Section: Anesthesiology

Research

NEUROMUSCULAR MONITORING AFTER ADMINISTRATION OF VECURONIUM IN PATIENTS WITH DIABETES MELLITUS

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Abstract

Background: In patients with diabetes mellitus there occurs degeneration of the peripheral nerves with segmental demyelination, some loss of axons and degeneration of terminal motor axons and end plates. In diabetic patients without neuropathy the ulnar, median and peroneal nerve conduction velocities were decreased significantly. We studied the supramaximal current for ulnar nerve stimulation during electromyographic monitoring of onset and recovery of neuromuscular block using a neuromuscular transmission module in patients with Type 2 diabetes undergoing general anaesthesia with nitrous oxide, oxygen, isoflurane and vecuronium. **Materials and Methods:** Thirty diabetic patients were randomly assigned to a post-tetanic count (PTC) group (n=15) or train-of-four (TOF) group (n=15). In addition 30 non-diabetic patients were divided into control PTC (n=15) and TOF groups (n=15). Following vecuronium 0.1 mg/kg, PTC was measured every 5 min in the diabetes and control PTC groups. A 50 Hz tetanic stimulation was delivered at the supramaximal current for 5 sec, and after a pause of 3 sec 20 single twitch stimuli of 0.2-ms duration square waves were given every 1 second at the supramaximal current. The time from vecuronium administration to the return of PTC1 was compared between the diabetes and control PTC groups. The PTC measured every 5 min was compared between the two groups. The time from vecuronium to the return of T1, T2, T3 and T4 were measured in every 5 minutes and compared between the diabetes and control TOF groups in every 15 minutes. After the dose of vecuronium, T1/T0 and T4/T1 were recorded every 15 min and compared between the two TOF groups. **Result:** In the diabetic patients the mean supramaximal stimulating current was significantly higher than in the non-diabetic patients. Time to return of PTC1 did not differ significantly between the diabetes and control PTC groups (9.33±1.75) vs (9.00±2.07) min (P=0.775). Time to return of T1 and T4 in the diabetes TOF group were significantly longer than in the control TOF group (T1: 29.33±1.75) vs (24.33±3.2) min, P=0.0001; T4: 57.33±5.30 vs 48.00±2.53 min, (P=0.0001). During recovery PTC in the diabetes PTC and TOF groups was similar to those in the control PTC and TOF groups respectively. T1/T0 and T4/T1 in the diabetes TOF group were significantly less than in the control TOF group after administration of vecuronium (P<0.05). **Conclusion:** The supramaximal stimulating current was higher and the onset of neuromuscular block and time course of recovery of PTC was similar between diabetic and non-diabetic patients receiving vecuronium.

INTRODUCTION

Diabetes mellitus, a common metabolic disorder has a prevalence of 537 million cases worldwide by the years 2021. During surgical procedure both anesthesia and surgery can adversely affect the metabolic status of the poorly controlled diabetes. In diabetes mellitus there occurs degeneration of the peripheral nerves with segmental demyelination, some loss of axons (probably both "A" and "C"

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fibres), degeneration of terminal motor axons and end plates. Demyelination of sensory roots and tracts with degeneration in the spinal cord, mainly the dorsal columns may occur. Sometimes combined dorsolateral sclerosis and denervation in joints and muscles may occur.[1] There is loss of motor units and denervation in the skeletal muscles of the upper and lower extremities. Electromyography shows the mean duration of action potential and amplitude of the evoked response in the skeletal muscles decreased by 20% and 50% respectively.[2] The analysis of the response to neural reflexes indicated that the ratio of neurological disorders in nerves of the ankle and knee were generally higher in the abnormal group (the patients with nerve conduction disorder) compared to the normal (the patients with normal nerve conduction).[3]

In most diabetic patients, the quadriceps femoris muscle have been denervated to such an extent that femoral motor responses recorded from it are unelectable or of low amplitude.[4] The adductor pollicis muscle is also denervated in diabetic patients and the supramaximal stimulating current is higher. In diabetic patients without neuropathy, the ulnar, median and peroneal nerve conduction velocities were decreased significantly. Also the nerve conduction velocities in diabetic patients with neuropathy were less than in diabetic patients without neuropathy.[5] However, no studies have yet evaluated the supramaximal stimulating current or the onset and recovery of neuromuscular block specifically in diabetic patients. This study was undertaken to investigate these variables when vecuronium was used in anaesthetized patients for operation under GA with Type 2 diabetes mellitus.

MATERIALS AND METHODS

After obtaining approval from ethical committee of SCB Medical College and Hospital, Cuttack the study was conducted on 60 patients with ASA grade I and II of both sexes aged between 18 to 60 years undergoing elective surgery under general anesthesia with endotracheal intubation from September 2018 to October 2020. Patients who refuse to give consent, pregnant women/breast-feeding mothers, patients with known allergy to drugs, psychiatric patients, patients with neuropathy, patients with muscular dystrophy were excluded from the study. Thirty adult patients with Type 2 diabetes mellitus for more than 5 yrs and thirty adult non diabetic patients were included in the study. Out of 30 diabetes patients, 15 patients each were randomly assigned to a post-tetanic count (diabetes PTC) group (n=15) or train-of-four group (diabetes TOF). The 30 patients without diabetes were randomly divided into control PTC and TOF groups with 15 patients in each group. All the diabetes PTC and TOF groups were free from diabetic neuropathy and nephropathy. All diabetes patients were maintained on regular insulin to keep the blood sugar level between 70 to 200 mg/dl. Premedication glycopyrrolate 0.005 mg/ kg; midazolam 0.05 mg/kg; nalbuphine 10-20 mg i.v. was given 30 min before induction of anesthesia.

Two stimulating electrodes were positioned over the ulnar nerve at the wrist and two recording electrodes were attached over the adductor pollicis muscle. The forearm and the four fingers were immobilized with vinyl belts but the thumb was allowed to move freely. After induction of anesthesia with Propofol 2mg/kg, TOF stimuli was applied to the ulnar nerve using the electrical nerve stimulator. Four twitch stimuli consisting of 0.2-ms square waves were applied at 2 Hz. The corresponding electromyographic amplitudes were measured using the neuromuscular transmission module. The height of the electromyographic responses were recorded from the anesthetic monitoring system. In each patient the monitoring system automatically searched for the stimulus current needed to achieve the maximal response of the adductor pollicis muscle. The search began with 10-mA single twitch stimuli of 0.2 ms duration applied every 1 second and was increased in steps of 5 mA until the increase in current did not increase the electromyographic response. The stimulating current was then automatically increased by 15% to produce a supramaximal current. If the supramaximal current was not found or the response was too weak to determine the current, the current was set at 70 mA and such patients were excluded from the next part of the study i.e. the comparisons of the onset of neuromuscular block and of recovery of the PTC or TOF responses. In such circumstances, an additional patient was studied in order to maintain the numbers of patients in each group.

Once the supramaximal current had been established, the electromyographic amplitude of T1 was considered to be the control response T0. The value of T1 was determined again 15 min after starting TOF stimuli which was applied every 5 minutes. During the stabilization of neuromuscular monitoring, the patient's lungs was ventilated using a facemask with oxygen 6 litre/min and isoflurane 1% inspired concentration. After recording T0, vecuronium 0.1 mg/kg i.v. was administered to facilitate tracheal intubation. Following vecuronium 0.1 mg/kg, the PTC was measured every 5 min in the diabetes and control PTC groups. A 50 Hz tetanic stimulation was delivered at the supramaximal current for 5sec and after a pause of 3sec, 20 single twitch stimuli of 0.2-ms duration square waves were given every 1 second at the supramaximal current. The number of detectable electromyographic responses to single twitch stimuli were regarded as the PTC. The time from vecuronium administration to the return of PTC1 was compared between the diabetes and control PTC groups. In addition the PTC measured every 5 min was compared between the two groups. The time from vecuronium to the return of T1, T2, T3 and T4 responses were measured every 5 minutes

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and compared between the diabetes and control TOF groups in every 15 minutes. After the dose of vecuronium, T1/T0 and T4/T1 were recorded every 15 min and compared between the two TOF groups. A thermometer probe to measure surface skin temperature was positioned over the adductor pollicis muscle in each patient.

Anesthesia was maintained with nitrous oxide 66% in oxygen and isoflurane 1% end tidal concentration. Ventilation was controlled to maintain normocapnia. The end-tidal concentrations of anesthetics and ETCO2 were measured continuously using a multiple gas monitor.

Statistical analysis- Patient characteristics were compared using the Kruskal–Wallis test and Mann–Whitney U-test with Bonferroni's adjustment. The stimulating currents, the times to return of PTC1 and times to the return of T1, T2, T3 and T4 were compared between the diabetic (diabetes PTC and diabetes TOF) and non-diabetic (control PTC and control TOF) groups using the Mann–Whitney U-test.t. The time course of recovery of the PTC was compared using the Mann–Whitney U-test with Bonferroni's adjustment. P<0.05 was considered statistically significant. Statistical analyses were performed using a statistical package (SPSS Inc., Chicago, USA) running on a personal computer.

RESULTS

In the diabetic groups, the mean supramaximal stimulating current at which the maximal response could be elicited was significantly higher than in the non-diabetic groups. In PTC group case (49.67±4.42 mA) to control (33.33±3.62 mA) which was statistically significant (P value=0.0001). In TOF group case (50.00±4.22 mA) to control (34.67±3.99 mA) which was again statistically significant (P value=0.0001). As shown in [Table 4], time from vecuronium administration to the return of PTC1 did not differ significantly between the diabetes and control PTC groups (9.33±1.75 to 9.00±2.07 minutes with P value=0.775 which is statistically insignificant. However, as shown in [Table 5], time to the return of T1, T2, T3 and T4 were significantly longer in the diabetes TOF group than in the control TOF group (P<0.05). Recovery of the PTC in the diabetes PTC and control PTC groups followed similar time courses as shown in [Table 4]. [Table 6] showing T1/T0 was significantly lower in the diabetes TOF group than in the control TOF group after administration of vecuronium (P<0.05). T4/T1 in the diabetes TOF group also compared in [Table 7] differ significantly from that in the control TOF group which was statistically significant (P<0.05).

Table 1: Demographic Data

		PTC Diabetic	PTC Control	TOF Diabetic	TOF Control	P Value
Sex Distribution	Male	8	8	8	7	
	Female	7	7	7	8	
Age in Years	Maximum	38	24	47	39	0.020
	Minimum	60	60	60	60	
	Mean±SD	52.67±6.76	44.13±10.38	54.33±3.86	53.27±6.00	
Height in cm	Maximum	142	144	141	148	0.332
	Minimum	169	176	169	167	
	Mean±SD	154.60±8.12	160.27±9.33	156.13±9.65	156.00±7.89	
Weight in Kg	Maximum	47	54	51	52	0.510
	Minimum	80	86	86	78	
	Mean±SD	60.07±8.07	65.20±10.93	64.27±10.02	64.20±7.38	

Table 2: Comparison of duration of diabetes

Duration of Diabetes (in yrs)		Minimun	Maximum	Mean	SD	P value
PTC	Diabetic	5	10	6.93	1.90	0.624*
TOF	Diabetic	5	10	7.20	1.74	

Table 3: comparing supramaximal stimuli

SMSC	Diabetic	Non-Diabetic	P value
PTC Group	49.67±4.42	33.33±3.62	0.0001
TOF Group	50.00±4.22	34.67±3.99	0.0001

Table 4: Comparing Return of PTC1 and PTC over time.

	Diabetic PTC		Control PTC		P value
	Mean	SD	Mean	SD	
PTC1	9.33	1.75	9.00	2.07	0.775*
PTC at 5min	0.13	0.35	0.20	0.41	0.775*
PTC at 10min	1.20	0.56	1.47	0.64	0.250*
PTC at 15min	3.07	0.79	4.80	0.67	0.0001*
PTC at 20min	5.00	0.65	6.53	0.64	0.0001*
PTC at 25min	6.53	0.74	8.20	0.86	0.0001*
PTC at 30min	9.40	0.82	11.13	0.63	0.0001*
PTC at 35min	13.13	0.834	14.87	1.12	0.0001*
PTC at 40min	15.20	1.01	17.13	0.91	0.0001*
PTC at45min	16.53	0.74	18.13	0.91	0.0001*

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PTC at50min	17.33	0.48	18.87	0.74	0.0001*
PTC at55min	18.27	0.45	18.40	0.63	0.0001*
PTC at60min	19.33	0.48	19.87	0.35	0.011*

Table 5: comparing return of T1, T2, T3 and T4.

	Diabetic TOF		Control TOF		P value
	Mean	SD	Mean	SD	
T1	29.33	1.75	24.33	3.2	0.0001*
T2	42.33	2.58	31.00	2.07	0.0001*
T3	44.00	3.87	35.00	5.00	0.0001*
T4	57.33	5.30	48.00	2.53	0.0001*

Table 6: T1/T0 comparison.

	Diabetic TOF		Control TOF		P value
	Mean	SD	Mean	SD	
T1/T0 at 15min	0	0	0	0	-
T1/T0 at 30min	0.041	0.007	0.079	0.008	0.0001*
T1/T0 at 45min	0.106	0.009	0.219	0.009	0.0001*
T1/T0 at 60min	0.248	0.009	0.501	0.014	0.0001*
T1/T0 at 75min	0.316	0.010	0.568	0.011	0.0001*
T1/T0 at 90min	0.414	0.012	0.701	0.020	0.0001*
T1/T0 at 105min	0.448	0.015	0.737	0.017	0.0001*
T1/T0 at 120min	0.467	0.016	0.803	0.023	0.0001*

Table 7: (T4/T1 Comparison.

	Diabetic TOF		Control TOF		P value
	Mean	SD	Mean	SD	
T4/T1 at 15min	0	0	0	0	--
T4/T1 at 30min	0	0	0.013	0.051	*-
T4/T1 at 45min	0.020	0.052	0.028	0.035	0.198*
T4/T1 at 60min	0.175	0.031	0.206	0.012	0.002*
T4/T1 at 75min	0.227	0.020	0.320	0.011	0.0001*
T4/T1 at 90min	0.309	0.019	0.465	0.010	0.0001*
T4/T1 at 105min	0.383	0.017	0.504	0.010	0.0001*
T4/T1 at 120min	0.508	0.016	0.596	0.009	0.0001*

DISCUSSION

In patients with diabetes, demyelination and axon loss occurs in the nerve endings. The motor nerve conduction velocity is also decreases in diabetic patients. Not only impairment of the motor nerve fibre, but also muscle damage and muscular infarction has been demonstrated in skeletal muscles. Additionally aseptic myonecrosis, ischaemic myonecrosis, tumoriform focal muscular degeneration and muscular atrophy have been observed.

This study shows that the supramaximal stimulating current is higher in diabetic than in non-diabetic patients. The onset of neuromuscular block caused by vecuronium 0.1 mg/kg did not differ significantly between diabetic and nondiabetic patients. Also time to return of the PTC1 and recovery of PTC did not significantly differ between diabetic and control patients. However, in diabetic patients time to the return of T1, T2, T3 and T4 were significantly longer and recovery of T1/T0 and T4/T1 was significantly slower than in non-diabetic patients. When the diabetic and non-diabetic groups were compared the supramaximal current was found to be 49.67±4.42 mA and 50.00±4.22 mA in Diabetic PTC and TOF group respectively vs 33.33±3.62 mA and 34.67±3.99 mA in control PTC and TOF group respectively [Table 3] which was found to be statistically significant (P=0.0001). Yuhji Saitoh, Yuhji & Kaneda et al6 had reported that in most diabetic patients the quadriceps femoris might have been denervated to such an extent that femoral motor responses recorded from it were un elicitable or of low amplitude. We assumed that because the adductor pollicis muscle was denervated in diabetic patients, the supramaximal stimulating current would be higher than in non-diabetic patients.

Geoffrey D. Clarke, Marjorie Molina-Wilkins et al,[7] reported a decrease in cardiac output, left ventricular dysfunction and impaired stroke volumes in diabetic patients. In the current study five patients in the diabetic groups had hypotension during induction of anesthesia suggesting significant cardiovascular disease. Burgos and colleagues8 reported that diabetic patients often suffered from hypertension, but might have hypotension for some minutes following tracheal intubation. As the cardiac output decreases, the onset of neuromuscular block is delayed. Even if diabetic patients are more sensitive to neuromuscular blocking drugs, onset of block may not be faster because the sensitivity is offset by a decrease in cardiac output.[8]

Time from administration of the neuromuscular blocking drug to return of the PTC or T4/T1 is thought to represent the degree of pre-junctional neuromuscular block. In our study the return of T1, T2, T3 and T4 were significantly prolonged in

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diabetic TOF group vs control TOF group (P value<0.05) [Table 8]. The return of PTC1 was also found to be 9.33±1.75 min in diabetic PTC group vs 9.00±2.07 min in control PTC group which was found to be clinically insignificant (P=0.775) and return of PTC was significantly less in Diabetic PTC group against control PTC group [Table 4]. Y Saitoh 1, H Toyooka, K Amaha,[9] had studied recovery of post-tetanic twitch (PTT) and train-of-four (TOF) responses after administration of vecuronium in 60 patients under different inhalation anesthetics and neurolept anesthesia with (droperidol and fentanyl) and halothane, isoflurane, enflurane or sevoflurane 1 MAC in nitrous oxide and oxygen. The time from initial administration of vecuronium 0.2 mg/kg to the first appearances of T1, T2, T3 and T4 differed significantly between groups. However, the intervals to the first appearance of PTC1 did not differ significantly between groups. We also found similar results in our study.

Time from administration of the neuromuscular blocking drug to return of T1, T2, T3, T4 single twitch response and T1/T0 demonstrate the level of block at the post-junctional region of the neuromuscular junction.[10,11] This study demonstrated that time from vecuronium to the return of PTC1 was comparable between diabetic and non-diabetic patients but time to return of T1, T2, T3 and T4 and recovery of T1/T0 and T4/T1 were significantly slower in the diabetic patients than in nondiabetic patients. These findings suggest that damage to the post-junctional region was greater than that at the prejunctional region in diabetic patients.

In this study the degree of neuromuscular block was monitored electromyographically. It has been reported that when the level of neuromuscular block is profound, the height of T1/T0 measured electromyographically tends to be slightly greater than that assessed mechanically using a force transducer. The electromyograph is more sensitive than the mechanical twitch response in detecting a TOF response during profound neuromuscular block.[12] When the degree of block is slight, the neuromuscular response evaluated electromyographically may also be different from that assessed mechanically. We observed that the average T1/T0 was statistically significantly less in diabetic TOF group against control TOF group [Table 6]. In the present study, diabetic patients without diabetic neuropathy were examined. Kopman AF et al,[13] reported that even if recovery from neuromuscular block was significant, T1/T0 recorded electromyographically tend to return to only 0.8.

In this study we observed that the TOF ratio T4/T1 was significantly less in Diabetic TOF group against control TOF group after 60 minutes onwards which was compared in every 15 minutes [Table 7]. Y. Saitoh H. et al,[14] studied for reversal of vecuronium-induced neuromuscular blockade with neostigmine in two groups with Type 2 diabetes mellitus and normal controls. Within fifteen minutes of reversal, the number of patients in whom recovery from neuromuscular blockade was judged insufficient to guarantee good respiratory function (train-of-four ratio <0.74) did not differ between the groups. However 15 min after reversal, the number of patients with a train-of-four ratio <0.9 was significantly higher in the Diabetic Group than in the Control Group (15 vs. 10, p = 0.033).

CONCLUSION

When neuromuscular block is monitored in diabetic patients the supramaximal stimulating current is higher than in non-diabetic patients. The onset of neuromuscular block and time course of recovery of PTC were similar between diabetic and non-diabetic patients receiving vecuronium. However, time from administration of vecuronium to return of T1, T2, T3 and T4 was significantly longer, and recovery of T1/T0 and T4/T1 were significantly slower in diabetic than in non-diabetic patients. These findings might be the result of the more marked impairment of the post-junctional region than the pre-junctional nerve endings in the neuromuscular junctions.

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