td>
<td>2.46 (0.66)</td>
<td>1.99 (0.45)</td>
<td>2.17 (0.59)</td>
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<td><strong>SVR (dyn s cm⁻⁵)</strong></td>
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<tr>
<td>Awake</td>
<td>1433.7 (455.1)</td>
<td>1561.1 (331.7)</td>
<td>1642.5 (317.4)</td>
</tr>
<tr>
<td>Before sound</td>
<td>1596.8 (457.4)</td>
<td>1731.5 (446.4)</td>
<td>1865.0 (359.4)</td>
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<tr>
<td>After sound</td>
<td>1674.9 (490.8)</td>
<td>1842.7 (564.3)</td>
<td>1913.4 (485.1)</td>
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Auditory evoked potential

**BAEP**

**MLAE**

**FIG. 1. Original tracing of an auditory evoked potential (AEP) of an awake patient. V belongs to the brainstem generated potentials (BAEP), which demonstrates that auditory stimuli were transduced correctly. Na, Pa, Nb, P1 (MLAE) are generated in the primary auditory cortex of the temporal lobe. The mid-latency auditory evoked potential (MLAE) has a characteristic periodic waveform.**

Figure 1 shows an original tracing of an AEP of an awake patient. V belongs to the brainstem generated potentials (BAEP), which demonstrates that auditory stimuli were transduced correctly [1]. Na, Pa, Nb, P1 (MLAE) are generated in the primary auditory cortex of the temporal lobe. The mid-latency auditory evoked potential (MLAE) has a characteristic periodic waveform.

The same picture was seen in the interindividual grand averages of the individual AEP of the three groups (fig. 3A-C). Generally, the large average rates (greater signal to noise ratio) and the interindividual differences, especially in the later latency range (P1), led to a slight flattening of the potential. In every upper trace the AEP of the awake patients can be seen. BAEP can be identified easily. MLAEP showed high amplitudes and a periodic waveform. During general anaesthesia in all patients, BAEP can be identified as similar to that in the awake state. In contrast, in patients in groups II and III, MLAEP showed markedly increased latencies and decreased amplitudes or were suppressed completely. MLAEP in patients in group I were different during general anaesthesia. There were only slight increases in latencies and decreases in amplitudes. The early cortical potentials, Na, Pa and Nb especially were similar to the awake state.
FIG. 2. Original tracings of AEP are shown for two patients in the flunitrazepam–fentanyl group (A), two patients of the isoflurane–fentanyl group (B) and two patients of the propofol–fentanyl group (C) for the following situations: awake; before presentation of the sound during sternum preparation (Before); and after presentation of the sound (After). In the AEP of the awake patients, BAEP can be identified easily. MLAEP show high amplitudes and periodic waveform. During general anaesthesia in all patients, BAEP are similar to those in the awake state. In contrast, in patients in groups II and III, MLAEP show markedly increased latencies and decreased amplitudes or are suppressed completely. MLAEP in patients of group I are different during general anaesthesia. There are only slight increases in latencies and decreases in amplitudes.

FIG. 3. Interindividual grand averages of the individual AEP in the flunitrazepam–fentanyl group (A), the isoflurane–fentanyl group (B) and the propofol–fentanyl group (C). In the AEP of the awake patients, BAEP may be identified in every potential. MLAEP show large peak-to-peak amplitudes and a periodic waveform. During general anaesthesia in all patients, BAEP remained stable. In contrast, MLAEP showed markedly increased latencies and decreased amplitudes or complete suppression in patients in groups II and III, whereas MLAEP of patients in group I showed only slight increases in latencies and decreases in amplitudes. The early
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Table 1. Mean (SD) latencies of the peaks V, Na and Pa for the three groups while awake and before and after presentation of the sound. *P < 0.05 compared with awake state

<table>
<thead>
<tr>
<th></th>
<th>Flunitrazepam-fentanyl group</th>
<th>Isoflurane-fentanyl group</th>
<th>Propofol-fentanyl group</th>
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<tbody>
<tr>
<td>V (ms)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Awake</td>
<td>6.89 (0.85)</td>
<td>6.45 (0.45)</td>
<td>6.73 (0.80)</td>
</tr>
<tr>
<td>Before sound</td>
<td>7.05 (1.39)</td>
<td>7.09 (0.76)</td>
<td>6.77 (0.46)</td>
</tr>
<tr>
<td>After sound</td>
<td>7.15 (1.26)</td>
<td>7.19 (0.37)*</td>
<td>6.82 (0.50)</td>
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<tr>
<td>Na (ms)</td>
<td></td>
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</tr>
<tr>
<td>Awake</td>
<td>19.13 (1.44)</td>
<td>19.54 (2.40)</td>
<td>19.21 (3.14)</td>
</tr>
<tr>
<td>Before sound</td>
<td>21.91 (2.81)*</td>
<td>44.86 (16.26)*</td>
<td>53.97 (23.06)*</td>
</tr>
<tr>
<td>After sound</td>
<td>21.74 (2.12)*</td>
<td>43.91 (17.68)*</td>
<td>58.57 (17.56)*</td>
</tr>
<tr>
<td>Pa (ms)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Awake</td>
<td>31.55 (2.91)</td>
<td>31.72 (2.47)</td>
<td>31.39 (2.10)</td>
</tr>
<tr>
<td>Before sound</td>
<td>35.45 (3.77)*</td>
<td>70.06 (24.12)*</td>
<td>75.70 (26.38)*</td>
</tr>
<tr>
<td>After sound</td>
<td>36.75 (4.00)*</td>
<td>63.67 (23.52)*</td>
<td>83.83 (19.71)*</td>
</tr>
</tbody>
</table>

Fig. 4. Correlation between changes in latencies (ΔPa = the difference in latency of the early cortical potential, Pa, during anaesthesia minus Pa awake) with changes in arterial pressure (ΔSAP = systolic arterial pressure after presentation of the sound minus systolic arterial pressure before presentation of the sound). Systolic arterial pressure remained stable when Pa latencies increased markedly. In contrast, increases in arterial pressure were observed only when Pa increased only slightly during general anaesthesia. This correlation was statistically significant (P < 0.001).

DISCUSSION

Mean latencies of peaks V, Na and Pa correspond well with peak latencies obtained under experimental conditions [1]. No anaesthetic agent blocked stimulus transformation, stimulus conduction and processing in the brainstem. Only isoflurane caused a slight prolongation of the brainstem components of a few milliseconds. In contrast with brainstem responses, latencies of Na and Pa increased significantly and peak-to-peak amplitudes were reduced with isoflurane and propofol. The early cortical potentials Na and Pa were suppressed completely. These results indicate that general anaesthesia with isoflurane or propofol blocked stimulus transmission at the level of the midbrain.

A similar increase in latencies and decrease in amplitudes of the MLAEP has been found also with halothane [7], enflurane [7], etomidate [8], propofol [17] and Althesin [18]. There was also a dose-related effect with isoflurane [6]. Results obtained from sensory evoked potentials measured in other modalities are in accordance with these findings. The early and late cortical components of visual and somatosensory evoked potentials are suppressed during anaesthesia with isoflurane, enflurane and halothane [19-24]. These results suggest that volatile anaesthetic agents induce general suppression of afferent conduction and processing within different sensory channels in the central nervous system.

In contrast with volatile anaesthetic agents, anaesthesia with a combination of flunitrazepam and fentanyl had a different effect on auditory evoked potentials. There were only slight increases in latencies of the MLAEP and amplitudes were cortical potential, Pa, during anaesthesia, minus Pa awake) with changes in arterial pressure (ΔSAP = systolic arterial pressure after presentation of the sound minus systolic arterial pressure before presentation of the sound). Systolic arterial pressure was stable when Pa latencies were increased markedly. In contrast, increases in arterial pressure were observed only when Pa increased only slightly during general anaesthesia. This correlation was statistically significant (P < 0.001).

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reduced only slightly. The early cortical potentials Na and Pa remained nearly unchanged. We conclude that the neural generators in the primary auditory cortex of the temporal lobes are not suppressed completely and cortical processing of auditory stimuli remains intact to some extent [1–5].

There is also evidence that evoked potentials of other sensory modalities are not suppressed by these anaesthetic agents. Administration of diazepam, for example, results in a slight reduction in amplitudes of visually evoked potentials (VEP), without an increase in peak latencies [25–27]. In addition, latencies of subcortical and cortical responses of the somatosensory evoked potentials also remain unchanged with benzodiazepines [21, 23, 26–28].

Kileny, Dobson and Gelfand [29] investigated the effect of hypothermia on auditory evoked potentials during cardiac surgery. They showed that increasing hypothermia and reduced perfusion pressure induced prolongation of peak latencies of MLAEP, whereas high-dose opioid analgesia did not change MLAEP or brainstem generated components [29, 30]. In accordance with these results, somatosensory and visual evoked potentials are known to remain stable with opioids [21, 23, 26, 31].

In the present study, we demonstrated that increases in heart rate, systolic arterial pressure and pulmonary capillary wedge pressure were induced during general anaesthesia by loud auditory stimuli (the running sternotomy saw). Hyperdynamic circulatory responses were observed predominantly in the flunitrazepam–fentanyl group. Furthermore, 10 of 20 patients in the flunitrazepam–fentanyl group were already receiving antihypertensive medication after skin incision, but before investigation of the effects of auditory stimulation. Therefore, haemodynamic responses were blocked, at least partially, in these 10 patients and one might expect that responses to auditory stimulation would have been even more marked in a uniform group of patients receiving flunitrazepam–fentanyl without antihypertensive medication.

Inadequate analgesia as a possible explanation for cardiovascular responses in this situation can probably be excluded for two major reasons. First, the patients had no further nociceptive stimulus when the sound was presented, as surgical stimulation was discontinued for this period. Second, the patients in the flunitrazepam–fentanyl group had received a total dose of fentanyl 20 μg kg⁻¹ when the sound was presented, while a dose of 6 μg kg⁻¹ is thought sufficient to allow toleration of surgical stimuli or tracheal intubation [32, 33]. Furthermore, no cardiovascular responses were observed in the isoflurane or propofol groups, who had received only fentanyl 10 μg kg⁻¹ up to that moment.

Hypertension, tachycardia and myocardial ischaemia may be observed frequently during cardiac surgery, especially during sternotomy. Therefore, many investigators have studied this period. Waller, Hug and Nagle found that fentanyl 60 μg kg⁻¹ alone produced no significant haemodynamic changes. However, during operation and especially during sternotomy, fentanyl 60 μg kg⁻¹ could not reliably prevent significant increases in arterial pressure, systemic vascular resistance, cardiac work and myocardial oxygen consumption [16]. Sonntag and colleagues gave fentanyl 100 μg kg⁻¹ at induction of anaesthesia and observed no haemodynamic changes, but significant increases in arterial pressure, systemic vascular resistance, cardiac work and myocardial oxygen consumption and myocardial lactate production were seen during sternotomy [14]. After sufentanil 40 μg kg⁻¹, increases in arterial pressure, systemic vascular resistance, pulmonary capillary wedge pressure and myocardial lactate production and a decrease in cardiac index, in addition to myocardial ischaemia, were observed in more than 50% of patients during or shortly after sternotomy [15]. Philbin and co-workers studied haemodynamic changes and endocrine stress responses during sternotomy with different doses of fentanyl (50–100 μg kg⁻¹) and sufentanil (10–40 μg kg⁻¹) [13]. In every group, approximately 50% of patients showed significant increases in arterial pressure and increased plasma concentrations of renin and aldosterone. The haemodynamic changes were correlated neither with the doses of opioid nor with plasma concentrations of endocrine stress hormones. It is important to note that in every case the doses of opioid in Philbin’s study exceeded the doses required to achieve surgical tolerance.

The incidence of myocardial ischaemia during cardiac surgery was investigated by Kotter and colleagues [12]. The highest incidence was found with high-dose opioids alone (20–25%), the lowest (2–3%) when opioids were combined with volatile anaesthetics (isoflurane). Hyperdynamic circulatory responses were not observed when opioids were supplemented with volatile agents. A combination of fentanyl 32 μg kg⁻¹ and 0.74% isoflurane led to decreased arterial pressure and stroke volume compared with awake values. Heart rate, arterial pressure, pulmonary capillary wedge pressure and systemic vascular resistance did not increase during sternotomy [34]. Furthermore, cardiovascular responses and coincident myocardial ischaemia during sternotomy could be attenuated using isoflurane to supplement fentanyl analgesia [35]. With pacemaker-induced tachycardia, the ischaemia threshold increased when isoflurane was used [36].

Several studies in cardiac surgical patients have compared high-dose opioid analgesia with balanced anaesthetic techniques (opioids and volatile anaesthetics). In several studies, arterial pressure and systemic vascular resistance increased and cardiac index decreased during sternotomy, predominantly in the high-dose opioid groups. The percentage increase in myocardial lactate production in the opioid groups was three times that in the balanced anaesthesia groups [10, 11]. Using enflurane or isoflurane as sole anaesthetics for cardiac surgery, some investigators have observed marked haemodynamic stability during sternotomy. In contrast, high-dose isoflurane caused increased myocardial lactate production, which was thought to be the result of coronary steal phenomenon [37–39].

Several explanations have been proposed for the haemodynamic, myocardial and endocrine stress
perception during anaesthesia is low. Clinical importance of the phenomenon of auditory anaesthetics such as isoflurane or propofol, the anaesthesia. When opioids are combined with general anaesthesia, cardiovascular changes when auditory stimulus processing may contribute to the primary auditory cortex were suppressed, cardiovascular changes could not be evoked by loud sounds. We conclude that inadequate suppression of auditory stimulus processing may contribute to the hyperdynamic cardiovascular reactions during anaesthesia. It is a general experience that loud sounds may cause panic, fear and restlessness in the awake individual which may be accompanied by increases in heart rate and arterial pressure. It is particularly noteworthy that there was a correlation between hyperdynamic changes after loud sounds and MLAEP. During anaesthesia with flunitrazepam-fentanyl, a loud auditory stimulus induced increases in heart rate, arterial pressure and pulmonary capillary wedge pressure and MLAEP showed a similar pattern to that in the awake state. This implies that during flunitrazepam-fentanyl anaesthesia, auditory stimuli could be processed to some extent at a cortical level. In contrast, with isoflurane-fentanyl and propofol-fentanyl, when MLAEP and auditory stimulus processing in the primary auditory cortex were suppressed, cardiovascular changes could not be evoked by loud sounds.

We conclude that inadequate suppression of auditory stimulus processing may contribute to the hyperdynamic changes during sternotomy. At this time there is only one case report in the literature on coincidental auditory perception and a hypertensive crisis during anaesthesia with diazepam-fentanyl. Our study indicates that auditory stimuli may induce cardiovascular changes when auditory stimulus processing in the primary auditory cortex is not blocked sufficiently. This is relevant clinically only when receptor specific drugs, such as opioids and opioids combined with benzodiazepines, are used for anaesthesia. When opioids are combined with general anaesthetics such as isoflurane or propofol, the clinical importance of the phenomenon of auditory perception during anaesthesia is low.

REFERENCES


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