Review Article

Haemodynamic Effects of the Use of the Intraaortic Balloon Pump

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Treatment with the intraaortic balloon pump (IABP) is the most common form of mechanical support for the failing heart. The augmentation of diastolic pressure during balloon inflation contributes to the coronary circulation. According to Christenson et al,[1] the presystolic deflation of the balloon reduces the resistance to systolic output. Consequently, the myocardial work is reduced. The overall effect of the IABP therapy is an increase in the myocardial oxygen supply/demand ratio and thus in endocardial viability.[2]

Development of the concept of the IABP

Kantrowitz[3] conceived of the idea of altering the temporal course of pressure events during a cardiac cycle. An external impulse was applied to the aorta by "wrapping" the hemidiaphragm around the distal part of the thoracic aorta. The "trained" muscle would contract during each diastole. The diastolic pressure showed a significant increase in comparison with control studies.

At the same time, other researchers[4-6] used the femoral artery for the removal of blood during systole and its restoration during diastole. A haemodynamic effect was observed, but a practical application of the technique was not possible.

Moulopoulos et el[7] performed studies with an IABP. A latex balloon was tied around the tip of a polyurethane catheter with multiple side holes. The distal end of the catheter was blocked, so that the balloon could be inflated and deflated through the side holes. The catheter and balloon formed a closed system that was filled with carbon dioxide. Periodically, using a piston like effect, the carbon dioxide was expelled so as to inflate the balloon. The pulse length and delay after the R wave on the ECG were preadjusted so that the latex balloon would inflate during diastole and remain deflated during systole. Testing their pump on a canine aorta, the investigators concluded that it was possible to increase the diastolic blood flow in the arterial system and to reduce blood pressure at the end of the diastolic phase.

In 1970 Mundth et al[8] reported the case of a patient who suffered cardiogenic shock following an infarction and was stabilised with an IABP. The patient subsequently underwent coronary reperfusion and with the support of the pump survived. This was the first report, and the IABP went on to find wider application, including support of heart failure following surgical coronary reperfusion.

At the beginning of 1971 Krakauer et al[9] reported their experience from use of the IABP in 30 cases who were treated for cardiogenic shock due to acute myocardial infarction. They concluded that the group of patients who were given IABP support in the early stages after the onset of the shock had a significantly better prognosis.

In 1973, two different groups[10,11] reported the successful use of the IABP technique in patients who could not be weaned from cardiopulmonary bypass. This ushered in a new era in the perioperative care of patients with ventricular dysfunction.

Continuing development led to the discovery of percutaneous catheter introduction.[12,13] This completely transformed the field of application of the IABP, because of the potential proliferation in the indications for use of the device in different subgroups of patients with advanced coronary artery disease, who were not expected to be able to tolerate conventional medical manipulations (Figure 1).

Function of the IABP

Following the principle of counterpulsation, the IABP is decompressed during systole, which coincides with the QRS-T interval (the R wave always triggers deflation of the balloon). In this way balloon inflation is prevented during cardiac systole. The IABP is inflated during diastole, which corresponds to the T-P interval (Figure 2).

Haemodynamic changes during IABP therapy (Table 1)

The cardiovascular effects of the IABP are due mainly to its effect on preload and afterload.[15] From a

mechanical point of view, inflation of the balloon causes "volume displacement" resulting in a change in the coronary circulation, with a redistribution of blood flow and an alteration in oxygen consumption.[16,17]

Deflation of the balloon takes place at the end of diastole, precisely at the start of isovolumic contraction. As a result, resistance to left ventricular output is reduced, thus reducing afterload.[18]

Reduction in systolic pressure

If one compares the systolic pressures of non-augmented beats with those following beats augmented by the IABP, one concludes that IABP therapy results

Figure 2. Haemodynamic function of the balloon. A: ECG. B, C: Balloon deflation, corresponding to systole during the cardiac cycle, and inflation, corresponding to diastole. E: Aortic pressure curve during balloon function.
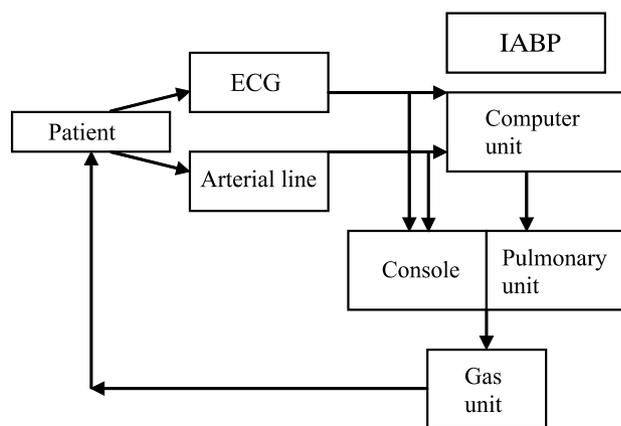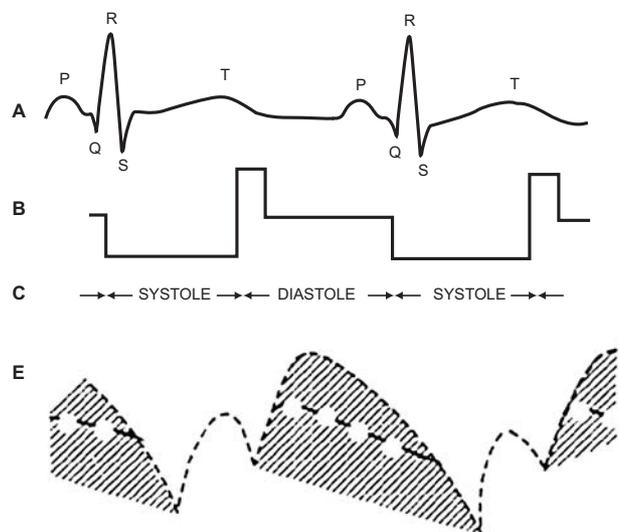

Figure 1. Description of the circuit between the patient and the intraaortic balloon pump (IABP). The IABP console includes the following: 1) A gas cylinder (usually helium, which has a theoretical advantage according to Hendrickx et al[14]); 2) a gas supply unit; 3) a monitoring system for recording the ECG and blood pressure; 4) a control unit that processes the ECG and generates a triggering signal. The latter unit is used for the timing of inflation and deflation of the balloon via activation of the valve unit, either opening the valve to supply gas or closing it to interrupt the gas flow.

Table 1. Haemodynamic effects of the intraaortic balloon pump.

Reduction in blood pressure
Fall in end-diastolic aortic pressure
Shortening of the isometric phase of left ventricular contraction
Reduction in left ventricular wall stress
Increase in left ventricular ejection fraction
Reduction of preload/afterload
Increase in DPTI/TTI ratio
Improvement of coronary flow
During haemorrhagic shock, an improvement in vasoregulatory control of splanchnic blood flow

DPTI – diastolic pressure time index; TTI – tension time index

in a reduction in systolic pressure of up to 10%,[19,20] implying a reduction in afterload.

Fall in presystolic (end-diastolic) aortic pressure

During IABP therapy the end-diastolic aortic pressure is reduced by up to 30%, implying systolic unloading.

Shortening of the isometric phase of left ventricular contraction

During IABP function the aortic valve opens prematurely, thus shortening the isometric phase of left ventricular contraction. This interval is proportionally related with myocardial oxygen consumption.[21]

Reduction of left ventricular wall tension and rate of increase in left ventricular pressure (dp/dt)

According to Urschel et al,[22] the rate of increase in left ventricular pressure is reduced by IABP therapy by up to 20% in comparison with control values.

Effects on ejection fraction, cardiac output, and the Frank-Starling law for the left ventricle

An increase in left ventricular ejection fraction has been observed during IABP therapy.[23] In addition, there is an increase in cardiac output of between 0.5 and 1.0 L/min, or up to 30%.[24,25] The Frank-Starling curve is also affected by IAPB therapy.[26] The curve is displaced towards the left, indicating an improvement in left ventricular function.

Reduction in preload

The left ventricular diastolic volume is reduced because of systolic unloading. Also, the relation between the change in left ventricular diastolic pressure and the change in left ventricular diastolic volume shows a tendency towards lower values,[27] which translates into an improvement in left ventricular compliance.

IABP and myocardial oxygen supply and demand

The effect of the IABP in improving myocardial oxygen supply may best be appreciated via an examination of the diastolic pressure time index (DPTI) and the tension time index (TTI) (Figure 3).

The DPTI reflects the diastolic and subendocardial blood flow. It depends on the aortic diastolic pressure, the left ventricular end-diastolic pressure, and the duration of diastole. The DPTI is increased by the IABP because of the increase in diastolic blood pressure and the reduction in end-diastolic pressure.

The TTI corresponds to the region below the left ventricular systolic pressure curve, which reflects myocardial oxygen demand. The TTI is reduced during balloon deflation because of the reduction in systolic blood pressure.

The ratio DPTI/TTI reflects the relation between myocardial oxygen supply and consumption and is called the endocardial viability ratio (EVR). The maximum value of 1.0 is equivalent to a normal balance between oxygen supply and demand. An EVR <0.7 suggests severe myocardial ischaemia.

Nanas et al[28] and Bolooki et al[29] reported that use of the IABP resulted in an increase in EVR. The EVR index is useful as a criterion for deciding on the
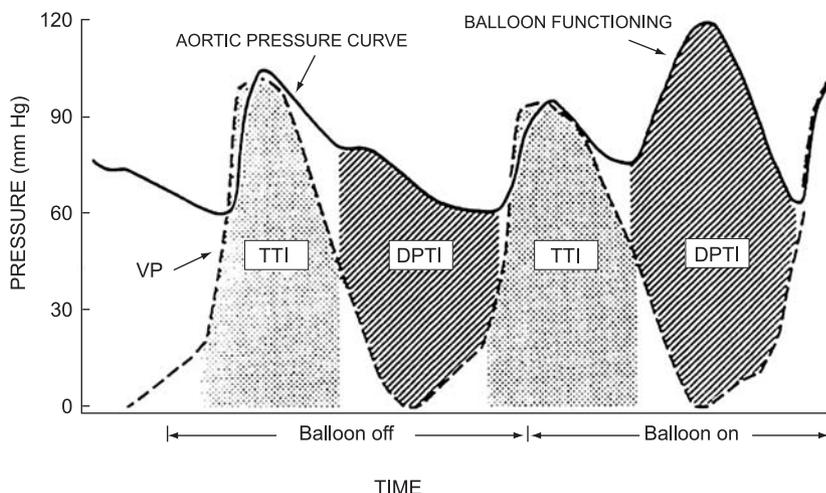

Figure 3. Effect of the intraaortic balloon pump on myocardial oxygen supply (DPTI) and demand (TTI). DPTI - diastolic pressure time index; TTI - tension time index

prompt use of the IABP method in cases of intraoperative heart failure.

IABP and perfusion of the coronary circulation

Dilatation of the balloon causes displacement of the surrounding blood, increasing coronary perfusion via an increase in diastolic pressure and in the diastolic perfusion pressure gradient.[30,31]

There are a number of animal studies that evaluated the contribution of the IABP to myocardial perfusion. The results were varied. Kern et al[32] reported that in animals with normal systemic blood pressure the intraaortic pump reduced myocardial oxygen consumption without causing a significant change in the overall coronary flow. In ischaemic animal models with low systemic blood pressure, the myocardial oxygen consumption depended on coronary flow. The IABP method had a small effect on the perfusion of myocardial regions supplied by obstructed coronary vessels.

Gewirtz et al[33] concluded that during IABP therapy the blood flow peripheral to the stenosis remained unchanged.

McDonald et al[34] found that the IABP caused an increase in pre-stenotic as opposed to post-stenotic flow.

However, Folland et al[35] reported conflicting findings. They observed relief of symptoms of angina in a population with coronary artery disease and severe aortic disease and concluded that the improvement in coronary flow must have been due to the IABP.

Ohman et al[36] described IABP therapy as "artificial myoconservation". The investigators concluded that the action of the balloon stimulates the collateral circulation in the region around the ischaemic core of the myocardial lesion. Fuchs et al[37] agreed that the collateral circulation is strengthened during the increase in diastolic pressure. Kern et al[38] measured the intracoronary flow velocity during catheterisation in 12 patients who were undergoing IABP treatment, recording the velocity time integral of diastolic flow. The greatest increase was seen in patients with baseline systolic pressure <90 mmHg. The investigators concluded that the IABP method increased the proximal coronary flow velocity via a doubling of the velocity time integral of diastolic flow.

During IABP therapy the end-diastolic pressure is reduced. By Laplace's Law, the fall in end-diastolic pressure will reduce the wall stress caused before the opening of the aortic valve, thus reducing myocardial oxygen demand.

Akyurekli et al[24] carried out a study in order to determine the effects of IABP therapy on systolic unloading, independently of the increase in diastolic pressure. This study involved using the pump in dogs whose coronary arteries were perfused by an extracorporeal source. The perfusion pressure was reduced, causing acute heart failure. When the IABP was applied, the systolic unloading (reduction in left ventricular systolic pressure) was apparent under normotensive conditions, but not under hypotensive conditions (coronary perfusion pressure <80 mmHg). During hypotension aortic compliance increases, causing expansion of the aortic wall during balloon dilatation, so that there is no displacement of blood volume. Furthermore, at the instant of balloon deflation aortic pressure is not reduced. Therefore, there is no decrease in either static work or myocardial oxygen consumption.

IABP and the peripheral circulation

Peripheral blood flow is determined by pressure, resistance, length, and internal friction. Balloon inflation during diastole increases blood pressure, with a consequent increase in arteriovenous pressure gradient and an improvement in flow. Also, balloon inflation during diastole causes displacement of the stroke volume, and hence activation of the aortic baroreceptors. This in turn blocks the medullary vasoconstrictive reflex. The peripheral resistance is reduced, thus improving blood flow.

Landreneau et al[39] studied the effect of IABP augmentation on splanchnic blood flow during prolonged haemorrhagic shock. They concluded that during haemorrhagic shock the IABP appears to im-

Table 2. Variables that affect the increase in diastolic pressure.

Balloon position: the nearer to the aortic valve, the greater the increase in diastolic pressure.

Volume: when the balloon volume equals the stroke volume, augmentation of diastolic pressure is maximised.

Diameter and obstructive capability of the balloon: the greatest possible increase is seen during complete aortic obstruction.

Optimal regulation of balloon gas flow and timing.

Stroke volume: if the stroke volume is <25 ml, no significant diastolic increase should be expected.

Blood pressure: the importance of aortic elasticity is shown by the fact that aortic volume doubles when mean blood pressure increases from 30 mmHg to a normal 90 mmHg.

prove the vasokinetic control of splanchnic blood flow, eliminating the phenomenon of hyperaemic reperfusion and thus resulting in less reperfusion injury.

The effects of the IABP on postoperative renal function in humans were studied by Hilberman et al,[40] who showed a postoperative improvement in human renal perfusion during counterpulsation.

Swartz et al[41] studied the effect of perirenal balloon placement on renal blood flow. They found a mean reduction of 66% while the balloon was in the renal site.

Table 2 shows the variables that influence the increase in diastolic pressure during balloon dilatation, according to Weber and Janicki.[15] The importance of aortic elasticity was shown by Kantrowitz et al,[42] who reported that the balloon is restricted by the blood volume contained within the aorta shortly before inflation, and that a further increase in the pump volume results only in swelling of the aorta and not in effective blood distribution.

Conclusions

An increase in the use of the IABP has been reported by many groups[43,44] and is due to its relative ease of placement via a percutaneous route, to the "freedom" of the indications, as well as to the increasing age of the population in recent years, which has led to a growth in the number of elderly patients with multi-vessel disease and a defective left ventricle.

To summarise, the use of the IABP is justified because an increase in diastolic pressure during balloon inflation augments the coronary circulation. Furthermore, pre-systolic deflation of the balloon reduces the resistance to systolic output; thus the myocardial work decreases and the EVR increases.

The efficacy of the IABP is reflected by the positive outcome of the large number of patients who are weaned from the device. The success rate is higher for patients in whom the device is deployed early, reflecting the reversible nature of ischaemic pathology and the positive contribution of the IABP to preventing the cascade of complications of ischaemic heart disease.

References

1. Christenson JT, Cohen M, Ferguson JJ, et al: Trends in intraaortic balloon counterpulsation complications and outcomes in cardiac surgery. Ann Thorac Surg 2002; 74: 1086-1090; discussion 1090-1091.

2. Ferrari G, Gorczynska V, Mimmo R, et al: IABP assistance: a test bench for the analysis of its effects on ventricular energetics and hemodynamics. Int J Artif Organs 2001; 24: 274-280.

3. Kantrowitz A: Origins of intraaortic balloon pumping. Ann Thorac Surg 1990; 50: 672-674.

4. Maccioli GA, Lucas WJ, Norfleet EA, et al: The intra-aortic balloon pump: a review, J Cardiothorac Anesth 1988;2:365-373.

5. Birtwell WC, Soroff HS, Well M, et al: Assisted circulation. An improved method for counterpulsation, Trans Am Soc Artif Intern Organs 1962; 8: 35-42.

6. Clauss RH, Birtwell WC, Albertal G, et al: Assisted circulation I. The arterial counterpulsator. J Cardiovasc Surg 1961; 41: 447-458.

7. Moulopoulos SD, Topaz S, Kolff WJ: Diastolic balloon pumping (with carbon dioxide) in the aorta—a mechanical assistance to the failing heart. Am Heart J 1962; 63: 669-675.

8. Mundth ED, Yurchak PM, Buckley MJ, et al: Circulatory assistance and emergency direct artery surgery for shock complicating acute infarction, N Engl J Med 1970; 283: 1382-1384.

9. Krakauer J, Rosenbaum A, Freed P, Jaron D, Kantrowitz A: Clinical management ancillary to phase-shift balloon pumping in cardiogenic shock. Am J Cardiol 1971; 27: 123-128.

10. Buckley MJ, Craver JM, Gold HK et al: Intra-aortic balloon pump assist for cardiogenic shock after cardiopulmonary bypass. Circulation 1973; 48: III-90-94.

11. Housman LB, Bernstein EF, Braunwald NS, Dilley RB: Counterpulsation for intraoperative cardiogenic shock. Successful use of intra-aortic balloon. JAMA 1973;224:1131-1133.

12. Bregman D, Casarella WJ: Percutaneous intraaortic balloon pumping: initial clinical experience. Ann Thorac Surg 1980;29:153-155.

13. Subramanian VA, Goldstein JE, Sos TA, McCabe JC, Hoover EA, Gay WA Jr: Preliminary clinical experience with percutaneous intra-aortic balloon pumping. Circulation 1980; 62: I123-129.

14. Hendrickx HH, Berkowitz D: The differences between intra-aortic balloon pumps and their use. Crit Care Med 1982; 10:796-797. (Letter to the editor).

15. Weber KT, Janicki JS: Intra-aortic balloon counterpulsation. A review of physiology principles, clinical results and device safety. Ann Thorac Surg 1974; 17: 602-636.

16. Kantrowitz A: Percutaneous intra-aortic balloon counterpulsation. Crit Care Clin 1992; 8: 819-837.

17. Williams DO, Korr KS, Gewirtz H, et al: The effect of intraaortic balloon counterpulsation on regional myocardial blood flow and oxygen consumption in the presence of coronary artery stenosis in patients with unstable angina. Circulation 1982; 66:593-597.

18. Folland ED, Kemper AJ, Khuri SF, et al: Intra-aortic balloon counterpulsation as a temporary support measure in decompensated critical aortic stenosis, J Am Coll Cardiol 1985; 5: 711-716.

19. Schottler M, Schaefer J, Schwarzkorpf HJ, et al: Experimentally induced changes of arterial mean and aortic opening pressure by controlled variation of diastolic augmentation. Basic Res Cardiol 1974; 69: 59-67.

20. Feola M, Haiderer O, Kennedy JG: Intraaortic balloon pumping (IABP) at different levels of experimental acute left ventricular failure. Chest 1971; 59: 68-76.

21. Mullins CB, Sugg WL, Kennelly BM, Jones DC, Mitchell JH: Effect of arterial counterpulsation on left ventricular volume and pressure. Am. J Physiol 1971; 220: 694-698.

22. Urschel CW, Eber L, Forrester J, Matloff J, Carpenter R, Sonnenblick E: Alteration of mechanical performance of the ventricle by intraaortic balloon counterpulsation. Am J Cardiol 1970; 25: 546-551.

23. Maddoux G, Pappas G, Jenkins M, et al: Effect of pulsatile and nonpulsatile flow during cardiopulmonary bypass on left ventricular ejection fraction early after aortocoronary bypass surgery. Am J Cardiol 1976; 37: 1000-1006.

24. Akyurekli Y, Taichman GC, Keon WJ: Effectiveness of intra-aortic balloon counterpulsation and systolic unloading. Can J Surg 1980; 23: 122-126.

25. Gabauer I, Nosalova V, Okalicanyl J: Distribution of cardiac output following 2-hour intraaortic balloon counterpulsation. Bratisl Lek Listy 1980; 73: 26-35.

26. Nichols AB, Pohost GM, Gold HK, et al: Left ventricular function during intraaortic balloon pumping assessed by multigated cardiac blood pool imaging. Circulation 1978; 58(3 Pt 2): I176-183.

27. Diamond G, Forrester JS: Effect of coronary artery disease and acute myocardial infarction on left ventricular compliance in man. Circulation. 1972; 45: 11-19.

28. Nanas JN, Nanas SN, Kontoyannis DA, et al: Myocardial salvage by the use of reperfusion and intraaortic balloon pump: experimental study. Ann Thorac Surg 1996; 61: 629-634.

29. Bolooki H: Clinical application of intra-aortic balloon pump, 2nd ed. Futura Publishing, New York, 1984: pp 73.

30. Port SC, Patel S, Schmidt DH: Effects of intraaortic balloon counterpulsation on myocardial blood flow in patients with severe coronary artery disease, J Am Coll Cardiol 1984; 3: 1367-1374.

31. Williams DO, Korr KS, Gewirtz H, Most AS: The effect of intraaortic balloon counterpulsation on regional myocardial blood flow and oxygen consumption in the presence of coronary artery stenosis in patients with unstable angina. Circulation 1982; 66: 593-597.

32. Kern MJ, Aguirre FV, Caraccido EA, et al: Hemodynamic effects of new intra-aortic balloon counterpulsation timing methods in patients: a multicenter evaluation. Am Heart J 1999; 137: 1129-1136.

33. Berne RM, Levy MN: Cardiovascular physiology, 6th ed. (chap. 8). Mosby-Year Book, St. Louis, 1992: pp 225.

34. MacDonald RG, Hill JA, Feldman RL: Failure of intra-aortic balloon counterpulsation to augment distal coronary perfusion pressure during percutaneous transluminal coronary angioplasty, Am J Cardiol 1987; 59: 359-361.

35. Folland ED, Kemper AJ, Khuri SF, Josa M, Parisi AF: Intra-aortic balloon counterpulsation as a temporary support measure in decompensated critical aortic stenosis. J Am Coll Cardiol 1985; 5: 711-716.

36. Ohman EM, George BS, White CJ, et al: Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocadial infarction. Results of a randomized trial. The Randomized IABP Study Group. Circulation 1994; 90: 792-299.

37. Fuchs RM, Brin KP, Brinker JA, Guzman PA, Heuser RR, Yin FC: Augmentation of regional coronary blood flow by intra-aortic balloon counterpulsation in patients with unstable angina. Circulation 1983; 68: 117-123.

38. Kern MJ, Aguirre F, Penick D, et al: Enhanced intracoronary flow velocity during intra-aortic balloon counterpulsation in patients with coronary artery disease. Circulation 1991: 84 (Suppl II): II-485 (abstract).

39. Landreneau R, Horton J, Cochran R, et al: Splanchnic blood flow response to intraaortic balloon pump assist of hemorrhagic shock. J Surg Res 1991: 51; 281-287.

40. Hilberman M, Derby GC, Spencer RJ, Stinson EB: Effect of the intra-aortic balloon pump upon postoperative renal function in man. Crit Care Med 1981; 9: 85-89.

41. Swartz MT, Sakamoto T, Arai H, et al: Effects of intraaortic balloon position on renal artery blood flow, Ann Thorac Surg 1992; 53: 604-610.

42. Kantrowitz A: Cardiac assistance by the intra-aortic balloon pump. 2nd Medical Physics Conference, Boston, 1969 (abstract).

43. Urban PM, Freedman RJ, Ohman EM, et al: In-hospital mortality associated with the use of IAB counterpulsation. Am J Cardiol 2004; 94: 181-185.

44. Stone GW, Ohman EM, Miller MF, et al: Contemporary utilization and outcomes of intra-aortic balloon counterpulsation in acute myocardial infarction: the benchmark registry. J Am Coll Cardiol 2003; 41: 1940-1945.