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The intra-aortic balloon pump in heart failure management: Implications for nursing practice

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Summary Management of acute heart failure is an important consideration in critical care. Mechanical support of the failing heart is crucial for improving health outcomes. The most common Australasian application of intra-aortic balloon counterpulsation (IABP) is in the setting of cardiogenic shock. High end users of IABP (>37/annum) demonstrate significantly lower mortality for cardiogenic shock managed with IABP ($p < 0.001$) in contrast to hospitals which employ limited IABP (<4/annum). This underscores the importance of proficiency in managing the patient receiving IABP support. Nurses play a crucial role in caring for patients with acute heart failure. This paper summarises care considerations for management of the IABP.

Introduction

Management of acute heart failure is an important consideration in critical care units. Following the initial application to practice in 1968, intra-aortic balloon counterpulsation (IABP) has become the most widely used mechanical support in the assistance of the failing heart. Australasian application of IABP is most predominant in the setting of cardiogenic shock.[1] Of interest, high end users of IABP (>37/annum) demonstrate significantly lower mortality for cardiogenic shock managed with IABP ($p < 0.001$) in contrast to hospitals which employ limited IABP (<4/annum). High volume facilities experience up to 150 fewer deaths per 1000 IABP recipients.[2] This marked reduction in mortality sug-

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gests IABP knowledge and modality proficiency to contribute toward improved IABP outcome.[3] Nurses play a crucial role in caring for people receiving IABP therapy. This paper describes the implications for nursing practice which result from IABP insertion, management and weaning from the device.

Nurses who care for patients managed with IABP require a knowledge of the mechanisms and actions of this therapeutic device. As well as addressing IABP physiology, benefits, potential complications and safety considerations, this paper discusses the importance of monitoring cardiac function and undertaking regular comprehensive patient assessment—core nursing responsibilities in the management of IABP. How assessment must be informed by an understanding of IABP complications and safety issues will be addressed, as will IABP timing and how this can maximise impaired cardiac function. Finally, discussion will describe patients ready to be weaned from IABP with consideration given to understanding weaning strategies and their implications for nursing practice.

Physiologic effects of intra-aortic balloon counterpulsation

The principal characteristic of acute heart failure is inadequate contractile force of the myocardium resulting in a failure to maintain adequate cardiac output.[4] This reduction in contractility is a product of inadequate myocardial oxygenation and increasing cardiac workload. The primary purpose of IABP is the support of the failing heart by simultaneously increasing myocardial oxygen supply and decreasing myocardial oxygen demand.[5] This is achieved by the positioning of an intra-aortic balloon (IAB) in the descending thoracic aorta. The IAB is located immediately inferior to the origin of the left subclavian artery and superior to the renal arteries and is attached to an external drive console which inflates and deflates the IAB in synchrony with cardiac contractions.[5] The IAB is inflated at the onset of diastole when blood ceases to eject from the heart. It displaces blood volume within the descending thoracic aorta. Proximal blood is returned to the heart to oxygenate the coronary arteries as well as being distributed through the branches in the aortic arch. Blood in the distal descending aorta is circulated systemically.[6] Balloon deflation is timed to occur immediately prior to the onset of systole before the heart commences ejection. Balloon deflation leaves the aorta partially empty thus reducing afterload, maximising left ventricular ejection fraction and reducing mitral regurgitation. By augmenting coronary artery and systemic perfusion pressures, IABP improves myocardial oxygen supply and decreases myocardial oxygen consumption by reducing cardiac workload.[5,7] Understanding these physiological effects enables the nurse to effectively manage patients treated with IABP with a specific focus on expected improvements in cardiac function.

Intra-aortic balloon counterpulsation timing

To optimise cardiac function in the setting of IABP inflation of the IAB must be synchronised with the cardiac cycle. Timing of IAB inflation and deflation is achieved by choosing a trigger which accurately predicts the open and closure of the aortic valve. The preferred trigger is the electrocardiogram (ECG) tracing. Other triggers such as aortic pressure or pacing spikes may be used if necessary.[8] The ECG is the favoured trigger as the R wave provides the most accurate reference point for signalling the onset of ventricular systole and the opening of the aortic valve.[5] Even in the context of irregular and paced rhythms the R wave will offer the clearest indication of the onset of systole. The importance of a reliable trigger for balloon inflation and deflation is crucial for optimised IABP.[8–10] Ideally ECG monitoring leads are attached directly to the IABP drive console. However, an option exists for the drive console to 'slave' the ECG trace from the cardiac monitor—as such, it is not unusual for a patient managed with IABP to have two sets of ECG leads.[8] Establishing and maintaining the quality of data from a trigger source is an important nursing responsibility.

To establish maximal IABP support careful monitoring of the aortic pressure waveform is critical. IAB inflation and deflation must be synchronous with the cardiac cycle. During systole (when blood is ejected from the left ventricle) the IAB should be deflated; during diastole (when there is no blood ejected from the left ventricle) the IAB should be inflated. The aortic pressure waveform represents systole and diastole. Systole occurs from the first point of positive pressure after the dicrotic notch (this immediately follows the lowest arterial pressure) to the highest arterial pressure and continues until the dicrotic notch (closure of the aortic valve). Diastole occurs from the advent of the dicrotic notch until the first point of positive pressure following this point. The IAB should be inflated on the dicrotic notch and deflated immediately prior to the first positive pressure following the dicrotic notch (Fig. 1). If IAB inflation and deflation is not

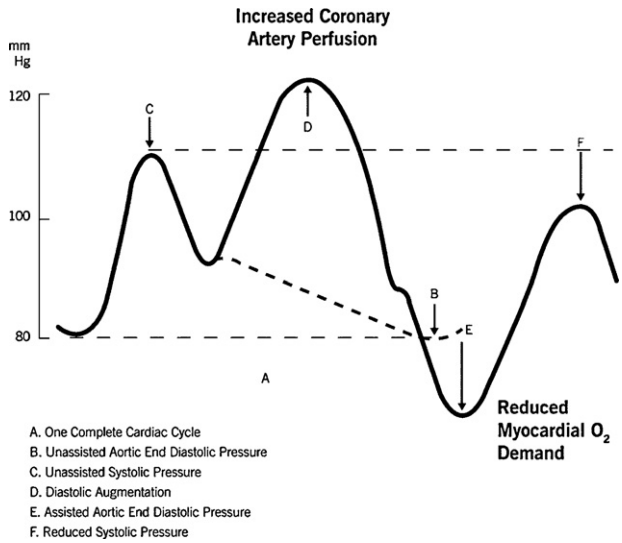

Figure 1 Schematic arterial pressure waveform illustrating correctly timed diastolic augmentation produced by intra-aortic balloon counterpulsation.

synchronised with the cardiac cycle optimal performance of IABP cannot occur. Late IAB deflation for example, would result in impedance of blood ejection from the left ventricle causing the heart to work harder and myocardial oxygen consumption to increase.[10] Fig. 2 presents waveform characteristics and physiologic effects of poorly timed IABP therapy.

Determining adequate IABP timing involves careful analysis of the pressure waveform displayed on the IABP drive console and assessment of the patient's haemodynamic response. Without synchrony, IAB inflation and deflation may increase myocardial oxygen demand and cardiac workload. The automatic timing functions on newer model IABP drive consoles facilitate timing optimisation even in the context of irregular rhythms such as atrial fibrillation. Given the variance in drive console functionality, an awareness of the timing features of the machine and model in use is important. Despite technological advance, it remains the responsibility of the nurse to maintain a reliable trigger and ensure timing is regularly checked to maximise physiologic benefit to the patient.

Observations

To optimise the physiologic benefit of IABP, minimise IABP risks, and allow cardiac support to be individualised in response to variations in heart function, patient observation must be a continual process. Thorough knowledge, continual assessment and prompt intervention are essential in determining the best patient response. Bolooki[11] advises haemodynamic variables requiring assessment to include: heart rate, arterial blood pressure, central venous pressure, pulmonary artery pressure, pulmonary capillary wedge pressure, and cardiac output. Clinical evaluation with ECG studies as well as chest X-ray may also be helpful in evaluating left ventricular function.[11] However, in practice interventions used routinely in conjunction with IABP for data acquisition fluctuate significantly.[1] To provide an accurate insight into the level of a patient's cardiac function, a combination of clinical measurements and observations would appear necessary. Presenting this global pic-

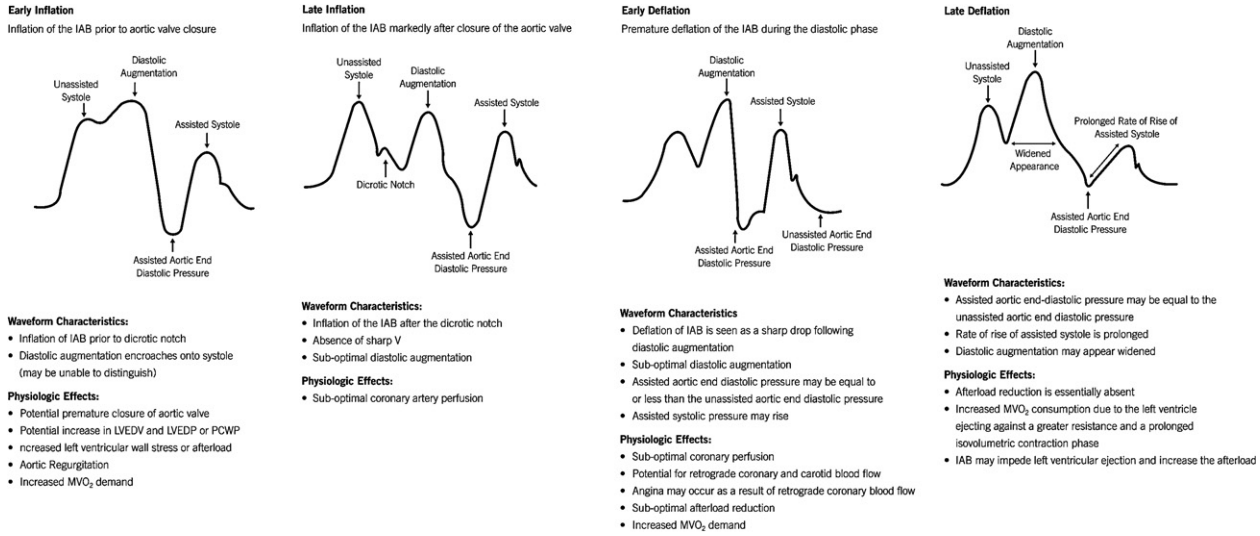

Figure 2 Schematic arterial pressure waveform illustrating incorrectly timed diastolic augmentation produced by intra-aortic balloon counterpulsation.

ture of a patient's cardiac function would generate the most effective approach to managing cardiac support.[12]

In Australasia, the primary routine intervention in the setting of IABP is a central venous catheter (used by 87% of Australasian units). Other interventions used to monitor the patient undergoing IABP are intra-arterial lines (72% of units), pulmonary artery catheters (70% of units), transthoracic echocardiography (41% of units), transoesophageal echocardiography (39% of units), continuous cardiac output monitoring (7% of units), and saturated venous oxygenation (2% of units).[1] Almost one-third of Australasian practitioners choose not to use a pulmonary artery catheter—a previous mainstay for the monitoring of IABP.[11] This wide variability in monitoring practices between institutions likely reflect the extensive cardiovascular effects of IABP and potentially the need to standardise practices to allow for benchmarking.[1] Irrespective of strategies used to monitor cardiac function, it is widely accepted that managing IABP encompasses many parameters, of which haemodynamic status is paramount.[11–14] Nurses managing IABP must be able to participate in the continual assessment of haemodynamic status.

Assessment of haemodynamic status begins with evaluation of the arterial pressure waveform. During IABP additional components exist in the pressure waveform resulting from balloon inflation and deflation and the consequent displacement of blood.[15] Normally systole is the highest pressure reached during the cardiac cycle. However, during IABP, balloon inflation at the onset of diastole (which coincides with closure of the aortic valve) displaces blood in the aorta increasing the peak pressure—this increase in pressure is called diastolic augmentation.[6,9] The IAB remains inflated throughout diastole until immediately prior to systole. Upon IAB deflation the aorta becomes partially empty. Aortic pressure will consequently drop lower than with an unassisted beat—the result is a dip in the waveform pattern called the assisted aortic end-diastolic pressure (which will result in an assisted systole).[9] Where IABP support is offered with each cardiac beat (frequency of 1:1), assisted components of the pressure waveform reflect improvements in myocardial oxygen supply and reduce myocardial oxygen consumption by decreasing cardiac workload.[5,7] These assisted pressures should be routinely documented as evidence of IABP effectiveness.

As with ECG and aortic pressure fluctuations, IABP assisted arterial pressures enable comparative analysis of cardiac function at different points in time. Any reduction in assist ratios of IAB inflation to cardiac contraction (1:2 or 1:4) requires careful assessment of the pressure waveform as unassisted contractions result in increased cardiac workload.[16] If cardiac function is insufficient, the increasing workload owing to a reduction in frequency of inflation will result in reduced ejection of blood into the aorta. As a result there will be less blood in the aorta to be displaced during IAB inflation, which decreases diastolic augmentation.[9] Alternatively when cardiac function improves and the systolic pressure achieved with myocardial contraction is higher, the difference between systolic pressure and augmented diastolic pressure will narrow. Interpretation of pressure waveforms is useful in understanding cardiac function and the support provided by IABP. However, a more comprehensive clinical picture regarding the degree of recovery or deterioration in cardiac function requires evaluation of other body systems.[12]

Cardiac function impacts widely on physiological processes throughout the body.[4] Factors other than mean arterial pressure considered to reflect cardiac function have been noted to include: heart rate, lung function, lactate levels, renal function, and metabolic measures.[1,17] Trends in respiratory assessment data can reflect improvements in cardiac function. A key factor in the development of cardiogenic pulmonary oedema is poor systolic ejection from the left ventricle. Assisted end-diastolic pressure serves to improve ventricular ejection and it can be expected that this will lead to reduced pulmonary congestion. Improving oxygen requirements, adventitious breath sounds, air entry and respiratory rate can all be considered useful measures of reduced congestion reflecting improved cardiac function.[12] Renal function may also be considered a good measure of cardiac function due to susceptibility of kidneys to low output states.[18] Where cardiac output is augmented by IABP, improvements in renal function such as improved urine output may be observed. It is important for nurses to consider a multi-organ approach in monitoring the effects of IABP.

Complications

The importance of clinician exposure to IABP and experience with its use should not be underestimated.[19] Mortality[2] and complications[20] have both been found to decrease in institutions with higher insertion rates. Mortality rates for patients requiring IABP are generally high and may increase if insertion is delayed.[3,20–22] However, mortality in the IABP cohort can largely be attributed to the acuity of this patient group,

underlying aetiology of heart failure and other co-morbidities.[3] It is rare for complications of IABP itself to directly contribute to mortality. Analysis of 669 insertions at the Prince Charles Hospital between 1994 and 2004 identified no mortality that could be directly attributed to IABP.[20] Recent literature suggests that overall IABP complications are low and decreasing overtime despite increasing application of IABP in clinical practice.[19,23–27] However, the observations related to procedural outcomes relating to volume underscores the importance of considering development and assessment of nursing competencies and undertaking regular training to ensure competencies are maintained in units where IABP is used infrequently.

Access site bleeding, limb ischemia, and balloon failure (IAB rupture or entrapment) [19,20,23–25,27] account for most reported complications.[20,28] Access site bleeding is the most common IABP complication. Bleeding is best managed with a gentle compressive bandage or primary suturing at the access site.[28,29] Excessive pressure in attempting to control bleeding should be avoided as this potentially narrows the arterial lumen reducing peripheral blood flow and increasing the risk of limb ischemia. Limb ischemia is most likely in patients with existing peripheral vascular disease, diabetes mellitus and those whose IAB insertion is sheathed.[30] Sheaths contribute to vascular complications even in patients without peripheral vascular disease and sheathless insertion is clinically indicated in most patients.[29] Recent technological advances leading to smaller catheter diameters may contribute to lower vascular complication rates.[23] The impact of the new seven French catheter may further contribute to reducing the risk of limb ischemia.[31] Most cases of limb ischemia occur early after balloon placement.[23] In any case the risk of ischemia exists for all patients and regular assessment of peripheral circulation is therefore mandatory. Checking pedal pulse strength, with pedal colour, warmth, movement, and sensation frequently enables the early detection of complications. Vascular assessment should include both legs and arms due to the possibility of IAB migration and the consequent occlusion of the subclavian artery.[28] Risk of ischemia is not isolated to limbs. Severe ischemic outcomes such as paralysis have also been reported.[28] Low urine output may also reflect distal migration of the balloon with subsequent occlusion of renal arteries. Lactate levels should also be monitored as a measure of tissue perfusion.[17] Patients who experience limb ischemia as a result of IABP require device removal. Despite removing the IABP, surgical intervention is still required for many of these patients.[29]

While repeated IAB inflation causes platelet damage, evidence suggests routine anticoagulation to be unnecessary.[32] Despite the damage to platelets a clotting risk still exists if the IAB remains stationary for any period of time. In the event of a cardiac arrest the balloon mode should be set to flutter to avoid balloon stasis.[33] In addition to platelet damage, the majority of IABP recipients experience a decrease in both red cell count and platelet count to the extent where IABP duration greater than 5 days may require blood transfusion.[11] Full blood count should me monitored daily and close observation of the insertion site undertaken. Early detection and rapid intervention are the best means to reduce the incidence of serious progression of any IABP complication.[32]

Weaning intra-aortic balloon counterpulsation

Given the level of cardiac compromise necessitating IABP application, mechanical ventilation and pharmalogical assistance are frequently employed simultaneously to further assist the ailing heart. Maximal IABP assistance consists full IAB inflation at the onset of diastole following each cardiac contraction.[34] As cardiac function improves support can be reduced. Withdrawal of IABP support, while progressive, is reliant on the degree of recovery in cardiac function and must be considered in the context of other support strategies. In practice the sequence for withdrawing cardiovascular supports such as mechanical ventilation, inotropes, or IABP is variable.[1] Evidence supports the view that inotropic support should be minimised or withdrawn prior to the withdrawal of IABP support.[11,14,15,34–37] Drugs such as adrenaline increase the work of the heart. Therefore by reducing the level of pharmalogical support, myocardial oxygen demand can be reduced.[37] Additionally, minimising drugs which support the heart allows an escalation of pharmacologic therapy following IABP withdrawal if the patient should deteriorate. Increasing pharmacologic support is an easier course of action than the reintroduction of IABP. Moving toward less aggressive drugs such dopamine or dobutamine ensures attempts to withdraw IABP are not made until there is evidence that cardiac function is improving.[11,14] A recent review of evidence concluded minimisation of pharmacological assistance should occur before considering any degree of withdrawal of IABP support.[34] However, Australasian data reveals an inconsistency with only 26% of intensive care units clearly identifying

pharmacological weaning as a strategy preceding reduction in IABP support and a further 21% having no clear sequence for withdrawing support.[1] Again this underscores the importance of evidence-based guidelines in promoting best practice and benchmarking outcomes.

The withdrawal of IABP support may be either by frequency weaning or volume weaning. In the absence of direct evidence to support either option, both are offered as justifiable options for the withdrawal of IABP support. [11,13,14,35,36,38] The use of frequency weaning holds clinical appeal in Australasia with 61% of intensive care units surveyed identifying this as their sole strategy [1]. Frequency reduction weaning involves the reduction in the total amount of assisted cardiac contractions from full assistance of 1:1 to partial assistance (IABP assistance less than every cardiac contraction). Initially this reduction is from 1:1 to 1:2 (IABP assistance of every second cardiac contraction) and may progress to 1:4.[34] Frequency reduction exposes the heart to large variations in afterload, impacting upon the amount of work the heart is expected to perform[13] and fails to increase cardiac vein flow.[38] Additionally, examination of haemodynamic outcomes of weaning IABP in heart failure found frequency reduction to result in greater haemodynamic suppression when compared with volume weaning.[39] When considering the patient's haemodynamic status, any reduction in IAB frequency could be the equivalent of complete IABP withdrawal. As such, the patient should be monitored carefully for cardiac decompensation.

Volume weaning may be seen as a more physiological approach to weaning IABP. The reduction of IAB volume will gradually increase cardiac workload; it has also been demonstrated that larger catheter inflation volumes may further augment cardiac output.[40] Moreover, diastolic pressures augmented with IABP result in the redistribution of coronary blood flow toward ischemic areas of the myocardium.[16,41] Considering arguments for frequency weaning and volume weaning, volume weaning would appear to be the method of choice when initiating the withdrawal of IABP support. Importantly, however, irrespective of the weaning strategy employed, having a clear plan for weaning is important. Intensive care units with a documented weaning policy demonstrate lower IABP reinsertion rates than those units without a weaning policy ($p = 0.06$).[1] This may reflect the value of carefully establishing a patient's readiness to wean and carefully monitoring physiologic markers of cardiac function.[14]

Considering patient response to IABP at all times is the key implication for nursing practice. The nurse must be constantly evaluating the patient response to decreasing IABP support with a view to identifying early cardiac decompensation.[12] With significant variation in IABP weaning strategies it is important for nurses to understand all current approaches and their implications. Strategies to standardise processes through evidence-based guidelines and consensus methods should be considered.

Conclusion

To achieve the best possible outcome for a patient managed with IABP nursing and medical staff require specialised skills. When caring for a patient managed with IABP the nurse must continually assess and measure the often subtle changes in patient condition and this requires expert knowledge of the cardiovascular system, therapeutic effects of IABP and potential adverse events. The heterogeneity of practice patterns across institutions underscores the importance of health professionals working together to collaboratively develop guidelines that promote best practice and allow benchmarking and monitoring of outcomes across centres. The lower mortality rates in high volume centres demonstrates the importance of ensuring maintenance of skills and competencies in IABP management. Developing and regularly assessing competencies through simulation techniques may be a strategy to ensure equitable outcomes for individuals receiving IABP.

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References

1. Lewis PA, Mullany D, Courtney M, Coyer F. Australasian trends in intraaortic balloon counterpulsation weaning: results of a postal survey. *Critical Care and Resuscitation* 2006;**8**(4):361—7.
2. Chen EW, Canto JG, Parsons LS, et al. Relation between hospital intra-aortic balloon counterpulsation volume and mortality in acute myocardial infarction complicated by cardiogenic shock. *Circulation* 2003;**108**(8):951—7.
3. Meco M, Gramegna G, Yassini A, et al. Mortality and morbidity from intra-aortic balloon pumps: risk analysis. *Journal of Cardiovascular Surgery* 2002;**43**(1):17—23.
4. Berne R, Levy M, Koeppen B, Stanton B. *Physiology*. 5th ed. St. Louis: Mosby; 2004.
5. Overwalder P. Intra aortic balloon pump (IABP) counterpulsation [Web document]. *The Internet Journal*

of Thoracic and Cardiovascular Surgery 1999;**2**(2), http://www.ispub.com/journals/IJTCVS/Vol2N/iabp.html.
6. Ryan E, Foster E. Augmentation of coronary blood flow with intra-aortic balloon pump counter-pulsation. *American Heart Association Inc* 2000;**102**(3):364—5.
7. Bolooki H. Emergency cardiac procedures in patients in cardiogenic shock due to complications of coronary artery disease. *Circulation* 1989;**79**(1):137—48.
8. Tremper RS. Intra-aortic balloon pump therapy—a primer for perioperative nurses. *AORN Journal* 2006;**84**(1):34—44.
9. Metules T. IABP therapy: getting patients treatment fast. *RN* 2003;**66**(5):56—62.
10. Stamatis S, Spadoni S. Getting to the heart of IABP therapy. *RN* 1997;**60**(1):38.
11. Bolooki H. *Clinical application of the intraaortic balloon pump*. 3rd ed. New York: Futura Publishing Company Inc.; 1998.
12. Lewis PA. *Identification of early cardiac decompensation and the management of intraaortic balloon counterpulsation weaning*. Brisbane: Queensland University of Technology; 2007.
13. Kantrowitz A, Cardona RR, Freed PS. Weaning from the intraaortic balloon pump. In: Quaal S, editor. *Comprehensive intraortic balloon counterpulsation*. Sydney: Mosby; 1993.
14. Krau SD. Successfully weaning the intra-aortic balloon pump patient: an algorithm. *Dimensions of Critical Care Nursing* 1999;**18**(3):2—11.
15. Quaal SJ, editor. *Comprehensive intraaortic balloon counterpulsation*. 2nd ed. St. Louis: Mosby; 1993.
16. Gill C, Wechsler A, Newman G, Oldham H. Augmentation and redistribution of myocardial blood flow during acute ischemia by intra-aortic balloon pumping. *The Annals of Thoracic Surgery* 1975;**16**:445—53.
17. Davies A, Bellomo R, Raman J, et al. High lactate predicts the failure of intraaortic balloon pumpin after surgery. *Annals of Thoracic Surgery* 2001;**71**:1415—20.
18. Huether SE, McCance KL, editors. *Understanding Pathophysiology*. 4th ed. St. Louis: Mosby Elsevier; 2008.
19. Elahi MM, Chetty GK, Kirke R, et al. Complications related to intra-aortic balloon pump in cardiac surgery: a decade later. *Eurpoean Journal of Endovascular Surgery* 2005;**29**:591—4.
20. Lewis PA, Mullany DV, Townsend S, et al. Trends in intra-aortic balloon counterpulsation: comparison of a 669 record Australian dataset with the multinational Benchmark Counterpulsation Outcomes Registry. *Anaesthesia and Intensive Care* 2007;**35**(1):13—9.
21. Chang SN, Hwang JJ, Chen YS, Lin JW, Chiang FT. Clinical experience with intra-aortic balloon counterpulsation over 10 years: a retrospective cohort study of 459 patients. *Resuscitation* 2008;**77**:316—24.
22. Ramnarine IR, Grayson AD, Dihmis WC, Mediratta NK, Fabri BM, Chalmers JAC. Timing of intra-aortic balloon pump support and 1-year survival. *European Journal of Cardiothoracic Surgery* 2005;**27**:887—92.
23. Arceo A, Urban P, Dorsaz PA, et al. In-hospital complications of percutaneous intraaortic balloon counterpulsation. *Angiology* 2003;**45**(5):577—85.
24. Azeem T, Stephens-Lloyd A, Spyt T, et al. Intra-aortic balloon counterpulsation: variations in use and complications. *International Journal of Cardiology* 2004;**94**(2—3):255—9.
25. Cohen M, Urban P, Christenson J, et al. Intra-aortic balloon counterpulsation in US and non-US centres: results of the Benchmark Registry. *European Heart Journal* 2003;**24**(19):1763—70.
26. Ferguson J, Cohen M, Freedman R, et al. The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry. *Journal of the American College of Cardiology* 2001;**38**(5):1456—62.
27. Urban P, Freedman R, Ohman E, et al. In-hospital mortality associated with the use of intra-aortic balloon counterpulsation. *The American Journal of Cardiology* 2004;**94**:181—5.
28. Mandak J, Lonsky V, Dominik J, Zacek P. Vascular complications of intra-aortic balloon counterpulsation. *Angiology* 2005;**56**(1):69—74.
29. Kocogullari CU, Emmiler M, Ayva E, Cekirdekct A. IABP-related vascular complications: who is responsible—the patient, the surgeon, or the sheath? Part I: sheath-related complications. *Advances in Therapy* 2008;**25**(3): 225—30.
30. Erdogan HB, Goksedef D, Erentug V, et al. In which patients should sheathless IABP be used? An analysis of vascular complications in 1211 cases. *Journal of Cardiac Surgery* 2006;**21**(4):342—6.
31. Cohen M, Ferguson JJ, et al. Comparison of outcomes after 8 vs 9.5 French size intraortic balloon counterpulsation catheters based on 9,332 patients in the Prospective Benchmark Registry. *Catheterization and Cardiovascular Interventions* 2002;**56**(2):200.
32. Jiang C, Zhao L, Wang J, San J, Mohammod B. Anitcoagulation therapy in intra-aortic balloon counterpulsation: does IABP really need anti-coagulation? *Journal of Zhejiang University Science* 2003;**4**(5):607—11.
33. Weil KM. On guard for intra-aortic balloon pump problems. *Nursing 2007* July 28, 2007.
34. Lewis PA, Courtney M. Weaning intraaortic balloon counterpulsation: the evidence. *British Journal of Cardiac Nursing* 2006;**1**(8):385—9.
35. Bavin TK, Self MA. Weaning from intra-aortic balloon pump support. *The American Journal of Nursing* 1991;**91**(9):54—9.
36. Sorrentino M, Feldman T. Techniques for IABP timing, use - and discontinuance. *The Journal of Critical Illness* 1992;**7**(4):597—604.
37. Vitale N. Mechanical cardiac assistance. *Intensive Care Medicine* 1999;**25**:543—5.
38. Fuchs R, Brin K, Brinkler J, et al. Augmentation of regional coronary blood flow by intra-aortic balloon counterpulsation in patients with unstable angina. *Circulation* 1983;**68**(1):117—23.
39. O'Malley P. *Hemodynamic outcomes of weaning intraaortic balloon counterpulsation in heart failure*. Ohio: The Ohio State University; 2000.
40. Cohen M, Fasseas P, Singh V, et al. Impact of intra-aortic balloon counterpulsation with different balloon volumes on cardiac performance in humans. *The Cochrane Controlled Trials Register 2002* 2002 (2).
41. Watson JT, Willerson JT, Fixler DE, Sugg NL. Temporal changes in collateral coronary blood flow in ischemic myocardium during intra-aortic balloon pumping. *Circulation* 1974;**49/50**(Suppl. II):249—54.