4. Otsu K, Khanna VK, Archibald AL, MacLennan DH: Co-segregation of porcine malignant hyperthermia and a probable causal mutation in the skeletal muscle ryanodine receptor gene in backcross families. Genomics 11:744–750, 1991
5. Gillard EF, Otsu K, Fujii J, Khanna VK, De Leon S, Derdemezi J, Britt BA, Duff CL, Worton RG, MacLennan DH: A substitution of Cysteine for Arginine 614 in the ryanodine receptor is potentially causative of human malignant hyperthermia. Genomics 11:751–755, 1991
6. Brownell AKW: Malignant hyperthermia: relationship to other disease. Br J Anaesth 660:303–308, 1988
7. MacKenzie AE, Korneluk RG, Zorzato F, Fujii J, Phillips M, Iles D, Wieringa B, Le Blond S, Bailly J, Willard HF, Duff C, Worton RC, MacLennan DH: The human ryanodine receptor gene, its mapping to 19q13.1, placement in a chromosome 19 linkage group and exclusion as the gene causing myotonic dystrophy. Am J Hum Genet 46:1082–1089, 1990
8. Levitt R, Nouri N, Jedlicka AE, McKusick VA, Marks AR, Shutack JG, Fletcher JE, Rosenberg H, Meyers DA: Evidence for genetic heterogeneity in malignant hyperthermia susceptibility. Genomics 11:543–547, 1991

(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 orfices 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.

PERTTI PESONEN, M.D.
Department of Anesthesiology
PEKKA LUUKONEN, M.D.
Department of Surgery
Mehilainen Hospital
P Hesperiankaty 17
00260 Helsinki, Finland

(Accepted for publication January 2, 1992.)

Anesthesiology
76:661–662, 1992

Afterload Dependence of Postischemic Myocardium

To the Editor:—The study by Buffington and Coyle[1] 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). However, 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.

TABLE 1. Recalculated Data from Tables 1 and 2[1]

	Preload (cmH_2O)	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

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.

MARIO A. INCHIOSA, JR., PH.D.
Professor of Pharmacology
Research Professor of Anesthesiology
CHARLES HER, M.D.
Associate Professor of Anesthesiology
MICHAEL V. HERMAN, M.D.
Professor of Medicine
Chief of Cardiology

New York Medical College
Valhalla, New York 10595

REFERENCE

1. Buffington CW, Coyle RJ: Altered load dependence of postischemic myocardium. ANESTHESIOLOGY 75:464–474, 1991

(Accepted for publication January 2, 1992.)

Anesthesiology
76:662, 1992

In Reply:—Inchiosa and colleagues suggest that the percentage change (in contrast to the absolute change) in regional myocardial contraction during a decrease in afterload is greater in postischemic regions than in control regions. We have performed the paired statistical comparison requested, and it yielded no firm support for this conclusion. The calculation of percentage change in thickneing as mean arterial pressure decreases from 110 to 70 mmHg involves small and, in some cases, negative numbers in the denominator. The resulting ratios are not normally distributed and thus violate the assumptions of a paired *t* statistic. The nonparametric analog, the sign test, was nonsignificant.

Although the statistics are inconclusive, the concept proposed by Inchiosa and colleagues seems valid. A change in a small number seems larger than the same absolute change in a large number. At issue, however, is interpretation rather than validity of the data. Patients with diseased hearts may, in fact, like to hear that their exercise tolerance has *doubled* from three steps to six with afterload reduction therapy. We prefer to think in absolute terms.

CHARLES W. BUFFINGTON, M.D.
Professor of Anesthesiology

RICHARD J. COYLE, M.D.
Assistant Professor

Department of Anesthesiology and Critical Care Medicine
University of Pittsburgh School of Medicine
Suite 910
3471 Fifth Avenue
Pittsburgh, Pennsylvania 15213

(Accepted for publication January 2, 1992.)

Anesthesiology
76:662–663, 1992

Prevention of Venous Air Embolism Related to Veno-venous Bypass during Orthotopic Liver Transplantation

To the Editor:—Although veno-venous bypass has been reported to decrease the morbidity and mortality associated with orthotopic liver transplantation (OLT),[1] venous air embolism has been described during veno-venous bypass using opaque, heparin-bonded tubing.[2] For this reason, clear polyvinyl tubing has been recommended,[3] allowing visual assessment of air in the system as well as an automated air detector. We report a case in which venous air embolism related to veno-venous bypass during OLT was prevented by the use of clear polyvinyl tubing.

A 9-yr-old, 17-kg boy with biliary atresia was scheduled for reduced-size OLT from a living related donor. Anesthesia was induced with isoflurane and was maintained with isoflurane and fentanyl. Muscle relaxation was achieved with of vecuronium, and the lungs were mechanically ventilated. Systemic arterial pressure (SAP), pulmonary arterial pressure (PAP), central venous pressure (CVP), ECG, peripheral hemoglobin oxygen saturation, and inspired and expired carbon dioxide tension were monitored.

Despite the loss of 4,000 ml blood loss during the 6 h of dissection, the patient was hemodynamically stable prior to venous bypass (SAP 120/70 mmHg, PAP 25/10 mmHg, and CVP 10 mmHg). The bypass circuit consisted of a Biomedicus model 520 console and a model 50 Bio Pump (48 ml volume) and clear polyvinyl tubing of 9.5 mm internal diameter. The bypass circuit was primed with lactated Ringer's solution without heparin. Cannulas, connected to pump tubing, were inserted into bilateral femoral veins, the portal vein, and the left axillary vein. Immediately after the initiation of veno-venous bypass, air bubbles were noted in the inlet cannula from the portal vein. The bypass pump was stopped, and a large amount of air was noted to be present in the pump head and inlet tubing, but not in the outlet tubing. SAP, PAP, CVP, and end-tidal carbon dioxide tension were unchanged.

The ligature around the portal vein cannula was noted to be loose, resulting in air entrainment. After reestablishment of the connection of the portal vein and disconnection of the outlet tubing from the axillary cannula, the entrained air was evacuated extracorporeally from the pump head with drained blood using the pump force. The 1,000-ml blood loss sustained during this procedure resulted in a decrease in SAP to 70/40 mmHg, but infusion of stored blood promptly restored normotension. After evacuation of air from the pump system, veno-venous bypass was instituted without further incident. The patient was discharged from the hospital 8 weeks later without neurologic, cardiac, or pulmonary sequelae.

We believe this case demonstrated that the use of clear polyvinyl tubing adds significantly to patient safety during veno-venous bypass. Surgeons must take special care to ensure that the cannulae connections are air-tight, and anesthesia personnel must continuously monitor tubing for entrained air.

MASAHIRO MURAKAWA, M.D.
Assistant of Anesthesia