OPTIMAL PRESSURES AND FLOWS DURING CARDIOPULMONARY BYPASS

Pro: A Low-Flow, Low-Pressure Technique Is Acceptable

J. Kent Garman, MD

IN 1976, A SURVEY of 26 cardiovascular centers was compiled for the Association of Cardiac Anesthesiologists (Garman JK: Unpublished data, 1976). That survey showed that there were wide differences in opinion concerning the flow rates and blood pressures used during cardiopulmonary bypass (CPB). Acceptable blood pressures varied from 30 to 120 mm Hg. Acceptable bypass flows varied from 1.2 to 3.5 L/min/m^2 (or 30 to 90 mL/kg/min). Obviously, when opinions vary as widely as this, the data on which decisions are made must not be definitive. Unfortunately, this is a very difficult subject to study because many variables confound the results.

Why is this controversy important? Many cardiovascular surgical and anesthesia teams prefer to use a low-flow, low-pressure bypass technique. There are very good reasons for choosing this technique. It is very important to understand these reasons in order to rationalize the choice of a technique as controversial as this one. The reasons are: (1) less trauma to blood elements; (2) less stress on pump tubing and connections; (3) slower rewarming of the heart due to less bronchial flow entering the heart; (4) a clearer operative field for the same reason; (5) less trauma to the aorta at the site of the cross-clamp; and (6) the ability to use smaller venous and arterial cannulae.

Given these advantages, many cardiovascular teams have decided that the risk-benefit ratio of using a low-flow, low-pressure technique is acceptable. Surgeons used to the advantages of this technique find it difficult to tolerate the bloodier operative field, the necessity for more vigorous cooling measures, and the resultant longer bypass times found with higher flows and pressures.

Is this a safe technique? Many studies have demonstrated no increased incidence of neurological dysfunction in groups of patients having cardiac surgery with low-pressure, low-flow bypass techniques. Kolkka and Hilberman[1] studied 204 patients and showed that low pressures during bypass did not correlate with cerebral injury. The average mean arterial pressure (MAP) in this group was 49 mm Hg with an average CPB flow of 42 mL/kg/min. Ellis et al[2] used preoperative and postoperative standard psychometric testing on 30 patients undergoing cardiac surgery with a low-flow, low-pressure bypass technique and showed a zero incidence of irreversible cerebral dysfunction at 6 months postoperation. The average MAP during CPB in these patients was 60 mm Hg. The average CPB flow was 39 mL/kg/min. Both of these studies can easily be criticized for experimental design flaws. However, they do establish that there is little difference in outcomes in these patient groups when compared with the rate of complications in high-flow, high-pressure bypass patient groups.

Even more interesting are some studies using prostacyclin (a prostaglandin used to prevent platelet aggregation during bypass). The major side effect of this agent is profound vasodilatation. When used during bypass, perfusion pressures consistently run below 30 mm Hg unless supported with vasopressors. In two studies, Aren et al[3,4] concluded that although CPB with hypothermia prolonged central brain conduction time, the hypotension produced by prostacyclin did not further impair conduction when compared with a nonhypotensive group. They also concluded that the hypotensive group did not have an increased risk of postoperative cerebral damage. Fish et al[5] studied 100 patients with detailed neuropsychiatric examinations and CT scans before and after CPB. Patients were randomized into groups receiving prostacyclin or saline. Perfusion pressures in the prostacyclin group were lower than the control group. There were no differences in the groups regarding perioperative cognitive changes.

If it is decided to use this technique, how can its safety be maximized? It is important to understand the effect of some easily manipulated variables on cerebral autoregulation. Several studies have examined the effect of using different methods of blood gas management during CPB. Murkin et al[6] randomized 38 patients undergoing CPB into two groups: one group managed with the pH-stat method that requires CO_2 to be added to maintain normal blood gases as measured in a temperature-corrected analyzer, and the second group managed with the α-stat method with no CO_2 added and blood gases maintained normal without temper-

From the Department of Anesthesia, Sequoia Hospital, Redwood City, CA, and Stanford University, Stanford, CA.

Address reprint requests to J. Kent Garman, MD, 27742 Stirrup Way, Los Altos Hills, CA 94022.

1053-0770/91/0504-0018$03.00/0

ature correction in the analyzer. The pH-stat method results in 50% higher $PaCO_2$ levels and the resultant effects on cerebral blood flow (CBF) and autoregulation. Murkin et al felt that autoregulation (flow/metabolism coupling) was not maintained with the pH-stat method, with CBF correlating with perfusion pressure over a range of 15 to 95 mm Hg. (In other words, CBF fell as pressure fell.) On the other hand, the α-stat method preserved autoregulation of CBF, resulting in correlation of CBF with $CMRO_2$ over a blood pressure range of 20 to 100 mm Hg. It would seem that if lower pressures during bypass are to be allowed, it would be advantageous to use the α-stat method of blood gas management because CBF would be better maintained at low bypass flows.

However, Bashein et al,[7] in a later article, examined neurobehavioral and cardiac outcome after CPB in two groups of patients, again randomized into pH-stat and α-stat management groups. They were unable to support the hypothesis that CO_2 management or hypotension had any measurable effect on outcome. However, they did not use a low-pressure CPB method, with perfusion pressures being maintained around 70 mm Hg by vasoactive agents. It still seems logical that if it is planned to allow low blood pressures during CPB, the α-stat method of CO_2 management offers some advantage because CBF should be better maintained at low pressures due to maintenance of autoregulation. Prough et al,[8] in an accompanying editorial, point out that the definitive study correlating neuropsychologic endpoints with measured cerebral circulatory and metabolic changes during CPB has not yet been completed. To further complicate the issue, Croughwell[9] recently reported that insulin-dependent diabetics had impaired cerebral pressure-flow autoregulation during CPB, thus requiring higher perfusion pressures to protect them from hypoxic insults.

The fact remains that low-flow, low-pressure CPB is widely practiced, with acceptable results. Why does it work? It is believed that several things allow these physiological transgressions.

1. The metabolic rate of the brain is significantly reduced by anesthesia and hypothermia. Hypothermia alone at levels used during CPB (28°C to 30°C) reduces metabolic needs by 50%. It has become more obvious that hypothermia is a powerful protective mechanism for hypoxic brain survival as evidenced by numerous cases of survival after prolonged cold water immersion.
2. Hemodilution changes flow characteristics and may allow areas of the brain beyond stenoses to continue to receive oxygenated diluted blood during low pressures.
3. A larger proportion of blood flow is redistributed to the brain during hypothermic bypass, again perhaps due to some of the same mechanisms operative during prolonged cold water immersion.
4. The brain is able to extract more oxygen from blood during low-flow states than during normal-flow states.
5. Govier et al[10] have also shown that cerebral autoregulation is preserved during hypothermic CPB down to pressures of 30 mm Hg. However, if volatile anesthetic agents are used, cerebral autoregulation is probably lost, a possible confounding factor in studies of cerebral autoregulation.

Recent advances in continuously monitoring venous and arterial blood oxygen saturations allow a more quantified approach to bypass flow management. The author now routinely sets CPB flow rates to maintain venous saturation (measured at the venous cannula) above 60%. In adults, this usually results in CPB flow rates of 2.5 to 3.5 L/min, and perfusion pressures stabilize between 35 and 60 mm Hg. This seems to be a better method of choosing a flow rate than just relying on a weight or body surface area multiplier.

However, the final word is not in on this subject. The problem with any study of cerebral dysfunction is the degree of validity of the measurement. The author knows several patients who have undergone CPB and who relate minor long-term complaints of slowed intellectual function and subtle memory loss. It is doubtful that these complaints would show up on most measurements of neuropsychiatric dysfunction. Yet, to these patients, the subtle changes are bothersome. It is still a concern when taking care of an older patient with known or suspected cerebrovascular disease. Our team tends to modify tolerance to prolonged hypotension during bypass in these patients. When an hour of a perfusion pressure of 40 mm Hg might be satisfactory in "normal" patients, the pressure will be increased to 55 to 65 mm Hg in this group of patients through increased flow or with low doses of vasopressors. This approach is obviously inconsistent, because it is not possible to identify every patient who is at risk from small vessel disease in the brain. However, the author has become much more comfortable with low-pressure, low-flow perfusion over the past 10 years with the accumulation of data supporting its safety. It is becoming more obvious that the benefits of low-flow, low-pressure CPB outweigh the risks.

REFERENCES

1. Kolkka R, Hilberman M: Neurologic dysfunction following cardiac operation with low-flow, low-pressure cardiopulmonary bypass. J Thorac Cardiovasc Surg 79:432-437, 1980

2. Ellis RJ, Wisniewski A, Potts R, et al: Reduction of flow rate and arterial pressure at moderate hypothermia does not result in cerebral dysfunction. J Thorac Cardiovasc Surg 79:173-180, 1980

3. Aren C, Blomstrand C, Wikkelso C, Radegran K: Hypotension induced by PGI_2 treatment during CPB does not increase the risk of cerebral complications. J Thorac Cardiovasc Surg 88:748-753, 1984

4. Aren C, Badr G, Fedderson K, Radegran K: Somatosensory evoked potentials and cerebral metabolism during cardiopulmonary bypass with special reference to hypotension induced by prostacyclin infusion. J Thorac Cardiovasc Surg 90:73-79, 1985

5. Fish K, Helms KN, Sarnquist FH, et al: A prospective, randomized study of the effects of prostacyclin on neuropsycholog-

ical dysfunction after coronary artery surgery. J Thorac Cardiovasc Surg 93:609-615, 1987

6. Murkin JM, Farrar JK, Tweed WA, et al: Cerebral autoregulation and flow/metabolism coupling during cardiopulmonary bypass: The influence of $PaCO_2$. Anesth Analg 66:825-832, 1987

7. Bashein G, Townes B, Nessly M, et al: A randomized study of carbon dioxide management during hypothermic cardiopulmonary bypass. Anesthesiology 72:7-15, 1990

8. Prough D, Stump D, Troost B: $PaCO_2$ management during cardiopulmonary bypass: Intriguing physiologic rationale, convincing clinical data, evolving hypothesis? Anesthesiology 72:3-6, 1990

9. Croughwell N, Lyth M, Quill T, et al: Diabetic patients lose pressure-flow cerebral autoregulation. Abstracts of the 12th Annual Meeting of the Society of Cardiovascular Anesthesiologist, May 1990, p 106

10. Govier AV, Reves JG, McKay RD, et al: Factors and their influence on regional cerebral blood flow during nonpulsatile cardiopulmonary bypass. Ann Thorac Surg 38:592-600, 1984