bypass reduces oxidative myocardial damage and nitric oxide during cardiac operations in the adults. J Thorac Cardiovasc Surg 1998;116:327-34.
2. Wachtfogel YT, Kucich U, Greenplate J, et al. Human neutrophil degranulation during extracorporeal circulation. Blood 1987;69: 324-30.
3. Butler J, Pillai R, Rocker GM, Westaby S, Parker D, Shale DJ. Effect of cardiopulmonary bypass on systemic release of neutrophil elastase and tumor necrosis factor. J Thorac Cardiovasc Surg 1993;105:25-30.
4. Gu YJ, van Oeveren W, Akkerman C, Huyzen RJ, Boonstra PW, Wildevuur CRH. Heparin-coated circuits reduce the inflammatory response to cardiopulmonary bypass. Ann Thorac Surg 1993; 55:917-22.
5. Korn RL, Fisher CA, Livingston ER, et al. The effect of Carmeda Bioactive Surface on human blood components during simulated extracorporeal circulation. J Thorac Cardiovasc Surg 1996;111: 1073-84.

12/8/96853

Reply to the Editor

We thank Dr Gu and his colleagues for their interest in our recent article.[1] They describe a prospective study in 60 patients in whom cardiopulmonary bypass (CPB) was used, but did not mention where these results have been published. They did not find a correlation between Po_2 on CPB and neutrophil elastase release. However, there are several methodologic differences between our two studies. First, they wrote, "a large variation in Po_2 appeared during perfusion," and their results showed that Po_2 varied between 107 and 440 mm Hg at the start of cooling, between 80 and 308 mm Hg during hypothermia, and between 128 and 317 mm Hg on average during CPB. It is not clear whether these disparities in Po_2 occurred randomly and within the same patient. Further, it is not evident whether normoxia was maintained at all times, as it was done in our study. This seems very important, because we also did not find a positive correlation between Po_2 and elastase at the beginning of CPB with low elastase levels, but when Po_2 was controlled to normoxic levels during the whole period of extracorporeal circulation, elastase release was reduced significantly compared with high levels after persistent hyperoxia. Second, they measured Po_2 levels only 5 times during CPB: Po_2 between these measurements is unknown and might have reached undesired levels. In our study, on-line Po_2 measurements were applied and the Po_2 during CPB was controlled at all times. Third, different types of oxygenators were used during their study. These differences might have influenced results, as our data (unpublished) indicate, namely, that elastase release varies between oxygenators. Fourth, blood samples in their study were obtained from the radial artery, whereas our blood samples were taken from coronary sinus blood and from the venous side of the CPB circuit. The significance of this detail, and whether this might have influenced results, however, remains unknown.

The authors mention that "elastase release during CPB has been known to be largely attributed to blood interaction with the artificial surface of the extracorporeal circuit."[2,3] These results confirm (as mentioned above) our own findings, in which membrane oxygenators cause less oxidative damage than bubble oxygenators. In their explanation, the authors reason: "It could well be that normoxic CPB had reduced the cardiac source of elastase but that the effect had been systematically counteracted by other factors, such as blood-material interaction." This statement is in contrast to our findings, in which values from coronary sinus blood and systemic circulation were not different and improved after normoxia. Independently from reduced leukocyte elastase levels, there are additional benefits from normoxic CPB.[4,5]

In summary, there are considerable methodologic differences between their study and ours. Our protocol of controlled normoxic CPB for the first time establishes a correlation between Po_2 and leukocyte elastase, whereas their investigation of "a large variation in Po_2" could not confirm this finding. I do agree with their conclusion that further studies are necessary to address this issue. Furthermore, the clinical significance of reduced leukocyte elastase after normoxic CPB has to be established.

Kai Ihnken, MD
Department of Surgery
Stanford University Hospital
Room H3680
300 Pasteur Dr
Stanford, CA 94305

REFERENCES

1. Ihnken K, Winkler A, Schlensak C, et al. Normoxic cardiopulmonary bypass reduces oxidative myocardial damage and nitric oxide during cardiac operations in the adult. J Thorac Cardiovasc Surg 1998;116:327-34.
2. Wachtfogel YT, Kucich U, Greenplate J, et al. Human neutrophil degranulation during extracorporeal circulation. Blood 1987;69: 324-30.
3. Butler J, Pillai R, Rocker GM, Westaby S, Parker D, Shale DJ. Effect of cardiopulmonary bypass on systemic release of neutrophil elastase and tumor necrosis factor. J Thorac Cardiovasc Surg 1993;105:25-30.
4. Ihnken K, Morita K, Buckberg GD, Sherman MP, Ignarro LJ, Young HH. Studies of hypoxemic oxygenation injury: with aortic clamping. XIII. Interaction between oxygen tension and cardioplegic composition in limiting nitric oxide production and oxidant damage. J Thorac Cardiovasc Surg 1995;110:1274-86.
5. Morita K, Ihnken K, Buckberg GD, Sherman MP, Young HH. Studies of hypoxemic oxygenation injury: Without aortic clamping. IX. Importance of avoiding perioperative hyperoxemia in the setting of previous cyanosis. J Thorac Cardiovasc Surg 1995;110: 1235-44.

12/8/96852

The extracardiac Fontan procedure using a pedicled pericardial roll without cardiopulmonary bypass

To the Editor:

We congratulate Okabe and his colleagues for their successful establishment of total cavopulmonary connection using a pedicled autologous pericardial roll without the aid of cardiopulmonary bypass.[1] We[2] used similar surgical maneu-

vers in our recent series of patients when we wanted to achieve a Fontan circulation.

We initially introduced total cavopulmonary connection without the use of cardiopulmonary bypass in April 1996. Overall, 47 patients have undergone the Fontan procedure at our institution since then, with no operative deaths. In 30 of these patients (61%), the Fontan circulation was established without the use of cardiopulmonary bypass. A pedicled autologous pericardial roll was used as an extracardiac conduit for draining the inferior caval vein in 9 of the 30 patients (30%), including 2 with isomeric right appendages. Age at operation ranged from 13 months to 4 years with a mean of 22 ± 12 months. Seven patients were younger than 2 years old. Postoperative angiography demonstrated smooth and unobstructed channels, as shown in our previous report.[2] No obstruction or dilatation was noted through the constructed extracardiac channel by consecutive postoperative echocardiograms, the longest follow-up being 30 months.

On the basis of our experience, the surgeon should pay particular attention to several points for successful achievement of this particular procedure. The temporary bypass placed between the caval veins and the atrial chamber should be appropriately designed so as to minimize resistance and turbulence through the tube. With an effective temporary bypass, caval venous pressures can be acceptable during crossclamping of the caval veins.[2]

The second technical point to be noted is the level of transection of the inferior cavoatrial junction. As described in the report of Okabe and colleagues,[1] it is crucial to leave a sleeve of the atrial musculature around the orifice of the inferior caval vein. If the inferior caval vein is divided exactly at the venoatrial junction, anastomosis of the extracardiac conduit to the orifice of the inferior caval vein is extremely difficult because of the very short distance between the site of cannulation and transection. In addition, our preference is an oblique division of the venoatrial junction,[2] differing from the square one shown in their schema.[1] The oblique incision can provide a sufficiently large anastomosis.

This surgical procedure can also be used in patients with visceral heterotaxy and abnormal connections of the hepatic and inferior caval veins.[2] In our 2 patients with isomeric right appendages undergoing the nonpump Fontan procedure using a pedicled roll, an independent hepatic vein was connected directly to the atrium. We placed dual temporary bypasses, one for the hepatic vein and the other for the inferior caval vein. In the setting of visceral heterotaxy, since pulmonary venous connection is frequently abnormal, construction of an extracardiac channel, particularly made of a flexible autologous tissue, can be advantageous to avoid obstruction of the pulmonary venous drainage.[3]

The presence of adhesions within the pericardial cavity may militate against use of a pedicled pericardial roll. In 2 of our 9 patients undergoing this surgical procedure, the pericardial cavity had been previously opened for banding of the pulmonary trunk. Although pericardial adhesions were moderate in these patients, a pedicled pericardial roll could be provided without major problems.

Obviously, potential for growth of the constructed channel remains controversial, and long-term results must be investigated to determine efficacy of this surgical procedure. As far as intermediate results are concerned, the nonpump extracardiac Fontan procedure using a pedicled autologous pericardial roll is an attractive option for establishing the Fontan circulation. In this respect, we agree with Okabe and colleagues.

Hideki Uemura, MD
Toshikatsu Yagihara, MD
Youichi Kawahira, MD
Department of Cardiovascular Surgery
National Cardiovascular Center
5-7-1 Fujishirodai, Suita
Osaka 565-8565, Japan

REFERENCES

1. Okabe H, Nagata N, Kaneko Y, Kobayashi J, Kanemoto S, Takaoka T. Extracardiac cavopulmonary connection of Fontan procedure with autologous pedicled pericardium without cardiopulmonary bypass. J Thorac Cardiovasc Surg 1998;116:1073-5.
2. Uemura H, Yagihara T, Yamashita K, Ishizaka T, Yoshizumi K, Kawahira Y. Establishment of total cavopulmonary connection without use of cardiopulmonary bypass. Eur J Cardiothorac Surg 1998;13:504-8.
3. Uemura H. The Fontan type procedure in patients with visceral heterotaxy. Cardiol Young 1998;8:419-22.

12/8/97379