

## Revista Mexicana de Anestesiología

Volumen **28**  
Volume

Suplemento **1**  
Supplement

**2005**

*Artículo:*

### Update of anesthesia for vascular surgery

Derechos reservados, Copyright © 2005:  
Colegio Mexicano de Anestesiología, AC

Otras secciones de  
este sitio:

-  [Índice de este número](#)
-  [Más revistas](#)
-  [Búsqueda](#)

*Others sections in  
this web site:*

-  [Contents of this number](#)
-  [More journals](#)
-  [Search](#)



[www.Medigraphic.com](http://www.Medigraphic.com)

## Update of anesthesia for vascular surgery

Alexandru Gottlieb, M.D. FACA\*

\* Professor of Anesthesia. Department of Anesthesia, The Cleveland Clinic Foundation, Cleveland, Ohio, USA.

### I. INTRODUCTION

Patients scheduled for aortic reconstructive surgery are often at increased risk due to:

#### A. Patient medical condition

Most of the pathology is due to diffuse atherosclerosis, a systemic progressive disease that can cause 1) Arterial plaque enlargement, 2) Arterial embolism, or 3) Complete occlusion of circulation. The plaque location is often at the bifurcation where turbulent flow might exist. Mostly, at the carotid bifurcation, coronary arteries, iliac arteries, aorta and superficial femoral artery. The symptoms might vary with the site and degree of the lesion. Intermittent claudication, rest pain, ulceration, or gangrene is all described. The patients are often older; most of these patients suffer from generalized atherosclerotic cardiovascular disease. Therefore, many of their other systems and organs may be affected as well. They have increased incidence of hypertension, coronary artery disease, myocardial disease, valvular heart disease, renal disease, diabetes and cerebral vascular disease.

#### B. Surgical procedures

Surgical procedures are often involved in potential for blood loss and major volume shifts. Organ preservation might also be of concern due to periods of interruption in perfusion. It is imperative, therefore, that the anesthesiologists perform special risk assessments on all these patients.

### II. PERIOPERATIVE EVALUATION

A complete history and physical examination are mandatory, including system review, selected laboratory tests, and medication review with special emphasis on daily medications because many can interact with anesthetics. Most anesthesia-in-

duced morbidity and mortality is related to the cardiovascular system<sup>(1-4)</sup>. Therefore, it is obligatory to perform a special risk assessment for this system.

#### A. Risk assessment

The American Society of Anesthesiologists, relying on the Drips classification, found that general physical condition correlates with surgical outcome. Similar correlation was found with the New York Heart Association classification where patients in Class 3 or 4 had a six- to ten-fold incidence of mortality<sup>(5)</sup>. Other factors included were history of nitrates intake, dyspnea on exertion, angina, aortic stenosis, advanced age, aortic surgery, emergency operation, dysrhythmias, CHF, and myocardial infarction<sup>(5-10)</sup>. According to Mangano, the last three dysrhythmias, left ventricular dysfunction and myocardial infarction are the most important prognostic factors in risk assessment<sup>(11)</sup>.

Others<sup>(12-18)</sup> revalidated the Multiple Risk Index (MRI) but modified the importance of factors such as angina, hypertension, diabetes, non-Q-wave infarction, uncomplicated recent MIs and extensive MIs. Also were added: left ventricular hypertrophy, digoxin intake or aortic reconstructive surgery, patient operation, anesthesia skill, surgical experience, new technology and hospital equipment, and other quality assurance factors.

#### B. Evaluation of ischemic heart disease

Patients with congestive heart failure and some of the signs and symptoms of chronic heart failure should first be treated with diuretics, digitalis, or inotrope until they attain optimum and stable function. Several authors<sup>(19-21)</sup> describe the association between CAD and vascular disease. As a result, perioperative myocardial infarction (PMI) can be as common as 6% to 20% during vascular surgery. The majority of pa-

tients, diabetic as well as non-diabetic, can have undiagnosed CAD or silent perioperative myocardial ischemia (SMI)<sup>(22,23)</sup>. Muir<sup>(24)</sup> recommended Holter monitoring to detect patients at high cardiac risk. Patients with SMI were more likely to have impaired left ventricular function (< 40% ejection fraction). Further heart investigation, such as ambulatory EKGs, or cardiac stress tests (exercise, dipyridamole, and thallium) were all suggested. A two-dimensional echocardiogram with dipyridamole, dobutamine, or color Doppler measurements, was also recommended. More sophisticated diagnostic methods, such as positron-emission tomography (PET) or single photon-emission computed tomography (SPECT) can provide information about the heart's metabolism as well as about its blood supply and mechanical function<sup>(25)</sup>.

C. "Guidelines for perioperative cardiovascular evaluation for noncardiac surgery"-  
(The American College of Cardiology/American Heart Association 1994, 1997, 2002)

These Guidelines are based on Medline search of the English literature from 1975 through 2001, review of selected journals from 1995 and expert opinions of 12 committee members representing various disciplines of cardiovascular care. The purpose of the guidelines was to provide a framework for considering cardiac risk of noncardiac surgery in various patients and operative situations.

1. General approach to cardiac patient undergoing noncardiac surgery necessitates careful teamwork and communication among patient, primary-care physician, anesthesiologist, surgeon, and the medical consultant.
2. Coronary revascularization before noncardiac surgery to enable the patient to "get through" the noncardiac procedure is appropriate only for a small subset of very high-risk patients. Preoperative testing should be limited to circumstances in which the results will affect patient treatment and outcomes.
3. A conservative approach to the use of expensive tests and treatments is recommended. Preoperative Clinical Evaluation.
4. It is essential to define severe stability and prior treatment of the disease. Patient's functional capacity, age, co-morbid conditions (DM, PVD, CRF, and COPD) and types of operation (AAA, TAA, and head and neck surgery) are considered higher risk.
5. **Clinical Predictors** of Increased Perioperative Cardiovascular Risk are:
  - a. *Major*: Unstable coronary syndromes, recent MI, decompensated CHF, significant arrhythmia or severe valvular disease.

- b. *Intermediate*: Mild angina pectoris, prior MI compensated or prior CHF or diabetes mellitus.
  - c. *Minor*: Advanced age, abnormal ECG findings (LVH, LBBB, ST-T abnormalities), rhythm other than sinus (i.e.: AF), low functional capacity, history of stroke and uncontrolled systemic hypertension.
6. **Surgical Cardiac Risk Stratification for Noncardiac Surgical Procedures:**
- *High*: Emergent operations, aortic and other major vascular operation, peripheral vascular operation, anticipated prolonged surgical procedures associated with large fluid shifts or blood loss (or both).
  - *Intermediate*: CEA, head and neck operation, intraperitoneal and intrathoracic operation, orthopedic operation, prostatectomy.
  - *Low*: Endoscopic procedures, superficial procedure, cataract operation, or breast operation.

In summary, numerous methods can be used to stratify high-risk cardiac patients before major vascular procedures. Most authors recommend a combination of history, physical examination, and non-invasive and invasive tests<sup>(26,27,27a)</sup>. Patients with diabetes, angina, a Q-wave on EKG, ventricular dysrhythmia, and age above 70 years are likely to have perioperative cardiac complications. Patients with all or none of these clinical risk factors will not benefit from further invasive or non-invasive tests; patients with two or three of these risk factors can benefit from further tests. There is no complete agreement as to the best tests to be performed on this group. A practical approach is to proceed from simple to more complex testing.

### III. ANESTHETIC MANAGEMENT OF AORTIC RECONSTRUCTIVE SURGERY

When determining the equipment, monitoring, and anesthetic to be used in this procedure, the risk status of the patient and the potential intraoperative acid-base, coagulation, and hemodynamic changes, including excessive blood loss, must be considered. Anesthesia for emergency or symptomatic AAA, can be more challenging due to higher incidence of surgical and anesthetic perioperative potential complications.

#### A. Monitoring

Del Guericco's<sup>(28)</sup> study in 148 patients has shown that more invasive preoperative care, as well as delay in surgery, can improve surgical outcome. Slogoff and Keats<sup>(17)</sup> and Rao<sup>(10)</sup> had better results with improved perioperative hemodynamic and cardiac monitoring. Whitmore<sup>(29)</sup> found that mortality decreased after aortic surgery from 5% to 0% when a preop-

erative pulmonary artery (PA) catheter was used to establish a Starling curve.

In a prospective randomized study of 89 patients, Berlauk<sup>(30)</sup> found a significant decrease in mortality (from 9.5% to 1.5%), cardiac morbidity, and early graft thromboses in patients who were 'tuned up' either in surgical intensive care or the preinduction room. The 'tuning up' consisted of combinations of fluid loading, after load reduction, and inotropic support.

This finding was not replicated by Pull Ter Gunne<sup>(31)</sup>, who found no improvement in hemodynamics or renal function after preoperative hydration guided, by a PA catheter. Preparation in the operating room should include large; multiple intravenous accesses, a five-lead EKG, and computerized continuous ST-T segment evaluation. Arterial blood pressure should be monitored with an in-dwelling arterial catheter that will also allow periodic measurements of arterial blood gases. A ST change indicating myocardial ischemia is strong predictors of perioperative MI. Postoperative ischemia is a significant predictor of long-term MI and cardiac death. Use of computerized ST-segment analysis in appropriately selected high-risk patients may improve sensitivity for detection of myocardial ischemia.

#### *Central venous pressure catheters or Swan-Ganz catheters*

Are used to monitor adequate volume replacement. The ASA believes that the following 3 variables are particularly important in assessing benefit *versus* risk of use of PAC:

1) Disease severity. 2) Magnitude of anticipated surgical procedure. 3) Practice setting. The patients most likely to benefit from use of PAC perioperatively are those with: 1) Recent MI complicated by CHF, 2) Severe CAD who is undergoing procedures associated with pronounced hemodynamic stress, 3) Systolic or diastolic LV dysfunction, 4) Cardiomyopathy, 5) Severe valvular disease. We recommend cannulating with a Swan-Ganz catheter every patient with a) poor left ventricular function, b) recent myocardial infarction, c) unstable angina, d) intractable CHF, e) preoperative renal insufficiency, f) renal revascularization, or g) thoracoabdominal aneurysm. With the improvement of transesophageal echocar-

diography, some suggest that echocardiography is more sensitive than ST-segment analysis in detecting myocardial ischemia<sup>(32)</sup>. Left ventricular regional wall motion and ejection fraction correlated well with the incidence of myocardial ischemia. But the largest experience to date suggests that the incremental value of this technique for myocardial ischemia risk prediction is minimal.

#### B. Choice of anesthetic

But as far as other drugs, there is not scientific evidence to support the use of any one anesthetic technique for major vascular procedures. Intravenous narcotics are better at preserving cardiac output and blood pressure<sup>(33)</sup>, and adding inhaled anesthetic helps to control hypertension but can cause myocardial depression (important for patients with borderline LV reserve). Sufentanil had better results than isoflurane in one study Benefiel ASA 1986. Most drugs should be titrated carefully because their effect on the geriatric atherosclerotic patient is unpredictable. Similarly, evidence favors neither general nor neuraxial techniques over the other for vascular surgery. One should still decide on the basis of personal clinical experience. Spinal/Epidural cause sympathetic blockade. Abdominal operations that necessitate a high dermatomal level of anesthesia may result in hypotension and reflex tachycardia. Monitored Anesthesia Care- failure to produce complete local anesthesia or analgesia can lead to increased stress response, which may produce myocardial ischemia or depression.

The American College of Physicians has recommended administration of  $\beta$ -adrenoceptor antagonists to patients with coronary artery disease undergoing surgery. To date,  $\beta$ -adrenoceptor antagonists are the only well-established means of prophylaxis against myocardial ischemia that demonstrates a reduction of morbidity and mortality in this patient population<sup>(32a)</sup>. (Table I).

#### C. Aortic cross-clamping and declamping

One of the most destabilizing events during aortic reconstructive surgery is the clamping and unclamping of the aorta. Clamping the aorta interrupts blood flow to major or-

**Table I.**

Operation	Time	Drug	Outcome
Stone JG 1988 Vasc Abd	Post-op	Labetolol, atenolol	11/39 vs 2/89 ↓ Ischemia
Hammon JW 1984 CABG	Pre/Intra/Post	Propranolol	↓ BP, dysrhythmias, ischemia
Pastermack PF 1989 Vasc	Pre	Metoprolol	↓ Ischemia
Raby KE 1999 Vasc	Intra post	Esmolol	↓ Ischemia
Poldermans D. 1999 Vasc	Pre/Intra/Post	bisoprolol	↓ MI/Death/Ischemia

gans, directly or indirectly causing hemodynamic and metabolic changes. These changes are highly visible with more proximal occlusion<sup>(34,40)</sup>.

**1. Hemodynamic changes.** In the 70s and 80s Silverstein and Attia described in detail the physiologic cardiovascular changes occurring during aortic cross-clamping. Increase in after-load, MAP, SVR, LVEDP. Depression of cardiac output and LSWI are usually happening while pre-load, CVP, PEDP can go up, down or remain unchanged. Peripheral vasodilatation with nitroglycerin or sodium nitroprusside has been suggested to attenuate the hemodynamic response.

**2. Metabolic changes.** Gelman described in a review article in 1995 all the metabolic changes that might occur with aortic cross clamping and declamping (Table II). Most metabolic changes are reversed gradually after declamping, but the acidosis that increases during the clamping time can reach new highs with the flushing of ischemic tissue. Several authors have suggested the use of continues bicarbonate drip throughout the clamping specially in TAAA repairs. "Declamping shock" appears to be caused mostly by volume depletion and decreased venous return but many mediators were implicated in the hemodynamic and metabolic changes. The gradual release of the aortic clamp, volume loading, and correction of acidemia, hyperkalemia, hypocalcemia, and inotropic support of cardiac output are all effective during declamping.

D. Effect of aortic cross- clamping on organ systems

**1. Spinal Cord Preservation.** The mechanism by which the insult to the spinal cord occur is in the table II. Several

techniques have been suggested to minimize anterior spinal cord ischemia: hypothermia, spinal cord drainage, arterial shunting, and pharmacological agents including papaverine, scavengers, and 'spinalplegic' solutions. Several monitoring techniques can detect patients at risk, including somatosensory-evoked potentials, motor-evoked potentials, and angiographic or other visualization methods for identifying the arterial radicularis magna.

**2. Renal preservation.** Is as prevalent as in 7% of aortic surgery (Table III). The use of mannitol, dopamine or

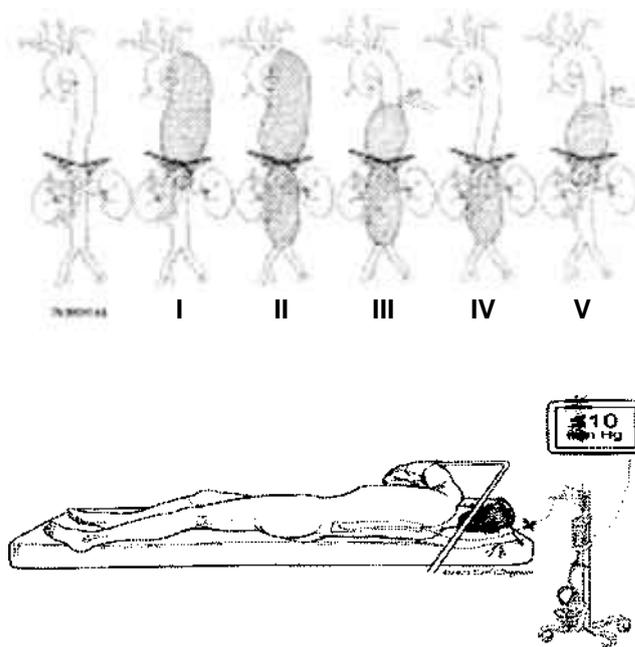


Table II.

Acidosis	Oxygen free Radical	Myocardial-Depressant Factor	Angiotensin	Interleukin-1,6
Lactate	Hypoxanthine	Endotoxins	Renin	Hypophosphatemia
Hypercarbia	Xanthine Oxidase	Cytokine	Epinephrine	TxA2 Synthesis
Hypocarbia	Purines	Tumor Necrosis Factor (TNF)	Norepinephrine	Prostaglandin
Bradykinin Endothelin-1		Anaphylatoxins	Histamine	

Table III.

AX-c Time(min)	Paraplegia%	Renal Failure%	Anterior-Spinal-Artery-Syndrome (Mechanism)
0-15	0	0	1. Injury to radicular artery by clamp
16-30	3.5	4.2	2. Radicular artery is distal to Ax-c
30-45	10	7.8	3. Arteria Magna is inside the aneurysm
40-60	12.5	6.3	4. Prolonged Ax-c
> 60	25		5. Prolonged hypotension
		Livesy 1985	6. Thrombosis of lumbar collateral

verapamil and fenolopam<sup>(41)</sup> were all suggested, but the most widely accepted method is to maintain adequate volume throughout the surgical procedure.

3. **Lungs.** Postoperative respiratory failure is as common as 26% after TAA. The pathophysiology is not always clear and it is multifactorial. Direct instrumentation, ventilatory barotrauma or lung atelectasis due to increase intra abdominal pressure and hypoventilation as a result of pain, can all contribute to organ failure. Microembolism of thrombi, atheromas or tissue debris can cause increase pulmonary vascular resistance both directly and through humerale factors such as TxA2 or anaphylatoxin. Increase in vascular permeability and congestive heart failure can bring increase in lung-water content and pulmonary edema.

Intraoperative Nitroglycerin data are insufficient to determine whether prophylactic intraoperative IV administration of nitroglycerin is helpful or harmful in high-risk patients vasodilating properties of NTG may be potentiated by anesthetic agents, leading to severe hypotension and even myocardial ischemia.

4. **Perioperative surveillance.** Surveillance for Perioperative MI: In patients with any history of CAD- Clinical symptoms, postoperative ECG changes, increase of MB-CPK, increase of troponin-I and troponin-T should all be followed closely. But in patients without known CAD, surveillance should probably be restricted to those who have signs of cardiovascular dysfunction. In patients with known or suspected CAD undergoing high-risk procedures, obtaining ECG at baseline, immediately after the procedure, and for the first 2 POD is cost-effective. Measurement cardiac enzymes are done only in patients with clinical, ECG, or hemodynamic evidence of cardiovascular dysfunction.
5. **Postoperative management.** Should include assessment and management of modifiable risk factors for-CAD, CHF, HTN, stroke and other cardiovascular disorders (Hyper-

cholesterolemia, Smoking, Hypertension, Diabetes, Physical inactivity, Peripheral vascular disease, Cardiac murmur, Arrhythmia, Conduction abnormalities, Perioperative ischemia, Postoperative MI). Patients who experience repetitive postoperative myocardial ischemia or sustain a superior MI have substantially increased risk of MI or cardiac death during long-term follow-up. These patients should especially undergo risk factor interventions and future risk stratifications and therapy. Effective perioperative pain management (I.V. or epidural analgesia) leads to: Reduction in postoperative catecholamine surges and controls hypercoagulability response-Both of which can theoretically affect myocardial ischemia.

In summary, there is no complete agreement about the best preoperative evaluation techniques for risk assessment in major vascular procedures. Our emphasis is returning, perhaps, to the basic history and physical, although more sophisticated scanning techniques are also being developed. Silent preoperative and postoperative ischemia is closely related to cardiac outcome. Myocardial oxygen demand should be kept low; oxygen supply is gaining interest, as favorable lower complication rates after CABG and PTCA are reported<sup>(42)</sup>. In addition, the less complex endovascular aortic reconstruction surgery is gaining acceptance. These surgeries demonstrate better short-term outcome<sup>(43,44)</sup>. Anesthesia and monitors are much improved and allow us to have more stable patients during induction, aortic cross clamping, and emergence from anesthesia. Complication rates are decreasing in general, but no significant differences were found between regional and general anesthesia, in spite of numerous studies. Techniques for organ preservation are not yet satisfactory but the problem is heavily investigated and there is hope for breakthroughs in physiology and pharmacology.

## REFERENCES

- Hicks GL, Eastland MW, DeWeese JA, May AG, Rob CG. *Annals of Surg* 1975;181:863-9.
- Crawford ES, Bomberger AR, Glaeser DH, Saleh SA, Russell WL. *Surg* 1981;90:1055-67.
- Diehl JT, Cali RF, Hertzner NR, Beven EG. *Ann Surg* 1983;197:49-56.
- Yaeger MP, Glass DD, Neff RK, Brinck-Johnsen T. *Anesthesia* 1987;66:729-36.
- Goldman L, Caldera DL, Southwick FS, Nussbaum SR, Murray B, O'Malley TA, Goroll AH, Caplan CH, Nolan J, Burke DS, Krogstad D, Carabello B, Slater EE. *Medicine* 1978;57:357-70.
- Foster ED, David KB, Carpenter JA, Abele S, Fray D. *Ann Thorac Surg* 1986;41:42-50.
- Kennedy RD, Andrews GR, Caird FI. *Br Heart J* 1977;39:1121-7.
- DeBusk RF, Blomqvist CG, Kouchoukos NT, Luepker RV, Miller HS, Moss AJ, Pollock ML, Reeves TJ, Selvester RH, Stason WB, et al. *N Engl J Med* 1986;314:161-6.
- Steen PA, Tinker JH, Tarhan S. *JAMA* 1978;239:2566-70.
- Rao TL, Jacobs KH, El-Etr AA. *Anesthesia* 1983;59:499-505.
- Mangano DT. *Anesthesiol* 1990;72:153-84.
- Zeldin RA. *Canadian Journal of Surg* 1984;27:402-4.
- Goldman L. *Ann Intern Med* 1983;98:504-13.
- Jeffrey CC, Kunsman J, Cullen DJ, Brewster DC. *Anesthesiol* 1983;58:462-4.
- Gerson MC, Hurst JM, Hertzberg VS, Doogan PA, Cochran MB, Lim SP, McCall N, Adolph RJ. *Ann Intern Med* 1985;103:832-7.
- Gibson RS. *Ann Rev Med* 1989;40:395-410.
- Slogoff S, Keats AS. *Anesthesiol* 1985;62:107-14.
- Hollenberg M, Mangano DT, Browner WS, London MJ, Tubau JF, Tateo IM. *JAMA* 1992;268:205-9.
- Hertzner NR, Young JR, Kramer JR, Phillips DF, deWolfe VG, Ruschhaupt WF III, Beven EG. *Arch Surg* 1979;114:1336-44.

20. Tomatis LA, Fierens EE, Verbrugge GP. *Surg* 1972;71:429-35.
21. Hertzner NR, Beven EG, Young JR, O'Hara PJ, Ruschhaupt WF III, Graor RA, deWolfe VG, Maljovec LC. *Ann Surg* 1984;199:223-33.
22. Raby KE, Goldman L, Creager MA, Cook EF, Weisberg MC, Whittemore AD, Selwyn AP. *N Engl J Med* 1989;321:1296-300.
23. Ouyang P, Gerstenblith G, Furman WR, Golueke PJ, Gottlieb SO. *Am J Cardiol* 1989;64:1113-6.
24. Muir AD, Reeder MK, Foex P, Ormerod OJ, Sear JW, Johnston C. *Br J Anaesth* 1991;67:373-7.
25. Cahalan MK, Litt L, Botvinick EH, Schiller NB. *Anesthesiol* 1987;66:356-72.
26. Eagle KA, Brundage BH, Chaitman BR, et al. *Circulation* 1996;93:1278-1317.
27. Fleisher LA, Barash PG. *Anesth Analg* 1992;74:586-98.
- 27a. Eagle KA, et al. *Circulation*. 2002;105:1257-67.
28. Bunt TJ. *J Vasc Surg* 1992;15:626-34.
29. Whittemore AD, Clowes AW, Hechtman HB, Mannick JA. *Annals of Surg* 1980;192:414-21.
30. Berlaak JF, Abrams JH, Gilmour IJ, O'Connor SR, Knighton DR, Cerra FB. *Ann Surg* 1991;214:289-97.
31. Pull ter Gunne AJ, Bruining HA, Obertop H. *Neth J Surg* 1990;42:113-7.
32. Smith JS, Cahalan MK, Benefield DJ, Byrd BF, Lurz FW, Shapiro WA, Roizen MF, Bouchard A, Schiller NB. *Circulation* 1985;72:1015-21.
- 32a. Poldermans D, et al. *European Heart Journal* 2001;22(15):1353-8.
33. Roizen MF. Does Choice of Anesthetic Significantly Affect Cardiovascular Surgery? In *Opioids in Anesthesia*. Edited by Estafanous FG, Boston: Butterworths; 1984.pp. 180-89.
34. Roizen MF, Beaupre PN, Alpert RA, Kremer P, Cahalan MK, Shiller N, Sohn YJ, Cronnelly R, Lurz FW, Ehrenfeld WK, et al. *J Vasc Surg* 1984;1:300-5.
35. Silverstein PR, et al. *Anesthesiol* 1979;50:462-6.
36. Attia RR, Murphy JD, Snider M, Lappas DG, Darling RC, Lowenstein E. *Circulation* 1976;53:961-5.
37. Meloche R, Pottecher T, Audet J, Dufresne O, LePage C. *Can Anaesth Soc J* 1977;24:20-34.
38. Carroll RM, Laravuso RB, Schauble JF. *Arch Surg* 1976;111:740-3.
39. Gelman S. *Anesthesiology* 1995;82:1026-60.
40. Ling E, et al. *Anesthesiology* 2000;93:1115-22.
41. Gilbert TB, et al. *J Cardvasc Pharm Therp* 2001;6:31-36.
42. Gottlieb A, Banoub M, Sprung J, Levy PJ, Beven M, et al (Accompanied by Editorial comments). *J of Cardiothor Vasc Anesth* 1998;12(5):501-05.
43. Gottlieb A, Sprung J, Mehrabi J, Antony DG, Bhandari G, Greenberg RK. *Anesth Analg* 2001;92:S26.
44. Gottlieb A, Westbrook NB, Marks TN, Mascha EJ, Ouriel K. *Anesthesia for Endovascular Stent vs Open Aortic Reconstruction - An Update Outcome Study* *Anesthesiology* 2004;101:A227.

