Hypertensive episodes during and after open heart surgery

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Since our initial description of hypertension following myocardial revascularization in 1973,[1] hypertension has been recognized more frequently as a complication before, during, and after surgery for myocardial revascularization and heart valve replacement. A classification based upon the timing of occurrence of the hypertension in relation to surgery is more feasible than a classification based on a specific mechanism[2] *(Table).*

Systemic arterial pressure is determined by several variables, cardiac output, peripheral resistance, blood volume, aortic compliance, autonomic activity, and other humoral factors. All of these are affected by premedication, anesthesia, surgery on the heart itself, cannulation of the big vessels, cardiopulmonary bypass, and the postoperative setup.

Preoperative stress, discomfort, and inadequate premedication can cause a sudden rise in blood pressure more pronounced in preoperatively hypertensive patients, particularly if antihypertensive treatment and beta blockers were discontinued. A sudden rise in blood pressure at this stage increases myocardial oxygen consumption, can cause anginal pain and vice versa, therefore, a vicious circle of hypertension and myocardial ischemia. Reflex sympathetic activity due to endotracheal intubation, nasopharyngeal, rectal, or urethral manipulation

Table. Hypertension associated with cardiac surgery

Preoperative
Anxiety, anginal crisis, etc.
Discontinuance of antihypertensive therapy
"Rebound" from beta-blockers
Intraoperative
Induction of anesthesia
Specific drugs
Tracheal intubation, nasopharyngeal, urethral, rectal manipulation
Precardiopulmonary bypass
During sternotomy and chest retraction
Cardiopulmonary bypass
Postcardiopulmonary bypass (during surgery)
Postoperative
Early: within 2 hr
Obvious cause
Hypoxia, hypercarbia, ventilatory difficulties, hypothermia, shivering, arousal from anesthesia
No obvious cause
35%–40% following myocardial revascularization
8%–12% following valve replacement
40%–50% following resection of aortic coarctation
Intermediate: 12–36 hr following surgery (Sealy type II) following repair of aortic coarctation
Late: weeks to months following aortic valve replacement by homografts (rare)

causes tachycardia and a rise in blood pressure, which can be complicated by acute impairment of left ventricular function as well as electrocardiographic signs of ischemia.

Not infrequently, acute hypertension develops during median sternotomy possibly due to reflex sympathetic stimulation, despite the expected decrease in venous return and cardiac output due to loss of negative intrapleural pressure.

On initiation of cardiopulmonary bypass, arterial mean pressure drops due to the nonpulsatile flow and the effect of hemodilution. Subsequently, the mean arterial pressure rises to levels even higher than the prebypass levels. This rise reflects an increased total peripheral resistance as the cardiac output is controlled mechanically. Increased antidiuretic hormone, catecholamines, and activation of the renin angiotensin system were reported as the cause of this hypertension. However, surgery and manipulation of the heart and cannulation of the large vessels lead us to postulate that pressor reflexes may also be activated. In our experience the total peripheral resistance continues to rise following cardiopulmonary bypass despite hemodilution and anesthesia; such a rise is extended to the postoperative period.

Postoperatively, hypertension is reported in 35% to 45% of myocardial revascularization patients and 8% to 12% of valve replacement patients. The rise in blood pressure occurs 1 to 2 hours postoperatively and lasts 8 to 12 hours. Usually, it is not accompanied by signs of hypovolemia or slowing of the heart rate. As the blood pressure rises, there is no significant change in cardiac output, central venous pressure, or left ventricular end-diastolic pressure. These findings are associated with significant increase in total peripheral resistance and are suggestive of increased sympathetic drive.[3] In 1973 we hypothesized that this increased sympathetic drive can be

due to pressor reflexes from the heart and great vessels. Later this hypothesis was emphasized by the control of hypertension by unilateral stellate ganglion blockade. Reports that increased plasma renin activity and catecholamines play a role in the pathogenesis of postoperative hypertension[4] do not contradict this hypothesis, as sympathetic stimulation is a potent stimulant to the release of catecholamines and renin.

Whatever the causes or the timing of hypertension, it is an undesirable complication in patients with heart disease as it increases myocardial oxygen consumption and predisposes to vascular, cerebral, and renal complications. Continuance of antihypertensive therapy and beta blockers, preoperative preparation, and sedation of the patient minimize these hypertensive episodes. Use of peripheral vasodilators is a safe and universal way of controlling the blood pressure during various stages of surgery. However, in our experience, in the postoperative period, unilateral stellate[5] ganglion blockade was successful in controlling the rise in blood pressure on those patients who were resistant to the use of peripheral vasodilators or required large undesirable doses.[5]

References

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