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## The Hemodynamic Response to Traction on the Abdominal Mesentery

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An association between traction on the abdominal mesentery and decreases in systemic blood pressure is reported in several major textbooks and review articles.<sup>1-4</sup> While opinion varies about the etiology of this response and available literature is confusing and self-contradictory, the majority of sources state mesenteric traction causes an afferent sympathetic stimulation that results in vasodilation of the splanchnic system and venous pooling in the splanchnic capacitance vessels.<sup>1,3,4</sup> This splanchnic pooling is assumed to result in a decreased venous return to the heart and a subsequent decreased stroke volume and cardiac output. All agree that this response is not uniform in that hypotensive episodes do not universally occur. One text suggests that bradycardia is sometimes present.<sup>1</sup> Although there seems to be an agreement on the basic mechanisms, we were unable to find studies that documented this sequence of events. We therefore examined the hemodynamic changes associated with traction on the abdominal mesentery.

### METHODS

Twenty adult patients ASA Class II-IV scheduled for elective aortic surgery gave their informed consent to participate in this study, which was approved by our Institutional Review Board. The patients ranged in age from 55 to 77 yr ( $62.8 \pm 7.3$  yr, mean  $\pm$  SD), in weight from 48 to 109 kg ( $76 \pm 14$  kg, mean  $\pm$  SD) and in surface areas from 1.54 to 2.35 m<sup>2</sup> ( $1.94 \pm 0.18$  m<sup>2</sup>, mean  $\pm$  SD). Nine had histories of coronary artery disease, eight of hypertension, one of treated congestive heart failure, and two of diabetes mellitus. Four patients

were receiving  $\beta$ -blockers, while four were receiving non- $\beta$ -blocking antihypertensive or diuretics. Three patients were taking cimetidine. Data for all patients entered into the study are used in this report. All patients were premedicated with morphine sulfate 0.08 mg/kg and atropine 0.4 mg in 45-90 min before the induction of anesthesia. These premedicants were chosen to duplicate standard clinical conditions. After insertion of intravenous, radial artery, and thermodilution pulmonary artery catheters, baseline awake measurements were obtained. Heart rate (HR); systolic, diastolic, and mean arterial pressure (MAP); systolic and diastolic pulmonary artery pressure; and pulmonary capillary wedge pressure (PCWP) and central venous pressure (CVP) were recorded from a Bentley Trantec<sup>®</sup> transducer (model 800) and a Hewlett-Packard<sup>®</sup> monitor (modules 782050). Cardiac outputs were determined in triplicate at end expiration using 10 ml room-temperature 5% dextrose in water injected into the right atrial lumen of an Edwards Laboratory 7F triple-lumen pulmonary artery catheter connected to an Edwards Laboratory Model 9520A cardiac output computer. Stroke volume (SV), stroke index (SI), systemic vascular resistance (SVR), pulmonary vascular resistance, and left and right ventricular stroke work index were calculated according to standard formulae.<sup>5</sup> Anesthesia was induced with thiopental 4-6 mg/kg iv, fentanyl 10  $\mu$ g/kg iv, and 60% N<sub>2</sub>O in O<sub>2</sub>. Following succinylcholine 1.5 mg/kg iv, the trachea was intubated and ventilation was controlled. Adequacy of ventilation was confirmed in all patients by arterial blood gas analysis prior to the study period. Metubine 0.3 mg/kg was administered to relax the abdominal muscles. Additional increments of thiopental 50 mg iv and fentanyl 25  $\mu$ g iv were given when clinically indicated by a 20% increase in the heart rate or the systemic blood pressure. However, no additional anesthetic drugs were administered for 5 min before the start of the study period or during the study. At the time the study measurements were begun, thiopental  $6.1 \pm 1.5$  mg/kg, fentanyl  $10.2 \pm 2.2$   $\mu$ g/kg, and metubine  $0.34 \pm 0.1$  mg/kg (mean  $\pm$  SD) had been administered. No vasoactive agents were given at any time

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TABLE 1. Hemodynamic Measurements and Derived Data (means  $\pm$  SD) for Awake, Premesenteric Traction and Postmesenteric Traction Periods (n = 20)

	Awake	Premesenteric Traction	<i>P</i> versus Awake	Postmesenteric Traction	<i>P</i> versus Premesenteric
Heart rate (beats $\cdot$ min <sup>-1</sup> )	76 $\pm$ 13.1	66.6 $\pm$ 13.7	<0.002	77.3 $\pm$ 16.8	<0.001
Mean arterial pressure (mmHg)	117 $\pm$ 19	105 $\pm$ 20	<0.02	81 $\pm$ 19	<0.001
Cardiac output (l $\cdot$ min <sup>-1</sup> )	7.74 $\pm$ 1.92	5.81 $\pm$ 1.93	<0.01	7.66 $\pm$ 2.29	<0.001
Cardiac index (l $\cdot$ min <sup>-1</sup> m <sup>-2</sup> )	4.0 $\pm$ 0.98	3.0 $\pm$ 0.94	<0.01	3.96 $\pm$ 1.22	<0.001
Stroke volume (ml)	103 $\pm$ 26	88 $\pm$ 25	<0.02	100 $\pm$ 22	<0.02
Stroke index (ml $\cdot$ m <sup>-2</sup> )	53 $\pm$ 12	45 $\pm$ 11	<0.01	51 $\pm$ 10	<0.02
Central venous pressure (mmHg)	5.2 $\pm$ 5.1	11 $\pm$ 4.7	<0.01	9.7 $\pm$ 5.1	<0.02
Mean pulmonary artery pressure (mmHg)	19.4 $\pm$ 7.3	20.9 $\pm$ 6.3	NS	19.8 $\pm$ 7.0	NS
Pulmonary capillary wedge pressure (mmHg)	15.1 $\pm$ 6.7	17.9 $\pm$ 6.2	<0.02	15.7 $\pm$ 5.8	<0.001
Systemic vascular resistance (dyn $\cdot$ s $\cdot$ cm <sup>-5</sup> )	856 $\pm$ 651	1,423 $\pm$ 504	<0.05	839 $\pm$ 405	<0.001
Pulmonary vascular resistance (dyn $\cdot$ s $\cdot$ cm <sup>-5</sup> )	45 $\pm$ 41	51 $\pm$ 45.2	NS	50 $\pm$ 58	NS
Left ventricular stroke work index (g $\cdot$ m <sup>-1</sup> )	72.7 $\pm$ 20.6	52.4 $\pm$ 15.6	<0.001	44.3 $\pm$ 11.6	<0.05
Right ventricular stroke work index (g $\cdot$ m <sup>-1</sup> )	3.2 $\pm$ 3.1	2.1 $\pm$ 1.7	NS	2.8 $\pm$ 3.1	NS

before or during the study. After the peritoneal cavity was opened, the surgeons refrained from further manipulations until the premesenteric traction measurements were made. Mesenteric traction was obtained by exteriorizing the small bowel and placing two retractors on the mesentery, one on the midportion and the other on the upper mesentery, and applying sufficient traction to expose the abdominal aorta. No further surgical maneuvers were performed until after the postmesenteric traction measurements were obtained. These were completed in 5–10 min after the onset of mesenteric traction when an obvious decrease in systemic blood pressure was observed. The changes that occurred between each of the three measurement periods were compared using paired *t* tests.

### RESULTS

Traction on the abdominal mesentery was associated with a significant decrease in MAP. The SV, SI, CO, cardiac index, and HR increased significantly. The SVR, left ventricular stroke work index, CVP, and PCWP all decreased significantly. Table 1 shows the means, standard deviations, and the associated *P* values for all the hemodynamic data. All patients had a decrease in MAP with mesenteric traction. In 19 of the 20 patients, the decrease was due to a decreased SVR, which was accompanied by an increased CO. The one patient whose decrease in MAP was apparently secondary to a decrease in CO actually showed very small changes. His CO decreased 0.2 l/min, while MAP decreased 3 mmHg, HR increased 4 beats/min, and SVR increased 81

dyn  $\cdot$  s  $\cdot$  cm<sup>-5</sup>. Changes in the remaining hemodynamic variables were less consistent.

In addition to the hemodynamic changes, 15 of the 20 patients developed marked flushing of the skin in the area observable, namely the head and neck. The flushing coincided with the decrease in MAP. No bronchospasm was noted at the time of the flushing. The patient in whom minimal hemodynamic changes were noted was one of the five who did not exhibit flushing with mesenteric traction.

The postmesenteric traction measurements were made after an obvious decrease in blood pressure was noted. This occurred in 8.6  $\pm$  2.7 min (mean  $\pm$  SD) (range 5–12 min) following the premesenteric traction measurements. No effort was made to time the duration of the response, but the flushing, low vascular resistance, and increased cardiac output persisted for more than 30 min in many cases. An increase in the rate of fluid administration was usually the only treatment needed to maintain acceptable systemic blood pressures. In one patient three 5-mg doses of ephedrine sulfate was given to rapidly restore the systolic arterial pressure and further increase cardiac output. This occurred in a patient with severe coronary artery disease whose systolic blood pressure decreased to 75 mmHg and whose cardiac output only reached 4.46 l/min from 3.44 l/min after mesenteric traction.

### DISCUSSION

In four representative texts the frequency of hypotension due to abdominal traction is: not noted<sup>1</sup>; said to

occur sometimes<sup>2</sup>; may occur<sup>3</sup>; or is not uncommon.<sup>4</sup> Our results suggest that there is a universal decrease in MAP under N<sub>2</sub>O, narcotic, relaxant anesthesia in this group of adult patients with cardiovascular and other systemic diseases when traction is applied to the abdominal mesentery. While the mean decrease in MAP was about 20 mmHg, the individual responses ranged from as little as 3 mmHg to as great as 57 mmHg. The small decreases in blood pressure might not be readily appreciated if blood pressure were not being recorded carefully. If it is assumed that the primary response to mesenteric traction is systemic vasodilation, then the variations in the responses of systemic blood pressure can be explained by the magnitude of compensatory increase in cardiac output.

It is easy to see why the mesenteric traction response was not considered to be universal. In fact, Stoelting *et al.*,<sup>6</sup> were unable to demonstrate any significant change in systemic blood pressure after gastric or gallbladder traction. They did not measure cardiac output and confined their study period to 30 ss of traction and additional 30 ss of blood pressure recordings. Since we did not note the maximum changes to occur until at least 5 min of traction, it is easy to see why these authors may have missed a response if one is associated with gastric or gallbladder traction.

Our study does not address the cause of the decreased SVR. Strunin<sup>2</sup> believes that hypotension due to abdominal traction may be similar to stimulation of the splanchnic plexus, which results in dilation of the splanchnic capacitance vessels with blood pooling and decreased venous return to the heart. Kaufman<sup>3</sup> simply states that systemic hypotension may be produced by traction on abdominal viscera due to afferent sympathetic stimulation. Batchelder and Cooperman<sup>4</sup> describe a reflex arc that they presume to be via sympathetic afferents, resulting in efferent mediated dilation of the splanchnic capacitance vessels. Smith<sup>7</sup> speculates, without supporting data, that the decrease in systemic blood pressure associated with abdominal manipulation is due to a celiac plexus reflex, which causes a fall in blood pressure secondary to vagal cardiac inhibition, resulting in decreased cardiac output. Burstein<sup>8</sup> reports that pressure on the celiac plexus of dogs causes a fall in systemic pressure. This response is enhanced by atropine and blocked by physostigmine. Further studies described by Burstein showed that elective stimulation of the celiac plexus caused an increase in blood pressure and a decrease in heart rate. The discussion in Dripps *et al.*<sup>1</sup> also speaks of a reflex arc and implicates the autonomic nervous system. However, these authors are not clear whether the afferent impulses ascend via the sympathetics, phrenic, vagus, or possibly the intercostal nerves. They suggest that the net result is an inhibition of

sympathetic activity and increased vagal tone on the heart, resulting in reduced myocardial contractility and stroke volume. They also note that bradycardia occasionally occurs as a result of mesenteric traction. Although not referenced, these conclusions are presumably based on the work of Rocco and Vandam,<sup>9</sup> who studied 68 patients with continuous systemic blood pressure monitoring obtained from an indwelling arterial needle. Using a variety of anesthetic techniques, they demonstrated a significant decrease in arterial pressure in 55 patients related to intraabdominal manipulation. However, when deliberate traction was applied to the mesenteries, some patients showed no change in blood pressure. Since the authors did not measure cardiac output in this study, we believe that in light of our data, lack of change in arterial pressure was probably due to compensation by an increased cardiac output. They also noted that no change in blood pressure could be obtained by direct stimulation of the celiac plexus. The heart rate response was varied in their study as it was in ours. The majority of their patients showed no change or a decrease in heart rate, while a few increased their heart rate. In our study 16 patients increased, three decreased, and one had no change in heart rate in response to mesenteric traction. The concepts of sympathetic mediated vasodilation<sup>2-4</sup> are challenged by Jacobson,<sup>10</sup> who describes constriction of splanchnic blood vessels with increased sympathetic activity, while splanchnic vasodilation takes place secondary to parasympathetic stimuli. While we cannot comment on the existence of any type of reflex arc, we believe that significant venous pooling, with decreased return to the heart, does not take place. Even though our data shows a statistically significant decrease in CVP ( $11 \pm 4.7$  to  $9.7 \pm 5.1$  mmHg) and PCWP ( $17.9 \pm 6.2$  to  $15.7 \pm 5.8$  mmHg), we consider these small decreases to be more likely due to generalized vasodilation, since we have demonstrated significant increases in cardiac output. Furthermore, many patients demonstrated a decrease in systemic MAP and SVR, in spite of increases or no changes in CVP and PCWP.

Flushing of the head and neck associated with mesenteric traction has not been previously described. Although the flushing is undoubtedly due to vasodilation of the skin vessels, the mechanism is unknown. It is reasonable to consider a hormonal mechanism. The parenchymal cells of the splanchnic organs are capable of producing nucleotides (cyclic AMP + ATP), polypeptides (bradykinins), amines (histamine), and fatty acid derivative (prostaglandins).<sup>10</sup> The time lapse (5-10 min) from initiation of mesenteric tractions to the marked response might also add support to the possibility of a hormonal mechanism.

In summary, our results are in contrast to published accounts of the hemodynamic effects of abdominal mes-

enteric traction during anesthesia that indicate the response to be an occasional decrease in systemic blood pressure due to venous pooling with decreased return to the heart and subsequent decrease in CO. Our data indicate that the response is a nearly universal decrease in MAP secondary to marked systemic vasodilation accompanied by a compensatory increase in CO. Changes in HR, CVP, and PCWP varied, and there seemed to be no significant effect on the pulmonary vascular system.

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## Failure of Metronidazole to Alter a Vecuronium Neuromuscular Blockade in Humans

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Neuromuscular blocking effects of muscle relaxants can be enhanced by some antibiotics, especially aminoglycosides, polymyxins, and tetracyclines.<sup>1-4</sup> Recently, in the last few years, prophylactic administration of large doses of nitroimidazole-type drugs have protected patients against systemic gram-negative bacterial infections

during and after elective colonic surgery.<sup>5</sup> Recently, McIndewar and Marshall found a marked potentiation of a neuromuscular blockade from vecuronium by metronidazole in  $\alpha$ -chloralose/pentobarbital-anesthetized cats.<sup>6</sup> Using routine neuromuscular monitoring procedures, we sought to determine whether a vecuronium neuromuscular blockade is enhanced in patients receiving a preoperative iv administration of metronidazole.

#### MATERIALS AND METHODS

The present study was approved by the Ethical Committee of Human Research of the Brussels Free University. Cumulative dose-effect curves were determined for vecuronium following metronidazole administration. Adult patients of both sexes (ASA Class I-II) were selected after their informed consent had been obtained. None of these patients had any clinical or biochemical evidence of renal or liver function abnormalities. The patients were free of neurologic abnormalities. One hour before anesthesia, all the patients received diazepam 200  $\mu\text{g} \cdot \text{kg}^{-1}$  orally.

Before the start of anesthesia, a displacement transducer (UC3 cell Statham), fitted with tension attenuator

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