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Haemodynamic response to induction, laryngoscopy and tracheal intubation in diabetic and non-diabetic patients

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Original Article

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Abstract

Objective: Presence of autonomic neuropathy in diabetic patients can alter the haemodynamic response to induction and tracheal intubation. This trial was conducted to compare this response in 30 non-diabetic (control group) and 30 diabetic patients including both insulin and non-insulin dependent.

Methods: A prospective, age matched case controlled study was conducted at the Aga Khan University Hospital, for one year. After pre-medication with tablet midazolam 7.5 mgs orally, patients received pethidine 0.8 mg kg-1, thiopentone sodium 4 to 5 mg kg-1 for induction and vecuronium 0.1 mg kg-1 to facilitate tracheal intubation. Following manual ventilation with isoflurane 0.8% in oxygen 33% and nitrous oxide 66%, laryngoscopy and tracheal intubation was performed. Blood pressure (systolic, diastolic and mean) and heart rate responses were measured for 10 minutes.

Results: The systolic arterial pressure dropped by 9% after induction and rose by 16% after intubation in non-diabetics compared to 12% drop after induction and a rise of 10% after intubation in diabetics. No difference was seen in diastolic blood pressure which increased by 27% in ND compared to 22% in DB groups. The heart rate rose by 27% in non-diabetics compared to 17% in diabetics after intubation.

Conclusion: The systolic, diastolic and mean blood pressure response was similar in the two groups. A greater fall in SAP was observed post intubation in the DB group. A significant difference was observed in the heart rate response which was less in the diabetic group (JPMA 59:27; 2009).

Introduction

Direct laryngoscopy and tracheal intubation is associated with reflex circulatory responses characterized by an increase in blood pressure and heart rate. The major cause of this sympatho-adrenal response is the stimulation of the supra-glottic region by the laryngoscope blade. Tracheal tube placement provides little additional stimulation.[1] In patients with diabetes mellitus both attenuated and enhanced pressor response to intubation has been reported.[2,3] Measurement of plasma concentration of adrenaline in diabetic patients has also shown both a normal or a reduced response.[4,5] Increased sensitivity to catecholamines has also been observed in patients with diabetic uropathy.[6] Autonomic neuropathy that can occur in both insulin dependent and non-insulin dependent diabetic patients may potentially modify the normal cardiovascular responses during anaesthesia and render the diabetic patients more susceptible to cardiovascular morbidity.[3] The present study was designed to compare the change in systolic (SAP), mean (MAP) diastolic blood pressures (DBP) and heart rate response to induction of anaesthesia and tracheal intubation following thiopentone and vecuronium induction in both diabetic and non-diabetic patients.

A power of more than 80% and a 5% significance level to detect a difference of more than 20% was used for sample calculation.

Patients and Methods

After approval from the Ethics Review Committee of the University and obtaining written informed consent, 60 adult surgical patients were enrolled in the study. The control group (ND) comprised of 30 non-diabetic patients while the second group included 30 diabetic patients, (DB group) both insulin and non-insulin dependent, of either sex. Patients were between ages of 16-65 years and ASA physical status of 1, 2 or 3. All patients were scheduled for elective surgical procedures requiring tracheal intubation in our operating rooms. Patients with cardiac disease, on cardiovascular medication like beta blockers and calcium channel blockers, anticipated difficult intubation, and emergency procedures were excluded.

The presence or absence of autonomic neuropathy was tested by taking a detailed history for symptoms of autonomic neuropathy, looking for resting tachycardia, and noting the cardiovascular response to Valsalva's maneuver, deep breathing, potential hypotension and sustained handgrip. The tests were done in the ward, a day before surgery and a positive response to two of the tests was taken as a sign for the presence of autonomic neuropathy.

Patients were premedicated with tablet midazolam 7.5 mg orally one hour preoperatively. Baseline values of non invasive blood pressure, heart rate with lead II configuration

and oxygen saturation were taken using Datex Cardiocap monitor. Preoxygenation was done for three minutes, using Magill's circuit and pethidine was administered for analgesia in a dose of 0.8 mg. kg-1 before induction. Thiopentone sodium 4 to 5 mg. kg-1 was given over 30-45 seconds and titrated to the loss of eyelash reflex as an endpoint. Vecuronium 0.1 mg. kg-1 was administered as soon as apnoea occurred. Manual ventilation was started with 0.8% isoflurane and nitrous oxide and oxygen in the ratio of 66% and 33% respectively. End tidal carbon dioxide level was kept between 35-40 mm Hg. After three minutes of manual ventilation, patients were intubated by the primary investigator using size 3 Macintosh blade for laryngoscopy and poly-vinyl chloride tracheal tube of internal diameter 7.5 mm for females and 8.5 mm for males. Systolic, diastolic and mean blood pressures were monitored after analgesic injection, during manual ventilation for three minutes and every minute for 10 minutes following tracheal intubation. Any complication like arrhythmias, hypoxia and hypercarbia were also observed.

Statistical Analysis was done by a repeated measure design (ANOVA) with two groups of 30 subjects. Each subject was measured 13 times.

All the values given in the results are presented as mean with standard deviation (SD). Numerical data were analyzed using the analysis of variance (ANOVA), Kruskal Wallis, Mann Whitney or Chi-square test where appropriate. A probability value of less than 0.05 was considered statistically significant in each case.

Results

Both groups were comparable with respect to age weight, height and gender (Table 1). Mean age of the diabetic patients was higher (50.8±10.4 years) compared to the control non-diabetic group (44.3±14.4 years), this was not statistically significant. Autonomic dysfunction was present in 20% of the diabetic patients.

Table 1: Demographic data.

	Group 1 (non-diabetic controls)	Group 2 (diabetics)	Sig (2 tailed)
Age (years)	44.37 ± 14.4	50.80 ± 10.4	0.53
Weight (kilograms)	64.6 ± 12.4	68.1 ± 12.7	0.28
Height (centimeters)	160.9 ± 8.6	160.9 ± 12.5	0.99
Gender (female: male)	20:11	13:18	Not significant

The baseline systolic and mean blood pressure in both groups showed a significant difference (p 0.0001) with the pressures in the diabetic group being higher (128 vs 147 mm of Hg for systolic and 94 vs 103 mm Hg for mean). Because of the difference in baseline values, a direct comparison of the post-induction values was not valid. We therefore compared the percentage change from the baseline between the two groups. A drop of 9% in group 1 (ND; non-diabetics) as compared to 12% in group 2 (DB; diabetics) was observed in the systolic blood pressure, immediately after induction. After intubation, group 1 showed a 16% rise from the baseline compared to group 2 which showed a 10% rise. Values returned to within 5% of the baseline in both groups by the third minute. In the DB group the drop in SBP after intubation was more compared to the ND group. The maximum drop seen was 25%, nine minutes after intubation before surgery was started. These changes are shown in Figure 1.

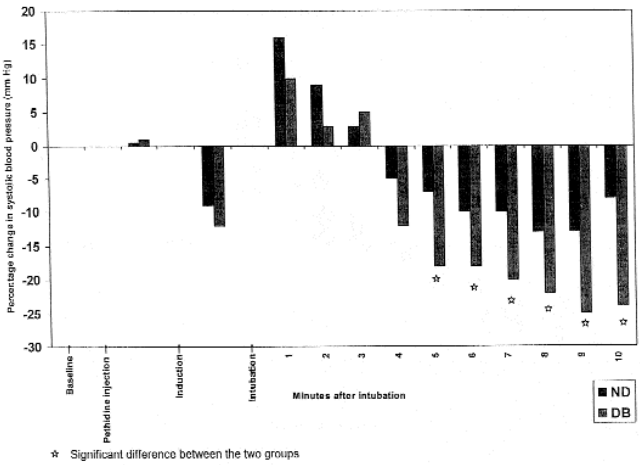

Figure 1: Comparison of percentage change from baseline systolic blood pressure (SAP) between non-diabetic and diabetic group after induction of anaesthesia and tracheal intubation.

When diastolic blood pressure was compared between the two groups, there was no statistical difference between the baseline reading (79 vs 83 mm of Hg). Both the groups showed a similar rise in diastolic blood pressure after intubation i.e. 27% vs 22% from the baseline at one minute post-intubation (99 vs 102 mm of Hg). The maximum fall in DBP was 17%, 10 minutes after intubation in ND group as compared to 19% in the DP group.

A statistically significant difference was observed in the baseline mean blood pressure (MAP) between the two groups, therefore a direct comparison was not done. A percentage change compared to baseline was calculated. The ND group showed a maximum rise of 20% one minute post-intubation compared to 16% rise in the DB group. The maximum fall was 16% in the ND group as compared to 22% in the DP group 10 minutes after intubation.

The heart rate (HR) showed no statistically significant difference in the baseline values between the two groups, however group 1 showed a greater increase in heart rate after intubation i.e. 27% as compared to group 2 which showed a 17% rise from the baseline. This difference was significant. There was a drop in heart rate of 11% in group 1 (ND) from baseline at ten minutes after intubation as compared to 13%

in group 2 (DB). These changes are shown in Figure 2.

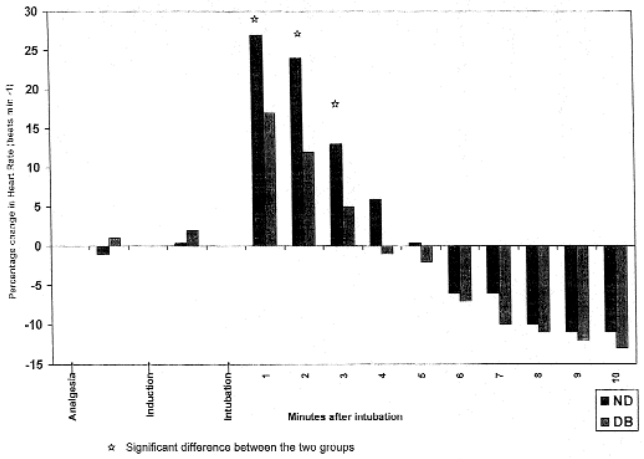

Figure 2: Percent changes in heart rate (HR) compared to the baseline in non diabetic and diabetic patients after induction of anaesthesia and tracheal intubation.

Discussion

An estimated 50% of diabetic patients will require surgery in their life time.[7] Haemodynamic stability is a requirement in these patients because of end organ involvement of heart, kidneys, brain and eyes. Haemodynamic instability is not well tolerated in this population because many of these patients have underlying coronary artery disease and may have suffered painless myocardial infarction.[8]

Studies on intubation response in diabetic patients have shown conflicting results. Increased sensitivity to catecholamines because of denervation induced hypersensitivity of receptors and decreased neuronal uptake of nor-adrenaline due to presence of autonomic neuropathy have been demonstrated in diabetic patients.[5,6] On the other hand some diabetics have shown decreased concentration of adrenaline[9] and absence of non-adrenaline response.[2] These different results have been attributed to the presence or absence of autonomic neuropathy in diabetic patients and merits further investigation of this response. Autonomic neuropathy is reported to be present in 20 to 40% of all diabetics[7] and can occur in both insulin and non-insulin dependent patients.[10] The effect is clinically obvious on the cardiovascular system.[11] In our study autonomic dysfunction was present in 20% of the diabetic group.

Our diabetic population also exhibited a higher baseline SAP compared to non-diabetic controls. This is not surprising and has been reported before. The incidence of hypertension in diabetic patients is 29-54%[7] and may be related to autonomic imbalance, atherosclerotic changes in vasculature or abnormal sodium retention. Induction is a period of haemodynamic instability because of effect of induction agents on vagal outflow, on peripheral vascular resistance and absence of stimulation. Both groups showed a fall in SAP in our study 19% in non-diabetics vs 12% in diabetics but this difference was not statistically significant and was within 20% of baseline, an acceptable change in clinical practice of anaesthesia. Post induction DAP, MAP and HR remained stable in both groups. All SAP, DAP and MAP showed a clinically but not significantly lower response to tracheal intubation in the diabetic group (6% lower for SAP, 5% lesser for DAP and 3% lower for MAP) and was within 20% of baseline.

A significant difference was observed in the heart rate response which was less exaggerated in the diabetics compared to the non-diabetic group (27% vs 17%) The mechanism for this difference could be similar to that seen in Valsalva's maneuver and head up tilt where diabetics have less response to, both these tests and show less tachycardia.[7] Both sympathetic and parasympathetic nervous system may be involved and heart rate response may be a balance between cardiac sympathetic excitation and cardiac vagal withdrawal.[7] Ageing is also know to be associated with decreased autonomic reflex function.[12] Elderly patients have lesser chronotropic effect associated with tracheal intubation.[13] The age effect probably did not influence our results since no significant difference was present in the age of patients in the two groups.

After the intubation response had settled, BP and HR in all groups showed a fall in the unstimulated patient. This fall was within 20% of the baseline in both groups except for SAP in the DB group where it showed a significantly greater fall than ND group and was more than 20% of baseline values.

Our findings are in concurrence with the findings of Burgo et al[7] who showed a lesser increase in HR in diabetic patients following intubation. On the other hand Vohra et al[3] have shown an increased response in diabetics compared to controls in both post-intubation HR and MAP but they used pancuronium as the muscle relaxant in their study, a drug known to have sympathomimetic effects. We used Vecuronium, a more cardiostable muscle relaxant. The mean heart rate in Vohra's study was also higher in the non-diabetic group during the intra-operative period.

One of the limitations of our study was the use of pethidine as an analgesic. Pethidine is know to have atropine like action and can have an effect on the response. However it was used in a similar dose in both groups. The reason for using Pethidine was the non-availability of more cardio stable short acting narcotics like fentanyl in our country. Another limitation could be that we included both insulin and non-insulin dependent diabetic patients in our diabetic group and the incidence of autonomic neuropathy

may vary between the two subgroups. We did not test this difference because the number of patients in each subgroup would be too small for a meaningful statistical comparison.

Based on our study we conclude that the rise in blood pressure was similar in both diabetic and non-diabetic groups. The SAP and MAP response was within 20% of baseline but the DAP response was higher than 20% in both groups. A greater fall in SAP was observed in the DB group after the BP response to intubation had settled. A difference was seen in the heart rate response which was less in the diabetic group. These findings may have clinical implications and though less tacchycardia was observed after intubation in the diabetics, this may promote post intubation hypotension in the period prior to stimulation due to inability to compensate.

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