

# Effects of Mesenteric Ischemia and Reperfusion on Small Bowel Electrical Activity

Sanjay S. Hegde, M. D.,\* Scott A. Seidel, M. D.,\* J. K. Ladipo, M. D.,\* L. Alan Bradshaw, Ph. D.,§  
Susan Halter, M. D.,‡ and William O. Richards, M. D.†,\*1,2

†Department of Surgery at the Veterans Administration Medical Center and \*Vanderbilt University School of Medicine; ‡Department of Pathology at Vanderbilt University School of Medicine; and §Living State Physics Group, Department of Physics and Astronomy, Vanderbilt University, Nashville, Tennessee 37232

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**Previous studies involving basic electrical rhythm (BER) have not been carried out far enough to fully characterize the relationship between mesenteric ischemia and BER. The phenomenon of reperfusion injury has also not been correlated with BER activity. The goal of this study was to characterize changes in BER during mesenteric ischemia and reperfusion and to correlate them with changes in pathology. Methods.** Serosal electrodes were used to record the electrical activity of rabbit jejunum ( $n = 20$ ) at baseline, during ischemia (90-210 min), and during reperfusion (120-240 min). BER frequency and amplitude were monitored, and biopsies were taken at the end of ischemia and reperfusion. A pathologist blinded to the specimen identity graded the histology on a scale of 0 (no changes) to 6 (transmural necrosis). Paired  $t$  test, the Kruskal-Wallis test of non-parametric ranks, and Fisher's  $r$  to  $z$  test were used for statistical significance where appropriate. **Results.** BER frequency and amplitude fell significantly after 15 min of ischemia and became undetectable by 90 min of ischemia in all animals. The likelihood that BER would return during reperfusion was highly correlated with length of ischemia ( $r = 0.99$ ). Longer periods of reperfusion were associated with increasing pathologic grade. **Conclusions.** BER frequency and amplitude are very sensitive to ischemia and their changes occur well before histopathologic changes. The variation in electrical activity of the small bowel during ischemia and reperfusion is a dynamic process that reflects the metabolic state of the smooth muscle. If electrical activity of the bowel is to be used for assessment of viability, continuous recordings more accurately reflect the metabolic state of the smooth muscle. © 1998 Academic Press

**Key Words:** mesentery; ischemia; electrophysiology, GI motility; electrodes; small intestine; reperfusion injury.

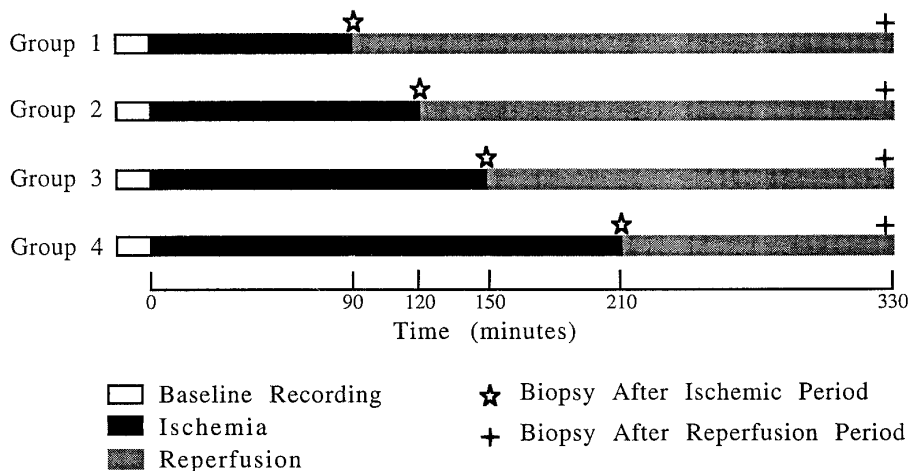
## INTRODUCTION

It is well known that the morbidity and mortality of mesenteric ischemia are high largely due to delayed diagnosis and the advanced stage of disease at laparotomy [1]. Once the diagnosis is made, additional hurdles remain, such as how to make the decision of which segments of intestine are viable or should be resected. The accuracy of various methods for assessing bowel viability at laparotomy available to the surgeon today, including fluorescein dye, doppler flow examination, and clinical judgment, lacks sensitivity and specificity [2, 3]. Second-look laparotomies have become routinely recommended in order to preserve as much bowel length as possible while not leaving behind nonviable bowel [2].

Studies in our lab have focused on the utilization of the basic electrical rhythm (BER) of small intestine as a possible means of not only detecting noninvasively mesenteric ischemia [4-6] but of assessing bowel viability as well. BER is the omnipresent slow wave of the gastrointestinal tract that characterizes the underlying electrical activity of the bowel. As such, it is present even in the absence of detectable mechanical activity. Unlike other methods of assessing bowel viability such as quantifying the migrating myoelectric complex or measuring transit times, measurements of BER are passive, do not require the presence of peristalsis, and can yield useful information during very short periods of analysis. In addition, there are only three known causes of decreased BER frequency: hypothermia, hypothyroidism, and hypoxia. The frequency and amplitude of small bowel BER have been previously shown to fall with arterial ischemia [7-10]. This decrease has also been shown to occur before the onset of pathologic changes [11]. However, studies have not been carried out far enough for the BER frequency to fall to undetectable levels in order to fully characterize the relationship between mesenteric ischemia and BER. Additionally, reperfusion has not been performed in these studies to assess whether BER frequency returns after it is completely lost. The purposes of this study were

<sup>1</sup> To whom reprint requests should be addressed at D5203 Medical Center North Vanderbilt University School of Medicine, Nashville, 37232. Fax: (615) 343-9485.

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**FIG. 1.** Study design. Baseline, ischemia, and reperfusion times are shown for all groups, as well as biopsy times.

to (1) correlate the effects of ischemia on small bowel basic electrical rhythm by inducing intestinal ischemia in an animal model for varying lengths of time; (2) allow ischemia to continue long enough for BER to become undetectable; (3) restore perfusion to the ischemic segment of bowel while measuring BER; (4) correlate changes in BER with histopathology, and (5) develop a more effective assessment of bowel viability.

## MATERIALS AND METHODS

The study was performed on 20 New Zealand rabbits weighing approximately 4 kg each, divided into four study groups (Groups 1, 2, 3, and 4 with five animals in each) depending on the length of intestinal ischemia and subsequent reperfusion. Induction of anesthesia was performed with acepromazine (0.5 mg/kg), xylazine (3 mg/kg), and ketamine (40 mg/kg) intramuscularly, after which an intravenous catheter was placed for fluids and subsequent doses of intravenous ketamine as needed to maintain anesthesia. Tempera-

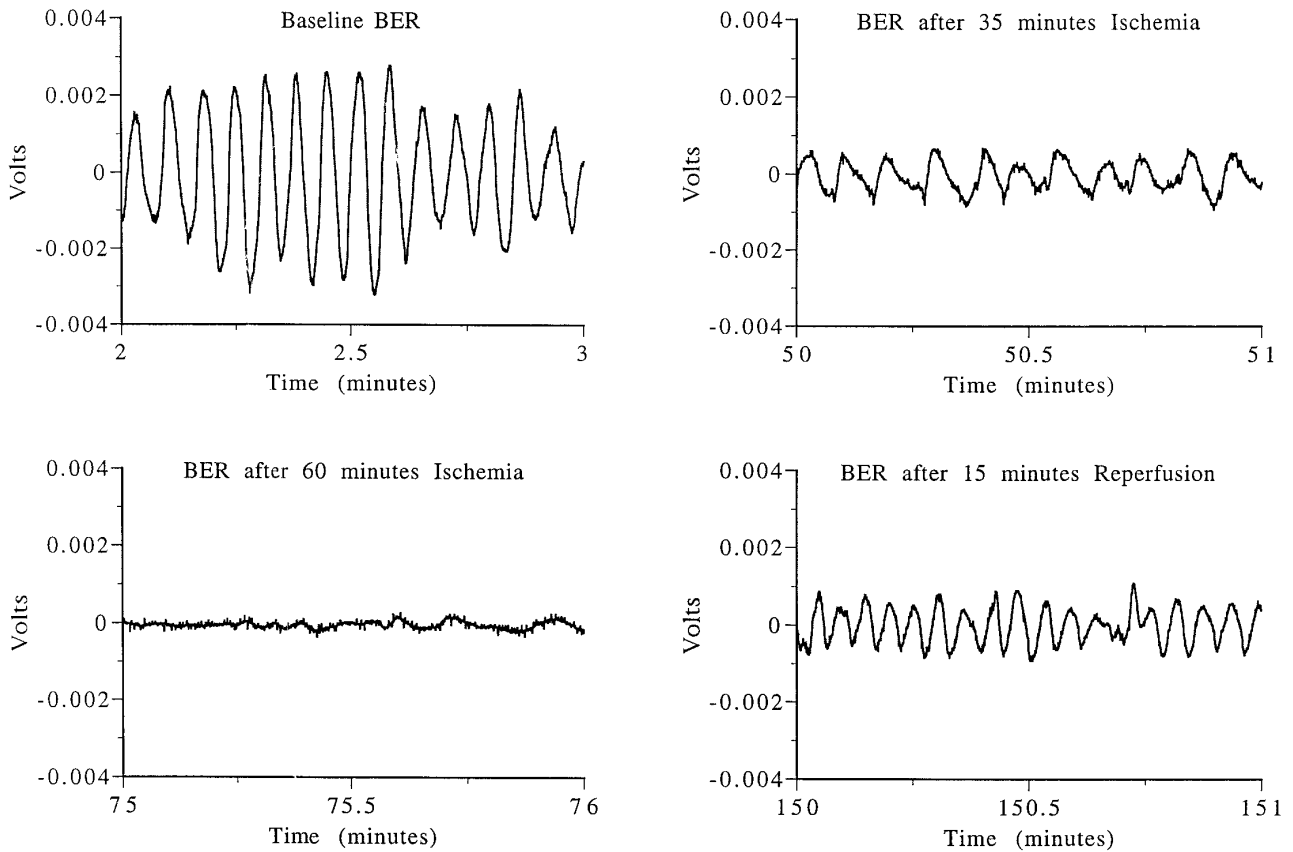
**TABLE 1**

### Grading Scale Based on the Swerdlow and Antonioli System [12] Used to Score Degree of Pathologic Injury

Grade	Histology
0	No pathologic change
1	Focal loss of surface epithelium
2	Mucosal infarction Extensive loss of surface epithelium Loss of variable amounts of lamina propria Sparing of basal glands Intact muscularis mucosae
3	Submucosal infarction Variable necrosis of submucosa Complete mucosal necrosis Intact muscularis mucosae
4	Mural infarction Loss of muscularis mucosae Complete necrosis of mucosa and submucosa
5	Mural infarction Involvement of inner layer of muscularis propria Complete necrosis of mucosa and submucosa
6	Transmural infarction Complete necrosis of entire bowel wall

ture was maintained using a heating blanket and periodically checked with a rectal thermometer. Intravenous heparin (125 u/kg q4h) was administered to prevent vascular thrombosis after occlusion and to ensure that reperfusion could be achieved. A midline laparotomy was performed and a segment of proximal jejunum was identified. A four-channel serosal electrode was sutured to the loop of jejunum and baseline BER recordings were performed for 15 min. BER signals were recorded using a Beckman amplifier (Beckman Instruments, Inc., Model R612) equipped with an analog to digital converter (Biopac Systems, Model MP100) and an Apple Powerbook 170 (Apple Computer, Inc.) utilizing Acqknowledge 3.1.2 software (Biopac Systems). All experiments were performed in an electrically shielded room to prevent any background electrical interference. Arterial blood flow to the loop in question was assessed using a Doppler flow probe (Koven Technology, Inc., Model ES-1000SPM). Next, the segment of bowel was transected proximally and distally to prevent intramural blood flow and the segmental artery and vein were isolated and occluded using a vascular occluder. Complete occlusion was confirmed with the Doppler flow probe. The onset of ischemia was noted, after which the bowel was placed back into the abdomen in a nonconducting, latex glove (in order to prevent the recording of BER from adjacent loops of bowel). Varying lengths of complete ischemia were maintained (90, 120, 150, and 210 min for Groups 1, 2, 3, and 4, respectively) while continuous recordings of BER were made. At the end of ischemia, intestinal biopsies were taken and the vascular occluder was removed in order to restore blood flow. Reperfusion was documented with the Doppler flow probe. The bowel was then placed back into the abdomen (still in the nonconducting glove) and reperfusion was maintained (240, 210, 180, and 120 min, respectively) while BER recordings continued. The total study time for each group was therefore kept constant (Fig. 1). One animal in Group 4 did not undergo reperfusion due to anesthetic death. At the end of reperfusion biopsies were again taken and the animal was then euthanized.

BER frequency was determined using auto-regressive (AR) spectral analysis performed on a Power Macintosh 7200/75 (Apple Computer, Inc.) running MATLAB software (The Mathworks, Inc.) and is expressed as mean cycles per minute (cpm)  $\pm$  SEM. AR spectral analysis was used to determine BER frequency for several reasons. First, it provides an objective assessment of frequency, rather than simply counting peaks over a known time period. More importantly, AR analysis provides the ability to examine short segments of recordings, rather than the longer (5 min) segments required by other analysis methods (such as Fast Fourier Transformation). This is essential since BER frequency was constantly changing throughout the study and these changes would not be detected if longer segments of recordings were analyzed. One-minute segments were analyzed at 15-min intervals. Since looking at BER frequency alone may not be fully informative, (i.e., the BER frequency 30 min after initiation



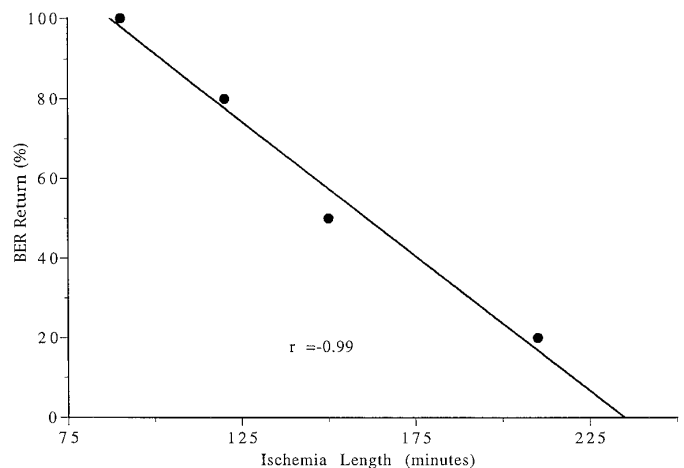
**FIG. 2.** Samples of BER recordings from the same animal at different time points during the study. After 35 min of ischemia, the frequency and amplitude of the BER signal are decreased. After 60 min of ischemia, the BER signal is no longer detectable. Fifteen minutes into reperfusion, the BER has returned to baseline frequency, but the amplitude of the signal is still diminished. Times represent continuous time during the study.

of ischemia can be the same as the frequency immediately after reperfusion) BER amplitude ( $A$ ) was also measured and is expressed as mean volts  $\pm$  SEM. Statistical analysis was performed using the paired two-way  $t$  test with significance defined as  $P < 0.05$ , and Fisher's  $r$  to  $z$  for correlation analysis with significance defined as  $P < 0.05$ . Histologic assessment of biopsies was performed by a pathologist blinded to the identity of the specimen utilizing a modification of the classification system devised by Antonioli and Swerdlow [12] as shown in Table 1. Analysis of pathologic grades was performed using a nonparametric one-way analysis of variance, the Kruskal-Wallis test, to determine if groups were significantly different from each other.

## RESULTS

BER recordings at baseline and at different time-points during a representative study are shown in Fig. 2. BER frequency fell from  $17.8 \pm 0.4$  cpm at baseline to  $14.0 \pm 0.5$  ( $P < 0.05$ , paired  $t$  test) after 15 min of ischemia. There was a complete loss of BER by 90 min of ischemia in all animals. The BER remained undetectable in all animals until reperfusion was initiated at which time it returned in at least one animal in each group. There was a high degree of correlation ( $r = 0.99$ ,  $P < 0.05$ , Fisher's) between the likelihood of BER returning during reperfusion and the length of ischemia, as shown in Fig. 3. The BER was noted to return with reperfusion and then become undetectable later in re-

perfusion and remain so for the rest of the study in one animal from both Group 1 and Group 2. Figures 4–9 display the data for the BER frequency and amplitude in graph form superimposed on one another. BER fre-



**FIG. 3.** Percentage of animals with a return of BER plotted against length of ischemia. The likelihood that BER frequency will return to baseline after an ischemic episode is highly correlated with the length of the ischemic episode with  $P < 0.05$  using Fisher's  $r$  to  $z$  test.

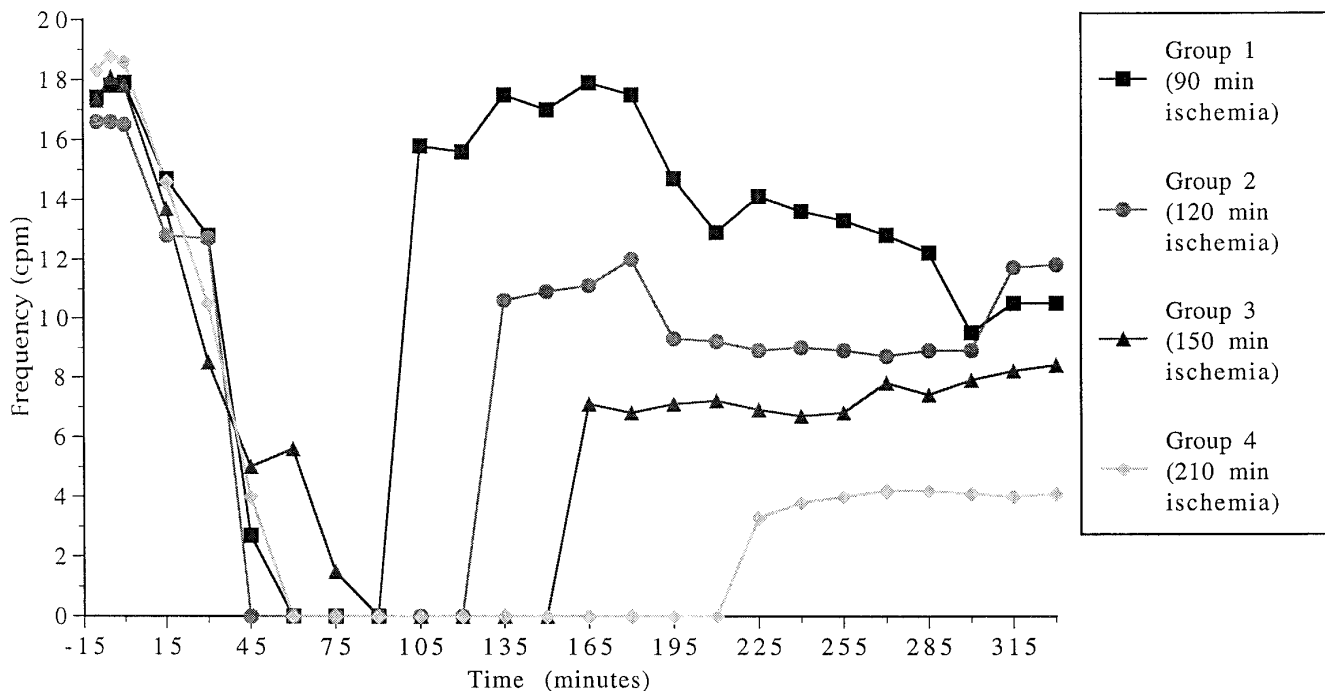


FIG. 4. Mean BER frequency plotted for all groups.

quency in Groups 1 and 2 returned to preischemic levels (no statistical difference from baseline) during reperfusion. The BER frequency in Group 3 fluctuated between levels significantly lower than baseline and levels comparable with baseline, whereas BER frequency in Group 4 did not return to baseline levels ( $P$

$< 0.05$ , paired  $t$  test). The data are shown in Table 2a. There was a high correlation ( $r = -0.95$ ,  $P < 0.05$ , Fisher's) between the frequency with which BER returned and the length of the ischemic insult: the longer the ischemic insult, the lower the average BER frequency immediately after reperfusion, as shown in Fig.

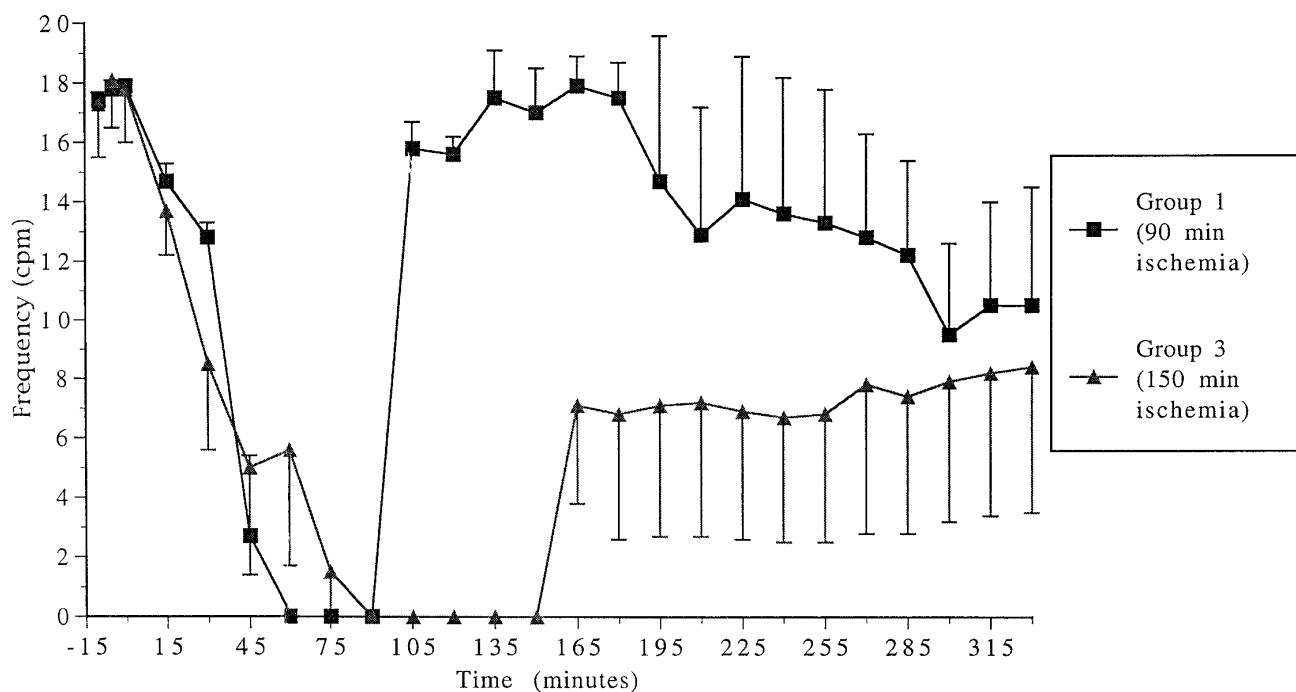
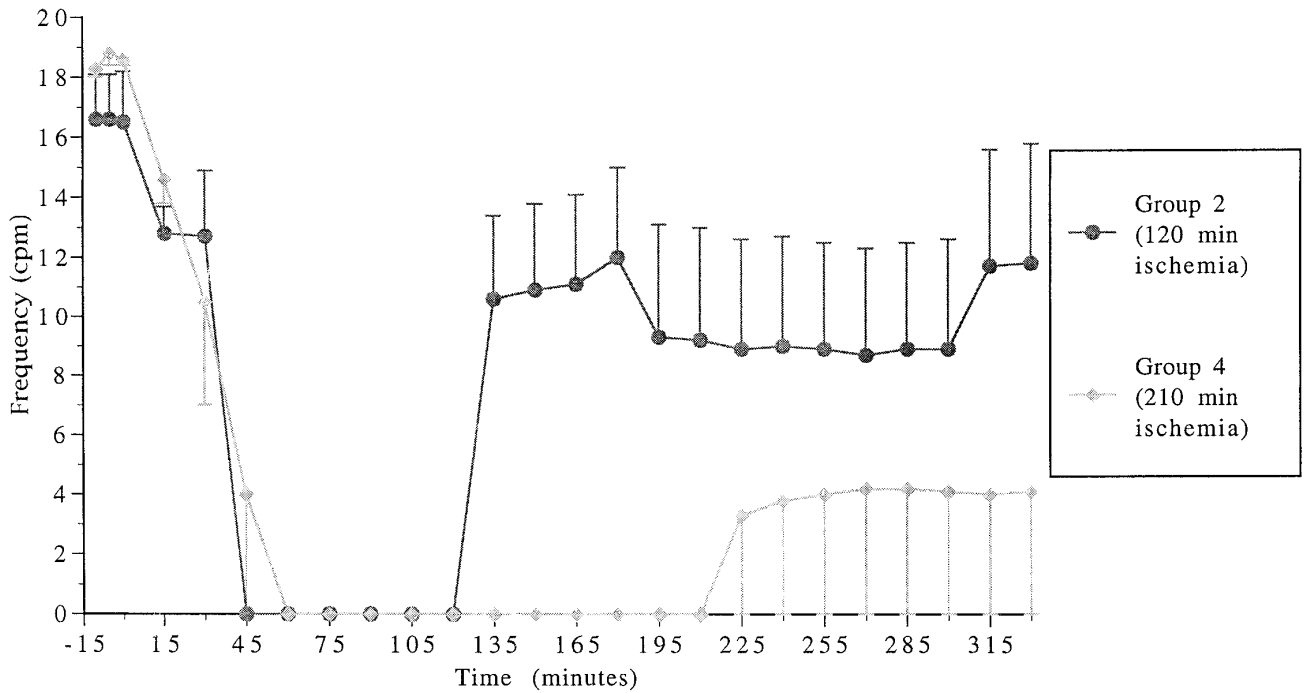


FIG. 5. Mean BER frequency plotted against time for Groups 1 and 3. BER falls to undetectable levels by 90 min of ischemia and remains so until reperfusion is initiated. Group 1 BER frequency returns to baseline levels, while Group 3 BER frequency varies in significance when compared to baseline. The error bars represent SEM. The data are separated into two figures to clarify presentation of the results.

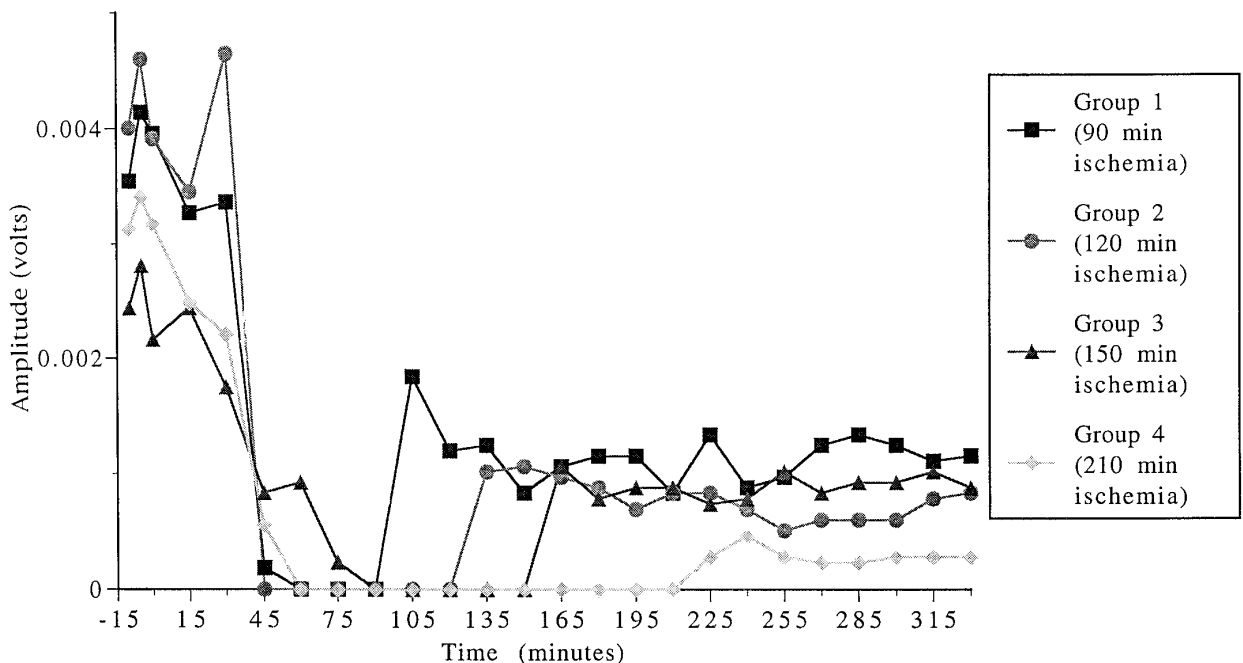


**FIG. 6.** Mean BER frequency plotted against time for Groups 2 and 4. BER falls to undetectable levels by 60 min of ischemia and remains so until reperfusion is initiated. Group 2 BER frequency returns to baseline levels, but Group 4 returns to a level lower than baseline ( $P < 0.05$  by paired  $t$  test).

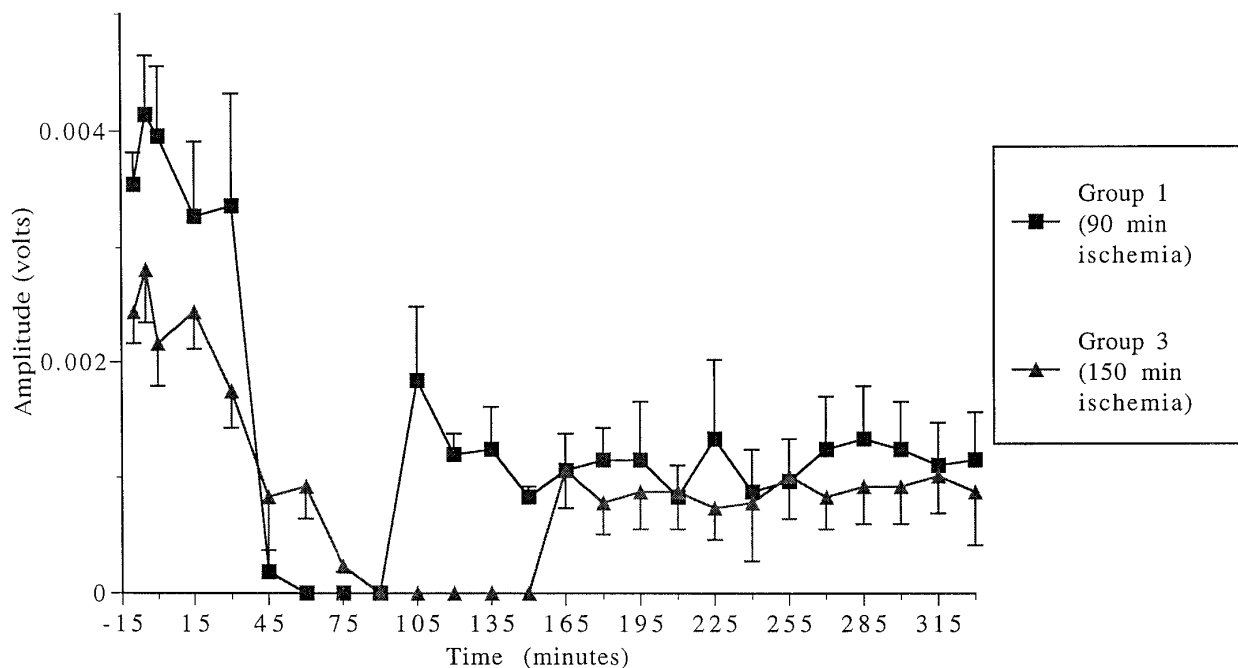
10. Examination of Figs. 7–9 reveals that once the BER signal returns during reperfusion, the average amplitude is significantly decreased ( $P < 0.05$ ,  $t$  test) from baseline (Table 2b). There is also a high correlation ( $r = -0.97$ ,  $P < 0.05$ , Fisher's) between the amplitude with which the BER returns and the length of ischemia: the longer the ischemic episode, the lower the average

amplitude of the signal immediately after reperfusion (Fig. 11).

The mean pathologic scores after ischemia and reperfusion for each group can be seen in Table 3. There was no statistical difference in the average pathologic grade immediately after ischemia among any groups despite the longer lengths of ischemia. The pathologic grade



**FIG. 7.** Mean BER amplitude plotted for all groups.

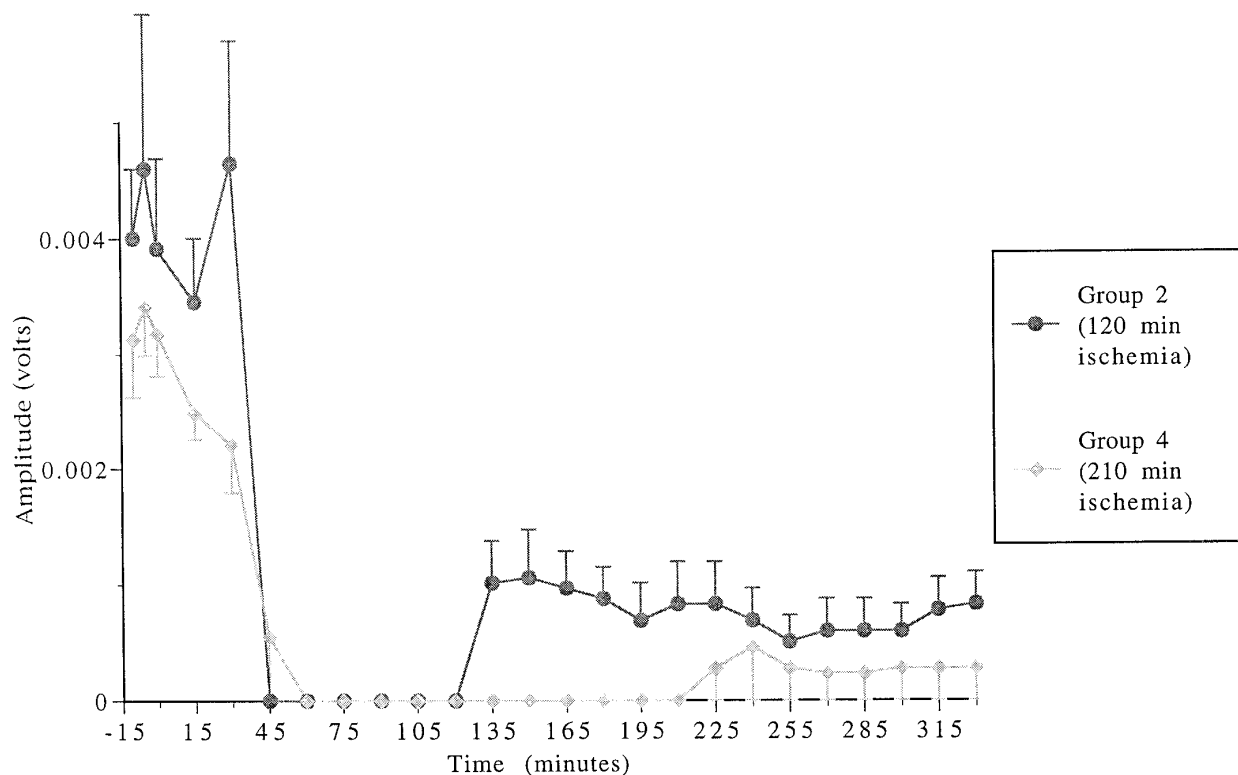


**FIG. 8.** Mean BER Amplitude plotted against time for Groups 1 and 3. Once the BER signal returns during reperfusion, the amplitude is significantly decreased from baseline (see Table 2b). Error bars indicate SEM.

worsened, however, after reperfusion for each group. The mean pathologic grade change from the end of ischemia to the end of reperfusion assumed a logarithmic relationship, increasing as reperfusion time increased with  $R^2 = 0.99$  (Fig. 12).

## DISCUSSION

This study reveals that frequency and amplitude of the small bowel BER signal are both significantly reduced with acute ischemia. BER amplitude and fre-



**FIG. 9.** Mean BER Amplitude plotted against time for Groups 2 and 4. Again, once BER signal returns during reperfusion, the amplitude is significantly diminished from baseline (see Table 2b). Error bars indicate SEM.

**TABLE 2**  
**Changes in BER Frequency and Amplitude Compared with Baseline Recordings**  
**for All Groups and for All Animals**

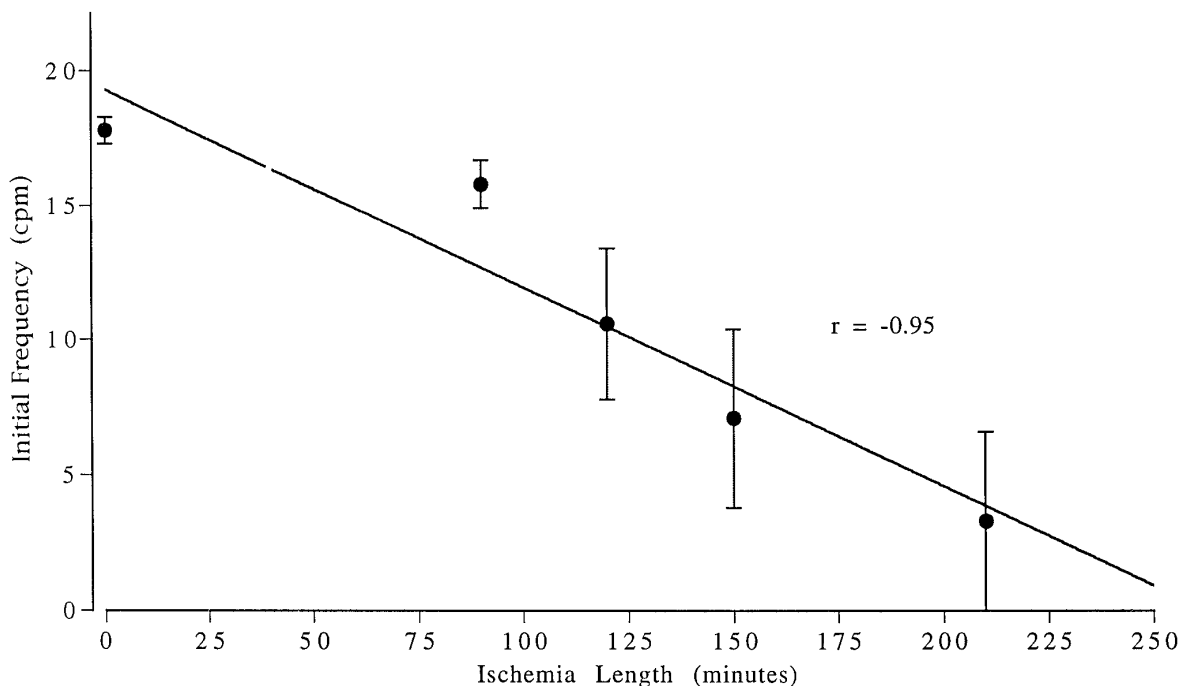
a. Reperfusion BER Frequency Change from Baseline by Group					
	Group 1	Group 2	Group 3	Group 4	All animals
Ischemia length	90 min	120 min	150 min	210 min	
Mean baseline BER frequency (cpm)	17.7 ± 0.4	16.6 ± 1.6	18.4 ± 0.6	18.6 ± 0.3	17.8 ± 0.4
Reperfusion length	240 min	210 min	180 min	120 min	
Mean BER frequency during first 120 min reperfusion	16.0 ± 1.5	10.0 ± 3.1	12.5 ± 3.5	4.0 ± 4.0*	11.0 ± 1.7*
b. Reperfusion BER Amplitude Change from Baseline by Group					
	Group 1	Group 2	Group 3	Group 4	All animals
Ischemia length	90 min	120 min	150 min	210 min	
Mean baseline BER amplitude (V)	0.0039 ± 0.0003	0.0042 ± 0.0009	0.0025 ± 0.0003	0.0033 ± 0.0004	0.0035 ± 0.0003
Reperfusion length	240 min	210 min	180 min	120 min	
Mean BER amplitude during first 120 min reperfusion	0.0013 ± 0.0003*	0.0008 ± 0.0003*	0.0010 ± 0.0003*	0.0003 ± 0.0003*	0.0009 ± 0.0002*

*Note.* For statistical comparison, reperfusion time means were calculated using only the first 120 min of reperfusion, as this was the time common to all groups. Although the reperfusion frequency values in each group are all lower than baseline, statistical significance is reached only in Group 4 and when all animals are compared as a larger group. Amplitude decreases were statistically significant in all groups.

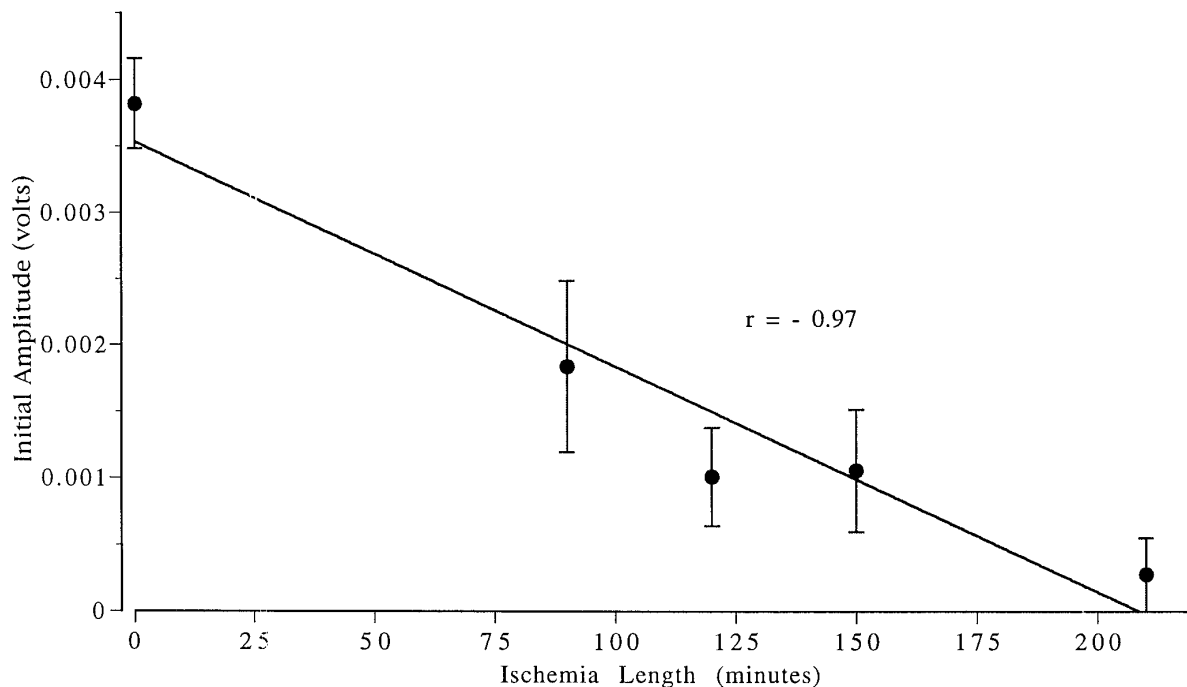
\*  $P < 0.05$  by paired  $t$  test.

quency are so sensitive to the effects of ischemia that they decline significantly within 15 min of the onset of ischemia. Our lab has previously shown that superconducting quantum interference device (SQUID) magnetometers may be used not only to noninvasively detect small bowel BER but to detect the changes in BER

frequency due to ischemia [4, 5, 13]. Thus, this noninvasive technology holds promise for the use of BER as a means of detecting mesenteric ischemia. The data presented also bolster our belief that intra- and postoperative recording of BER using surgically implanted electrodes may be able to quantitate bowel viability



**FIG. 10.** Initial BER frequency following reestablishment of blood flow (i.e., following onset of reperfusion) plotted against ischemia length. There is a high correlation between how low the BER frequency will be immediately after reperfusion and the length of the ischemic insult ( $P < 0.05$  by Fisher's  $r$  to  $z$ ).



**FIG. 11.** Initial BER amplitude following onset of reperfusion plotted against ischemia length. There is also a high correlation between how low the BER amplitude will be immediately after reperfusion and the length of ischemia ( $P < 0.05$  by Fisher's  $r$  to  $z$ ).

and thus assist the surgeon in the timing of second look operations.

Prolongation of an ischemic insult eventually causes the BER to fall to undetectable levels. BER amplitude and frequency correlate well to the length of ischemia and reperfusion and appear to accurately reflect the viability of the bowel. Complete loss of BER during episodes of ischemia does not preclude a subsequent recovery of BER during restoration of blood flow, in some cases to baseline levels. The likelihood that BER will return is highly correlated with the length of ischemia. Conversely, the recovery of BER during reperfusion does not necessarily preclude a subsequent loss of BER despite ongoing blood flow. In addition, the longer the ischemic episode, the lower the average BER frequency will be when it returns during reperfusion (Fig. 10).

The amplitude of the BER signal during reperfusion is significantly reduced from baseline. BER amplitude

is a more sensitive indicator of muscle ischemia than BER frequency, and the amplitude correlates well with the length of ischemia (Fig. 11).

Several observations can be made from the results of the histologic analysis. First, not only was there no significant increase (or worsening) in pathologic grade after ischemia among the four groups, but the worst mean grade was only  $2.3 \pm 0.3$ . Thus, the pathologic changes which occur in small bowel due to ischemia must take place over longer periods of time than those seen in this study. Additionally, there was no evidence of transmural infarction in any of these biopsies, indicating that irreversible changes can only be detected histologically after longer lengths of time as well. Our findings are supported by several other authors, including Amano *et al.* [14], who demonstrated that transmural infarction and bowel wall disintegration were seen histologically only after 8 to 12 h of ischemia. Mitsudo

**TABLE 3**

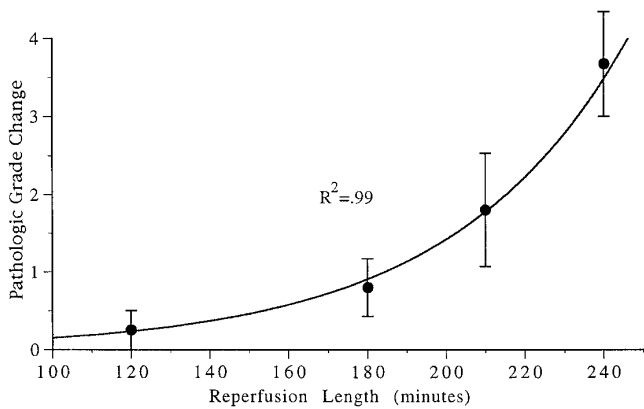
**Average Pathologic Grades for Each Group at the End of Ischemia and Reperfusion<sup>a</sup>**

	Group 1	Group 2	Group 3	Group 4
Ischemia length	90 min	120 min	150 min	210 min
Mean grade after ischemia	$1.2 \pm .3^\dagger$	$1.6 \pm .6^\dagger$	$2.3 \pm .3^\dagger$	$1.8 \pm .3^\dagger$
Reperfusion length	240 min	210 min	180 min	120 min
Mean grade after reperfusion	$5 \pm .6^*$	$3.4 \pm .6^*$	$3.5 \pm .5^*$	$2 \pm 0^*$

<sup>a</sup> The pathologic grade after reperfusion worsens with longer reperfusion time.

<sup>†</sup> No statistical difference among groups by the Kruskal-Wallis test (nonparametric one-way analysis of variance by rank; grouping variable = Group),  $P = 0.10$

\* Groups are statistically different by Kruskal-Wallis test,  $P < 0.05$ , except as noted. (Groups 2 and 3 are not statistically different from each other.)



**FIG. 12.** The increase in pathologic grade from the end of ischemia to the end of reperfusion as a function of reperfusion length. This is a logarithmic relationship with a high degree of correlation.

and colleagues [15] state that total ischemia of 6 to 8 h duration "can produce reversible intestinal damage." If one takes into account that the BER frequency and amplitude dropped significantly by 15 min of ischemia and became absent in all animals by 90 minutes of ischemia (and remained so until the end of ischemia when the biopsies were taken), we can also conclude that not only does the BER frequency and amplitude decrease significantly before pathologic changes can be seen, but they will in fact fall to undetectable levels far before even a significant worsening in the histopathology can be detected.

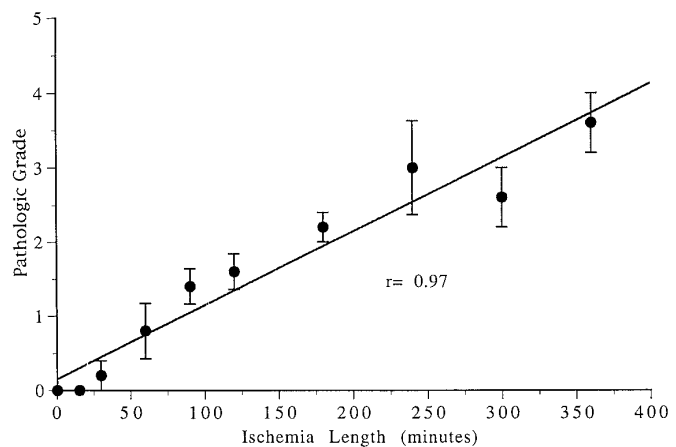
The average histopathologic grades after reperfusion were worse for each group compared to the grades after ischemia. The fact that each group underwent differing lengths of ischemia makes it somewhat difficult to compare pathologic grades after reperfusion among groups, but as can be seen in Fig. 12, there was a high degree of correlation between increasing pathologic grade and reperfusion time. In fact, the only statistically significant worsening in pathology from ischemia to reperfusion occurred in the group which underwent the longest period of reperfusion and the shortest period of ischemia. This worsening in histopathologic grade during reperfusion is compatible with reperfusion injury.

Another explanation for the worsening in pathology after reperfusion relates to the time course over which pathologic changes occur. If transmural necrosis is only seen 8–12 h after the ischemic insult, and the histopathologic changes in ischemic bowel lag changes in BER [11], it is thus possible that the worsening pathologic grades after longer periods of reperfusion simply reflect the increased time for the pathologic changes to occur. To investigate this further, we performed serial 1-h jejunal biopsies on five animals during mesenteric ischemia to a loop of jejunum for 6 consecutive hours without reperfusion. In all other respects, this group underwent the same procedures as the other four groups as outlined under Materials and Methods. The data are presented in Fig. 13. During continuous ischemia without reperfusion, the increase in pathologic grade was linear ( $r = 0.97$ ,  $P < 0.0001$ , Fisher's). Since

the increase in pathologic grade during reperfusion for Groups 1 through 4 was logarithmic, this suggests that the larger increase in pathologic grade during longer periods of reperfusion is indeed due to reperfusion injury and not solely the progression of previous ischemic pathology.

There was only one episode of transmural infarction with a concomitant absence of BER at the time the biopsy was taken. Thus, we can also conclude that the absence of small bowel BER does not correlate with transmural necrosis. However, since irreversible pathologic changes are only apparent long after changes in BER occur, it remains to be seen whether the bowel in question will actually remain viable with reperfusion if ischemia has lasted long enough for BER to be undetectable. This study suggests that reperfusion injury plays a great role. It is possible that if ischemia has lasted long enough for BER to become absent, then the bowel will eventually become nonviable even if it appears viable at the time of laparotomy because of significant reperfusion injury. If it is left in the abdomen, irreversible pathologic changes may not become apparent over the intervening time until a second look laparotomy is performed.

This study raises many questions, but the potential clinical usefulness of utilizing small bowel BER as a means of assessing bowel viability is apparent. Since the other two causes of decreased BER frequency (hypothermia and hypothyroidism) can easily be ruled out, we conclude that decreases in BER frequency and amplitude must be due to mesenteric ischemia. This study demonstrates that BER activity fluctuates in response to ischemia and reperfusion. This is a dynamic process and more than one data point is needed to evaluate the viability of the bowel. Other methods of assessing bowel viability (fluorescein dye, doppler examination, and clinical judgment) are unreliable because they are sin-



**FIG. 13.** Data for the continuous ischemia group. Following the study protocol, five animals underwent laparotomy and occlusion of a segmental artery to the jejunum. While ischemia was maintained, biopsies were taken at 1-h intervals for 6 h. The mean pathologic grade increased in a linear fashion for this group while the reperfusion pathology grade for the other groups increased logarithmically with time (despite reestablishment of blood flow). This is evidence of reperfusion injury.

gle timepoint measurements, whereas the process of ischemia, reperfusion, and recovery is a dynamic one. Further ischemic insults and reperfusion injury may occur and dramatically affect bowel viability hours after the assessment. Continuous recordings of intestinal BER in humans with mesenteric ischemia would enable the physician to monitor response to treatment and degree of reperfusion injury, and thus to intervene appropriately. Further investigations must be performed to assess the time course over which pathologic changes occur during ischemia as well as to evaluate the viability of ischemic bowel after longer lengths of reperfusion in order to determine whether a correlation exists between absence of BER after ischemia and irreversible pathologic changes apparent after longer lengths of reperfusion (i.e., either because more reperfusion injury has occurred or because enough time has passed for these changes to become detectable). Such a correlation not only will prove invaluable to the surgeon who must decide whether to resect or not but also may obviate the need for second-look laparotomies and no doubt lead to better treatment algorithms and thus a better prognosis in patients with mesenteric ischemia.

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