Defining the critical limit of oxygen extraction in the human small intestine

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Although animal models have been used to characterize the relation between oxygen consumption and blood flow, reliable data have not been generated in the human small intestine. We perfused segments of human small intestine by using an ex vivo perfusion circuit that allowed precise manipulation of blood flow and perfusion pressure. Our goal was to define the critical level of intestinal blood flow necessary to maintain the metabolic needs of the tissue. Human small intestine (n = 5) tissue obtained at transplantation harvest was transported on ice to the laboratory. A 40-cm mid-jejunal segment was selected for perfusion, and appropriate inflow and outflow vessels were identified and cannulated. Perfusion with an autologous blood solution was initiated through an extracorporeal membrane oxygenation circuit. After a 30-minute equilibration period, arterial and venous blood gases were measured at varying flow rates while maintaining a constant hematocrit level. Arterial and venous oxygen content, arteriovenous oxygen difference (A-$\dot{V}O_2$ diff), and oxygen consumption ($\dot{V}O_2$) were then calculated. Our results demonstrated that at blood flows >30 ml/min/100 g, $\dot{V}O_2$ is independent of blood flow (1.6 ± 0.06 ml/min/100 g), and oxygen extraction is inversely related to flow. Below this blood flow rate of 30 ml/min/100 g, oxygen extraction does not increase further (6.3 ± 0.3 vol%), and $\dot{V}O_2$ becomes flow dependent. This ex vivo preparation defines for the first time a threshold value of blood flow for small intestine below which oxygen consumption decreases (30 ml/min/100 g). Previous animal studies have correlated such a decrease in oxygen consumption with functional and histologic evidence of tissue injury. This "critical" flow rate in human intestine is similar to that found previously in canine and feline intestine, but lower than that of rodent species. (J Vasc Surg 1996;23:832-8.)

The clinical syndrome of mesenteric ischemia results in significant morbidity and mortality from frequent delays in diagnosis and difficulty in identifying patients at risk before a catastrophic event. Integral to our ability to improve the outcome of these patients is an understanding of the regulation of intestinal circulation, particularly the regulation of intestinal oxygen consumption. Oxygen consumption can be affected by changes in blood flow and oxygen extraction; the interrelation of these factors has been described in animal models. An essential extension of this work would be the definition of the relation of blood flow to oxygen consumption in the human small intestine. This definition would allow better design of treatment strategies in low-flow ischemia.

Studies in canine, feline, and rat intestine have indicated that initial decreases in blood flow result in reciprocal changes in oxygen extraction by the tissue that allow initial maintenance of oxygen consumption.[1-3] Decreases in oxygen consumption occur only after a "critical limit" of blood flow for a given organ is reached, and further increases in oxygen extraction cannot compensate for further decreases in blood flow. At this level tissues are unable to maintain normal aerobic metabolism, and cellular dysfunction and death occur when limited cellular anaerobic sources are no longer able to provide energy for critical metabolic processes.[4]

Because this important relation between blood flow and oxygen consumption is both organ- and species-specific, our experiments were undertaken to delineate this relation for human tissue. We hypothesized that basal oxygen consumption would be less in human intestine than in rat and other small animals,

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but that the relation of oxygen consumption, oxygen extraction, and blood flow would be qualitatively similar. A unique ex vivo perfusion preparation allowed the controlled study of isolated segments of human small intestine obtained during organ procurement for transplantation. The qualitative and quantitative relation between blood flow and oxygen consumption was defined, providing for the first time the option of ex vivo testing of treatment strategies in human intestinal ischemia.

METHODS

Ex vivo perfusion preparation. Human small intestine tissue was harvested after removal of transplantable organs at human organ procurements when consent for use of nontransplantable tissues for research was obtained. Initial dissection of the transplantable organs was performed in donors who were brain-dead but had a beating heart and systolic blood pressure maintained at 90 mm Hg or greater with fluid or pressors. After this initial dissection, the supraceliac abdominal aorta was cross-clamped just inferior to the diaphragm while perfusing the entire splanchnic bed with 2 to 3 liters cold Belzer UW-CSS solution through a cannula placed in the abdominal aorta just proximal to the iliac bifurcation. The donor was also simultaneously exsanguinated through the intrathoracic vena cava, and the abdominal contents were cooled by placement of normal saline slush into the abdominal cavity. Transplantable organs were then removed. The superior mesenteric artery was ligated near its point of origin, and the hepatic graft was taken en block with the proximal abdominal aorta. The small bowel was then harvested with its mesentery resecting from the ligament of Treitz to the terminal ileum. The bowel was stored cold and transported to the laboratory.

On arrival to the laboratory, a 40-cm mid-jejunal segment was selected for perfusion; appropriate inflow and outflow vessels to this segment were identified. The mid-jejunal segment to be perfused was weighed, biopsied, and placed on a 37° C isotonic bath of normal saline to prevent dessication. A thermistor-controlled incandescent lamp was used to facilitate rewarming and to maintain the preparation at 37° C throughout the perfusion.

The mesenteric vessels were cannulated (PE240 tubing), the bowel was clamped proximally and distally, and cannulas were placed at both ends of the selected segment with small enterotomies. The bowel segment was perfused with a perfusate consisting of heparinized blood obtained from the organ donor at the harvest by cannulation of the inferior vena cava. The blood was washed twice with normal saline and centrifuged at 4000 rpm for 10 minutes (Megafuge 1.0, model 3492; Haraeus Instruments, Boston) to remove any Belzer solution. The packed red blood cells were then reconstituted in modified Krebs buffer solution, including albumin (3%) and 5 mmol/L glucose to produce an osmolality of 290 mOsm/L and a hematocrit level of 20% to 25%. The perfusate was placed in a reservoir from which it was pumped through an extracorporeal membrane oxygenator supplying 20% oxygen, 75% nitrogen, and 5% carbon dioxide. The perfusion pressure was maintained at 60 to 90 mm Hg; pressure was continuously monitored by a transducer placed in the arterial line. Arterial inflow and venous effluent were also diverted through an arteriovenous oxygen difference (A-V O_2) analyzer (AVOX Systems, San Antonio, Texas) to provide continuous A-V O_2 difference (Fig. 1).[5]

Vascular perfusion was initiated as the intestinal segment was rapidly rewarmed to 37° C. The segment was initially allowed to equilibrate for 30 minutes at perfusion pressures of 60 to 90 mm Hg and blood flows of 20 to 30 ml/min/100 g. Viability was indicated by serosal hyperemia, the return of active peristalsis, and baseline oxygen consumption >1.5 ml/min/100 g.

After this initial equilibration period, the blood flow was varied by the pump at 15-minute intervals. At the end of each 15-minute period, blood flow was determined by measuring the volume of venous effluent over 60-second period in a graduated cylinder. Additionally, arterial and venous blood gas samples were obtained through ports in the arterial inflow line and venous outflow line. Perfusate hematocrit was measured at the onset of perfusion and at 30-minute intervals throughout the preparation.

The perfusion was continued for 90 minutes, at which time the segment was opened, blotted dry, and weighed. Histologic integrity of the intestinal tissue was confirmed by hematoxylin and eosin stained biopsies obtained at the beginning and end of perfusion.

Calculations and statistics. The measurements of intestinal blood flow, perfusate hemoglobin, and arterial and venous blood gases and the weight of the intestinal segment were used to calculate arterial and venous oxygen contents, arteriovenous oxygen difference, and oxygen consumption. Blood flow was expressed as milliliters of venous outflow/min/100 g tissue. Arterial (C_aO_2) and venous (C_vO_2) oxygen contents and arteriovenous oxygen difference were calculated from the partial pressure of oxygen (PO_2) oxygen saturation (O_2sat), and hemoglobin (hb) by the following formulas:

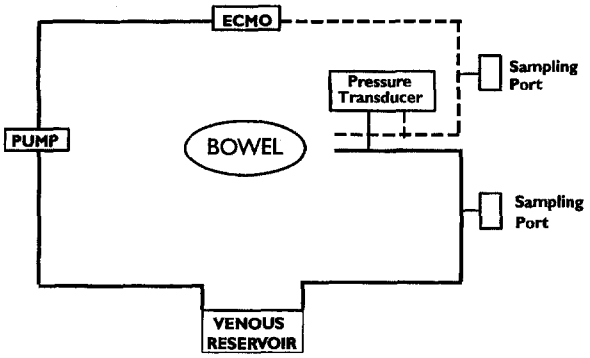

Fig. 1. Circuit for ex vivo perfusion of human small intestine includes extracorporeal membrane oxygenator, roller-type pump, venous reservoir, arteriovenous oxygen difference analyzer, pressure transducer, and arterial and venous sampling ports.

$$cO_2 = (pO_2 \times 0.003) + (hb \times O_2\ sat)$$

$$A\text{-}VO_2\ diff = c_aO_2 - c_vO_2$$

Oxygen consumption was calculated from the arteriovenous oxygen difference and the intestinal blood flow:

$$\dot{V}O_2 = Blood\ flow \times A\text{-}VO_2\ diff$$

Data were expressed as mean ± SEM. All curves were drawn by inspection.

RESULTS

Histologic sections of intestinal mucosa before and after perfusion revealed intact villous morphologic characteristics. All specimens demonstrated grade 1 or 2 histologic characteristics unassociated with necrosis of villi or crypts.[6] Intestinal blood flow in these ex vivo perfused mid-jejunal segments ranged from 70.6 to 11.4 ml/min/100 g. At blood flows >30 ml/min/100 g, arteriovenous oxygen difference varied inversely with blood flow (Fig. 2). Arteriovenous oxygen difference at blood flows <30 ml/min/100 g was maintained at 6.3 ± 0.3 vol%.

Oxygen consumption of the mid-jejunal segments was maintained at 1.6 ± 0.06 ml/min/100 g at all times when blood flow remained >30 ml/min/100 g. With increments in blood flow below this level, oxygen consumption varied proportionally (Fig. 3).

DISCUSSION

These experiments describe a unique intestinal perfusion preparation that allows the precise characterization of the regulation of oxygen consumption in the face of decreasing blood flows. Such studies of human intestine provide a basis for the ex vivo evaluation of treatments designed to lessen the considerable morbidity and mortality associated with intestinal ischemia.

The intrinsic regulation of intestinal blood flow may be attributed to metabolic or myogenic mechanisms.[7] The "metabolic theory" proposes a regulation of intestinal blood flow by metabolites resulting from an imbalance between tissue oxygen delivery and tissue oxygen demands. These metabolites modulate local vascular smooth muscle tone to maintain a balance between oxygen supply and demand. The precise metabolite that may be responsible for this control of intestinal blood flow has not been clearly identified; however, metabolites that have been shown to affect intestinal blood flow include potassium,[8-10] hydrogen ion,[11-14] adenosine,[15] and adenine nucleotides.[16-18] The release of one or more of these nucleotides during anaerobic metabolism is thought to cause local vasodilatation and subsequent increased intestinal blood flow and oxygen delivery. This release serves to locally redistribute blood flow and augment oxygen extraction.[19]

In a variation of the metabolic theory of intestinal blood flow control, Granger et al.[20] has proposed that changes in interstitial partial pressure of oxygen drive the local mechanism by which intestinal blood flow is controlled.[20] The high oxidative requirements of intestinal vascular smooth muscle are thought to render this tissue particularly susceptible and reactive to changes in local partial pressure of oxygen.

The "myogenic theory" of local control supposes a control of vascular resistance (by vascular smooth muscle) by arteriolar transmural pressure.[21] This mechanism of control is operative when arterioles maintain a constant wall tension in the face of changes in perfusion pressure (tension ≈ pressure × radius). Myogenic mechanisms can be demonstrated by altering venous pressure in isolated perfusion preparations and demonstrating a vasoconstrictive response at the arteriolar level.

The predominance of metabolic or myogenic control mechanisms may be affected by a variety of factors, including the level of blood flow at which these mechanisms are active, the metabolic state of intestinal tissue (ie, postprandial vs fasting),[22] and degree of arterial hypoxemia. The ability of our ex vivo perfused intestinal segments to maintain stable oxygen consumption at flows that are subnormal yet >30 ml/min/100 g is most consistent with a metabolic mechanism of control. This mechanism is supported by canine studies performed by Shepherd.[7] Assessment of the vasoconstrictive response to elevations in venous pressure revealed that at subnormal blood flows, venous pressure elevations resulted in

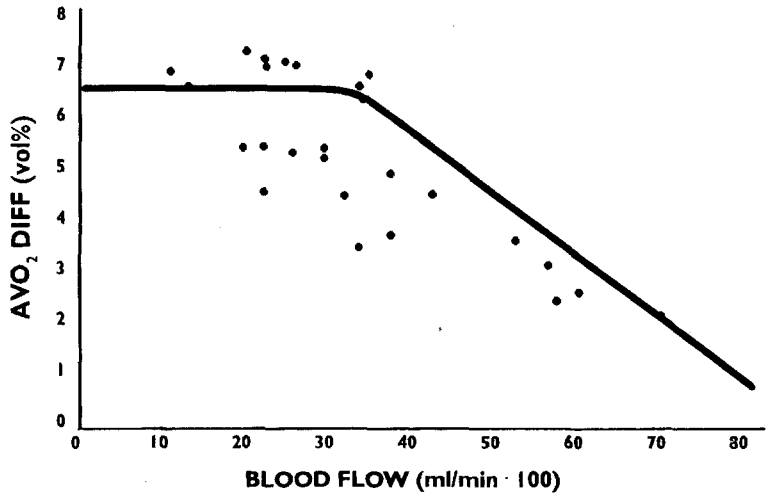

Fig. 2. Relation between intestinal blood flow and arteriovenous oxygen difference.

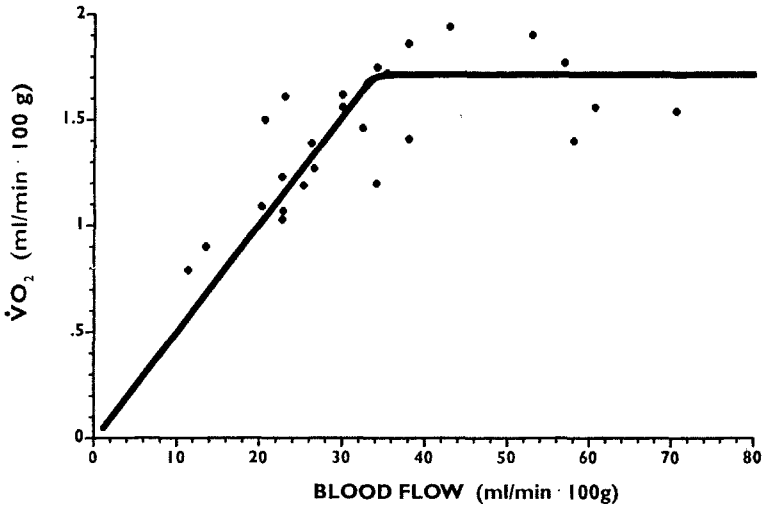

Fig. 3. Relation between intestinal blood flow and oxygen consumption.

vasodilatation rather than the vasoconstriction that would be predicted by a myogenic local regulatory mechanism.[23] Additional mechanisms that affect the regulation of intestinal blood flow include neural input as well as systemically released vasoactive substances (hormones, catecholamines, systemic metabolites). The effects of these factors in low-flow ischemia were not determinable in this denervated isolated perfusion preparation but have been described in a variety of animal models.[24]

As defined by Kvietys and Granger,[1] the "critical blood flow" is the level at which oxygen extraction by the tissue is maximized such that further decrements of blood flow result in corresponding decreases in oxygen extraction. This series of experiments estabishes this critical blood flow for the human small intestine at 30 ml/min/100 g. At this point, capillary density is maximized and local regulatory mechanisms cannot further increase oxygen extraction (Fig. 4). The concept of a "critical blood flow" above which oxygen consumption is relatively constant and below which oxygen consumption progressively decreases has been consistently noted across species.

The blood flow–oxygen consumption relation defined in these ex vivo perfused human intestinal segments is qualitatively similar to previous animal studies (Fig. 5). Canine studies performed by Kvietys and Granger[1] examining the relation of blood flow to oxygen consumption in isolated in vivo perfused intestinal segments indicated that oxygen consumption is maintained at 1.75 ml/min 100 g above blood flows of 30 ml/min/100 g by changes in oxygen

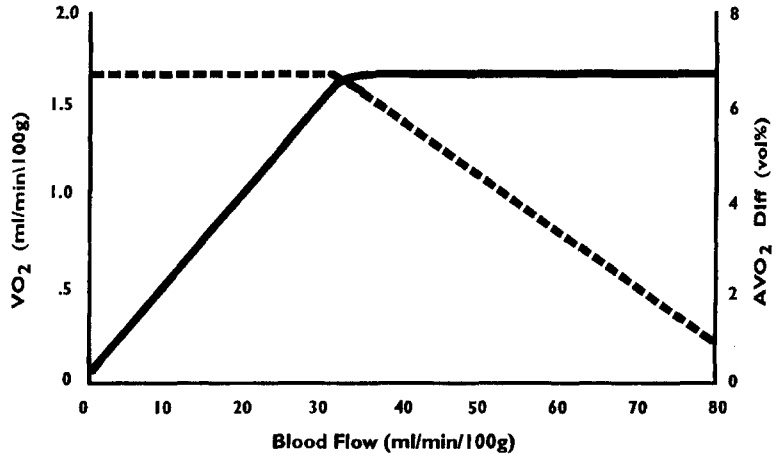

Fig. 4. Graph of intestinal blood flow, oxygen consumption (left axis, *solid line*), and arteriovenous oxygen difference (right axis, *dotted line*) demonstrates critical blood flow of 30 ml/min/100 g. Below this value arteriovenous oxygen difference is maximized and oxygen consumption decreases with blood flow.

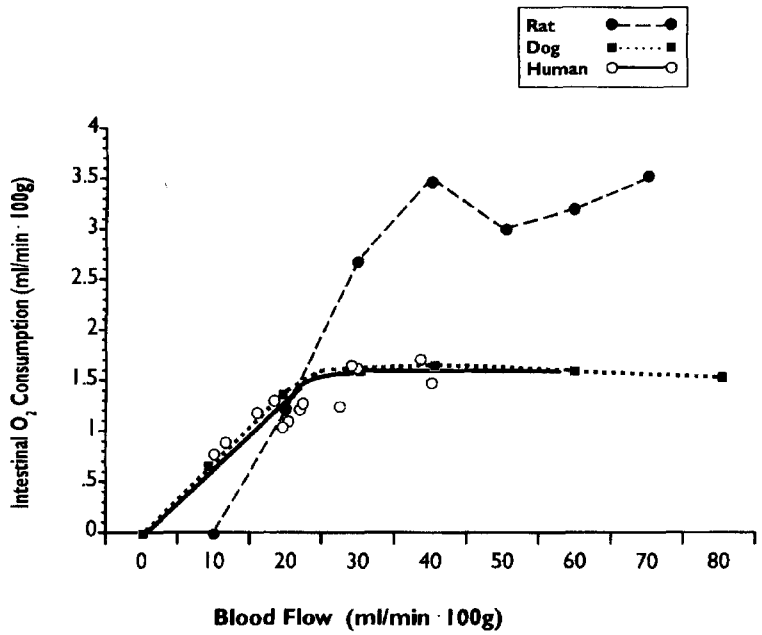

Fig. 5. Blood flow–oxygen consumption relation depicted for rat, dog, and human small intestine. Note similar qualitative relation but differing quantitative levels of blood flow and oxygen consumption among species.

extraction. Only in blood flows of <30 ml/min/100 g does oxygen consumption decrease with blood flow. Similar experiments performed in rats revealed decreases in oxygen consumption only when intestinal blood flow decreased <60 ml/min/100 g.[3]

The importance of these observations in the human small intestine, to a large extent, depends on earlier animal studies that have delineated a clear relation between intestinal oxygen consumption and tissue injury. Prolonged reductions in intestinal blood flow results in progressive histologic changes ranging from separation of the epithelial layer from the lamina propria to massive epithelial lifting and, finally, to complete denudation of the villus. While these changes are often associated with loss of mucosal barrier function,[6] the converse is not necessarily true.

That is, preservation of normal morphologic characteristics (as in our specimens) does not preclude disruption of mucosal function. In the rat, ischemic histologic changes have been seen when intestinal oxygen consumption decreases <2 ml/min/100 g.[25] Bulkley et al.[26] performed similar studies in canine intestine with mucosal albumin clearance as a functional indicator of the loss of mucosal barrier function. Significant increases in mucosal albumin clearance were seen when oxygen consumption fell <50% of control levels. Grum et al.[27] have shown decreases in canine mucosal pH when blood flow reductions are sufficient to decrease oxygen consumption <60% of control, presumably reflecting a conversion to anaerobic metabolism. Although we did not specifically measure mucosal function in our ex vivo preparation, our description of the blood flow–oxygen consumption relation in the human small intestine predicts such a disruption of normal barrier function at blood flows <25 ml/min/100 g.

In conclusion, this study performed in human small intestine provides a basis for the testing of treatment strategies in low-flow ischemia. For example, digitalis and various vasopressors have been implicated in the pathogenesis of nonocclusive mesenteric ischemia. These medications are often used in treating generalized "low-flow" shock states. Better understanding of the effects of these agents on intestinal oxygen consumption would provide an important means of evaluating their effectiveness in preserving tissue integrity. Although these effects have been characterized in animals,[25,28-31] ex vivo testing in human intestinal segments will have much greater clinical relevance.

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DISCUSSION

Dr. Peter Gloviczki (Rochester, Minn.). Acute mesenteric ischemia continues to be a problem that is difficult to predict, hard to diagnose, and challenging to treat.

There are several factors that determine intestinal oxygen consumption in addition to blood flow. These include luminal distention of the bowel, oxygen-carrying capacity and oxygen content of the blood, and the duration of low-flow state. Would changes in any of these factors influence the level of critical blood flow in patients? In this denervated isolated perfusion model, the authors could not study the effect of neural controls or that of the systematically released vasoactive substances. Would the lack of these control mechanisms have an effect on critical blood flow? Is there an ischemic penumbra in the mesenteric circulation? In other words, what is the critical flow rate that causes irreversible ischemic injury? How would these experiments ultimately help us determine which patients are at high risk to develop acute mesenteric ischemia? Finally, Kvietys and Granger from Alabama found that in dogs critical blood flow in a similar model was 30 ml/min/100 g, exactly what this study confirmed in human tissue. Is the dog then an ideal model to study mesenteric ischemia, or do you still have to stay up and do these experiments during the night after harvesting the tissue from transplant donors?

Dr. Tina Desai. There are numerous factors that can affect oxygen consumption. Luminal distention and oxygen-carrying capacity are a few of them. In this study we did not measure those particular factors, but my prediction would be to start with luminal distention. The effects of this would most likely be on venous pressure in the system; if we buy that the control of oxygen consumption in this model is from metabolites released from the intestine, then luminal distention would have a negative control in terms of oxygen consumption. Distention would probably cause a decrease in the level of oxygen consumption. The venous pressure would more likely affect the arterials and capillaries and cause an increase in pressure built back through the capillaries, which would affect the capillary density and might actually again reduce oxygen consumption by this mechanism. Oxygen-carrying capacity will obviously affect the oxygen consumption, and that was a variable that we actually tried to control so that it would not change in our particular model. There are known to be organ-specific differences as well as species differences in hemoglobin, the factor of hemoglobin and oxygen-carrying capacity and affecting oxygen consumption. Additionally, another factor we have to consider in a pump perfused preparation weighing an increase in viscosity with increasing hemoglobin and increasing oxygen-carrying capacity versus increasing the oxygen-carrying capacity per se. The effects of denervation and sympathetic and other vasoactive agents in the systemic circulation are obviously lacking in this model. It is very difficult to study the human small intestine in any way in which you can add these particular agents. One future study might, which we have been planning, be to add vasoactive agents into the perfusate and see what those effects are. I think the limitations of the neural input are going to be there in any ex vivo preparation. Finally, the concept of an ischemic threshold in the mesenteric circulation is sort of the $100,000 question. If we could identify what is reversible versus irreversible ischemia, it would be again a prize winner. One of the things that I think this model can help in identifying is figuring out the level of oxygen consumption decrease in which tissue injury starts beginning. We can do this with functional indicators and histologic indicators. Even more importantly, if we can find some sort of clinically relevant measure such as tonometry or mucosal pH that correlates with these decreases in oxygen consumption and with tissue injury, it might help us to at least determine where tissue injuries begin in whole humans. Finally, before accepting any animal model as a good model for humans on the basis of one simple similar factor, I think we really have to study all the factors that we have just talked about.

Dr. Lazar J. Greenfield (Ann Arbor, Mich.). I have some basic questions about the preparation in terms of reactive hyperemia because mucosa and muscle differ in that regard. You were looking at global oxygen uptake. Do you have any idea of what was actually happening in terms of perfusion of mucosa versus muscle?

Dr. Desai. I do not know exactly what the nature of the mucosal perfusion was versus the muscle. I do know that at the end of the procedure all the layers were at least histologically grossly intact.

Dr. Mark B. Adams (Milwaukee, Wis.). Were these pancreas donors, and if they were, are you sure that the mesentery was not ligated before the flush or at some point in the early stages of it? Did you get peristalsis in these segments once they were rewarmed?

Dr. Desai. I did not use any pancreas donors in these particular specimens, and we did get peristalsis.