CORRESPONDENCE

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Primary Closure of Omphalocele/Gastroschisis in Newborns

To the Editor:—We read with interest and concern the article "Hemodynamic effect of primary closure of omphalocele/gastroschisis in human newborns."[1] The study hypothesized that development of increased intragastric pressure (IGP) during primary surgical repair would be associated with postoperative organ system failure in infants with ventral wall defects, and sought to determine other physiological parameters that might provide objective, predictive criteria for safe primary closure. Yaster's finding of a critical IGP of 20 cm water confirms the earlier report of Wesley *et al.* that IGP should not exceed 20 cm of water with or without a silastic silo in repair of omphalocele/gastroschisis.[2] Wesley also established experimentally in a series of five puppies that cardiac output and mean blood pressure were compromised most notably between IGP of 22 and 28 cm of water. He found IGP to be an objective and reliable parameter, as opposed to the previous practice of observation of color, respiratory rate, and lower-extremity skin turgor.

The current report does not differentiate between omphalocele and gastroschisis. Recent literature, especially as regards *in utero* assessment of ventral wall defects, indicates that while the bowel in omphalocele is normal, that in gastroschisis is often edematous and inflamed.[3] If the bowel in gastroschisis is not forced into the abdomen, nor into a tight silastic chimney under pressure, the edema subsides in 24 to 48 h, allowing replacement into the abdominal cavity with ease and safety.[4,5] Of the other physiologic parameters which Yaster *et al.* measured, only CVP and cardiac index (CI) also predicted which infants could be safely closed primarily. Both CI and CVP are more difficult to measure, require invasive technology, and proved to be no more accurate than simple measurement of IGP in predicting safety of closure.

In this series, 50% of infants in whom the abdomen was closed primarily required re-exploration for oliguria, anuria, and/or compromised cardiac output postoperatively. In light of the established safety of silastic pouch technique and previous reported series, this morbidity should be considered excessive.[2,4,5] Since this was neither a blinded nor random study, we question the wisdom of continuing the experimental protocol after the first unsuccessful forced primary closure, and certainly after the second.

In the interests of expanding knowledge for the eventual betterment of medical practice and patient care, research on human subjects must continue, but we must remain constantly vigilant that individual patient welfare not be jeopardized.

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In Reply:—The treatment of choice for abdominal wall defects (either omphalocele or gastroschisis) in newborn infants is primary repair whenever possible.[1,2] In our institution, the alternative therapy, a staged repair utilizing a silastic pouch, is used only when, in the surgeon's clinical judgement, the herniated viscera can not be reduced because of the size of the defect or if reduction compromises respiratory and cardiovascular function. Unfortunately, clinical observation of the infant's respiratory rate, blood pressure, skin color, and tissue turgor are not reliable in (fentanyl) anesthetized, paralyzed patients.[3] Because our anesthetic practice changed from spontaneous ventilation of anesthetic vapors to balanced anesthesia with controlled ventilation, we sought to determine if specific, physiologic, intraoperative measures could provide our surgeons with objective, predictive criteria for safe primary closure of either omphalocele or gastroschisis.

In our study, published in ANESTHESIOLOGY,[4] we found that intraoperative changes in intragastric pressure, central venous pressure, and cardiac index could successfully distinguish infants who successfully or unsuccessfully underwent primary surgical closure. These were retrospectively determined data. Based on our results, we set up a treatment algorithm to determine prospectively if measurement of either

intragastric and/or central venous pressure could successfully predict safe primary closure. We have recently completed this study[5] which showed (in 11 patients) that it could.

Although we suspected from the study of Wesley *et al.*[6] that intragastric pressure measurement could serve as a guide in determining whether to close an infant's defect primarily, there were also data to suggest that this might not work. Several laboratory studies[7,8] have shown that increasing intra-abdominal pressure above 20 mmHg may seriously compromise cardiac output and organ blood flow. In these same studies, however, there was no correlation between intra-abdominal and intragastric pressure. Furthermore, Wesley made his measurements through a gastrostomy tube connected to a water manometer.[6] He used 20 cm of water as his therapeutic cutoff, which is less than our finding of 20 mmHg measured through a fluid-filled nasogastric tube.

We agree with Drs. Sacks and Gongaware that human research demands that an individual patient's welfare not be jeopardized for "study purposes." In our study, the decision to close an infant's abdominal wall defect was a clinical decision made by our surgeons. Once it became clear from our data that intraoperative measurements could help separate those children who successfully underwent primary closure from those who failed, we immediately stopped our study and set up a treatment algorithm to test our hypothesis.

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Standards for Oxygen Analyzers

To the Editor:—At a meeting of ASTM Committee F-29.03.08 held March 22, 1989, on the harmonization of the international standard on oxygen analyzers for breathing mixtures[1] and the relevant United States standard,[2] it was unanimously decided to recommend withdrawal of the United States standard forthwith. This United States standard was written by F-29's predecessor committee, American National Standards Institute (ANSI) Z79.[3]

The international standard should be followed until a new United States standard that is in concordance with the new edition of IEC 601–1[4] and ISO 7767[1] is published by ASTM, probably in 1990. Any queries about this supercedence should be addressed to Ms. Beth K. Moran, Staff Manager, ASTM Committee F-29 on Anesthetic and Respiratory Equipment, 1916 Race Street, Philadelphia, PA 19103–1187.

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