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(Accepted for publication December 27, 1991.)

Anesthesiology
76:661, 1992

Use of a Capnometer to Detect Leak of Carbon Dioxide during Laparoscopic Surgery

To the Editor:—In laparoscopic surgery, carbon dioxide is insufflated into the abdominal cavity to make space for the procedure. Sometimes a leak in one of the connectors or orifices of the instruments allows the distending gas to escape and results in poor visibility. Localization of the leak is not always straightforward, and operating time is lost. Capnography can be used to solve this problem by having the surgeon examine the suspected sites with a sterile end of a gas sampling tube. The capnometer will sense the leaking carbon dioxide, and the indicator will rise to the top of its scale.

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(Accepted for publication January 2, 1992.)

Anesthesiology
76:661-662, 1992

Afterload Dependence of Postischemic Myocardium

To the Editor:—The study by Buffington and Coyle¹ on the load dependence of postischemic myocardium provides valuable data on the residual functional capacity after perfusion is reestablished. As pointed out by the authors, the marked decrement in the contractile response of the postischemic myocardium to increases in preload, compared with its control response, is of great interest and importance. The authors also contend that the response of the postischemic myocardium to changes in afterload is not different from the control response and suggest that afterload reduction therapy would not be expected to have the same particular benefit as when this type of therapy is used in cardiac failure.

In fact, the data support the opposite conclusion. The authors base their conclusion on the fact that the *slope* of the relationship between systolic wall thickening and mean arterial pressure (MAP) was the same for control and postischemic myocardium (fig. 2 of their paper). How-

ever, although the slopes of these relationships are parallel, the absolute values of the data are different; at all combinations of left atrial pressure and MAP, the systolic thickening of the postischemic myocardium is less. As a result, the *percent* improvement in systolic wall function as MAP is reduced from 110 to 70 mmHg is much greater in the postischemic myocardium than in the control state. This is borne out by the data of tables 1 and 2 from the paper, which present systolic wall thickening as a percent of end-diastolic thickness or in absolute units (millimeters), respectively.

Using the mean values presented in these tables (either as percents or absolute units), we have calculated the percent *increases* in thickening as MAP is decreased from 110 to 70 mmHg at different left atrial pressures; the data are for the test zone before (control) and postischemia. Percent increases (table 1) were calculated as follows:

$$\frac{\text{systolic thickening at 70 mmHg MAP} - \text{systolic thickening at 110 mmHg MAP}}{\text{systolic thickening at 110 mmHg MAP}} \times 100$$

It can be seen that the postischemic myocardium demonstrates a much greater relative improvement in systolic wall thickening with decreases in MAP from 110 to 70 mmHg than does the control state. Thus, the trends in the data suggest that the increased sensitivity of the failing heart to afterload and the value of afterload reduction therapy are concepts that also apply to the postischemic myocardium. Statistical tests of these trends would require analysis of the paired control and postischemic data for individual animals.

TABLE 1. Recalculated Data from Tables 1 and 2¹

	Preload (cmH ₂ O)	Control (% increase)	Postischemic (% increase)
Table 1 data	3	7.1	17.6
	6	26.7	38.9
	9	18.9	57.9
Table 2 data	3	11.1	25.0
	6	33.3	53.3
	9	21.9	56.2