Effects of Mean Arterial Pressure on Cerebral Perfusion during Cardiopulmonary Bypass: A Review

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Cardiopulmonary bypass (CPB) remains an essential component of many cardiac surgical procedures. The non-physiologic nature of CPB flow creates a complex array of challenges, which can threaten optimal perfusion and patient well being. It is easy enough to assert that the perfusionist’s responsibility is to provide enough blood flow to meet the metabolic requirements of the patient’s tissues. In practice, however, it is difficult to ensure that this condition is always being met. The essence of the problem is that we lack the capability of accurately monitoring the metabolic status of individual tissue beds. Although there have been some promising technological advancements in this regard, “adequacy of perfusion” on CPB is still assessed essentially as it has always been. Systemic measures, such as partial pressure of oxygen and carbon dioxide, venous O2 saturation, pH, and base deficit, provide valuable information and can reveal profound systemic mismatches in flow/metabolism. Perfusionists are well aware, however, that normal values in these parameters can mask regional tissue malperfusion severe enough to result in morbidity and mortality.

The determination of the appropriate range of arterial pressure must be made each time the cardiac surgical team puts a patient on cardiopulmonary bypass. Decades of experience have taught us that the right answer is not always as simple as trying to mimic what is “physiologic”. Indeed, CPB flow has been demonstrated to have a number of decidedly “nonphysiologic” characteristics. It is typically nonpulsatile. It is high in embolic content. It is diluted—at times significantly so. Its coagulation and immune system constituents are in a state of generalized activation. Added to these variables is the fact that oxygen requirement in the brain, both regionally and globally, is profoundly attenuated by anesthesia and hypothermia. Moreover, the limitations imposed on flow rate by the physical characteristics of the CPB circuitry (especially the cross-sectional area of the tip of the arterial cannula) render it difficult, if not impossible, to maintain “normal physiologic” pressure on bypass. Indeed, it can be said that virtually all centers that perform CPB are accepting of pressures that are, to some degree, below what is considered “normal physiologic” (MAP = 90–95 mmHg). Is it possible to determine the pressures below which any patient will be at risk of cerebral hypoperfusion and subsequent injury? Second, are there specific subsets of patients for whom this threshold (assuming we can answer the first question) is different?

Experience over the last 40 years has shown that ischemic injury can occur—especially in the brain—despite CPB flow rates that were presumed adequate. Such occurrences led to the hypothesis that arterial hypotension on bypass can result in cerebral hypoperfusion, causing tissue injury and necrosis. Determination of the limits of safe and adequate mean arterial pressure (MAP) during CPB has proved to be a complex problem involving core temperature, arterial CO2 management, and cerebral “autoregulation.” It remains an area of controversy, and practice varies widely. This paper reviews the literature investigating the effect of MAP on cerebral perfusion during CPB.

1960–1980: CORRELATIVE DATA

The 20 years between 1960 and 1980 constitute a period during which numerous clinical studies were published implicating low arterial pressure during CPB as a cause of postoperative neurologic dysfunction. One of the first was Gilman (1) in 1965. This study retrospectively analyzed 35 CPB cases. The author reported that 12 of the 35 demonstrated some postoperative neurologic deficit and went on to point out that 3 of those 12 had periods of “hypotension” on bypass. The category was not rigidly defined but seemed to involve instances where MAP was below 40 mmHg for several minutes or more. The author also noted 3 patients who had similar hypotensive periods but did not demonstrate subsequent neurologic dysfunction. All 12 patients with cerebral injury had either valve replacement or septal defect repair. Arterial PCO2 management was not reported, and aortic cross-clamping technique was not discussed. Although the author stated that pre-existing cerebrovascular disease was probably the preeminent risk factor, this was one of the first papers to attribute a role for low pressure on bypass in the etiology of postoperative neurologic dysfunction.
In 1970, Tufo and associates published a prospective descriptive investigation, which reported postoperative neurologic deficits in 43% of the 85 survivors out of an original cohort of 100 patients (2). All cases were adult cardiac surgery involving aortic valve, mitral valve, or aortic aneurysm procedures.

Chi-square analysis showed a trend toward increased incidence of cerebral damage with increased duration of “arterial hypotension” (MAP < 50 mmHg). These differences reached statistical significance only in patients aged 40–49 years. The factor that correlated most strongly with injury was age. The authors admitted that air or particulate emboli could not be excluded as the cause of cerebral injury (as opposed to inadequate perfusion secondary to hypotension), but cited the finding of “anoxic lesions in the hippocampus”—presumably from autopsies of the 15 deceased patients—as evidence against the emboli hypothesis. Once again, aortic cross-clamp technique was not discussed, nor was arterial blood gas management. Notably, systemic hypothermia was used in the first 38 cases but was discontinued thereafter.

In the early to mid-1970s, the work of James J. Stockard (3) became influential. In 1974, he and associates reported a series of 75 CPB cases (40 CABG, 35 valve and other) during which EEG monitoring was used. Fifteen of these patients were subjected to “significant hypotensive stress,” defined as a drop in mean cerebral perfusion pressure (MAP—jugular venous pressure) of greater than 100 mmHg-minutes below 40 mmHg. The duration of this event (in minutes) multiplied by the pressure deficit (mmHg below 40) yielded a category termed mmHg-minutes. Those patients categorized as experiencing significant hypotensive stress had periods of cerebral perfusion pressure < 40 mmHg of at least 100 mmHg-minutes. Of these 15 patients, 8 demonstrated postoperative cerebral dysfunction. EEG disturbances coincided with the onset of hypotension in each of these 8 patients, persisted postbypass, and correlated with the severity of the deficit. The authors did not report the EEG findings for the 7 patients who experienced hypotension but did not sustain neurologic injury. They stressed that 53.3% of the hypotensive patients demonstrated CNS sequelae, whereas, the incidence was only 10.5% in those who experienced no hypotension. Age and preexisting cerebrovascular disease were the patient characteristics most strongly associated with cerebral injury. CPB flow rates were not reported. pH was maintained at 7.40 corrected for temperature (pH-stat). The authors reported that “some degree of hypothermia” was used during “long perfusions.”

The investigations above (and others similar in design and results) influenced the practice of many cardiac surgical teams in the late 60s, 70s, and early 1980s. It was argued that arterial pressure of at least 50 mmHg should be maintained to ensure effective cerebral perfusion (4). Not all practitioners held to this tenet, however, and some investigations during this period reported data suggesting that low flow and/or pressure was well tolerated during CPB. Ellis et al. (5) reported a prospective, descriptive study looking at a series of 30 coronary artery bypass-grafting (CABG) patients. CPB flow averaged 39 mL/kg, while arterial pressure ranged from 40 mmHg in some patients to 85 mmHg in others. Psychometric testing was done pre- and postoperatively. Five of the 30 patients demonstrated “mild impairment” postoperatively, which resolved within 6 months. The authors stated that “no correlation was identified” between psychometric change scores and MAP on bypass but did not report a correlation coefficient. Systemic hypothermia (28°C) was used in all cases. Arterial PCO₂ management was not reported.

Kolkka and Hilberman (6) studied a series of 204 cardiac surgical patients using a low flow (30–50 mL/kg), low pressure (30–60 mmHg) CPB protocol. Pre- and postoperative neurologic and neuropsychologic testing identified 35 patients with dysfunction postoperatively. The authors dichotomized the cohort based on the existence (vs. lack) of CNS sequelae and performed t-tests for differences in group means on a range of parameters. The only statistically significant differences found between patients with no dysfunction and those with dysfunction were in, respectively, age (55 ± 12 vs. 65 ± 10 years, p < .001), BUN on admission (18 ± 8 vs. 25 ± 13 mg/dL, p < .01), and percentage of patients undergoing CABG only (55% vs. 29%, p < .01). No difference was found in MAP (49 ± 7 vs. 51 ± 7) or in mmHg-minutes below 50 mmHg (554 ± 478 vs. 589 ± 591). These data stood in marked contrast to the work of Stockard et al., who had suggested that pressures below 50 resulted in neurologic injury (4).

Although the studies above (and other similar ones not mentioned) were intended to shed light on the relationship between perfusion pressure and neurologic injury on CPB, they share numerous flaws, which should be mentioned. With very few exceptions, they were retrospective, meaning that important confounding variables were not controlled for. Arterial PCO₂ management was seldom mentioned. The failure to control this variable alone renders conclusions regarding mechanisms of neurologic injury suspect. Other critical factors that were unaccounted for were patients’ age, diabetes, and previous stroke. Recent work (7) has identified these characteristics as having high association with CPB-related stroke. In addition, a wide variety of operative procedures were generally included in these studies. Procedures requiring myotomy, myectomy, and debridement of calcified valvular structure are more highly associated with neurologic injury than is primary CABG. In light of all this, it must be stated that these early studies shed little light on the possible role of arterial pressure in neurologic injury during CPB.

It can be said that by the early 1980s, considerable un-
certainly existed regarding appropriate arterial pressure on CPB. This led to calls for controlled studies evaluating cerebral perfusion under a variety of conditions during bypass. Beginning in the mid-1980s, investigators published studies that formed the foundation of the current understanding of the regulation of cerebral blood flow on bypass.

1984–PRESENT: PACO₂, TEMPERATURE, AND AUTOREGULATION

By this time it was widely held among both physiologists and physicians that under normal physiologic conditions, cerebral blood flow is subject to intrinsic regulation via at least two mechanisms. Lassen (8) first demonstrated the existence of a myogenic mechanism whereby CBF remains unchanged over a wide range of perfusion pressures (50–150 mmHg). Although not well understood, this is thought to be a localized response to stretch by vascular smooth muscle. Harper was among the first to describe the exquisite sensitivity of CBF to PaCO₂ (9). Shapiro documented profound cerebral vasodilatory responses to PaCO₂ below a certain hypoxic threshold (10,11,12). Taken together, these data strongly suggest a mechanism of “flow–metabolism coupling” in the brain. Cerebral vascular smooth muscle seems to be sensitive to the metabolic status of surrounding tissue and thus dilates or constricts to permit adequate (but not excessive) flow to meet oxygen demand.

Assessment of these mechanisms on bypass was dependent on the development of a technique to measure CBF reliably under these conditions. Clearance of tagged Xe (¹³³Xe) became widely accepted as the “gold standard,” but it should be noted that all techniques are estimates subject to error. Moreover, measurements of total brain blood flow can miss potentially important regional flow changes.

Prough and co-workers (13) published an important study in 1986, which yielded much information on the critical issue of CBF sensitivity to PaCO₂ during CPB. Twelve CABG patients were randomly assigned to either pH-stat or α-stat blood gas management. On CPB, patients were cooled to 28°C, and MAP was maintained between 65 and 70 mmHg, and flow index was 2.0–2.2 L/m²/min. PaCO₂ was manipulated in both groups, and CBF was determined via ¹³³Xe clearance twice on each patient: once at a low PaCO₂ and again at a high PaCO₂. In the α-stat group, CBF averaged (±SD) 13 ± 5 mL/100g/min at a mean PaCO₂ of 36 ± 4 mmHg. Increasing PaCO₂ in these patients to 42 ± 4 mmHg resulted in an increase in CBF to 19 ± 10 mL/100g/min. The pH-stat group had a similar response: at the lower PaCO₂ of 47 ± 3 mmHg CBF was 20 ± 8 mL/100g/min. Increasing PaCO₂ to 53 ± 3 mmHg yielded a CBF of 26 ± 9 mL/100g/min. Thus, CBF increased linearly with PaCO₂ in both groups, and differences were statistically significant within groups.

During the same year, Henrikson (14) published a study that tended to confirm Prough’s findings. He reported the relationship between CBF (¹³³Xe clearance) and MAP at three levels of PaCO₂—low (<40 mmHg), medium (40–50 mmHg), and high (>50 mmHg). CO₂ values were corrected to 25°C. At any arterial blood pressure (range 30–90 mmHg) CBF was higher at medium than low, and highest at the high CO₂ level. He also noted that at higher PaCO₂:1, the autoregulatory plateau shifts to a higher flow, and 2, the lower limit of autoregulation shifted to a higher pressure.

These studies had profound implications both clinically and from a research standpoint. It now seemed clear that CBF could be increased on bypass by adding CO₂ to the perfusate. Whether this was a salutary effect remained undetermined. Moreover, investigations purporting to evaluate the effects of MAP (or any parameter) on cerebral perfusion during bypass were now seen as uninterpretable unless PaCO₂ management technique was controlled for and reported.

Lundar and associates used transcranial Doppler (TCD) to measure flow velocity in the middle cerebral artery (MCA) in five CPB patients (15). Patients were cooled to 28–32°C, flows were maintained at 1.5 L/min, and pH-stat CO₂ management technique was used. The authors reported a positive linear relationship between cerebral perfusion pressure (CPP = MAP–CVP) and MCA flow velocity. They interpreted this as evidence that CBF (as assessed by MCA flow velocity) is pressure-passive during CPB with pH-stat management. It must be emphasized that there exists considerable debate as to the validity of MCA velocity as a measurement of cerebral blood flow during CPB. The preponderance of data suggests that it does not correlate well with more accepted techniques of CBF measurement. The usefulness of TCD measurements in this setting may be limited to detecting changes in flow trends and emboli in the MCA.

Grovier et al. (16) reported on a series of 67 CABG patients without cerebrovascular disease or hypertension. CPB parameters were: flow of 1.0–2.0 L/min, temperature of 28°C, and α-stat management. CBF was determined via ¹³³Xe clearance at various timepoints before, during, and after CPB. Two important findings were reported. First, there was a close relationship between nasopharyngeal temperature and CBF throughout bypass. Thus, CBF decreased in concert with (a presumed) decrease in cerebral O₂ demand as the patients were cooled. Second, there was no correlation between MAP (range = 30–110 mmHg) and CBF (r = −0.13). If the myogenic mechanism were not operative, we would have expected CBF to show a positive linear relationship with perfusion pressure. These data suggest that both mechanisms of ce-

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cerebral autoregulation—the myogenic mechanism and flow-metabolism coupling—are maintained during CPB when α-stat technique is used. Furthermore, this was the first evidence that the lower limit of pressure-flow autoregulation may be reduced to as low as 30 mmHg at mild to moderate hypothermia during CPB using α-stat.

In 1987, John Murkin and associates (17) shed considerable light on these issues with a prospective study of 38 elective CABG patients. Subjects were assigned to either α-stat or pH-stat management. The groups were otherwise identical, both demographically and in terms of interoperative management. Moderate hypothermia (26°C) was used, and 133Xe clearance was measured before CPB, three times on CPB, and after CPB. Cerebral oxygen consumption (CMRO₂) was calculated (CBF × arterial–jugular O₂ diff) at each of these timepoints. CMRO₂ decreased significantly from prebypass to hypothermic CPB, and then returned toward normal but was still significantly reduced from baseline values after rewarming in both groups. There were no differences in CMRO₂ between groups at any timepoint. CBF, however, differed markedly between groups: in the α-stat groups, CBF decreased with temperature and was significantly reduced as compared to pH-stat at both hypothermic measurements. CBF in the pH-stat group remained at pre-CPB levels throughout CPB. There was a positive linear relationship between CMRO₂ and CBF in the α-stat group (r = 0.50, p < .005), indicative of maintained metabolic autoregulation. CBF was unrelated to CMRO₂ in the pH-stat group (r = −0.11, p > .2), suggesting that the higher level of PaCO₂ abolished flow-metabolism coupling.

In the pH-stat group, CBF was highly correlated with CPP (r = 0.59, p < .002). This supports the hypothesis that myogenic autoregulation is also abolished at higher PaCO₂. There was no relationship between perfusion pressure and CBF in the α-stat group (r = −0.16, p > .2). Pressure-flow autoregulation was maintained over a range of 20–100 mmHg. Murkin’s paper still stands as strong evidence of the maintenance of cerebral autoregulation during CPB with α-stat management, and, thus, of the relative nonimportance of perfusion pressure as a determinant of CBF under these conditions.

Three subsequent investigations evaluated cerebral autoregulation and possible effects of vasoactive agents commonly used during CPB. Rogers and associates published two studies looking at the effects of phenylephrine and sodium nitroprusside on CBF during α-stat and pH-stat conditions (18,19). Increasing MAP by ≥25% had no effect on CBF in the α-stat group but increased flow by 41% in the pH-stat group (p < .001). Similarly, lowering MAP by ≤20% left MAP unchanged in the α-stat patients and slightly reduced in the pH-stat group.

Aladj and co-workers (20) measured CBF during CPB under conditions of control, 0.6% isoflurane, and 1.2% isoflurane. In each group, measurements were obtained at an initial MAP, then again after raising MAP by ≥25% via phenylephrine infusion. CBF was significantly reduced at both measurements in the isoflurane groups as compared with control (p > .05). However, the 25% increase in MAP had no effect on CBF in any of the groups. The authors argued that the reduced CBF in the isoflurane groups was secondary to reduced CMRO₂ although they did not measure that parameter.

These three studies made two important contributions. First, they reaffirmed the maintenance of cerebral autoregulation during CPB with moderate hypothermia and α-stat CO₂ management, the precise characteristics (i.e., range and slope) of the autoregulatory plateau are not agreed upon. Data from Grovier, Murkin, and Soma suggest a low-end arterial pressure threshold of 30 mmHg (16,17,21). Henrikson’s data, however, showed pressure-passive CBF at ≤50 mmHg (14). The latter might well be viewed with caution because a PaCO₂ of 31 mmHg at 27°C is not consistent with a patient properly managed using the α-stat protocol. Schwartz and co-workers (22) measured CBF (133Xe) in baboons during moderately hypothermic CPB at MAP = 60, and again at MAP = 20 mmHg. Flow index for both measurements was 2.25 L/m²/min. This protocol was repeated at a flow index of 0.75 L/m²/min. CBF was significantly reduced at 20 mmHg under both flow regimens. Under these circumstances, autoregulation was abolished at MAP = 20 mmHg.

The preponderance of data that exists now suggests that there is, in fact, a positive slope to the CBF autoregulatory “plateau”. Newman (23), employing a study design using patients as their own controls, calculated that CBF increased by 0.86 mL/100g/min for each 10 mmHg increase in MAP. This was at 29.7°C. In 1996, Newman and associates calculated an autoregulatory slope of twice that at normothermia (1.78 mL/100g/min). This may be important information given recent trends toward normothermic CPB. Mutch et al. (24) published results similar to those of Newman’s group. The former were confounded, however, because the technique used to vary MAP was alteration in the CPB flow rate. More recently, Sungurtekin and co-workers (25) published data suggesting a much steeper slope in the MAP–CBF relationship (on the order of 7 mL/100g/min of CBF per 10 mmHg increase in MAP) using α-stat and mild hypothermia. This work used a canine model, however, and should be replicated in humans to allow more definitive interpretation.
This review of literature reveals certain points that have been demonstrated repeatedly and, in some cases, quite elegantly. These are outlined below.

1. CBF increases with increasing PaCO₂. This relationship holds over a broad range of perfusion pressure and core temperature.

2. pH-stat technique results in a relationship between perfusion pressure and CBF that is direct and linear. There exists no “autoregulatory plateau,” as has been demonstrated repeatedly under “normal physiologic” conditions.

3. When alpha-stat technique is used, pressure-flow autoregulation has been demonstrated. That there is a small positive slope to this autoregulatory “plateau” comes as no surprise. Indeed, it is characteristic of most physiologists understanding of this type of autoregulation. That the slope increases with temperature is of interest and concern because it means that a given decrease in perfusion pressure results in larger decreases in CBF when a patient is normothermic.

4. Flow-metabolism coupling is obviated under pH-stat, but not under α-stat, conditions. The lack of relationship between local oxygen demand and blood flow during pH-stat can presumably be ascribed to CO₂-mediated vasodilation.

**CAN MANAGEMENT OF MAP OR ARTERIAL BLOOD GASES INFLUENCE OUTCOMES?**

Well-designed clinical outcome trials involving systematic manipulation of arterial pressure and/or arterial PCO₂ form only a small body of literature. Interpretation can prove difficult because definitions used for morbidity range from stroke to neurologic and neuropsychologic dysfunction. Use of different testing methods to identify dysfunction creates a further problem. Finally, the degree of dysfunction considered to be significant varies with investigators. Agreement on some of these issues would help advance our understanding.

The question of whether arterial pressure on bypass influences outcomes has yet to be definitively answered. Gold and associates (26) attempted to answer this question, among others, in the Cornell Coronary Artery Bypass Outcome Trial (CABOT). In this study, a total of 248 primary CABG patients were randomly assigned to either low (51.8 ± 5.2 mmHg) or high (69.5 ± 7.1 mmHg) mean arterial pressure on bypass. Patients were cooled to 28–30°C and α-stat blood gas management was used. The authors assessed mortality, cardiac morbidity, neurologic morbidity, cognitive dysfunction, and changes in quality of life prospectively to 6 months postsurgery. They reported no statistically significant differences between groups in any of these parameters. They went on to make the argument, however, that higher pressures effectively improve outcomes. The evidence they used to support this were trends toward higher mortality (4.0% vs. 1.6%, $p = .25$), higher incidence of stroke (7.2% vs. 2.4%, $p = .076$), and higher incidence of cardiac morbidity (4.8% vs. 2.4%, $p = .3$) in the low pressure group. They also summed the number of incidences for these three separate parameters in each group and determined that the low pressure group was significantly higher (12.9% vs. 4.8%, $p = .026$).

It is important to evaluate this investigation thoroughly because it remains the only one that directly assesses the influence of CPB pressures on outcomes. From a design standpoint, it holds up well, with the possible exception that the difference in MAP between the two groups might, perhaps, have been larger. The conclusions the authors drew from the data, however, must be questioned. Rejection of a null hypothesis based upon nonsignificant trends is not an acceptable procedure in the western scientific method. Furthermore, the post hoc grouping of separate dependent variables into one obfuscates the effects of CPB pressures on cerebral outcomes. Whether the reported difference in cardiac morbidity reflects a true treatment effect depends on the level of control of the myriad parameters that can affect that variable. Cardiac morbidity can clearly be secondary to numerous mechanisms unrelated to those that underlie neurologic injury on CPB. A conservative, judicious evaluation of the data presented in this study must lead to the conclusion that improved neurologic outcomes secondary to higher MAP was not demonstrated in this study.

**DISCUSSION**

Ample evidence has been presented that CBF autoregulation is maintained—perhaps to pressures as low as 30 mmHg when patients are moderately hypothermic—during cardiopulmonary bypass with alpha-stat blood gas management. The lower end of the autoregulatory range—especially at varying core temperatures—has not been adequately defined. It would, therefore, seem imprudent to maintain perfusion pressures purposely at levels where the literature suggests that uncertainty exists. No study has demonstrated failure of autoregulation at pressures of 50 mmHg or above. That would seem a good place to begin in establishing protocols because Henrickson (27) has argued that pressures at or just below the autoregulatory threshold are unlikely to result in neural injury.

Another area of uncertainty centers on the effects of periods of hypotension during CPB. Such periods typically occur at the initiation of CPB and when venous return is obstructed, such as during heart retraction for right or
circumflex anastomoses. Henrikson cites data, much of it from studies using oligemic models, suggesting that such insults may not be well tolerated and, thus, should be avoided during CPB whenever possible. It is in precisely this situation that a reliable cerebral ischemia monitoring technology would be extremely valuable. Cerebral oximetry may represent such a technology (28). More research must be done to establish its uses and limitations in the cardiac surgical theater.

It has been argued that maintenance of higher perfusion pressure—within the autoregulatory range—might protect against neurologic injury during bypass. However, a large, prospective, randomized trial designed to test this very hypothesis (26) failed to show a significantly lower incidence of stroke in the high-pressure group. If we accept that the vast majority of strokes are embolic in nature, then these results are not surprising (29). Moreover, higher intravascular pressure could mitigate toward capillary filtration and the buildup of interstitial fluid pressure. The “tissue pressure hypothesis” has been clearly implicated as a mechanism in attenuated or blocked regional flow in cerebral hemorrhage (30). MRI data (31) demonstrate that postbypass cerebral swelling can be significant. Whether this is in and of itself a mechanism of injury during or after CPB has not been determined.

Certain circumstances may call for heightened concern about adequate perfusion pressure during CPB. Data have been published that are suggestive of a rightward shift in the pressure-flow autoregulatory curve in hypertensive patients (32). These and other data also suggest that the autoregulatory threshold tends to normalize in patients with pharmacologically controlled hypertension. Whether these relationships hold during cardiopulmonary bypass remains to be determined. We also know little about autoregulation and adequacy of cerebral perfusion on bypass in patients with known carotid or cerebrovascular disease. Prospective, randomized trials focusing on these patients have not been done. Clinicians tend to surmise that higher pressure is “safer” in this scenario, but we do not really know if this is true, or if it is, what pressure is optimal.

It is likely that future efforts to reduce the incidence of postoperative neurologic injury will not be focused upon manipulation of arterial pressure. The literature would suggest that clinicians should strive to maintain pressure above the low end of the autoregulatory range. There are scant, if any, data documenting that within that range higher is better than lower. Reduction in stroke and other brain injury will likely result from techniques and materials designed to reduce delivery of emboli to the central nervous tissues. These may include evaluation and identification of high-risk patients, such as echo analysis of aortic atheromata. Surgical techniques associated with embolism (aortic cross clamping, valvular debridment, de-airing after myotomy closure) will evolve. Foreign surface materials will be modified to attenuate activation of platelets, coagulation factors, and the immune system. There is mounting evidence that the latter may play a significant role in neurologic injury. Techniques of cerebral blood flow monitoring may provide helpful information on acute changes in flow trends, guiding modification of CBP technique to minimize injury.

REFERENCES


