# Attenuation of the haemodynamic response to placement of the Mayfield skull pin head holder: alfentanil versus scalp block

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**Key words:**
Brain; anaesthesia; Mayfield skull pin head holder, haemodynamic response, attenuation; scalp nerve block versus alfentanil.

**Summary**
*Introduction: Application of the Mayfield clamp causes a significant haemodynamic response. Different methods have been used to attenuate this response. We compared two of these methods, namely alfentanil bolus (Group A) and nerve block of the scalp (Group B). METHOD: Twenty-two patients entered the study. Anaesthesia was standardised using thiopental, sufentanil, vecuronium, isoflurane, oxygen and air. Group A patients received alfentanil 10 mg kg$^{-1}$ 90 seconds before clamp placement and group B patients received a scalp block with lignocaine 4-5 mg kg$^{-1}$ as a 1% solution after intubation . Blood pressure and pulse rate were recorded before, during and 30 s, 60 s, 120 s, 240 s and 480 s after clamp placement. RESULTS: For group A, the mean maximum changes in systolic, diastolic and mean arterial blood pressure, and heart rate were, 34%, 39%, 35% and 20% respectively. The corresponding values for Group B were 9% (p=0,004), 16% (p=0,009), 13% (p=0,0066) and 10% (p=0,0901) respectively. CONCLUSION: The scalp block is significantly more effective in attenuating the blood pressure response to clamp placement (p<0.05).*

During neurosurgery, the head often needs to be stabilised by means of the Mayfield skull pin head holder or clamp. This clamp consists of a C-shaped metal piece with three sharp interchangeable metal pins, arranged triangularly to one another. These pins are forced through the layers of the scalp and periosteum into the external lamina of the skull, by manually pushing the two arms of the C-clamp towards each other and tightening it with a calibrated pressure screw. Despite the fact that placement of the clamp is always done under general anaesthesia, patients show a significant hypertensive response to this stimulus.[1 2 3 4]

Increased arterial blood pressure may cause a rise in intracranial pressure. Patients with intracranial pathology have abnormal auroregulation of cerebral blood flow and are particularly prone to increased intracranial pressure when systemic arterial pressure rises.[5] The patients with intracranial vascular lesions are at increased risk, because an abrupt increase in arterial blood pressure can cause rupture of a thin walled lesion. Schutta has shown experimentally that arterial hypertension can lead to acute cerebral oedema and herniation of the brain within two minutes.[6] In patients with intracranial vascular lesions (cerebral aneurysms or arterio-venous malformations), an acute increase of blood pressure may cause rupture or rerupture and present with subarachnoid or intracerebral haemorrhage. Not only can an acute increase in blood pressure disrupt the intracranial milieu, but also cause extracranial complications, for example, pulmonary oedema. [7]

Shapiro and colleagues studied the changes in intracranial pressure (ICP) in patients who had normal or increased ICP's preoperatively. The latter group showed acute elevations in ICP ranging from 6 to 43 mmHg during application of the Mayfield clamp. The patients with normal ICP have shown no marked increase in ICP. Changes in mean arterial blood pressure (MAP) in both groups were similar and reported to be of "moderate magnitude".[8] The change in ICP in the group with high ICP was most probably due to lack of volume compensation and of ICP autoregulatory mechanisms.

The response to clamp placement must be attenuated. This

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can be achieved by interrupting the sensory conduction somewhere along the sensory pathway or by intravenous agents. The intravenous agents that have been used for this purpose include the potent opioid alfentanil. Alfentanil has a rapid onset of action with a peak effect within 1 to 2 minutes. The short distribution half life may explain the lack of hypotension after the stimulus has subsided.[1] A dose of 10 mg $kg^{-1}$ 180 seconds before clamp placement has proved to be highly effective to attenuate the haemodynamic response. A dose of 10 mg $kg^{-1}$ has been found to suppress noxious stimuli during anaesthesia.[9]

Other drugs that have been used include esmolol,[1] and clonidine.[10] Clonidine 3 mg $kg^{-1}$ has been given intravenously 10 minutes before induction of anaesthesia. MAP increased less in the clonidine group, but intracranial pressure (ICP) increased in both clonidine and placebo patients. This resulted in a cerebral perfusion pressure of less than 60 mmHg in 66% of patients.[10]

A deeper level of anaesthesia has been used, either by increasing the halothane concentration, or by administering thiopental (100 to 200 mg) and fentanyl (50 to 100 mg). In all these patients the mean increase in MAP was 62% within 30 to 60 seconds.[11] The anaesthetic vapours impair autoregulation of cerebral blood flow (CBF). Increasing the concentration of these drugs causes a drop in arterial blood pressure and CBF decreases progressively as autoregulation is lost. It is accepted that autoregulation is maintained with isoflurane up to a concentration of 1,5 MAC.[12] Therefore, increasing vapour concentration is not recommended to attenuate the clamp response.

Local infiltration of the pin area has proved to be effective, provided that the pins were placed exactly into the infiltrated areas. If one or more of the pins were placed outside these areas, the same response occurred as in controls.[13 14]

The well-defined anatomy of the nerves to the scalp[15 16] provides for a relatively simple block.[17] A scalp block using bupivacaine 0,5% and done 5 minutes before pinning was effective in blocking the response.[18] The scalp block has the advantages that pins can be placed anywhere in the scalp, and that the pins can be repositioned if necessary.

The aim of this study was to compare the efficacy of two methods, namely alfentanil bolus (Group A) and nerve block of the scalp (Group B) to attenuate the haemodynamic response to placement of the Mayfield skull pin head holder.

## Subjects and methods

This was a prospective, open, randomised, parallel, comparative study. Approval to conduct the study was obtained from the Ethics Committee of the University of Pretoria. The study sample consisted of 22 ASA I or ASA II patients with a Glasgow Coma Scale count of more than 12/15, scheduled for any procedure in which the Mayfield clamp would be used. Besides the ASA status of the patient, no other exclusion criteria (age, preoperative treatment) were applied as a patient was, within the groups (A or B), his or her own control (relative change in own blood pressure and pulse rate[a]). The sample of 22 patients gave a power of 80% and significance level of 0.05, based on the assumption that a change of at least 15% in blood pressure or pulse rate is clinically significant. Patients were randomly divided into two groups: Group A (n=11) patients received an intravenous bolus of alfentanil, and Group B (n=11) patients received a nerve block of the scalp. For ethical reasons, a control group was not included. The study was not blinded for obvious reasons.

Patients in both groups were premedicated with midazolam 7.5 mg one hour preoperatively. An intravenous canula was placed and Lactated Ringer solution 15 ml $kg^{-1}$ was infused before induction. A canula was inserted under local anaesthesia into the nondominant radial artery at the wrist prior to induction. Anaesthesia was induced with sufentanil 0.2 mg $kg^{-1}$ and sodium thiopental 3 to 5 mg $kg^{-1}$. This was followed by vecuronium 0.15 mg $kg^{-1}$. Patients were ventilated with isoflurane 1.5 MAC end tidal concentration (ETC) in oxygen 100% and intubated when the neuromuscular response to the train of four stimulus was 0/4. Patients were ventilated with oxygen and air. Arterial $PCO_2$ was kept between 33 and 37 mmHg. Anaesthesia was maintained with isoflurane 1 MAC ETC. A central venous catheter, urinary catheter and, when necessary, a lumber drain were inserted. Positioning of the head for application of the clamp took place approximately 20 minutes after intubation.

Group A patients received an alfentanil bolus of 10 mg $kg^{-1}$ 90 seconds before clamp placement. In Group B a scalp block was performed with lignocaine 4 to 5 mg $kg^{-1}$ as a 1% solution (without adrenaline) after intubation. The clamp screws were tightened over a period of approximately 10 seconds.

The scalp block: The supraorbital and supratrochlear nerves are blocked from the point where the supraorbital artery can be palpated above the eyebrow, to the medial end of the eyebrow. The auricular-temporal nerve is blocked 1.5 cm anterior to the ear at the level of the tragus. The lesser occipital and greater auricular nerves are blocked from a point 1.5 cm posterior to the ear at the level of the tragus in the direction of the occiput. The greater and third occipital nerves are infiltrated along the superior nuchal line approximately midway between the external occipital protuberance and the mastoid process. Palpation of the occipital artery helps to locate the adjacent greater occipital nerve.

Base line (BASE) systolic blood pressure (SAP), MAP, diastolic blood pressure (DAP), and heart rate (HR) were recorded while preparing for clamp placement and before the alfentanil bolus (Group A), during clamp placement, and 30, 60, 120, 240 and 480 seconds thereafter. A change in blood pressure and heart rate of 15% (in relation to the blood pressure and heart rate before placement of the clamp) was considered significant. Hereafter, the anaesthesiologist could use the anaesthetic technique of his or her choice.

Blood pressure and heart rate were measured before (baseline), during and 30, 60, 120, 240 and 480 seconds after head clamping. The groups were compared at different times, with respect to the changes relative to the baseline measurement. The maximum change during the study period of 480 seconds, as well as the overall response, expressed by the area under the curve (AUC), was recorded. Comparison was done using an appropriate t-test, taking into account whether the group had equal variance or not, as assessed by Bartlett's test for equal variance. In view of the small sample, the comparison was also confirmed employing the non-parametric Mann-Whitney U-test. All testing was done at the 0,05 level of significance. To facilitate interpretation, the 95% confidence intervals for the differences between the means of the two groups were calculated for the relative as well as for the maximum change. For the AUC, the 95% confidence interval for the ratio of alfentanil relative to the scalp block was calculated.

[a] Relative change in blood pressure or heart rate = (value after placement of clamp/value before placement of clamp)

**Table I Mean (SD) for observed SAP, DAP, MAP (mmHg), and heart rate (HR) at different times for Group A and Group B**

| *Group* | *Time* | *BASE* | *30 s* | *60 s* | *120 2* | *240 s* | *480s* |
|---|---|---|---|---|---|---|---|
| A | SAP | 105,5 (12,2) | 132,2 (26,6) | 139,7 (33,0) | 134,9 (26,3) | 122,1 (18,9) | 110,6 (20,9) |
| B | | 107,7 (17,7) | 113,8 (18,7) | 113,0 (19,6) | 113,3 (21,8) | 108,9 (19,8) | 107,2 (17,2) |
| A | DAP | 61,7 (7,4) | 79,1 (14,4) | 81,3 (13,0) | 78,6 (8,9) | 70,3 (7,5) | 62,9 (12,0) |
| B | | 65,3 (11,5) | 72,5 (9,8) | 71,6 (12,1) | 69,3 (11,4) | 65,9 (10,7) | 64,3 (13,3) |
| A | MAP | 76,8 (8,0) | 97,1 (17,7) | 100,8 (18,7) | 97,6 (13,4) | 87,3 (9,7) | 79,3 (13,10 |
| B | | 79,3 (12,5) | 86,2 (12,0) | 85,5 (14,2) | 84,0 (14,2) | 80,2 (12,8) | 78,5 (13,8) |
| A | HR | 70,2 (15,1) | 80,9 (16,5) | 81,5 (16,6) | 78,9 (17,2) | 75,4 (15,2) | 72,0 (17,0) |
| B | | 70,9 (11,5) | 76,6 (13,9) | 76,2 (14,9) | 74,6 (14,6) | 72,1 (11,8) | 69,0 (9,8) |

## Results

Descriptive statistics, mean and standard deviation, for the observed heart rate and blood pressures, over time and by group are reported in Table I. In Table II the descriptive statistics for the ratios with respect to baseline are given along with the p-values for the relevant t-tests (Student / Welch) and the 95% confidence intervals for the group means as well as for the differences between group means. Similar to Tables II, III and IV, report on maximum values over time and area under the curve (AUC).

In the alfenanil group the mean maximum change for SAP, DAP, MAP and heart rate was 34 mmHg (34%), 20 mmHg (39%), 24 mmHg (35%) and 11 beats/minute (13%), respectively. The corresponding values for the scalp block was 6 mmHg (9%), 8 mmHg (16%), 7 mmHg (13%) and 6 beats/minute (10%), respectively. A significant difference was demonstrated between the two groups considering the mean maximum change for SAP (p=0.004), DAP (p=0.009) and MAP (p=0.006). The difference in the mean maximum change in heart rate was only marginally not significant

**Table II Group comparison for ratio to baseline for Group A (n=10) and Group B (n=11)**

| Variable | Group | Mean | SD | 95%CI for Group mean | 95% CI for difference | p-value |
|---|---|---|---|---|---|---|
| RSAP30 | A | 1.248 | 0.158 | 1.135; 1.361 | 0.069; 0.309 | 0.0047* |
| | B | 1.059 | 0.080 | 1.006; 1.112 | | |
| RSAP60 | A | 1.316 | 0.192 | 1.179; 1.454 | 0.123; 0.410 | 0.0016* |
| | B | 1.050 | 0.085 | 0,993; 1.107 | | |
| RSAP120 | A | 1.276 | 0.170 | 1.154; 1.398 | 0.097; 0.355 | 0.0023* |
| | B | 1.050 | 0.086 | 0.992; 1.107 | | |
| RSAP240 | A | 1.162 | 0.170 | 1.040; 1.284 | 0.027; 0.278 | 0.0216* |
| | B | 1.010 | 0.060 | 0.969; 1.050 | | |
| RSAP480 | A | 1.052 | 0.192 | 0.915; 1.189 | –0.095; 0.200 | 0.4580 |
| | B | 0.999 | 0.107 | 0.927; 1.071 | | |
| RDAP30 | A | 1.286 | 0.224 | 1.126; 1.446 | –0.0075; 0.330 | 0.0600 |
| | B | 1.125 | 0.140 | 1.030; 1.219 | | |
| RDAP60 | A | 1.325 | 0.190 | 1.189; 1.461 | 0.072; 0.367 | 0.0057* |
| | B | 1.106 | 0.129 | 1.019; 1.193 | | |
| RDAP120 | A | 1.285 | 0.174 | 1.161; 1.410 | 0.086; 0.348 | 0.0026* |
| | B | 1.068 | 0.109 | 0.995; 1.141 | | |
| RDAP240 | A | 1.146 | 0.117 | 1.063; 1.230 | 0.043; 0.222 | 0.0069* |
| | B | 1.014 | 0.064 | 0.971; 1.057 | | |
| RDAP480 | A | 1.017 | 0.143 | 0.915; 1.119 | –0.080; 0.125 | 0.5660 |
| | B | 0.986 | 0.099 | 0.919; 1.053 | | |
| RMAP30 | A | 1.263 | 0.189 | 1.128; 1.339 | 0.029; 0.308 | 0.0206* |
| | B | 1.095 | 0.110 | 1.021; 1.169 | | |
| RMAP60 | A | 1.314 | 0.190 | 1.178; 1.449 | 0.092; 0.371 | 0.0025* |
| | B | 1.082 | 0.109 | 1.008; 1.155 | | |
| RMAP120 | A | 1.276 | 0.157 | 1.163; 1.388 | 0.093; 0.334 | 0.0015* |
| | B | 1.062 | 0.104 | 0.993; 1.132 | | |
| RMAP240 | A | 1.146 | 0.135 | 1.050; 1.243 | 0.031; 0.235 | 0.0144* |
| | B | 1.013 | 0.065 | 0.970; 1.057 | | |
| RMAP480 | A | 1.033 | 0.151 | 0.925; 1.141 | –0.073; 0.157 | 0.4560 |
| | B | 0.992 | 0.098 | 0.926; 1.057 | | |
| RHR30 | A | 1.165 | 0.152 | 1.056; 1.274 | –0.033; 0.199 | 0.149 |
| | B | 1.082 | 0.099 | 1.016; 1.148 | | |
| RHR60 | A | 1.176 | 0.173 | 1.052; 1.300 | –0.027; 0.236 | 0.109 |
| | B | 1.072 | 0.087 | 1.013; 1.130 | | |
| RHR120 | A | 1.131 | 0.110 | 1.052; 1.210 | –0.016; 0.175 | 0.096 |
| | B | 1.051 | 0.099 | 0.985; 1.118 | | |
| RHR240 | A | 1.080 | 0.066 | 1.032; 1.127 | –0.0028; 0.124 | 0.060 |
| | B | 1.019 | 0.072 | 0.970; 1.067 | | |
| RHR480 | A | 1.025 | 0.067 | 0.978; 1.073 | –0.0093; 0.105 | 0.096 |
| | B | 0.977 | 0.059 | 0.938; 1.017 | | |

*p-value < 0.05 is significant

**Table III Comparison of Group A (n=10) and Group B (n=11) with respect to mean maximum relative change of haemodynamic parameters**

| Variable | Group | Mean | SD | 95%CI for Group mean | 95% CI for difference** | p-value |
|---|---|---|---|---|---|---|
| RSAPMax | A | 1.338 | 0.196 | 1.198; 1.478 | 0.091; 0.387 | 0.0040* |
| | B | 1.099 | 0.097 | 1.033; 1.164 | | |
| RDAPMax | A | 1.392 | 0.227 | 1.229; 1.554 | 0.066; 0.402 | 0.0090* |
| | B | 1.158 | 0.133 | 1.069; 1.247 | | |
| RMAPMx | A | 1.350 | 0.196 | 1.210; 1.490 | 0.074; 0.375 | 0.0066* |
| | B | 1.126 | 0.107 | 1.054; 1.198 | | |
| RHRMax | A | 1.197 | 0.155 | 1.086; 1.308 | 0.017; 0.213 | 0.0900 |
| | B | 1.099 | 0.092 | 1038; 1.161 | | |

* p-value < 0.05 is significant
**Confidence interval for ratio of Group B relative to Group A

**Table IV: Comparison of Group A (n=10) and Group B (n=11) with respect to AUC of relative change of SAP (RSAPAUC), DAP (RDAPAUC), MAP (RMAPAUC) and heart rate (RHRAUC)**

| Variable | Group | Mean | SD | 95% CI for group mean | 95% CI for the ratio** | p-value† |
|---|---|---|---|---|---|---|
| RSAPAUC | A | 9.365 | 1.230 | 8.485; 10.244 | 0.795; 0.966 | 0.0145* |
| | B | 8.169 | 0.493 | 7.838; 8.501 | | |
| RDAPAUC | A | 9.288 | 0.770 | 8.737; 9.838 | 0.833; 0,956 | 0.0025* |
| | B | 8.258 | 0.580 | 7.868; 8.647 | | |
| RMAPAUC | A | 9.286 | 0.940 | 8.615; 9.958 | 0.824; 0.957 | 0.0048* |
| | B | 8.225 | 0.552 | 7.854; 8.647 | | |
| RHRAUC | A | 8.700 | 0.639 | 8.243; 9.157 | 0.887; 1.001 | 0.0530 |

†p-value associated with the t-test (Student/Welch)
*p-value < 0.05 is significant
**Confidence interval for ratio of Group B relative to Group A

(p=0.0901). The mean change of SAP, DAP and MAP from baseline, differed significantly between the groups at 30, 60 120 and 240 seconds after clamping. Mean changes in blood pressure at 480 seconds, and mean changes in heart rate at all times, did not differ significantly. The magnitude of these differences is expressed in terms of 95% confidence intervals (CI) (Table 4).

Considering the AUC, there is a significant difference between the groups for SAP (p=0.0145), DAP (p=0.0025) and MAP (p=0.0048) (Figure 1), but not for heart rate (p=0.053) (Figure 2). The 95% CI for the ratio of the AUC of Group A relative to the AUC for Group B, displays the variation of the relative overall response.

## Discussion

Patients show a significant haemodynamic response to placement of the Mayfield clamp. In patients with intracranial pathology, sudden increases in blood pressure can increase the blood flow and volume in intracranial blood vessels, and consequently increase intracranial pressure (ICP).[19 7 8] Administration of drugs to attenuate this response may cause hypotension. A sudden drop in MAP below the lower autoregulatory margin will result in a lower CPP, and possibly cerebral ischaemia. The response to placement of the clamp should therefore be accompanied by minimal haemodynamic changes.

In this study a change of more than ±15% in mean arterial pressure was regarded as clinically significant. Although this change is well below a change of ± 25% that is tolerated in normotensive patients[20] (approximately 60 mmHg to 150 mmHg mean arterial pressure),[21] autoregulation is less effective in the presence of intracranial pathology.[5] Moreover, it should be kept in mind that autoregulation has a lag time of 3 to 4 minutes to be effective in keeping cerebral blood flow constant.[22] Cerebral ischaemia may thus occur whenever the blood pressure remains above or below some critical set point. Therefore, patients with raised ICP are more predisposed to complications following acute changes in haemodynamic parameters.

Our results demonstrate that the scalp block is more effective than an intravenous bolus of alfentanil in blocking the blood pressure response to application of the Mayfield clamp. The alfentanil was administered 90 seconds and not 180 seconds before application of the clamp. Waiting for 180 seconds could cause hypotension and earlier placement would ensure that the hypotensive effect of alfentanil has worn off by the time that the painful stimulus has subsided. The short acting drug remifentanil may be particularly useful in this regard but had not been registered in this country by the time this study was being done.

In one patient (Patient No 9 in Group A), a problem was encountered during placement of the clamp, which delayed clamping for 180 seconds instead of 90 seconds after the alfentanil bolus. A 13.2% drop in MAP was noted at the time of clamp application, probably due to the longer period before the stimulus. The blood pressure recovered to baseline values immediately after placement, where it stabilized until the end of the study period. In this case, it could be expected that the blood pressure would remain low were it not for

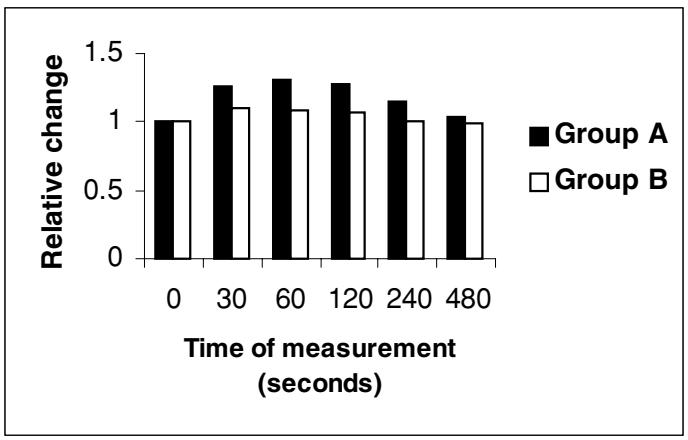



**Figure 1 Relative changes in mean arterial pressure at different times.**

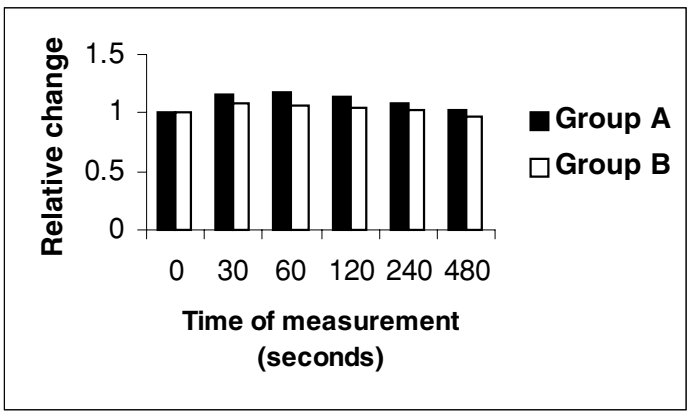



**Figure 2 Relative changes in mean heart rate at different times. Differences were not significant.**

placement of the clamp as blood pressure returned to normal after placement of the clamp. If there were a delay in application of the clamp, hypotension would probably persist for some time, jeopardising cerebral perfusion. The decrease in blood pressure in this patient was however not significant (< 15%) and probably not enough to cause a decrease in cerebral blood flow. The mean maximum change in blood pressure in group A occurred after 60 seconds, which also suggests that clamp placement should be delayed for at least 150 seconds after alfentanil administration, instead of 90 seconds.

The rate of clamp placement has also been studied.[23] Placement over a period of 10 seconds (as it is usually done) was compared with a more timeous placement of over 60 seconds. The mean increases in blood pressure was almost the same. The increase in blood pressure however took place within 10 seconds in the 10-second group as compared to 60 to 70 seconds in the 60-second group. The advantage of the more timeous placement is that it allows time for action by the anaesthesiologist, as well as time for autoregulation of CBF.

The scalp block was found to be easily done and to be effective in blocking the blood pressure response. It also offers the opportunity for repositioning of the pins if necessary. Adrenaline was not used as systemic absorption could increase blood pressure and pulse rate. Lignocaine was used, as the onset of block is faster than that of the other local anaesthetics available in this country, namely bupivacaine and ropivacaine. The dose of 4 mg/kg to 5 mg/kg is perhaps on the high side, especially in a relatively vascular area as the scalp. It is however within the dose regarded as safe for infiltration anaesthesia.[19] It is also below the cardiotoxic dose and after induction with thiopentone and maintenance with isoflurane most unlikely neurotoxic. The cardiotoxic dose of lignocaine is 7,1 ± 1,1 times the neurotoxic dose.[19] No haemodynamic changes were noted during infiltration of the scalp. This may be explained by the anaesthetic level (Isoflurane 1 MAC). The only critique against the block is that open needles are used, which exposes the anaesthesiologist to needle stick injury.

Movement of the head during positioning may cause movement of the endotracheal tube in the trachea. This may cause haemodynamic instability. As alfentanil has a systemic affect, it may be more effective than the scalp block in preventing hypertension that follows tracheal stimulation. We tried to limit head movement during positioning, which may explain the haemodynamic stability in both groups during this period.

Apart from ASA status, no other exclusion applied. As patients were randomised, the effect of factors that might have influenced the individual response, i.e. age and preoperative treatment, were not recorded, Inclusion of these data in the analysis of the present data would however give insight into the effects of age and preoperative treatment, e.g. beta-blockers. These data were not recorded, as these factors were not the focus of this study. It is concluded that the scalp block is more effective in attenuating the haemodynamic response to placement of the Mayfield clamp. Alfentanil is less effective when administered 90 seconds before clamp placement. Further investigation is necessary to confirm that alfentanil might be more effective when given 150 seconds before clamp placement. A definite advantage of the alfentanil technique is that it does not expose the anaesthetist to needle stick injury.

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