Anesthesiology
46:366–368, 1977

Comparison of Metocurine and Pancuronium—Myocardial Tension–Time Index during Endotracheal Intubation

JAMES W. BASTA, M.D.,* AND MONTE LICHTIGER, M.D.†

The anesthetic management of patients undergoing coronary-artery revascularization procedures must be conducted with meticulous attention to factors that increase myocardial oxygen demand. The three major determinants of myocardial oxygen utilization are heart rate, myocardial contractility, and intraventricular wall tension.[1] A readily measurable indicator of myocardial oxygen demand, the tension–time index (TTI), has been extensively studied by Sarnoff and Braunwald[2]; it is described as the product of the heart rate and the area beneath the systolic portion of the aortic pressure curve. With the use of the TTI it is possible to assess effects of anesthetic techniques on myocardial oxygen demand.

The cardiovascular effects of pancuronium bromide[3] and metocurine iodide[4] have been studied in intubated patients during moderate levels of halothane–N_2O anesthesia. Both relaxants produce good clinical levels of relaxation, but only metocurine has minimal cardiovascular effects. The purpose of this study was to compare the effects of metocurine and pancuronium on changes in the myocardial TTI evoked by tracheal intubation in patients with coronary-artery disease.

METHODS

Thirty-three adult patients who had histories of angina pectoris (New York Heart Association cardiac status 3–4) and angiographic evidence of significant occlusive coronary-artery disease were randomly divided into two groups. Both groups of patients were undergoing coronary-artery revascularization procedures and were free of valvular heart disease. All were given scopolamine (0.4 mg, im), diazepam (5–15 mg, im) and morphine (7–15 mg, im) 60 minutes prior to induction of anesthesia.

In the operating room, peripheral intravenous, central venous and radial arterial catheters were inserted using local anesthesia. The intra-arterial and central venous catheters were connected to Statham P23DB pressure transducers. Electrocardiograph leads were also applied. The electrocardiographic and pressure data were displayed on an Electronics for Medicine VR-6 monitor with photographic recorder.

General anesthesia was induced with halothane and equal concentrations of N_2O and oxygen, delivered through a semiclosed circle system with CO_2 absorber. After loss of the lid reflex, the patients were given pancuronium, 0.1 mg/kg, iv (22 patients) or metocurine, 0.3 mg/kg, iv (11 patients). Induction of anesthesia was continued with controlled ventilation until the systolic blood pressure had been lowered to 95–125 torr. Inspired halothane concentrations were usually 1–1.5 per cent at this time. Simultaneous recordings of the electrocardiogram, blood pressure, and central venous pressure were made immediately before and after tracheal intubation. The TTI was calculated from analysis of the radial arterial pressure recordings. Student's t test for paired data was used to analyze the data obtained before and after intubation within each group; Student's t test for unpaired data was utilized to compare the pancuronium group with the metocurine group.

RESULTS

The data obtained from the pancuronium and metocurine groups are presented in tables 1 and 2, respectively. The pancuronium group had a mean preintubation heart rate of 89/min and a mean arterial blood pressure of 78 torr. After intubation the average heart rate increased by 16 per cent to 103/min and the mean blood pressure increased 33 per cent to 104 torr.

The metocurine group had a mean preintubation heart rate of 74/min and a mean blood pressure of 73 torr. After intubation the average heart rate increased 19 per cent to 88/min and the mean arterial blood pressure 29 per cent to 94 torr.

The tension-time index values for the two groups are presented in table 3. In the pancuronium group the TTI increased 49 per cent from a mean of 1,888 to 2,803 torr sec/min. In the metocurine group the TTI increased 41 per cent from a mean of 1,567 to 2,214 torr sec/min.

Analysis of the data reveals that prior to intubation there were differences ($P < 0.05$) between the heart rates and TTI values in the two groups. There was no significant difference in the mean arterial

* Fellow in Cardiovascular Anesthesia.
† Clinical Associate Professor of Anesthesiology.

Received from the Department of Anesthesiology, Mount Sinai Medical Center, Miami Beach, Florida, and the University of Miami School of Medicine, Miami, Florida. Accepted for publication December 18, 1976.

Address reprint requests to Dr. Lichtiger.

pressures. After intubation there were significant increases in heart rate, blood pressure, and TTI in both groups ($P < 0.001$). However, the post-intubation heart rate, mean blood pressure, and TTI were significantly greater in the pancuronium group.

We also found that arrhythmias frequently developed after administration of pancuronium; 14 per cent of the patients given pancuronium developed nodal rhythms; in two-thirds of these patients ventricular rates of 120–130/min developed following intubation. S-T segment depression of 1 mm or more occurred in 18 per cent of patients given pancuronium. No arrhythmia or S-T segment change was seen following administration of metocurine.

Discussion

More patients with ominous anginal symptoms and/or angiographic evidence of severe coronary-artery disease are being anesthetized.[5,6] As the preoperative evaluation and operative indications are further refined, anesthetic management of this group of high-risk patients must also be systematically improved if we are to continue to meet the anesthetic challenge they represent.

The primary consideration in the management of these patients is the avoidance of myocardial ischemia, which may precipitate congestive heart failure, arrhythmias, or myocardial infarction. Significant coronary-artery disease reduces the ability of the coronary vessels to autoregulate in response to increased myocardial oxygen demand. This produces relatively fixed limits of oxygen supply to the myocardium. With a fixed oxygen supply, the moment-to-moment adequacy of myocardial oxygenation will depend upon the oxygen demand. This oxygen demand, in turn, is governed primarily by the heart rate, myocardial contractility, and intraventricular wall tension.[1] The anesthesiologist has at his disposal a clinically valuable indicator of myocardial oxygen demand, the TTI. By utilizing the TTI as a guide to proper anesthetic technique, improvements in anesthetic management are possible.

Halothane anesthesia is commonly used for patients undergoing coronary-artery surgery because it reduces heart rate, myocardial contractility and blood pressure.[7] Anything obviating these oxygen-sparing effects is detrimental. Laryngoscopy and intubation of the trachea, even under favorable conditions, may produce reflex increases in heart rate and blood pressure.[8,9] This response, which increases myocardial oxygen demand, can be minimized by deepening anesthesia. However, this may result in unacceptable levels of hypotension with decreased coronary blood flow. Topical anesthesia of the trachea prior to intubation does not eliminate the cardiovascular response[10] and can, by

TABLE 1. Pre- and Post-intubation Heart Rates and Blood Pressures, Pancuronium Group

Patient	Pre-intubation		Post-intubation	
	Heart Rate (Beats/Min)	Mean Blood Pressure (torr)	Heart Rate (Beats/Min)	Mean Blood Pressure (torr)
1	90	87	90	103
2	105	82	130	115
3	80	80	90	102
4	70	63	80	102
5	90	82	110	120
6	60	77	90	97
7	95	73	115	100
8	80	65	100	108
9	70	72	80	115
10	90	75	90	92
11	92	80	120	93
12	120	77	135	105
13	120	72	130	97
14	120	93	110	113
15	115	80	120	93
16	80	73	105	102
17	70	73	80	87
18	90	77	110	115
19	75	70	80	126
20	70	78	70	77
21	90	88	130	120
22	90	93	95	97
Mean ± SD	89 ± 18	78 ± 8	103 ± 20	104 ± 2

TABLE 2. Pre- and Post-intubation Heart Rates and Blood Pressures, Metocurine Group

Patient	Pre-intubation		Post-intubation	
	Heart Rate (Beats/Min)	Mean Blood Pressure (torr)	Heart Rate (Beats/Min)	Mean Blood Pressure (torr)
1	95	72	100	120
2	70	67	90	103
3	70	73	80	93
4	70	80	90	97
5	80	73	90	95
6	70	70	85	77
7	80	83	90	92
8	78	80	80	87
9	60	58	90	90
10	70	77	90	87
11	75	75	80	92
MEAN ± SD	74 ± 9	73 ± 7	88 ± 6	94 ± 11

TABLE 3. Pre- and Post-intubation Tension–Time Indexes (torr sec/min)

	Number of Patients	Pre-intubation	Post-intubation	P	Per Cent Change
Pancuronium	22	1,888 ± 498	2,803 ± 620	<0.001	49
Metocurine	11	1,567 ± 188	2,214 ± 277	<0.001	41
P	—	<0.05	<0.01	—	—

itself, produce reflex cardiovascular stimulation.[9] Thus, the use of moderate levels of anesthesia with meticulous airway management and good muscle relaxation to facilitate intubation is a reasonable approach to the anesthetic management of these patients. The choice of relaxant, however, must also be appropriate for the patient.

Pancuronium bromide has vagolytic properties and is reported to produce 20–50 per cent increases in heart rate when administered during halothane anesthesia.[11] These findings are consistent with our observations. Pancuronium did not produce a significant increase in blood pressure. However, in our study the reported preintubation blood pressures were purposely maintained at comparable levels in the two groups by the use of halothane. The vagolytic properties of pancuronium seem to exaggerate the sympathetic response to intubation, which increases myocardial oxygen demand.

Metocurine iodide in intubated patients during halothane–N_2O anesthesia has been found to be without significant cardiovascular effects when administered iv in doses of 0.2 mg/kg.[4] This study demonstrates that metocurine iodide, compared with pancuronium in the clinical setting described, is also associated with less increase in myocardial oxygen demand. For this reason we believe metocurine is especially useful for management of patients who have minimal cardiac reserves, in whom increases in heart rate and blood pressure should be minimized. Additional study of the effects of anesthetic techniques on myocardial TTI (oxygen demand) should point toward further improvements in management of the high-risk patient who has coronary-artery disease.

References

1. Bland JHL, Lowenstein E: Halothane-induced decrease in experimental myocardial ischemia in the non-failing canine heart. ANESTHESIOLOGY 45:287–293, 1976
2. Sarnoff SJ, Braunwald E, Welch GH, et al: Hemodynamic determinants of the oxygen consumption of the heart with special reference to the tension–time index, Work and the Heart. Edited by FF Rosenbaum, EL Belknapp. New York, Paul B. Hoeber, Inc., 1959, pp 18–33
3. Stoelting RK: The hemodynamic effects of pancuronium and *d*-tubocurarine in anesthetized patients. ANESTHESIOLOGY 36:612–615, 1972
4. Stoelting RK: Hemodynamic effects of dimethyltubocurarine during nitrous oxide–halothane anesthesia. Anesth Analg (Cleve) 53:513–515, 1974
5. Bonchek LI, Rahimtoola SH, Anderson RP, et al: Late results following emergency saphenous vein bypass grafting for unstable angina. Circulation 50:972–977, 1974
6. Miller DC, Cannom DS, Fogarty TJ, et al: Saphenous vein coronary artery bypass in patients with "preinfarction angina." Circulation 47:234–241, 1973
7. Vatner SF, Smith NT: Effects of halothane on left ventricular function and distribution of regional blood flow in dogs and primates. Circ Res 34:155–167, 1974
8. King BD, Harris LC, Greifenstein FE, et al: Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. ANESTHESIOLOGY 12:556–566, 1951
9. Wycoff C: Endotracheal intubation: Effects on blood pressure and pulse rate. ANESTHESIOLOGY 21:153–158, 1960.
10. Dewlinger JK, Ellison N, Ominsky AJ: Effects of intratracheal lidocaine on circulatory responses to tracheal intubation. ANESTHESIOLOGY 41:409–412, 1974
11. Miller RD, Eger EI, Stevens WC, et al: Pancuronium-induced tachycardia in relation to alveolar halothane, dose of pancuronium, and prior atropine. ANESTHESIOLOGY 42:352–355, 1975

Anesthesiology
46:368–370, 1977

Anesthesia for Repair of a Pulmonary-artery Sling in an Infant with Severe Tracheal Stenosis

CHARLES H. MCLESKEY, M.D.,* AND WAYNE E. MARTIN, M.D.†

The combination of pulmonary-artery sling and severe tracheal stenosis has been reported three times previously. This paper reports the only known survivor to date with this combination of congenital defects.

The infant's marked obstructive airway problem was thought to be due to both severe tracheal stenosis and a pulmonary-artery sling. It was decided to attempt definitive repair of the pulmonary-artery sling, since results of surgical correction of stenotic tracheas in infants are universally poor. A solution to the difficult problem of intraoperative and postoperative airway management utilizing a Cole‡ tube with amputated tip, positive end-expiratory pressure (PEEP), and systemic and topical steroids is described in this report.

* Resident.

† Associate Professor.

Received from the Department of Anesthesiology and the Anesthesia Research Center, University of Washington School of Medicine, Seattle, Washington 98195. Accepted for publication January 5, 1977.

Address reprint requests to Dr. McLeskey: Department of Anesthesiology, Naval Regional Medical Center, Oakland, California 94627.

‡ Cole endotracheal tube manufactured by Foregger Labs, Division of Air Products, Inc., Allentown, Pa. 18105.