at these induction times. Because all operations began at essentially the same time of day, diurnal variation could not have contributed to the differences between groups 30 min after induction and later.

The data derived in this investigation were suggested from *in vitro* studies by Kenyon *et al.*,[9] Lambert *et al.*,[10] and Robertson *et al.*,[11] who found that the concentration of etomidate required to suppress adrenocortical steroidogenesis is obtained following typical clinical doses. The concentrations of thiopental and propofol required to achieve adrenocortical suppression were greater than those produced clinically.

The side effects seen after propofol induction were similar to those seen after thiopental induction; they did not include the myoclonic movements or pain seen after etomidate injection.

In conclusion, the ability of the adrenal cortex to secrete cortisol and aldosterone in response to surgical stress or ACTH stimulation was not blocked after 2.5 mg/kg of propofol iv. Larger doses of propofol might have a different effect, but they were not studied. Therefore, induction doses of propofol have a similar non-suppressive adrenocortical effect as induction doses of thiopental, and are different than the adrenocortical suppressive effect of induction doses of etomidate.

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Unequal Effects of Cardiopulmonary Bypass-induced Hypothermia on Neuromuscular Blockade from Constant Infusion of Alcuronium, d-Tubocurarine, Pancuronium, and Vecuronium

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Recent publications suggest that hypothermia may either attenuate or enhance nondepolarizing neuromuscular blockade, depending on the specific muscle relaxant involved.[1–4] However, within these studies, the possible significance of organ bath cooling[1] *versus* hypothermic cardiopulmonary bypass[2–4] is difficult to ascertain. We, therefore, examined the neuromuscular blockade induced by four nondepolarizing muscle relaxants for its response to cardiopulmonary bypass-induced hypothermia in the clinical setting.

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Key words: Neuromuscular relaxants: alcuronium; d-tubocurarine; pancuronium; vecuronium. Surgery: cardiopulmonary bypass. Temperature: hypothermia.

Materials and Methods

Forty patients, ASA Physical Status III and IV, undergoing coronary artery bypass grafting or aortic or mitral valve replacement gave informed consent to participate

in this study. Anesthesia was achieved with flunitrazepam, fentanyl, and nitrous oxide. The trachea was intubated following topical anesthesia without the aid of a muscle relaxant. Details on premedication, anesthesia, cardiopulmonary bypass, and monitoring have been described previously.[3,5] Simultaneous recording of the evoked compound electromyogram (EMG) and the evoked twitch tension[5] were used for the assessment of neuromuscular transmission. The patients were randomly assigned to four groups, according to the muscle relaxant used. Muscle relaxation was initiated 30 min after induction of anesthesia by an iv loading dose and maintained by constant infusion of the particular muscle relaxant, as described by previous workers.[4,6,7] The infusions were started concurrently with the injection of the loading doses and were discontinued upon closure of the pericardium (figs. 1, 2). The doses of the muscle relaxants were chosen to achieve 30–60% neuromuscular blockade before the institution of bypass: alcuronium 0.15 $mg \cdot kg^{-1}$ and 0.03 $mg \cdot kg^{-1} \cdot h^{-1}$, d-tubocurarine 0.3 $mg \cdot kg^{-1}$ and 0.06 $mg \cdot kg^{-1} \cdot h^{-1}$, pancuronium 0.05 $mg \cdot kg^{-1}$ and 0.01 $mg \cdot kg^{-1} \cdot h^{-1}$ and vecuronium 0.05 $mg \cdot kg^{-1}$ and 0.05 $mg \cdot kg^{-1} \cdot h^{-1}$. The recordings were evaluated for EMG amplitudes (peak to peak) and twitch tension, read in per cent of control. The speed of recovery of the EMG amplitudes and twitch tension, expressed in per cent of the respective control values per minute, was used to quantify the effect of particular stages of the cardiopulmonary bypass on neuromuscular blockade. All data were calculated as means and standard deviations (mean ± SD). Statistical significance was assessed by paired and unpaired Student's *t* test for within and between group comparison, respectively, and by analysis of variance. Correlation statistics were performed by means of the least squares method.

Results

The patients' overall age, body weight, and duration of cardiopulmonary bypass were 49 ± 10 yr, 75 ± 10 kg, and 89 ± 25 min, respectively. These figures did not differ significantly between the four groups. With respect to both EMG and twitch depression, analysis of variance did also not reveal significant differences between the four muscle relaxants, neither following the administration of the loading doses, nor before the initiation of bypass, nor at the end of surgery. Significant differences between the four muscle relaxants were associated with changing body temperature (table 1).

Before the initiation of cardiopulmonary bypass, neuromuscular blockade had reached a constant level in five patients who received pancuronium and in all patients receiving vecuronium (table 2). In the patients receiving alcuronium, d-tubocurarine, or pancuronium, upon active cooling, the speed of recovery of the EMG action potentials increased 2.5- to fourfold ($P < 0.05$), and, during sustained hypothermia, returned to its pre-bypass level (table 2). As a result, at the lowest temperature (27.7 ± 2.3° C; 18 ± 5 min after the initiation of bypass), the EMG amplitudes were 56%, 37%, and 38% greater, respectively, than at the start of the bypass (table 1). By contrast, with none of the three muscle relaxants was the recovery of twitch tension significantly accelerated by cooling (table 2). Figure 1 illustrates these features using alcuronium neuromuscular blockade as an example which is also typical for d-tubocurarine and pancuronium. Vecuronium neuromuscular blockade with a 10–15-min delay relative to active cooling was reversibly augmented by hypothermia (lowest temperature: 26.3 ± 1.2° C). Prior to rewarming the EMG action potentials and twitch tension had decreased significantly to 34% and 23%, respectively, of their pre-bypass levels (table 1, 2; fig. 2). The hypothermia-related depression of neuromuscular transmission disappeared when nasopharyngeal temperature returned to 28–30° C.

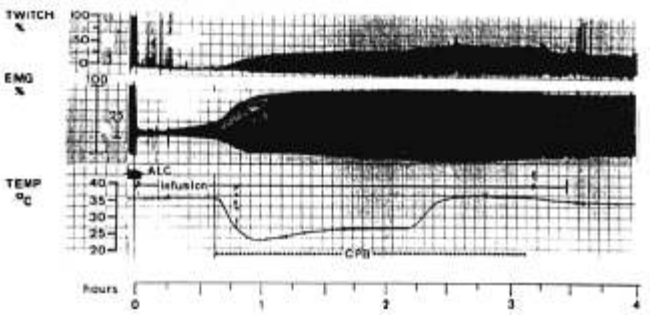

FIG. 1. Cardiopulmonary bypass (CPB)-induced hypothermia attenuating neuromuscular blockade from an iv loading dose (arrow: 0.15 $mg \cdot kg^{-1}$) and constant infusion (0.03 $mg \cdot kg^{-1} \cdot h^{-1}$, of alcuronium (ALC). Individual recording. Chart speed: 1 mm per min. (Similar tracings were obtained with d-tubocurarine and pancuronium.)

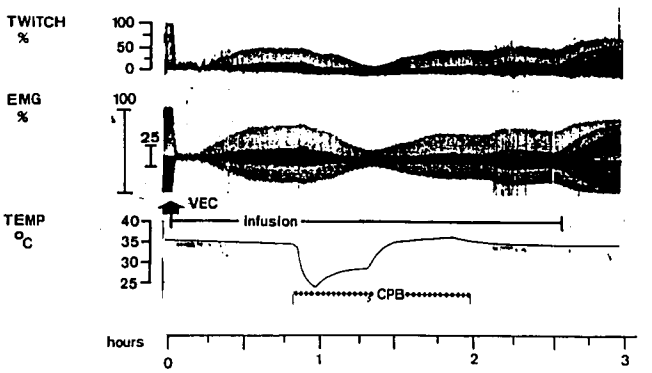

FIG. 2. Cardiopulmonary bypass (CPB)-induced hypothermia enhancing neuromuscular blockade from an iv loading dose (arrow: 0.05 $mg \cdot kg^{-1}$, and constant infusion (0.05 $mg \cdot kg^{-1} \cdot h^{-1}$, of vecuronium (VEC). Individual recording. Chart speed: 1 mm per min.

TABLE 1. The Intensity of d-Tubocurarine, Alcuronium, Pancuronium, and Vecuronium Neuromuscular Blockade at Particular Stages of Surgery and Cardiopulmonary Bypass (CPB). Figures Represent EMG Amplitudes and Twitch Tension in % of Control (Mean ± SD)

	Alcuronium, n = 10		d-Tubocurarine, n = 10		Pancuronium, n = 10		Vecuronium, n = 10	
	EMG	Twitch	EMG	Twitch	EMG	Twitch	EMG	Twitch
Maximum effect of loading dose	22 ± 13	8 ± 8†	12 ± 5	4 ± 6†	19 ± 18	6 ± 8†	12 ± 10	9 ± 4
Start CPB	50 ± 28	29 ± 22†	30 ± 19	17 ± 12†	37 ± 30	29 ± 28†	41 ± 20	26 ± 22†
Lowest temp.	78 ± 27*	40 ± 20*†	41 ± 24*	22 ± 15*	51 ± 35*	37 ± 31*†	35 ± 26*	20 ± 18†
Start rewarming	83 ± 21	50 ± 19†	58 ± 27	40 ± 23†	55 ± 34	46 ± 35†	14 ± 11	6 ± 5*
End CPB	77 ± 28	64 ± 32	54 ± 24	50 ± 31†	59 ± 28	59 ± 40	24 ± 18	21 ± 25†
End surgery	76 ± 23	59 ± 30†	41 ± 21	49 ± 27	72 ± 25	64 ± 31	50 ± 26	45 ± 37

* $P < 0.05$ *vs.* corresponding figure at start CPB; †$P < 0.05$ *vs.* corresponding amplitude of EMG (paired *t* test).

With all muscle relaxants, a greater intensity of neuromuscular blockade was indicated by the depression of twitch tension than by depression of the EMG amplitudes (table 1), with a significant positive correlation between the two variables (r = 0.73 − 0.96). Yet, significant effects of the cardiopulmonary bypass on neuromuscular transmission were reflected by the EMG rather than by the twitch (table 1, fig. 1, 2). With alcuronium, during hypothermia, the correlation between EMG and twitch was irreversibly lost and, after cessation of bypass, the twitch failed to parallel the recovery of the EMG.

DISCUSSION

In humans, both surface cooling and cardiopulmonary bypass-induced hypothermia were shown to attenuate a d-tubocurarine neuromuscular blockade.[8,9] Our data with d-tubocurarine, alcuronium, and pancuronium agree with these reports. Vecuronium, on the other hand, was the only drug with a consistently augmented neuromuscular block during hypothermic bypass. This finding agrees with previous results with vecuronium after repeated injections.[5] Before the initiation of bypass, constant block was obtained from both vecuronium and pancuronium. Hence, the presence or absence of constant block does not explain the different effects of hypothermia on neuromuscular blockade from different nondepolarizing muscle relaxants.

TABLE 2. Speed of Recovery of Neuromuscular Transmission, Expressed in % of Control per Minute (Mean ± SD), at Different Stages of Cardiopulmonary Bypass (CPB)

	Before CPB	Active Cooling	Constant Hypothermia
Alcuronium (n = 10)			
EMG	0.5 ± 0.3	2.1 ± 1.1*	0.3 ± 0.5*
Twitch	0.4 ± 0.4	0.8 ± 0.6	0.4 ± 0.5
d-Tubocurarine (n = 10)			
EMG	0.4 ± 0.3	1.0 ± 1.0*	0.5 ± 0.4*
Twitch	0.3 ± 0.2	0.4 ± 0.4	0.6 ± 0.4
Pancuronium (n = 10)			
EMG	0.3 ± 0.3†	1.2 ± 1.1*	0.1 ± 0.4*
Twitch	0.5 ± 0.5†	0.6 ± 0.6	0.3 ± 0.3
Vecuronium (n = 10)			
EMG	0‡	−0.4 ± 1.2*	−0.7 ± 0.6*
Twitch	0‡	−0.5 ± 0.6*	−0.5 ± 0.4

* Significant difference from previous number ($P < 0.05$; paired *t* test).

† Figures include five patients with constant neuromuscular blockade before the institution of bypass.

‡ Constant neuromuscular blockade in all patients.

In the absence of cardiopulmonary bypass, twitch tension has been known to correlate with the voltage of the evoked compound EMG.[10,11] During the pre-bypass period, we also found a significant positive correlation between the two parameters. However, in a previous study, we found that, in the absence of muscle relaxants, hypothermic bypass depressed twitch tension, while facilitating the EMG.[5] Such opposing effects of hypothermia on the EMG *versus* twitch tension were not recorded in the present study on neuromuscular blockade.

Hemodilution has been proposed as the reason for the rapid decrease of d-tubocurarine and pancuronium neuromuscular blockade following the initiation of bypass and cooling.[12–14] The acute reversal of neuromuscular blockade following the institution of the bypass suggests that this mechanism also applies to alcuronium. However, during active cooling, previous authors found sharply rising plasma concentrations of both d-tubocurarine and alcuronium.[6,15] Unfortunately, their first blood samples were drawn as late as 10 min after the institution of bypass. Thus, these results do not exclude hemodilution as an early cause of the bypass-associated decrease in block. However, they are not compatible with hemodilution as the cause of the attenuation of neuromuscular blockade following active cooling. Regrettably, also, monitoring of neuromuscular blockade was not part of these pharmacokinetic studies, nor was constant block established before the initiation of bypass.[6,7,15] More recently, with metocurine, the same authors found the EMG action poten-

tials to increase with active cooling, despite a rising metocurine plasma concentration.[4] Comparable data were published on pancuronium.[12] Having measured the urinary excretion of pancuronium following hypothermia, these authors were able to exclude compromised kidney function as a source of accumulation of pancuronium in the plasma.

Like vecuronium, atracurium has been shown to have a greater neuromuscular blocking potency during hypothermic cardiopulmonary bypass than at normal body temperature.[2,16] The increasing neuromuscular blocking potency was attributed to a reduced rate of drug metabolism during hypothermia.[2] Yet, in pharmacokinetic studies at normal body temperature, a slow disappearance of the atracurium metabolites from the plasma failed to correspond with a steep decay curve of the parent compound. Hence, contrary to current concepts, reversal of neuromuscular blockade being controlled by redistribution, rather than by metabolic degradation of the muscle relaxant, seems to apply not only to vecuronium,[17-19] but also to atracurium. Consequently, a reduced rate of metabolic degradation at low body temperature does not satisfactorily explain the increased neuromuscular blocking potency of vecuronium or atracurium. Likewise, in cats, Miller *et al.*[20] advocated an increased sensitivity of the neuromuscular junction to pancuronium to account for the increased neuromuscular blocking potency of this muscle relaxant at reduced body temperature. These authors' results are in contrast to our findings on pancuronium. However, our results on vecuronium support their interpretation. It may be hypothesized that, at the level of the neuromuscular junction, the facilitating or depressing effect of hypothermia on EMG voltage and twitch tension, respectively, may be either enhanced or inhibited by the particular muscle relaxant. Possible mechanisms include altered local diffusion and receptor affinity of the muscle relaxants as a function of temperature or the temperature gradient which were extensively studied by Holmes *et al.*[21] The present clinical results do not provide a basis for further analysis, which might explain the opposing effects of hypothermia on the neuromuscular blocking potency of different nondepolarizing muscle relaxants.

In summary, hypothermic cardiopulmonary bypass attenuated alcuronium, d-tubocurarine, and pancuronium neuromuscular blockade, while vecuronium neuromuscular blockade was enhanced. The different response of nondepolarizing neuromuscular blockade to hypothermic bypass may be explained by a direct effect of hypothermia at the neuromuscular junction, rather than by pharmacokinetic factors related to the bypass. The EMG is more sensitive than is twitch tension in reflecting the effect of changing body temperature on neuromuscular transmission. Following hypothermia, the contractile force of the skeletal muscle may fail to parallel the recovery of the evoked compound EMG.

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Evaluation of Intense Neuromuscular Blockade Caused by Vecuronium Using Posttetanic Count (PTC)

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Injection of a non-depolarizing relaxant in a dose sufficient for smooth tracheal intubation causes intense neuromuscular blockade of the peripheral muscles indicated by disappearance of the response to train-of-four (TOF) and single twitch stimulation.[1] It is possible to quantify part of this period of no response by applying tetanic stimulation (50 Hz for 5 s) followed by 1 Hz stimulation and observing the posttetanic single twitch response (posttetanic count or PTC). A close correlation was found to exist between PTC and recovery from intense blockade caused by pancuronium.[2,3]

The objective of the present study was to evaluate the relationship between PTC and recovery from intense neuromuscular blockade caused by vecuronium.

METHODS AND MATERIALS

The study plan was approved by the College of Medicine Research Center of our institution. Informed consent was not sought, because neuromuscular monitoring is considered routine, is non-invasive, and does not pose any risk to the patient. No patient had any neuromuscular disease or received any drug that might influence neuromuscular function.

Sixty adult patients (ASA physical status I-II) undergoing intraabdominal or orthopedic procedures were studied. The patients ranged in age from 16–65 yr (mean: 32 yr), and there were 20 females and 40 males. All patients were given lorazepam 1–3 mg orally 2–4 h before induction. The patients were divided randomly into two groups of 30 each. In all patients, anesthesia was induced with sodium thiopental 3–5 $mg \cdot kg^{-1}$ body weight. Patients in group 1 were then allowed to breathe halothane in 50% O_2 with N_2O sufficient to achieve surgical plane of anesthesia (0.6–2.0% inspired halothane concentration as indicated by a Fluotec Mark III vaporizer). Patients in group 2 breathed 30% O_2 with N_2O, supplemented with fentanyl 2–4 $mg \cdot kg^{-1}$. Following induction of anesthesia, the ulnar nerve was stimulated at the wrist through cutaneous electrodes, and the response of the adductor pollicis muscle was recorded using the Myograph 2000 neuromuscular transmission analyzer.[2] TOF nerve stimulation was used every 12 s. After supramaximal stimulation was achieved, 0.1 $mg \cdot kg^{-1}$ body weight vecuronium was given intravenously. The trachea was intubated when the response to TOF stimulation had disappeared, and the patients were ventilated with a tidal volume of 10 $ml \cdot kg^{-1}$ and a respiratory rate of 12 min^{-1}, the aim being to maintain normocapnia (arterial blood gas measurements were made periodically). As cooling of the patients was not a problem in Saudi Arabia, body temperature was not monitored routinely. Every 6 min, the mode of nerve stimulation was changed: 1 Hz twitch stimulation was given for 1 min, followed by tetanic stimulation (50 Hz) for 5 s. After a pause of 3 s, the single twitch stimulation was resumed. If there was no response to this stimulation (PTC = 0), or when the observed response had faded to zero, the TOF mode of stimulation was reinstituted until

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