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Abstracts

Contents

What's New in Heart and Lung Transplantation

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Heart and lung transplantation (Tx) remains the mainstream treatment to improve the prognosis and quality of life of patients with a variety of end-stage cardio-respiratory diseases. The process however, is constrained by critical shortages of suitable organs in addition to transplantation-related acute heart and lung injury that contributes to both postoperative mortality and the development of chronic rejection and ultimate demise of the recipient. There has been great progress in mechanistic and diagnostic aspects of these perioperative events at molecular, cellular, physiological and clinical domains, which is beginning to translate into some preclinical and early clinical therapeutic strategies. Furthermore, assist-device and ex vivo perfusion technologies have transformed the management of end-stage heart and lung disease, not only providing bridge to transplantation but also bridge to recovery.

Bridge to recovery and destination therapy for heart failure

Many of the patients waiting for heart Tx acutely deteriorate and their survival requires mechanical assist. Although traditionally bridge to transplantation has successfully been achieved, the Harefield heart failure programme using a combination therapy of mechanical assist and pharmacology demonstrated that more than two third of patients could be recovered to a sustained normal functional state by achieving full reverse cardiac remodelling and enabling explantation of assist devices and long term survival without the need for transplantation [1]. Also, selected patients who become ineligible for Tx may be treated with long term devices as destination therapy [2].

Beating heart transplantation

Reduction of ischaemia time and associated damage remains a primary goal in organ Tx. The potential role of warm blood perfusion in heart Tx has been explored by the European PROTECT I feasibility trial demonstrating the successful use of the Organ Care SystemTM (OCS) in maintaining and transporting donated hearts for transplant surgery. The trial concluded with excellent perioperative and early outcomes. The technology is also being evaluated in the USA in the form of a multi-centre trial (PROCEED).

Non-heart-beating lung Tx

The current shortages of suitable donor organs prompted utilisation of lungs from non-heart-beating donors. Our own experience and those of other centres indicate that perioperative outcomes are comparable between controlled non-heart-beating and heart-beating donors [3].

Ex vivo lung perfusion and reconditioning

Lungs deemed unsuitable for Tx may fully recover following a short period of ex vivo perfusion employing organ protective perfusion and ventilation strategies. The early clinical results with such technology are promising and may represent a new strategy to improve lung Tx [4].

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Mechanical Circulatory Support

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Continuous flow axial pumps were introduced in clinical use nearly ten years ago to support patients with advanced heart failure (HF) as a bridge to cardiac transplantation (CT). Nowadays, these devices are widely utilized to prevent impending organ damage and to support those patients who have temporary CT contraindications [1].
Axial pumps provide continuous flow by means of a single rotating impeller designed to achieve a blood flow of 5 to 10 L/min. The pump is connected to the left ventricular apex by an inflow cannula and to the aorta by an outflow graft. Continuous flow eliminates the need for valves and an internal compliance chamber that are required components in the previous pulsatile devices. Because of their smaller size and tubular configuration, axial pumps are less invasive and require less time to implant, there is less morbidity and they may be used in smaller patients including women and adolescents. Furthermore, they make little noise and because they generally consume less power than pulsatile pumps, these devices are suitable to be powered by implantable batteries.
In general, after exposing the heart through a standard median sternotomy, implantation is carried out with full cardiopulmonary bypass (CPB) support with or without cardioplegic arrest, in the following order: the apical inflow cannula is connected to the LV apex, the percutaneous cable is tunnelled out and the exit site is localized above the right iliac crest. The outflow cannula is connected to the ascending aorta, de-airing is done and the device is started during gradual weaning from cardiopulmonary bypass.
Bleeding and right ventricular (RV) dysfunction are the most frequent complications during surgery and in the first two postoperative weeks. These two complications may, along with end organ dysfunction, negatively impact on early survival.
Bleeding complications usually resolve in 24 hours post-implant. Special attention has to be paid for anticoagulation management in those patients with preoperative hepatic dysfunction because they can develop bleeding lasting beyond 72 hours post-implant. RV failure can become manifest immediately after the activation of the device in the operating room or in the subsequent postoperative hours. Preoperative and postoperative RV dysfunction is implicated as an important factor in postoperative multiple-organ failure and mortality.
The gold standard during circulatory support it to secure good left ventricle unloading with adequate pump flow but also to maintain right atrial pressure (RAP) <10 mmHg. In fact, too high an RAP is the only reliable haemodynamic parameter associated with end-organ failure and adverse outcome.
Device infection, pump thrombosis, thromboembolism and pump mechanical failure are the most frequent complications during long term support [2]. Infection involving the percutaneous lead is one of the major problems in long-term support. Thrombosis with or without thromboembolism is the second most important concern after device implantation, representing, after the first 15 days of support, the main causes of pump malfunction. Thromboembolism can involve the pump impeller with stoppage of pump function. Despite technical improvements in the new generation of axial flow devices and anticoagulation management protocols, the incidence of thrombosis and thromboembolism is still high and neurologic events are common.

Although axial pumps improve survival in patients with refractory HF there are still several problems to solve. Actual evolution of left ventricular support seems to follow two directions: (1) non-contact bearing design, and (2) centrifugal configuration. Further clinical evaluation will be necessary to determine if the improved hydrodynamic properties of centrifugal pumps and potential enhanced durability of a non-contact bearing design are associated with improvements in patient outcome.

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Anatomy of the Right Heart

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Atrial Component

The right atrium is composed of a venous component, (receiving the superior and inferior caval veins and the coronary sinus), a vestibule supporting the tricuspid valve, the septal wall and the appendage. The appendage has a broad junction with pectinate muscles that arise from the terminal crest. The septal surface of the right atrium comprises the floor of the oval fossa and its immediate muscular surround. The superior rim of the fossa (the septum secundum) is an infolding of the atrial wall between the superior caval vein and the right pulmonary veins entering the left atrium (Fig. 1).

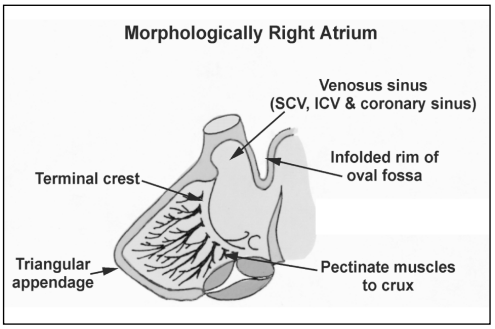

Figure 1 (© yen.ho@imperial.ac.uk)

Ventricular Component

The right ventricle is divided into arbitrary inlet, apical and outlet regions. In the inlet component the leaflets of the tricuspid valve occupy septal, antero-superior and inferior/mural locations. This valve has characteristic cordal attachments of the septal leaflet to the ventricular septum. The apical region has coarse trabeculations and from the base of the anterior papillary muscle a moderator band crosses the cavity of the ventricle. The septal surface is buttressed by a muscular structure called the septomarginal trabeculation. The pulmonary valve in the outlet region of the ventricle is supported by a complete muscular infundibulum (Fig. 2).

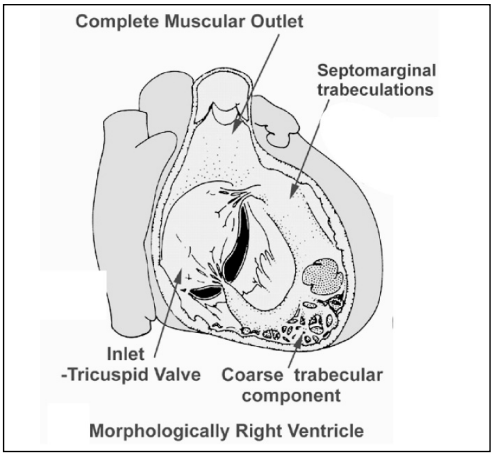

Figure 2 (© yen.ho@imperial.ac.uk)

Arterial Component

The pulmonary valve is the most superiorly located cardiac valve. It has three semi-lunar leaflets with sinuses that 'face' the aortic valve leaflets. The semi-lunar leaflets are separated by interleaflet triangles. The pulmonary trunk bifurcates into the left and right pulmonary arteries, which diverge to each lung. Characteristically, the right pulmonary artery passes inferiorly to the aortic arch.
Abnormalities of the right heart include Ebstein malformation of the tricuspid valve where the septal and mural leaflet are abnormally formed and apically displaced. The antero-superior leaflet is hinged at the atrioventricular junction but abnormal distal attachments are common leading to a reduced functional size of the right ventricle.
Pulmonary stenosis: this right heart abnormality can occur in isolation or associated with a congenital heart defect, such as tetralogy of Fallot. Stenosis of the valve can occur at various levels:
Subvalvar stenosis can affect the right ventricular outlet by hypertrophy of the infundubular region beneath the valve as well as hypertrophy of the septoparietal and septomarginal trabeculations. Subvalvar obstructions can also occur within the left ventricular outlet, including fibrous tags or ridges, septal hypertrophy or anomalous tendinous cords.
Valvar stenosis can be found where the individual leaflets have fused together along their zones of apposition creating a funnel orifice.
Supravalvar stenosis can occur from a narrowed arterial trunk. If the narrowing is discrete, this is referred to as a waist lesion. Fibrous ridges above the valve can also produce stenosis.

TOE Imaging of the Right Heart with Congenital Heart Disease

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The role of right ventricular (RV) morphology, function and dysfunction in cardiovascular disease has not attracted major interest until recently. Congenital heart disease (CHD) occurs in 0.5 to 1% of living births with a less frequent incidence of complex congenital disorders reaching 1.5‰ [1]. Due to major advances in paediatric cardiology and cardiac surgery over the past 50 years, the 15 years survival rate has reached 80% and 95% for complex and simple congenital heart disease respectively [2]. Consequently, the number of adolescents and adult patients with CHD increases continuously, creating a new patient population in which the RV has become the centre of attention to avoid the adverse outcomes including exercise limitation, RV failure and lethal ventricular arrhythmias [3] that may be associated with RV dysfunction and myocardial fibrosis. The timing of surgery remains extremely controversial. It is however clear that interventions performed earlier may have excellent long-term outcomes [4].
The complex, more triangular shape of the RV contrasts with the more conical shape of the left ventricle (LV). The muscular wall of the normal RV is usually 3 to 5 mm in thickness, but in case of pressure overload, it may even exceed that of the LV. The contraction of the RV myocardium relies more heavily on longitudinal shortening and, as a subpulmonary ventricle pumping to a low-resistance circuit, the RV functions at a lower ejection fraction (EF) than the LV. Although a morphologic RV seems inherently incapable of functioning as a subaortic systemic ventricular pump, it has a remarkable adaptation capacity and may function at systemic pressures for decades (congenitally corrected transposition, Senning or Mustard repairs).
Precise measurements of the RV are challenging because of its complex shape [5]. It must

be imaged in multiple planes and a qualitative visual assessment is usually applied, with the RV size being mostly described as normal, mildly, moderately (same size as the LV) or severely dilated (larger than the LV). Similarly the RV function is usually characterized as normal or mildly, moderately or severely dysfunctional. Rapid advancements in the field of magnetic resonance imaging (MRI) have established this technique as the „gold standard" for quantitative assessment of RV volume, mass and function, regardless of its position in the thorax (subpulmonary or systemic RV). There are however difficulties with its use including considerable inter-observer / intra-observer variability [6]. MRI with late gadolinium enhancement can detect myocardial fibrosis and is likely to make an important contribution to our understanding of the pathophysiology of RV dysfunction [7].
Volume-based indexes of RV function have limitations because of geometrical assumption and load-dependency. Other Doppler measurements may add insights into RV function such as dP/dt of the TR velocity or the index of myocardial performance or Tei-index of the RV [8]. Other measurements include tissue Doppler imaging (TDI) of the tricuspid annulus and myocardial acceleration during isovolumic contraction, which measure intrinsic contractility but are not used routinely [4]. RV diastolic function may also be assessed by Doppler interrogation of hepatic and superior vena cava inflow patterns as well as the RVOT.
We differentiate two broad contexts of RV adaptation to CHD: the volume loaded RV and the pressure loaded RV.

Volume loaded RV

The three most common lesions associated with RV volume loading are atrial septal defect (ASD), significant pulmonary regurgitation (PR) and significant tricuspid regurgitation (TR).

Atrial septal defect

There are three major types of ASD, ostium secundum, ostium primum and sinus venosus defects. An isolated ASD results in left to right shunting with progressive right atrial/ventricular and pulmonary artery dilatation. Paradoxical septal motion as a result of RV volume loading is evident both in M-mode and bidimensional echocardiography. The RV tolerates volume loading well for a long time. In older patients with long-standing RV volume overload, regional RV TDI may disclose early relaxation abnormalities. It is now accepted that long-standing right heart volume overload and dilatation in the setting of an ASD is detrimental and leads to morbidity (heart failure, arrhythmias, thromboembolic events) and increased mortality [3]. Early defect closure is warranted if a significant shunt is present.

Pulmonary regurgitation

Severe PR is very common after tetralogy of Fallot repair and is associated with RV dysfunction, diminished exercise capacity, atrial and ventricular arrhythmias and sudden death. Timely pulmonary valve replacement (PVR) may protect patients from these complications. The RV maintains systolic function for a long time unless there is an added haemodynamic burden such as peripheral PS that impedes forward flow, or another volume lesion such as a residual ventricular septal defect. Severe RV enlargement and severe RV dysfunction can antedate the onset of symptoms. An unfavourable ventriculo-ventricular interaction has been described in case of severe RV enlargement. If the RVEF was impaired, the LVEF was also worse. Cut-off values for RV volumes have been described beyond which the RV size will never normalize: end-diastolic RV volume $\leq$ 150ml/m^2 [4]. If the volume of the RV might normalize after PVR, no study could show that the RVEF improves. Although the window of opportunity for PVR is shifting earlier,

there is no evidence that earlier surgery might improve survival and decrease the incidence of arrhythmias.
MRI is considered the „gold-standard" for PR quantification and RV volumetric analysis. Doppler is a useful alternative for semi-quantitative PR assessment using the PR-index (PRi): ratio of PR duration to diastolic duration. A PRi <0.77 yields 100% sensitivity and 85% specificity for identifying patients with significant PR [9] A PR pressure half time <100 ms is also a good indicator of significant PR. Doppler detection of forward and laminar late diastolic pulmonary blood flow, coinciding with atrial systole and associated with a prominent retrograde superior vena caval flow, defines the restrictive RV physiology. A non-compliant hypertrophied RV along with low pulmonary arterial diastolic pressures, results in partial presystolic opening of the pulmonary valve during right atrial contraction, which contributes to forward flow. This physiology is frequently present early after tetralogy of Fallot repair, is associated with a low cardiac output and leads to longer intensive care stay. In contrast restrictive physiology late after repair counteracts the effects of chronic PR and is associated with smaller RV size, shorter QRS duration and better exercise capacity.

Tricuspid regurgitation

TR is mostly secondary to severe RV enlargement with resultant annular dilatation (RV dysplasia, free PR after tetralogy of Fallot repair).
Ebstein's anomaly is characterized by an apical displacement of both the septal and the posterior tricuspid leaflets exceeding 20 mm or 8 mm/m2 in adults. The right heart is then divided into three components: the true right atrium, the functional RV and the atrialized RV. In extreme cases, the atrialized portion of the RV can occupy more than one-half of the RV volume, and the RV dilatation may be so pronounced that the ventricular septum shifts leftward, compressing the LV. Not well recognised is the accompanying RV myopathy that adds to the propensity for RV dysfunction, in addition to that posed by the TR. The RV is usually very thin-walled and vulnerable to progressive dilatation and dysfunction.
Surgery should be performed for symptomatic adults. Repair includes transverse plication of the atrialized chamber and tricuspid valvuloplasty or tricuspid valve replacement or, in case of compromised RV function, a one and a half ventricular repair. A detailed preoperative assessment of RV size and function, of the valve leaflet attachments, commissures and surface is mandatory. Tridimensional echocardiography may have an important impact on the definition of the valves commissures and leaflets' surface.

Pressure loaded RV

The two most common lesions associated with RV pressure loading are: RV outflow tract obstruction (RVOTO) / pulmonary stenosis (PS) and the systemic RV.

RVOTO / PS

Isolated stenosis at the valvar level represents 80-90% of PS cases. Regardless of the level of the obstruction, the RV exerts a hypertrophic response. Echocardiography is the diagnostic method of choice, using continuous wave (CW) Doppler for the estimation of the pressure gradient across the RVOT. The RVOT instantaneous gradient correlates well with catheter based peak-to-peak gradient, in contrast to left sided stenoses. When scanning transoesophageal four-chamber views, the body of the right ventricle is demonstrated. From a low oesophageal scan position, the right ventricular apex is shown. Obstructions within the cavity of the RV are thus readily diagnosed. The relationship of the tricuspid valve apparatus to these obstructive lesions is well defined.
It is a misconception that the RV dilates and fails when exposed to high pressure. As long

as sinus rhythm is preserved and there is no additional volume lesion, the RV may maintain good function until the fifth decade. However, when the RV pressure exceeds 50% of the systemic pressure many patients develop symptoms.
The therapy of PS is closed or open valvotomy and a trans-annular patch in case of a small pulmonary annulus. These operations always result in PR, which is well tolerated for years. Initially the RV compensates by dilatation but maintains contractility and stroke volume. Often decades later, the RV systolic function deteriorates and a pulmonary valve replacement (PVR) is necessary. The PVR should be performed before the deterioration of RV function. Development or progression of secondary TR should prompt reoperation.

Systemic RV

Atrial switch operations (Mustard and Senning) have been performed for transposition of the great arteries for over 40 years. The atrial switch results in the RV supporting the systemic circulation. The assessment of RV is paramount but challenging in the absence of criteria for normal values. Volumetric methods have been the mainstay of RV assessment. Cumulative survival 25 to 30 years after the Mustard repair is as high as 80%. It seems however that there is a progressive deterioration of RV function along with residual lesions (baffle obstruction or leakage, residual VSD, PS) leading to reduced exercise capacity, heart failure, endocarditis, arrhythmias, reoperation and cardiac death. The cause of RV dysfunction is unclear. The single right coronary artery supplies the morphologic RV, making it vulnerable to perfusion mismatch and ischaemia in the context of severe RV hypertrophic response to systemic pressure loading. Focal fibrosis has been shown with MRI with late gadolinium enhancement [7]. Hypertrophy might be associated with fibrosis in some patients, and correlates inversely with RV systolic performance. Although accurate assessment of RVEF is important, the definition of normal systemic RVEF remains problematic. A RVEF >50% in the absence of significant valve regurgitation is considered as normal.
The congenitally corrected transposition demonstrates the remarkable ability of the RV to adapt to systemic pressure. The contraction pattern of a systemic RV may resemble that of the normal LV, with predominant circumferential shortening over longitudinal free wall shortening. This may represent an adaptive response to the systemic load. Survival to the seventh and eighth decade has been reported. However, an added volume lesion will precipitate ventricular dysfunction and failure of the morphologic RV occurs as a sequel to systemic atrio-ventricular valve regurgitation. An important marker for poor survival is a poor preoperative systemic EF. If systemic AV valve replacement (repair does not seem to work) is performed before the EF is depressed, systemic ventricular function may be maintained even in older patients.

Conclusion

Although MRI has been accepted as „gold-standard" for the assessment of volumes and function of the RV, echocardiography remains key to assessing disease progression, timing of late re-intervention and perioperative RV function.

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Echocardiographic Imaging of Right Heart Function

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Echocardiography has become an invaluable tool to diagnose right ventricular (RV) dysfunction in the acute clinical setting. A straightforward approach to differentially diagnose right from left ventricular (LV) failure is the direct comparison of cavity dimensions in the midoesophageal four-chamber and the transgastric short-axis view. Because these classic LV views often transect the RV in an oblique way, slight modifications of the probe's position and sector plane are often required to fully expose the RV. The primary response of the thin walled RV (normal thickness of the free wall = 5 mm) to any haemodynamic disturbance, be it an inappropriate loading condition or a primary contractile failure, is dilatation. In normal conditions the RV occupies less than 60% of the LV area. Mild dilatation exists when RV area and/or diameter is between 60 and 100% of the LV and severe enlargement is present when RV dimensions exceed the LV. With dilatation, the RV also changes its shape from triangular to globular in the longitudinal plane and from crescent-shaped to ellipsoid or circular in the short axis. In normal conditions, the RV long axis extends to only 2/3 the length of the LV and the apex of the heart is always formed by the LV. Formation of the apex by the RV rather than the LV is an additional sign of severe volume increase. Further clues to increased load on the right circulatory system are an enlarged coronary sinus (>1 cm) and leftward displacement of the interatrial septum.

The next step in the assessment of RV function is to study its motion characteristics during the ejection phase of systole. The RV is a volume pump whose performance is best characterized by the longitudinal piston-like displacement from base to apex. The absolute distance travelled by the tricuspid annular plane during systole (TAPSE = tricuspid annular plane systolic excursion) is easily quantified with (anatomical) M-mode echocardiography. Normal values range between 2-2.5 cm in spontaneously breathing subjects but are lower (1.5-2 cm) in anaesthetized and mechanically ventilated patients. More traditional measurements such as fractional shortening and fractional area change can also be used to assess systolic pump performance in the acute clinical setting. If ejection characteristics are normal the cause of RV dilatation is an isolated volume overload. This is usually not an immediate haemodynamic threat to the patient but prompts an active search for causal defects (left-to-right shunts, tricuspid or pulmonary valvular insufficiency). It is not uncommon to detect a previously unrecognized ASD of the sinus venosus type in this scenario.

If RV ejection is *not normal* the dilatation is caused by a mismatch of contractile performance and afterload. There is no simple way to differentiate between a primary contractile dysfunction or a dysfunction secondary to high afterload. Although several attempts have been made to construct a simple index of contractility, none of these variables is truly load independent. A pragmatic approach is to first measure the pressures in the right circulation using the modified Bernouilli equation on a tricuspid regurgitant jet. High pressures (RVPsystolic > 35 mmHg) indicate a pathological increase in RV afterload. Low RV pressures may result from low cardiac output and do not exclude excessive afterload. Simultaneous measurement of pressures and flows together with inspection of the pulmonary outflow signal are required to rule out increased pulmonary vascular resistance. Indices such as the myocardial performance index, maximum rate of pressure development in the RV (dP/dt max), strain and strain rate and isovolumic acceleration are used to assess contractile performance but their utility in the acute clinical setting is not clear. The various techniques will be discussed in the lecture. An algorithm for differential diagnosis of RV dysfunction with the intent to guide therapeutic decision making will be presented.

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Can Computerized Perioperative Data Improve Outcome from Cardiac Surgery?

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Introduction

The adoption of anaesthesia information systems (AIMS) provides a major opportunity for cardiac anaesthesia programmes to use data for quality purposes and research with the goal of improving outcomes. The following abstract serves to outline these approaches.

Enhancing performance improvement and patient safety

In addition to providing better documentation of clinical care, AIMS have the potential to improve quality of care. Several studies using simple reminders in an AIMS showed that this intervention could significantly improve compliance with prophylactic antibiotic administration timing [1-3]. Another group used a computer-generated reminder to enhance rates of re-dosing of antibiotics [4]. In addition to the contemporaneous reminders, data from AIMS may be extracted and analysed to generate daily reminders for US Physician Quality Reporting Initiative reports and communication with practitioners encouraging them to improve their performance.

Another use of an AIMS is to provide decision support. In one study, an AIMS-based algorithm that alerted the clinician if a patient had multiple risk factors for post-operative nausea/vomiting nearly doubled the use of antiemetic prophylaxis for these high-risk patients [5]. Using an AIMS to screen for intraoperative markers of complications may also be helpful in identifying cases for quality assurance reviews. Electronic screening yielded many more cases of interest than voluntary reporting by clinicians [6,7]. Overall, the evidence in the literature is just emerging, but the trend towards pay-for-performance and other quality measurement efforts will provide further impetus to foster systems that help health care practitioners adhere to guidelines proven to enhance patient safety.

Technologies in common use in other industries are also beginning to be used in healthcare and integrated into AIMS. Use of barcodes on medication labels in conjunction with a special scanning device may decrease medication errors and improve documentation [8-10]. Barcodes and radiofrequency identification tags can also be used to verify patient identity, locate patients and vital equipment, and ensure blood product compatibility. As the Society of Cardiovascular Anesthesiologists embarks on the FOCUS Project, the linkage of cardiac anaesthesia safety to technology will be an area of intense study.

Other areas of improvement possible with AIMS include the ability to retrieve records of prior anaesthesia encounters to identify previous problems, improvement of summary documentation for transfer of care (i.e. handoffs), guidance during emergencies (e.g. malignant hyperthermia or cardiac arrest), laboratory data interfaces that report and record pertinent lab values when they become available, alerts to worrisome trends in physiologic values beyond the simple limit alarms built into monitors, and the ability to monitor cases remotely by accessing live AIMS records from a remote workstation or PDA.

The possibility that an AIMS could actually jeopardize patient safety has been considered. With clinicians free of the need to record vital signs manually, there is potential for inattention to the vital signs that are both measured and recorded automatically. This issue was addressed in two studies that concluded that the use of an AIMS did not decrease vigilance in this regard [11,12].

Conclusion

The extensive functionality of AIMS and custom add-on systems in many of the „early adopter" centres developed over many years. Such systems are likely to be implemented more rapidly in the near future by others. Significant additional programming resources and initiative are needed for a full exploitation of the potential of an AIMS to improve outcomes in cardiac anaesthesia programs.

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Cognitive Outcome and CABG Surgery: A Case of the Emperor's New Clothes?

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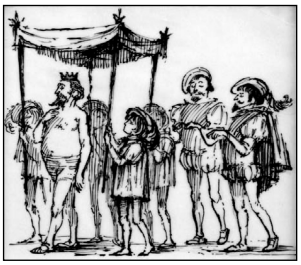

„The Emperor's New Clothes" is a short tale by Hans Christian Andersen about an Emperor who cares for nothing but his wardrobe. He hires two weavers who promise him the finest suit of clothes from a fabric invisible to anyone who is unfit for his position or „just hopelessly stupid". The Emperor cannot see the cloth himself, but pretends that he can for fear of appearing unfit for his position or stupid, and his ministers do the same.
When finally the Emperor parades before his subjects in his new clothes, a little boy cries out, „But he isn't wearing anything at all!" The Emperor cringes, suspecting the assertion is true, but holds himself up proudly and continues the procession.
Many researchers including myself have studied postoperative cognitive decline (POCD) following coronary artery bypass grafting (CABG) for years. During three decades we have obtained funding to set up psychometric test facilities and assess cognitive performance in thousands of CABG patients. POCD is diagnosed when patients have a lower psy-

chometric test performance after their surgery, than before. Again and again we found alarming incidences of POCD, not only in the first weeks after CABG, but also during long-term follow-up. Not surprising, we have been able to publish this disturbing finding in prestigious journals and newspapers [1].

We have always attributed the high incidence of POCD after cardiac surgery to the use of cardiopulmonary bypass (CPB). This seems reasonable because several studies have demonstrated potentially harmful side effects of CPB on the brain. This includes cerebral micro emboli during CPB and cerebral oedema after CPB. Over the last 8 years, however, randomized trials have compared cognitive outcomes after off-pump versus on-pump CABG. Surprisingly, these trials failed to show a clear benefit of avoiding CPB on the incidence of POCD.

The little boy who cried out, „But he isn't wearing anything at all!" is Dr Lars Rasmussen, who assessed the reliability of definitions of POCD [2]. Dr Rasmussen administered a baseline and a follow-up psychometric test battery to healthy volunteers, with a time interval of three months. He applied the most commonly used definition of POCD to the test results, and found that according to this definition, 29 percent of the volunteers had cognitive decline, three months after their baseline assessment. Apparently there is substantial natural variation in psychometric test performance and in contrast to what we thought, it is very difficult to distinguish true cognitive decline from random variation in test performance.

Although many people now suggest that the Emperor's new clothes in fact did not exist at all, we are still uncertain about POCD. How can we ignore the many transcranial Doppler studies showing hundreds of emboli during CPB, or the post-mortem examinations of the brains of CABG patients, also revealing evidence of cerebral emboli? And how can we close our eyes to the patients who are unable to return to employment after their CABG because of mental problems.

What we know, however, is that the incidence of true POCD is much lower than we always thought, and that the presence of advanced age and atherosclerotic disease are more important determinants of long-term POCD than the use of CPB.

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Immediate Recovery from Cardiac Anaesthesia: Increasing Patient Risks or the Way Forward?

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It is conventional to delay recovery following cardiac surgery for a number of hours. This was necessary, to ensure thorough re-warming following prolonged periods of hypothermic bypass; to treat pulmonary oedema and acute lung injury; as an adjunct in circulatory failure requiring high dose inotrope therapy and to provide immobility and blood pressure control to minimise postoperative blood loss. There have been advances in almost every aspect of the patient pathway associated with routine cardiac surgery. By using normothermic bypass [1], short acting opioids, minimal bypass prime volumes, cell salvage and refinements in post operative analgesia [2] it is possible to reverse anaesthesia at close of surgery.

Despite the feasibility of immediate recovery, a period of ventilation and sedation is generally perceived as a port of safety and provides a precautionary period in which to monitor the development of early complications. However mechanical ventilation is not innocuous. Increases in intrathoracic pressure from positive pressure ventilation reduce cardiac output and may increase the requirements for inotropic support. There are also risks of barotrauma, sputum retention and pulmonary collapse. Additional adverse effects of sedative drugs associated with mechanical ventilation include hypotension requiring vasopressors and depression of ciliary activity which may predispose to sputum retention. Ventilation-perfusion mismatch may also predispose to hypoxaemia. A reduction in metabolic rate associated with sedative drugs may compromise temperature management. A reduction in cardiac output associated with mechanical ventilation may prolong the clearance of opioids by the liver and reduce clotting factor production, both processes being highly perfusion related.
It follows that immediate recovery from cardiac anaesthesia avoids adverse effects associated with sedation and ventilation. However, in order to capitalise on this advantage, it is imperative that immediate recovery does not incur any additional adverse effects. Experimental evidence on gas exchange associated with immediate recovery shows surprisingly good ventilatory function with spontaneous tidal volumes of the order of 5-6 ml/kg within 10 minutes, and excellent gas exchange with extubation in a matter of minutes [3,4]. It can be concluded that positive pressure mechanical ventilation should not be considered as the position of default after uncomplicated cardiac surgery, and it should be justified in terms of specific clinical indications as with any other therapy. In view of the economic costs associated with critical care occupancy and the seemingly good outcomes associated with immediate recovery, a prospective trial comparing it with conventional Intensive Care is now an urgent imperative.

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TEE: Past, Present and Future

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This talk will give a 25-year perspective on the past, present, and future of TEE. TEE's first reported use was in 1975 using a homemade m-mode probe. Then in the early 80's Dr Oka reported the use of m-mode TEE to detect retained air after open heart surgery. In the mid 1980's, TEE was used to assess regional and global wall motion. The next major advance was the introduction of pulsed wave Doppler. This allowed TEE to be used to assess ventricular filling dynamics. Shortly thereafter, colour flow Doppler, biplane scanning, and continuous wave Doppler were developed. At this point TEE became a full diagnostic tool in the echo lab and the Operating Room. Since then there have been further technological enhancements including multiplane imaging, matrix probes, and now 3-D scanning.

In addition to these technical enhancements, there has been a tremendous change in the role of the anaesthesiologist with respect to TEE. In the early 1990's anaesthesiologists were the Rodney Dangerfields of intraoperative echocardiography. "We didn't get any respect". It was hard to be trained, credentialed, and paid for our services. Basically we had no way to prove our skills. Then in 1995, the Perioperative TEE (PTXe) exam was introduced. This joint project of the SCA and the ASE gave credibility to anaesthesiologists performing diagnostic imaging in the ORs ERs, and ICUs. Now in addition to the PTXe, we also have certification in Perioperative Echocardiography.
Listed below is a partial list of current TEE applications:
- Cardiac
- Mitral valve surgery
- Aortic surgery
- Coronary surgery
- Non Cardiac Surgery
- Haemodynamics
- Diagnosis

Non Cardiac Applications
- Augment pre-op evaluation
- Major blood loss cases
- Liver
- Ortho
- DDX of hypotension
- New non procedures

It's always hard to accurately predict the future, but broader application of 3-D scanning, more automated analysis, smaller, less expensive equipment, digital image storage, and better clinical algorithms are all very likely.

Guidelines and CPB

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The goal of this presentation is to introduce the audience to the application of clinical practice guidelines in the area of cardiopulmonary bypass. When talking about guidelines, these must be seen in a broader perspective:
- **Not all Evidence is Created Equally:** when two studies on the same subject come to conflicting conclusions, how do we know which is right [1]?
- **Guidelines are not Standards:** although different, both are extremely important in providing the medical profession (perfusion) the opportunity to provide the best possible outcomes for patients.
- **How to Distinguish the Good from the Bad from the Ugly:** details are important in the methods used for selecting, reviewing and synthesizing the evidence.
  When guidelines are developed, they should be *validated, reliable & reproducible, clinically applicable* but *flexible* as well, *clear* and developed in a *multidisciplinary process*, with *scheduled reviews*. Last but not least, guidelines should be developed with a detailed *documentation of the* ***methodology***.
- **Reviewing the current literature on CPB:** it is clear that the level of existing evidence is too small to serve as a basis for guidelines. Guidelines that do exist are mostly based on the lowest level of evidence, although a vast amount of literature is available. The guidelines that have been produced and published are small in number and with only a few strong recommendations, accompanied by a vast number of topics that need further clinical investigation. The existing published guidelines are grouped together on the website of the ICEBP, together with recommended literature. The International Consortium for Evidence-Based Perfusion (ICEBP) is a partnership and col-

laboration between perfusion societies, medical societies, clinicians and industry to improve continuously the delivery of care and outcomes for our patients. It seeks to identify gaps between current and evidence-based clinical practice, and in doing so, it reviews, comments and endorses evidence-based guidelines concerning the practice of cardiopulmonary bypass. There is a strong collaboration with medical societies (STS, ASA, in the development of these guidelines. www.bestpracticeperfusion.org)

– **Current Published Guidelines** make recommendations on 'optimal perfusion' [2], blood glucose management [3], perioperative blood transfusion and blood conservation in cardiac surgery [4], neurologic injury, glycaemic control, haemodilution, and the inflammatory response [5].

Currently, the ICEBP is developing a guideline document for the systemic inflammatory response, as well as partnering with The Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists to produce:

- An update of the current transfusion guidelines
- New guidelines for cardiopulmonary bypass

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Improving Outcome from CPB

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More than a million cardiac surgery procedures are performed annually worldwide. Despite this enormous number, a recent review on this topic states: "The scientific data concerning the effectiveness and safety of key principles of cardiopulmonary bypass are insufficient in both amount and quality of scientific evidence to serve as a basis for practical, evidence-based guidelines" [1]. Thus it is not astonishing that the strength of recommendation used in guidelines relevant for this field, e.g. for blood transfusion during CPB, are also rather weak [2] and only expert statements are available [3,4]. Consequently, any attempt to review how we can improve outcomes following CPB cannot be strictly "evidence based" and will rely on the description of concepts that, despite being sound, remain to be proven in future trials.

What are the adverse effects of CPB?

Until the advent of Off-Pump Coronary Artery Bypass (OPCAB) surgery, nearly all postoperative disturbances and complications following cardiac surgery were directly attributed to the use of CPB and cardioplegic arrest. This, however, may be regarded as an oversimplification.

Several studies have shown that the postsurgical inflammatory state as well as relevant complications after cardiac surgery (cognitive dysfunction, stroke, renal failure and pulmonary dysfunction) are also detectable in patients undergoing OPCAB surgery. Consequently, thoracotomy per se, manipulation of the heart and the ascending aorta, blood contact with the pericardium and the pleura are at least, in part, responsible for several adverse effects of cardiac surgery commonly attributed to the use of CPB [5,6].

Nonetheless, there is a relationship between the postoperative course of plasma markers of organ dysfunction and inflammation and the duration of CPB [7], suggesting that the CPB has adverse effects independent from the surgical insult per se. Thus, the manipulation of the coagulation system, the contact of blood with the artificial surfaces of the CPB, the consequences of arterial and venous cannulation, the physical stress exerted on the blood by the mechanical components of the system (blood pumps, reservoir, cannulas, etc.) as well as changes in blood flow characteristics, oxygen delivery, and acid-base balance, are unique features of the extracorporeal circulation that may be relevant in mediating adverse effects and may thus be relevant for improving outcomes from CPB. However, it is not possible to go into detail of all these aspects. Thus the present overview will be limited to a few potentially important aspects.

Artificial surfaces and management of shed blood

The contact of blood with artificial surfaces leads to activation of the coagulation system and indirectly to activation of pro-inflammatory mediators [8]. Consequently, measures to reduce the artificial surface area by reducing the length of the tubing and the surface of the oxygenator as well as coating the tubing with anticoagulants may be useful to reduce contact activation. Additionally, avoidance of air contact of the blood may reduce microembolic events by microbubbles and thereby reduce neurological dysfunction [9]. This has led to the development of minimized extracorporeal circulation systems [10] with shorter tubing and excluding the conventional venous reservoir. The data to show whether these systems have beneficial effects beyond a reduction in priming volume, leading to a reduction in the need for allogeneic blood products, are controversial [11,12].

Minimizing contact activation by using heparin-bonded circuits has been associated with reduced inflammation. Clinical data regarding the effects on postoperative morbidity and mortality are conflicting.

Reinfusion of cardiotomy suction blood is a major source of fat emboli during cardiac surgery and has been shown to be associated with neurological deficits and activation of coagulation, fibrinolysis, and inflammation. Thus, ideally, reinfusion of pericardial suction blood should be avoided and instead, the blood should be processed by a cell saver or at least be filtered before reinfusion. It is of note that the use of a cell-saver only for the processing of shed blood during CPB has been associated with a higher postoperative bleeding rate and an increased need for fresh frozen plasma. A recent meta-analysis comes to the conclusion that the use of cell-saver reduces exposure to allogeneic blood only if used throughout the entire procedure and for processing the residual blood of the CPB circuit [13].

Arterial cannulation and aortic cross-clamping

The severity of aortic atherosclerotic disease is related to an increased incidence of stroke and athero-embolic disease [14]. It is of note that the examination of the aorta by TOE is clearly better than direct palpation of the vessel by the surgeon. However, epi-aortic ultrasound has been reported to be superior to TOE and may thus be the method of choice [15].

Measures to detect the extent of atherosclerotic plaques have been reported to reduce the incidence of stroke in on-pump cardiac

surgery if the surgical strategy is changed according to the results of the ultrasound examination; i.e. aortic non-touch OPCAB surgery may be performed instead of a planned conventional on-pump procedure [16]. Unfortunately, aortic cross-clamping cannot be completely avoided in a relevant number of surgical procedures. Although the single cross-clamp technique reduces neurological complications and thus should be preferred to the side-clamp manoeuvre for insertion of proximal coronary grafts, it is still controversial.

Perfusion technique

Organ blood flow during cardiopulmonary bypass is dependent on the flow applied by the pump. Historically, pump flow has been set to a more or less fixed level comparable to a cardiac index of a patient at rest with reduced metabolic needs (as it is typically the case in an anaesthetized, moderately hypothermic patient), i.e. a cardiac index of 2.2 to 2.5 l min^{-1} m^{-1}. This is often associated with a decrease in arterial blood pressure below the preoperative baseline.

The optimal blood pressure during CPB is controversial. Reich and co-workers have shown an association between an increased arterial blood pressure during CPB and stroke rate and between a low mean arterial pressure and mortality [17]. Fischer et al. observed a clear correlation between a low mean arterial pressure during CPB and postoperative renal failure [18]. A prospective study by Gold and co-workers of 248 patients showed that optimizing perfusion pressure during CPB to a mean arterial pressure higher than 80 mmHg led to reduction in postoperative morbidity [19], a finding that could not be reproduced more recently in a larger study population [20]. It is of note, that a retrospective analysis of the first trial showed that patients with severe arteriosclerosis of the aorta benefited most from maintaining a higher mean arterial pressure [21]. Thus, a recent evidence based expert statement suggests that arterial blood pressure, at least in patients at high risk for cerebral embolic complications, should be kept at or higher than 70 mmHg [4].

When discussing arterial blood pressure during CPB, the role of pump flow cannot be ignored. Ranucci and co-workers have shown that a low oxygen delivery (DO2) leads to an increase in plasma lactate and hyperglycaemia and associated postoperative morbidity [22], and an increased incidence of renal failure [23]. In the latter studies, mean arterial blood pressure was targeted around 60 mmHg and was not associated with postoperative morbidity.

Conventionally, non-pulsatile blood flow during CPB is maintained during CPB. Experimental studies have shown improved organ perfusion with pulsatile blood flow [24]. The clinical effects of pulsatile blood flow, generated by modification of the CPB are controversial [4]. This may be related to the fact that the pulse pressure produced by the pump differs markedly from the physiological pulse wave form. Interestingly, a recent prospective trial investigating the effects of pulsatile perfusion generated by an intra-aortic balloon pump revealed improved organ function and clinical outcome [25].

Temperature

Historically, moderate hypothermia has been used to reduce oxygen consumption during CPB. This allows a lower pump flow, reducing potentially adverse effects of a higher flow rate, reducing cerebral and myocardial oxygen needs, improving neurological outcome and decreasing myocardial injury during cardioplegic arrest [26]. Yet these concepts have not been convincingly confirmed in clinical studies. With the exception of procedures needing periods of low flow or even circulatory arrest, it is at present not clear if the use of moderate hypothermia improves outcome from CPB. However several studies have clearly shown that hyperthermia >37°C during rewarming is associated with increased

cognitive dysfunction and thus should be avoided [27].

Transfusion management

Transfusion of allogeneic blood during cardiac surgery is associated with an increased rate of complications and a worse short and long term prognosis [28]. Unfortunately, the same is true for a low haematocrit during CPB and this effect is independent from the transfusion of blood products [28]. Interestingly, Ranucci and co-workers have shown that a low haematocrit during CPB is not associated with an increased incidence of postoperative renal failure if the pump flow is adjusted to maintain a high oxygen delivery and that the renal failure rate increased beyond a cut-off DO_2 of less than 270 ml min^{-1} m^{-2} [22]. It is not known if this cut-off level also applies to other types of postoperative morbidity (e.g. stroke rate) or mortality.

The question of what haematocrit may be optimal during CPB is complicated by the findings of a recent prospective trial aimed to analyse the effects of maintaining a haematocrit >27% during CPB in comparison with profound haemodilution (haematocrit 15-18%) [30]. While older patients undergoing profound haemodilution suffered from neurological dysfunction, the trial had to be stopped prematurely due to a higher rate of pulmonary complications in the high haematocrit group. It is of note that this cannot be explained by a higher overall transfusion rate in the high haematocrit group, and thus the reasons for this adverse effect remain unclear. Nonetheless, at present the optimal haematocrit as well as the ideal transfusion trigger during CPB, remain to be determined and any measures to conserve blood and to avoid unnecessary haemodilution remain pivotal.

Monitoring the effectiveness of perfusion

Besides monitoring pump flow and arterial pressure, the effectiveness of the perfusion has historically been derived from the trend of oxygen saturation in the venous effluent, the mixed venous saturation (SvO_2). Despite being a gold standard for evaluation of the adequacy of systemic oxygen delivery to demand, a normal and high normal SvO_2 does not rule out an oxygen deficit at organ level. This is also true for plasma lactate as a measure of anaerobic metabolism.

A promising alternative for the monitoring of the adequacy of organ specific perfusion, not only during CPB but also during other periods of both cardiac and general surgery, is the determination of cerebral oxygen saturation (ScO_2) by near-infrared-spectroscopy. Available evidence suggests that adjusting perfusion to maintain ScO_2 near the healthy preoperative range reduces neurological dysfunction and postoperative morbidity [31]. Additionally, ScO_2 has been shown to be especially useful during selective cerebral perfusion for major thoracic vascular surgery [32].

Glycaemic control

Besides these aspects, cardiopulmonary bypass management includes a bundle of variables that are far from being clearly settled, often already being of interest before bypass and extending not only into the post-bypass period but also into the period after surgery. For example, the extent of intraoperative hyperglycaemia is related to the degree of postoperative morbidity [33]. A recent trial on intraoperative normalization of glucose to levels between 4.44 to 6.05 mmol/L *(80-110 mg/dl)* showed an increased stroke rate in the intervention group [34]. Thus, current expert recommendation suggests maintaining a blood glucose of less than 8.25 mmol/L *(150 mg/dl)* [35].

These are only a few examples of aspects related to the treatment of patients undergoing cardiac surgery with CPB and the list of unsolved questions is very much longer. Thus, as a recent expert statement on the optimal use of CPB states, there is an urgent need for randomized controlled trials to improve the

safety of this technology to allow really evidence-based recommendations in the future [4].

Declaration of a potential conflict of interest

The author receives honoraria from COVIDIEN, the distributor of the INVOS® cerebral oxygen saturation monitor.

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Anaesthesia for Cardiopulmonary Bypass

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Historically, anaesthesia for cardiopulmonary bypass (CPB) relied on high dose opioids and the cerebral depressant effect of moderate hypothermia. Hypothermia decreases the Bispectral Index (BIS) value by around one BIS 'unit' per degree centigrade [1], and increas-

es the latency of the auditory evoked potential [2]. Volatile anaesthetic requirements are substantially reduced during hypothermic CPB [3], although they return to normal on rewarming. Modern cardiac surgery is increasingly performed within a 'fast-track' protocol at normothermia. This requires a change in the way that cardiac anaesthetists work, and increases the potential for unintended awareness during cardiac surgery. Health economic considerations and patient safety dictate that we provide careful titration of anaesthetic dose rate to physiological effect to minimize the adverse effects of anaesthesia.

At the onset of CPB there is rapid and significant haemodilution by the circuit prime, which reduces the circulating concentration of anaesthetic drugs but which also reduces the concentration of plasma proteins. The unbound fractions of both propofol and midazolam increase during CPB as a result of changes in protein binding [4,5], which compensates for the decrease in total concentration resulting from haemodilution. The blood solubility of volatile anaesthetics increases as temperature decreases, but this is antagonised by the reduction in solubility of volatile anaesthetic agents in crystalloid prime compared to blood [6]. In addition to the effects of CPB on anaesthetic agents, opioids are adsorbed and sequestered within the plastics of the extracorporeal circuit at physiological pH [7].

The dose rates of both isoflurane and propofol required to maintain a constant BIS value during CPB decrease by 15-20% [8,9], although one study found no change in BIS during normothermic CPB using a propofol-sufentanil anaesthetic [10]. That similar reductions in dose rate during CPB can be seen with both volatile and intravenous anaesthetics suggests that the mechanism is not pharmacokinetic but a direct result of extracorporeal circulation. We know that CPB affects neurocognitive function as a result of microemboli, inflammatory mediators and cerebral oedema [11,12], so it is maybe not surprising that the brain is more sensitive to the effects of anaesthetic drugs during and immediately after CPB.

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Double Lumen Tube (DLT)

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The most commonly used DLTs (left, right, more frequently without hook) are PVC single use only and are characterized by a wide "D" shaped lumen and high volume-low pressure cuffs.
They are made in several sizes (26, 28, 32, 35, 37, 39, 41 Fr) with some differences among producers.
Only one PVC double-lumen tracheostomy cannula, left or right shaped, is available (Tracheopart, -Teleflex). It is indicated in tracheostomized patients undergoing OLV.
Although many expert physicians emphasize a good degree of safety in the use of the right shaped DLT [1,2], the choice of this tube in routine procedures is limited by several factors.
The choice of the correctly fitting DLT requires a good knowledge of the anatomy of the patient's airways with particular attention to the size of trachea and the size and length of the main bronchus to be selectively intubated.
DLT intubation is usually carried out using the Macintosh laryngoscope, which ensures a wide direct view. The stylet, if used, must absolutely be removed when the tip of the DLT passes through the vocal folds, although different and potentially dangerous methods are reported in the literature [3]. At this point the intubating technique can be accomplished differently according to the kind of DLT being used and is accurately described in the literature.
DLT intubating techniques cannot ignore some fundamental recommendations necessary to produce better results with lower complication rates. As indicated, direct fibreoptic bronchoscopy (FOB) during DLT insertion or FOB control after DLT positioning can be considered a gold standard in selective bronchial intubation.
The most frequent complications from DLTs are displacement and trauma.
DLT displacement can be caused by several factors independent of the intubating technique. Among them, surgical manipulation and bronchial cuff overdistension can cause an outward movement of the tube, while flexion and extension of the head and neck can move the tip of the DLT an average of 2.7 cm, during flexion and 3.5 cm, during extension.
Cuffs overinflation can cause serious consequences both on tracheal and bronchial mucosa.
Airways injuries can be evident by air leak, subcutaneous emphysema, haemorrhage inside the airways, or haemodynamic instability due to possible tension pneumothorax.

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Univent Tubes and Bronchial Blockers

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In thoracic anaesthesia, "double lumen tubes" (DLTs) are used most commonly for one-lung ventilation (OLV). However, after the introduction of new blocker designs and because of the widening scope of surgical procedures, there has been a recent increase of interest in the use of bronchus blockers (BBs) such as the Arndt Blocker, Cohen Flexitip Blocker and Uniblocker. With this concept, the Univent tube, combining an endotracheal tube with a BB attached on one side, can also be considered within this group of devices.

Some of the advantages of the BBs (compared to DLTs) are:

1. In patients with abnormal upper or lower airway anatomy and in patients with difficult airways, BBs can offer a relatively easier method of airway management.
2. The use of DLTs requires a change to a single-lumen ETT at the completion of surgery if the patient requires postoperative ventilator support. This may present challenges in the presence of facial oedema, secretions, or laryngeal trauma from the initial intubation.
3. BBs (combined with the use of a normal single lumen tube) are associated with less airway trauma compared with DLT's, leading to less postoperative hoarseness and throat pain.
4. In patients with previous lung resections, blocking of more distal bronchi (i.e. lobar blockade instead of lung blockade) can be necessary and can be possible with BBs.
5. BBs can also be inserted through a tube after nasal intubation, or even through an LMA

The Arndt Blocker is a "wire-guided" BB, where the Fibre Optic Bronchoscope is inserted through the wire and advanced to the bronchus. After releasing the wire guide, the FOB can be pulled back and the position confirmed. The only disadvantage of this method is that the FOB can not "see" the wire guide, which is more proximal than its tip.

The Cohen Blocker is a BB with a flexible tip. The tip can be directed in two directions with a proximal circular device. The FOB can "see" the tip of the BB and also its direction during the process. The balloon at the tip has a huge capacity; and can block even the largest bronchus without any leak.

The Univent tube combines a single lumen tube with a bronchus blocker. The bronchus blocker is housed in a small anterior lumen containing a thin (2 mm internal diameter) tube with a distal balloon on the blocker. Univent tubes are available in sizes that range from 6 to 9 mm. However, that size refers only to the internal diameter of the single lumen tube.

The novel device, the Uniblocker, has a (similar) BB but this is not attached to the single lumen tube. Instead it is connected to a device on a normal single lumen tube (after intubation). The device is turned to the side of the operation site, and with the help of a fibreoptic bronchoscope (FOB), the BB is inserted into the main bronchus of the lung to be blocked. Uniblockers are available in sizes from 4.9 to 9 mm, including also sizes for paediatric patients.

After placement of the BB, and after confirmation of the location, the balloon at the tip of the BB is filled with 4 to 9 ml of air (depending on the type and the size of the BB). Almost all the BBs (with the exception of some paediatric types) have a lumen permitting suction of air (or secretions) from the blocked lung or lobe. Application of CPAP is also possible via this lumen.

When a BB is used in adults, the smallest acceptable single-lumen endotracheal tube size recommended is 8.0 mm internal diameter. It is important to have enough space between

the bronchus blocker and the flexible fibreoptic bronchoscope so that navigation can be achieved within the single-lumen endotracheal tube.

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How to Avoid Pneumonectomy: The Role of Bronchial and Vascular Sleeve Resection

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The indication for a sleeve resection is well established: a tumour arising at the origin of a lobar bronchus but not infiltrating as far as to require pneumonectomy. In addition, a sleeve resection may be indicated when N1 nodes infiltrate the bronchus from the outside, as is often the case in left upper lobe tumours requiring a combined reconstruction of the bronchus and the pulmonary artery.

From a functional point of view, sleeve lobectomy is strictly indicated in patients who cannot withstand pneumonectomy, but recent experiences have shown that the advantages of sparing lung parenchyma are evident also in patients without cardiopulmonary impairment. Oncologically, the primary goal is, in every case, the complete resection of the tumour with free resection margins.

When analysing survival data reported in literature of recent years, most of the studies show similar or better results for parenchyma sparing resections (including pulmonary artery reconstructions) compared with pneumonectomy. Moreover, in the analysis of 5-year survival according to stage and nodal status, sleeve lobectomy results in higher survival rates for stages I, II and III (Deslauriers '04, Kim '05, Ludwig '05), although the survival advantage in stage III appears to be limited and the benefit is not always confirmed for stage III-N2 patients. Therefore the role of parenchymal sparing operations in patients with N2 disease still remains not completely defined (Fadel'02, Yildizeli '07).

These results also justify the increasing use of parenchymal sparing procedures for lung cancer in patients with good cardiopulmonary function, as observed in recent years. Postoperative morbidity and mortality data reveal overall better results for patients undergoing sleeve lobectomy with respect to pneumonectomy. Looking at literature data, when morbidity is evaluated according to the type of complication, pneumonectomy patients appear to experience a higher rate of cardiac complications, while sleeve lobectomy patients show a greater incidence of pulmonary and airway complications.

The preservation of lung parenchyma has been indicated by some authors as the possible cause of a theoretical increased risk for locoregional recurrence after sleeve lobectomy. However, although in some experiences (Fadel'02) a higher local recurrence rate is reported for sleeve resection with advanced nodal status (N2), the few studies (Fadel'02, Terzi'02, Kim'05) analysing risk factors for recurrence, show that the tumour stage and the

nodal status are the only negative predictive factors, rather than the type of operation performed.
A number of studies indicate that lung parenchyma sparing improves postoperative quality of life because of a greater cardiopulmonary reserve, less pulmonary oedema and less right ventricular dysfunction as a result of a lower pulmonary vascular resistance (Terzi'02, Martin-Ucar'02).

Pneumonectomy Mortality and Morbidity: A Multicentre, Prospective, Observational Cohort Study

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Background

In order to assess the short term risks of pneumonectomy for lung cancer in contemporary practice, a one year prospective observational study of pneumonectomy outcome was made. Current UK practice for pneumonectomy was observed, to note patient and treatment factors associated with major complications.

Methods

A multicentre, prospective, observational cohort study was performed. All 35 UK thoracic surgical centres were invited to submit data to the study. All adult patients undergoing pneumonectomy for lung cancer between 1 January and 31 December 2005 were included. Patients undergoing pleuropneumonectomy, extended pneumonectomy, completion pneumonectomy following previous lobectomy and pneumonectomy for benign disease, were excluded from the study.
The main outcome measure was suffering a major complication. Major complications were defined as: death within 30 days of surgery; treated cardiac arrhythmia or hypotension; unplanned intensive care admission; further surgery or inotrope usage.

Results

312 pneumonectomies from 28 participating centres were entered. The major complication incidence was: 30-day mortality 5.4%; treated cardiac arrhythmia 19.9%; unplanned intensive care unit admission 9.3%; further surgery 4.8%; inotrope usage 3.5%. Age, American Society of Anesthesiologists physical status ≥ P3, pre-operative diffusing capacity for carbon monoxide (DLCO) and epidural analgesia were collectively the strongest risk factors for major complications. Major complications prolonged median hospital stay by 2 days.

Conclusions

The 30 day mortality rate was less than 8%, in agreement with the British Thoracic Society guidelines. Pneumonectomy was associated with a high rate of major complications. Age, ASA physical status, DLCO and epidural analgesia appeared collectively most associated with major complications.

Anterior Mediastinal Masses

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Anaesthesia and surgery for benign and malignant mediastinal masses can be life-threat-

ening because of compression of vital structures within the mediastinum; in particular the major airways (trachea, bronchi), superior vena cava, and major pulmonary vessels. In some patients induction of anaesthesia can result in failure to adequately ventilate the lungs and/or severe hypotension followed by cardiac arrest.
Mediastinal masses can be stratified into one of three groups:

1. those with minimal compression which can be managed according to routine practice
2. those with mild compression of adjacent structures which require some modification to induction, intubation and mechanical ventilation
3. those with potentially life-threatening loss of airway and/or cardiovascular collapse that demand substantial changes to the anaesthetic plan, or cancellation and consideration of alternative approaches to diagnosis and treatment of the mass.

A complete history, examination, and selected investigations are necessary to evaluate the likely impact of induction of anaesthesia and mechanical ventilation on these masses. Useful items on history include evidence of tracheal compression or postural change (especially when lying flat on the back). On examination, look for evidence of SVC obstruction, tracheal shift, or postural change, as well as atrial fibrillation and postural shifts in blood pressure. Essential investigations include chest x-ray, CT scan, and echocardiography. Pulmonary function tests with flow-volume loops are useful but cannot be relied upon to identify clinically important obstruction. MRI scans have become popular because of the capacity to create 3D images to guide surgical planning.
These steps, along with a discussion with the surgeon as to their expectations and surgical plan, can help the anaesthetist decide which induction technique is most appropriate.
High-risk lesions, in patients with limited reserve, should lead to a reassessment of the need for surgery. Some mediastinal masses do not require surgery for diagnosis, as they can be managed with CT-guided needle biopsy.
This presentation will include examples of patients from each of the above groups.

Preoperative Risk Assessment: Biomarkers versus Exercise Testing

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Preoperative cardiac risk assessment is traditionally based on three key elements, the patient history, their functional capacity and the risk of the surgical procedure. In case of poor functional capacity, the presence of several clinical risk factors and high risk surgery, non-invasive testing has to be considered [1]. Non-invasive testing for coronary artery disease consists of physical exercise tests (treadmill or bicycle ergometer) or pharmacological stress tests (nuclear perfusion imaging, dobutamine or dipyridamol stress echocardiography). Non-invasive testing for valvular heart disease consists of echocardiography as a first step. All these examinations are expensive and time consuming and, if not performed well in advance, call for postponing surgery.
Recent data suggest that biomarkers can play a substantial role in perioperative risk evaluation. So far, the interest has focused on natriuretic peptides (NP) and cardiac troponins (cTn).
NP, primarily brain natriuretic peptide (BNP) and the N-terminal fragments of its pro-hormone (NT-proBNP) are neurohormones. They are released from the myocardium in response to cardiac volume and pressure load

[3]. Both are of high diagnostic value in patients with congestive heart failure [4-6] or acute coronary syndromes (ACS) [7,8]. The information derived from BNP or NT-proBNP measurements seems to be similar.

In surgical patients, available data consistently show an association between elevated NP levels and adverse outcome after non-cardiac [9-13] and cardiac surgery [14]. A recent meta-analysis on 30-day outcome reported on an odds ratio (OR) of 19.3 (95% CI: 8.5 to 43.7) for the association between an elevated pre-operative BNP level and various cardiovascular outcomes (e.g. a composite of cardiac death and nonfatal myocardial infarction and atrial fibrillation) [15]. We performed a meta-analysis on the association between elevated NP levels and mid-term outcome (³6 months): the pooled OR for all-cause mortality was 4.97 (95% CI: 3.06-8.07) [16]. Of even greater importance is the very high negative predictive value (0.94. 95% CI: 0.88-0.97) of a low NP level before non-cardiac surgery [16]. This finding confirms the result of a smaller study in vascular surgical patients that reported on a negative predictive value for subsequent adverse cardiac events of 0.965 (95% CI: 0.879-0.996) in case of a preoperative BNP value below 50 pg/L [17] and of two other studies in patients undergoing non-vascular surgery [10] and vascular surgery [18].

In the light of the recent data, NP analysis should be considered in patients in whom the classic triad of risk assessment (history, physical capacity and type of surgery) does not unambiguously resolve the question of the necessity of further time consuming and expensive testing. If NP levels are below a discrimination threshold and the patient is clinically stable, the patient can proceed to surgery without the need for further testing. If NP levels are above the threshold, further testing, optimization of medical therapy and, rarely, invasive therapy should be performed [19]. In the recently published guidelines for the evaluation of patients at cardiac risk undergoing non-cardiac surgery, "NT-proBNP and BNP measurements should be considered for obtaining independent prognostic information for perioperative and late cardiac events in high-risk patients (Class IIa. level of evidence B)" [2].

The fact that NP are not mentioned in the AHA/ACC guidelines published in 2007 [1] demonstrates the rapidly growing evidence in this field of perioperative medicine. It has to be pointed out that, so far, the optimal cut-off value of BNP or NT-proBNP has yet to be determined and that the proposed approach is speculative and needs formal testing.

cTn are structural proteins of the contractile apparatus of skeletal and myocardial myocytes. cTn are released into circulation only upon the occurrence of cardiac cell death. This is typically the case in ACS [20,21] but can also take place in other acute conditions [22] e.g. in acute congestive heart failure [23] or in pulmonary embolism [24].

There is growing evidence demonstrating the association between elevated postoperative cTn levels and adverse short- and midterm outcomes after major non-cardiac [25-30] and cardiac surgery [31,32]. Accordingly, cTn analysis could be used for identification of patients whose risk was preoperatively considered to be intermediate but whose postoperatively elevated cTn levels, nevertheless, indicate a substantial risk for adverse short- and long-term outcome. Such identification will be the basis for future strategies of intensified observation and treatment. The development and validation of those strategies needs further research.

Currently, a large number of studies have been published describing the value of *new biomarkers* for risk assessment. In patients at high risk for coronary events, for example, measurement of markers of plaque instability (e.g. the pregnancy-associated plasma protein-A (PAPP-A) [33-35] or measurement of markers of platelets reactivity (e.g.. the platelet collagen receptor glycoprotein V1 [36- 37] could potentially prove to be useful. However, even more important than studies of new biomarkers are studies investigating how biomarker-guided management might

enhance care and outcome of this group of patients [38].

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The Safety of GA versus LA for Carotid Endarterectomy: What can we Learn from GALA?

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There is a widespread view amongst anaesthetists, surgeons, patients and other professionals that major surgery is inherently safer if it can be performed under local or regional anaesthesia (LA) rather than general anaesthesia (GA), particularly if the patient has multiple co-morbidities. Carotid endarterectomy

is one such procedure, where views are polarized, such that in some centres a GA is not offered. However is there any real evidence to support such a stance, either for this or other procedures? The GALA study tested this hypothesis in a large population undergoing carotid endarterectomy [1,2]. Key elements are summarised in the table (see next page) and further details are available on design [3] and website http://www.dcn.ed.ac.uk/gala

Arguments in favour of LA included assumptions that avoiding the physiological disturbances produced by GA is potentially beneficial in patients with significant comorbidities. In addition there may be potential benefits of LA specific to carotid surgery;

- Maintenance of definitive monitoring of brain function with an awake patient during carotid cross-clamping.
- The preservation of cerebral and systemic autoregulation to provide adequate cerebral perfusion pressure and thereby reducing the risk of low brain perfusion.
- As a result of the above, a reduced need for carotid shunts.

The GALA trial did not show a clear and unequivocal benefit from receiving LA rather than GA. It did however demonstrate that outcomes from carotid endarterectomy have improved over time. The findings in GALA follow other research in this area that has failed to show a consistent reduction in major complications or death with LA as compared to GA.

GA and LA both packages of care

It seems intuitive (but not proven) that a minor surface procedure under topical or infiltration anaesthesia alone should be safer than a GA (e.g. cataract under topical LA). The sicker and more frail the patient the more obvious the benefit might appear, even if only to have a patient who is destined to collapse on the day of surgery, collapsing under LA to the relief of anaesthetists! However as surgery gets more invasive with greater tissue injury and stress response, and local anaesthetic techniques become more complex and invasive, the picture is confused.

All nerve blocks are associated with finite risks and patients with regional anaesthesia may receive sedation or GA, either planned or unplanned. The use of vasopressors, opioids and antiemetic drugs will differ between LA and GA. Thus most studies compare packages of care that differ by multiple elements, apart from just GA or LA.

Table: The key features of GALA

Design	A parallel group, multicentre, randomized controlled trial of 3,526 patients with symptomatic or asymptomatic carotid stenosis from 95 centres in 24 countries. Participants were randomly assigned to surgery GA (n=1,753) or LA (n=1,773), 1999 - 2007. Analysis by intention to treat.
Primary outcome	The primary outcome was the proportion of patients with stroke (including retinal infarction), myocardial infarction, or death between randomization and 30 days after surgery.
Results	A primary outcome occurred in 84 (4.8%) patients under GA and 80 (4.5%) under LA; three events per 1000 treated were prevented with LA (95% CI -11 to 17; risk ratio [RR] 0.94 [95% CI 0.70 to 1.27]).
Findings	There was no definite difference in outcomes between GA and LA, nor for quality of life, length of hospital stay.

Longer Term Effects: The other Half of the Story

GALA suggests a minor long-term benefit from LA and other studies have suggested less cognitive impairment [4], health related quality of life and cancer recurrence [5]. There is further work to be done in these areas [6].

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Transoesophageal Echo for all

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Over the past twenty-five years transoesophageal echocardiography (TOE) has become an established and critical element of cardiac surgery. Cardiothoracic anaesthetists are now trained and certified in the application of TOE during surgery of the heart and great vessels, are recognized as the experts, and typically are the providers of TOE in this setting. But there is considerable and increasing interest by anaesthetists in the use of TOE during other types of surgery, often from practitioners with cardiothoracic experience who bring their TOE skills to the non-cardiac theatre, but also anaesthetists not involved with hearts cases at all. In this session we will consider some of the issues raised by this development: How can TOE be of benefit outside the cardiac theatre; what TOE skills and knowledge are needed to use TOE during non-cardiac surgery; should TOE be part of every anaesthetist's training, and if so, what needs to be included in that training?

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Renal Failure after Neonatal Heart Surgery, Prevention and Management

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As a result of advancement in surgical treatment, congenital heart disease (CHD) became one of the leading causes of acute kidney injury (AKI) in children in developed countries [1]. The incidence of AKI after CHD repair was assessed to range between 2.7 and 9%, and survival rates between 21% and 80% [2].

Previous research has focused mostly on the incidence and risk factors of severe AKI requiring renal replacement therapy (RRT). But recent studies in adult cardiac surgery showed the role of an even mild post-operative serum creatinine (SCr) rise (0.3-0.5 mg/dl (26.5-44 μmol/L) within 48 h) as an independent predictor of 30-day mortality [3,4]. An independent association was found between a small rise in SCr of 25% above baseline and length of intensive care stay and mechanical ventilation in children [5].

Neonates are at particularly high risk and young age is an independent predictor for AKI after CHD repair [2,5]. When referring to a Danish population, Pedersen et al found an adjusted OR of 4.34 for neonates requiring RRT when compared to children [2]. However, the incidence of neonatal AKI is likely to be higher than that reported, as neonates commonly have non-oliguric renal failure.

Preventive interventions in AKI have been largely disappointing. One cannot counteract all risk factors specific to CHD such as surgical complexity and consequently long CPB and cross-clamp durations, the need for hypothermic circulatory arrest (HCA), nor early age repair and perioperative multi-organ failure due to haemodynamic deterioration. Whatever has been tried, CPB flow remains non-pulsatile. Interventions such as 'renal-dose dopamine' and diuretic therapy have been shown not to alter the course of AKI. NIRS monitoring of renal perfusion seems promising but has not yet been shown to improve outcome. Early treatment of the post-operative low cardiac output is nowadays probably the best preventive intervention.

The use of large-dose aprotinin during CPB has been linked to postoperative AKI in adults. A very properly designed retrospective analysis using a propensity score was conducted at the Children's Hospital of Pittsburg and showed no association between the use of aprotinin and AKI [6]. Similarly we found no association between the need for RRT after CPB and the use of aprotinin in 116 neonates whether in a univariate analysis (up to 13% with aprotinin vs. 9% without) or when adjusting on weight, Aristotle score, length of CPB and the need for HCA.

The prognosis of severe AKI is highly dependent on the early start of RRT in adults. There is no paediatric study comparing early and late use of RRT but several publications pointed out the degree of fluid overload as a determinant of outcome [7,8], suggesting necessarily early intervention. When analysing 95 neonatal cases requiring RRT in our unit during the last decade, we noted that mortality related neither to postoperative SCr rise nor to postnatal age at operation, but to late onset of RRT, with a more than 2-fold rise in the risk of death when RRT was started after the 48th postoperative hour. Hence, our practice changed and nowadays we start RRT if urine output remains below 2 ml kg^{-1} h^{-1} during more than 4 h, when there is clinical evidence of fluid overload, when low cardiac output or metabolic complications occur despite an optimal preload, inotropic support and diuretics.

Early management requires early diagnosis. SCr is an unreliable marker for AKI and changes in SCr occur late after the onset of AKI. When analysing the postoperative SCr of all neonates we operated during two years and when considering the 28 patients requiring RRT, less than one half of them fell into RIFLE F or into pRIFLE F. Besides, in more than one half of them SCr rose after the 48th postoperative hour. New markers of AKI are needed. More than 50 articles on over 20 biomarkers of AKI were published in the last 7 years. The most promising early non-invasive biomarkers are serum and urinary neutrophil gelatinase-associate lipocalin (NGAL), urinary interleukin-18, kidney injury molecule-1 (KIM-1) and serum cystatin C. The most popular to date is NGAL; current research on NGAL emerged from CHD repair populations [9]. The systematic review by Haase et al [10] concluded that urine and serum NGAL are useful as early markers of AKI (2 h off-pump). However, the reference standard used to define AKI was a 50% increase in SCr, the number of patients was small, the measurement of NGAL was not standardized and no specific analysis was conducted in neonates. We still need larger studies based on "harder" outcomes, such as RRT requirement, cardiovascular events and death, before providing NGAL for intervention.

Data for RRT modality choice in the treatment of paediatric AKI are limited. Walters et al [11] recently overviewed the main modalities of RRT in paediatric patients: peritoneal dialysis (PD), continuous RRT (CRRT) and intermittent haemodialysis (IHD). The choice depends on the major goal of dialysis therapy, the solute clearance or primarily haemofiltration, haemodynamic stability, available resources and patient limiting factors. The only study that compared PD to CRRT after CHD repair by Fleming et al [12] gave advantage to CRRT over PD in terms of overall net urea or creatinine removal, management of fluid overload and caloric intake. The recent improvements in vascular access and control of the ultrafiltrate volume allows CRRT to be performed in haemodynamically instable patients. Surveys among US paediatric nephrologists demonstrate increased CRRT use over PD in paediatric AKI [13]. Experience is still highly limited after cardiac surgery in paediatrics. No ultrafiltration dose study has been performed in paediatric AKI., and a rate of 2000 ml min^{-1} $73m2^{-1}$ h^{-1}, an extrapolation based on IHD, was used for CRRT by many centres. Besides, a recent Polish publication found a 76% mortality rate and a 68% rate of complications in 25 patients aged 7 days to 11 yr managed with CRRT after CHD repair [14].

In the smallest neonates vascular access may not be achievable with double lumen catheters in the neck or groin and PD remains the traditionally recommended RRT after CHD repair. It allows a fluid depletion of 4ml kg^{-1} h^{-1} and a SCr clearance of 2 to 10ml min^{-1} $1.73m2^{-1}$ in the neonate [15]. Pedersen et al [16] found it feasible and safe after cardiac surgery in children, with a mortality rate of 20% and a complication rate of 1 per 12 days of PD. Our experience in neonates was similar during the last decade, with a 30-days mortality rate of about 30%. One complication occurred in every 12.8 days of PD, but when considering peritonitis, haemoperitoneum or bowel perforation they only occurred in every 45 days of PD. However, PD may be impossible in neonates with automated machinery due to tubing "dead space". Contraindications to PD include diaphragmatic hernia and recent intra-abdominal surgery, sepsis or malignancy.

Conclusion

There is clear association of early mortality and morbidity following cardiac surgery with minimal changes of plasma creatinine concentrations within 48 h and the clinician must avoid any precipitating factors for further renal impairment. When AKI occurs, the early onset of RRT is the only intervention that has been shown to improve outcome. Therefore we need to develop new AKI definitions us-

ing early biomarkers of renal injury in addition to functional markers which could ultimately predict morbidity and mortality, and early intervention. The choice between RRT methods is multifactorial, but experience with the oldest one, PD, proves safety and effectiveness in the neonate after CHD repair.

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Anaesthesia for Grown-ups with Congenital Heart Defects

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Background

Congenital heart defects (CHD) are a group of the most common birth defects, occurring in approximately 8 in 1000 births. Due to the advances in the field of paediatric cardiology, anaesthesia and surgery, about 90% of patients with CHD survive to adulthood. Estimates suggest that more grown-ups (GUCH) than children with congenital heart defects are now living in the USA.

Classifications of CHD

According to the American Heart Association (AHA), CHD can be classified into three categories:
Defects with left to right shunt: Atrial Septal Defect, Ventricular Defect, Patent Ductus Arteriosus
Cyanotic: Tetralogy of Fallot, Truncus Arteriosus, Ebstein Anomaly, Mono Ventricle, etc.
Obstruction defects: Aortic Stenosis, Pulmonary Stenosis, Coarctation of Aorta, etc.

Anaesthetic consideration and management

Even after corrective surgery, some CHD patients may remain with residual defects, which may have implications on the quality of their life and, more importantly, on perioperative management in future cardiac or non-cardiac interventions. In particular, patients with impaired or decreased ventricular function, arrhythmia or pulmonary hypertension present a considerable challenge. When dealing with GUCH, it is crucial for the anaesthetist to have insight into CHD and its haemodynamic consequences.

Preoperative assessment. Taking into consideration the underlying CHD (whether corrected or not) and the type of operation, the aim remains to provide adequate oxygenation, ventilation and a stable haemodynamic status. Preoperatively, a cardiologist should see the patients who have an implantable cardiac defibrillator (ICD) or pacemaker device for device evaluation and reprogramming. Many adults with CHD have long-life risk of infective endocarditis. This has been outlined in recent guidelines from the AHA (2007) and the European Society of Cardiology (2009).

Premedication. Premedication can be of value in managing anxiety in patients who have already undergone previous cardiac surgery. However, anxiolytic agents should be used with caution in patients with pulmonary hypertension due to the risk of hypoventilation and hypercapnia that may worsen the condition.

Anaesthetic technique. Generally, there are no specific guidelines for preferring one anaesthetic technique or agent to the other. Spinal or epidural anaesthesia is better avoided in some cases with right to left shunt. Most intravenous agents depress myocardial contractility and decrease systemic vascular resistance. Etomidate is considered to be a superior choice.

Intraoperative monitoring. Depending on the complexity of the case and the type of surgery, intraoperative invasive monitoring such as a PA catheter may be used. In addition, continuous monitoring of cardiac function with transoesophageal echo (TOE) may be required in some cases. Patients with complex lesions are preferably treated in a specialized centre with a multidisciplinary approach.

Conclusion

GUCH is a modern-day medical challenge. Optimal anaesthetic care requires excellent understanding of the pathophysiology in-

volved in the CHD with a careful selection of the anaesthetic technique and intraoperative monitoring.

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Blood Transfusion and Heart Surgery

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For the cardiac anaesthetist and those working in the cardiac intensive care environment, transfusion covers numerous areas of practice. Controversial topics still include:
- At what haemoglobin level should blood be transfused?
- How old should the blood be?

The major change in transfusion practice occurred after the publication of the TRICC trial in 1999 [1]. This trial took critically ill, stable patients who had been in an ICU for 72 hours and randomized them to either transfusion of blood or permissive anaemia. This showed that in stable ICU patients, anaemia was safe and mortality outcomes were the same. Patients undergoing cardiac surgery were excluded from this study, but much of the data has been translated into clinical practice including cardiac surgery and CICU, with many patients having operations being left anaemic. Post hoc analysis revealed a higher mortality in those patients randomized to anaemia who had known cardiovascular disease.

The outcomes for this change in practice are not fully known or understood. Most anaesthetists transfuse blood to improve oxygen delivery and whilst increasing oxygen delivery might have seen its day in the 1990s [2], in the ICU it has made a resurgence in the area of 'early goal directed therapy' [3]. One of the cornerstones of the River's papers was the transfusion of red blood cells to maintain a haemoglobin above 10 g/dL, in sick, septic, haemodynamically unstable patients. Those patients who had an improved survival had a greater RBC transfusion.

On face value the TRICC study is supported by data from three large scale retrospective propensity matched observational studies. Murphy, Koch and Engoren [4-6] have all shown that there is no benefit from transfusion of RBCs in patients undergoing cardiac surgery, even with a haematocrit as low as 21%, while the risk of death was six times higher in those patients receiving only one unit of blood.

The cardiology world has produced two conflicting database results. Wu et al. [7] reporting a Medicare administrative dataset, showed an improved outcome in patients older than 65 who received a transfusion for Hct <30% in patients admitted with acute myocardial infarction. This is almost the opposite of the outcome reported by Rao et al. [8].

It is clear that preoperative anaemia must be treated if we are to avoid unnecessary transfusions. There is no clear evidence for an optimal Hct on CPB. Postoperatively in stable, critically ill patients with few significant medical co-morbidities, haemoglobin should be allowed to drift down to 7g/dL before transfusion. This may not apply to patients who are unstable after surgery or with multiple medical co-morbidities.

Recently the age of transfused blood has come under scrutiny. A number of observational studies have suggested that 'old blood'

is bad for patients [9]. These studies need to be looked at carefully and in the cold light of day their findings may not stand up to randomized clinical trials. A major logistical dilemma that has arisen is the problem of conducting such trials since the provision of date correct blood is very difficult on an ad-hoc patient randomised basis, leaving clinicians only with observational data. When these are analysed correctly such trials do not show marked differences in outcomes related to the age of the blood transfused.

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Vasopressin in the ICU – Friend or Foe

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Vasopressin is a stress hormone and, like adrenergic agonists and renin/angiotensin, acts to maintain cardiovascular homeostasis.

Role of vasopressin in cardiovascular physiology

The cardiovascular system ensures blood kinetics (the heart as a pump) and blood distribution in order to satisfy oxygen and nutritional needs of organs. Homeostasis is then the result of two regulations, regional and systemic. Regional regulation (namely autoregulation) depends on organ oxygen demand. High metabolic demand and high oxygen extraction mean a strong autoregulation, such as the coronary circulation. Systemic regulation consists of three major components, the autonomic nervous system and two hormonal systems, renin-angiotensin and vasopressin (VP). Regional and systemic regulations may compete but strong autoregulation surpasses systemic regulation.

The systemic regulation can be observed in blood pressure control during hypovolaemia. Each major contribution to systemic regulation has been identified using specific blockers [1].

The main mechanism involved is the sympathetic system while hormonal contributions are implicated in more severe hypovolaemia or when there is sympathetic system failure. Renin-angiotensin shows a stronger effect than VP which appears as a backup. All three systems induce arterial vasoconstriction causing blood flow redistribution. Besides the arterial effect, norepinephrine and angiotensin

II cause venoconstriction, resulting in a decrease in venous capacitance, thus increasing venous return which supports cardiac output. VP is the strongest vasoconstrictor. However it has no significant venoconstrictive effect but induces improvement of venous return indirectly. VP-induced splanchnic vasoconstriction results in redistribution of blood flow from a long-time-constant vascular compartment to shorter-time-constant compartments which improves the venous return.
The short-term regulation mediated through acute vascular effects is completed by hormone-induced water and sodium-chloride reabsorption at kidney level in order to restore efficient volaemia. It is a mechanism of self-containment to avoid sustained production of VP or angiotensin II with undue vasoconstriction.

Excess of vasopressin has deleterious effects in heart failure

In chronic heart failure, low cardiac output results in a decrease in perfusion that stimulates the systemic cardiovascular regulation with activation of the sympathetic nervous system and possibly the hormonal systems. These stimuli may further alter cardiac output and perfusion, mainly by increasing loading conditions of the heart. An increased level of VP may deteriorate cardiac function through V1a receptor as well as V2 receptor action.
Plasma VP level is a proper marker for the presence and severity of congestive heart failure [2]. Generation of copeptin, that correlates with the release of VP since they are derived from the same precursor, is linked to excess mortality, and this link is maintained irrespective of the clinical signs of severity of the disease [3]. Actually, treatment of chronic heart failure consists in blockade of the sympathetic system with beta-blockers, antagonists of the renin-angiotensin-aldosterone system, and eventually, antagonists of vasopressin.

Conversely, VP deficiency has been described in critically ill patients

Inadequacy between plasma concentrations of VP and low blood pressure has been documented in septic shock [4]. A depletion of endogenous AVP stores following a vigorous stimulation of AVP release from the neurohypophysis has been suggested. Similarly lower response of vasopressin after cardiac surgery has been observed. In some cases, a vasodilatory postcardiotomy shock characterized by a decreased vascular tone has been reported with an inappropriate low-plasma concentration of the vasoconstrictor hormone arginine VP [5]. Accordingly, first clinical retrospective reports showed beneficial effects of VP therapy on cardiovascular function in cardiac surgery patients with severe vasodilatory shock that corroborates results observed in septic shock [4-6].
However, administration of VP to compensate the relative deficit is not always successful [7]. Infusion of low to moderate doses of VP in patients with norepinephrine-dependent vasodilatory shock after cardiac surgery may induce intestinal and gastric mucosal vasoconstriction [8]. Similar results have been observed in hypotensive episode after anaesthesia induction. Terlipressin, a lysine VP precursor, is effective in the treatment of hypotension episodes in anaesthetized patients chronically treated with renin-angiotensin system inhibitors. However, besides long lasting effect through slow conversion into lysine-VP, terlipressin is a fast acting vasopressor peptide per se that has an impact on the coronary circulation and myocardial function, and may induce negative effects on gastric mucosal perfusion [9,10].

Conclusion

Lessons from physiology tell us that VP is only a backup system engaged in cardiovascular regulation for short-term unstable conditions. It is a strong mesenteric vasoconstrictor with potential deleterious effects. VP treatment

cannot thus be considered as a first choice vasopressor treatment in ICU but only as a backup treatment which should be used cautiously, possibly through monitoring of VP deficiency.

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The Operation was a Success but the Patient Died – What is a Meaningful Outcome?

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Small clinical studies have dominated the anaesthetic research agenda. Anaesthesia studies focus mostly on drug pharmacology, respiratory and cardiovascular physiology, and analgesia. Severe complications leading to death or chronic disability are rare, but it is these that patients and their families are concerned about. Such low event rates have important implications for trial design.

Because most improvements in anaesthesia are modest and incremental, large numbers of patients need to be studied in order to have adequate statistical power to detect a clinically important difference in serious adverse outcomes [1-3]. Such studies require a sample size in the many 1,000s to provide sufficient statistical power and reliable estimates of effect.

The use of surrogate, or intermediate, outcome measures in anaesthesia is widespread [4]. Measurement of myocardial ischaemia rather than myocardial infarction, cardiac output rather than cardiac failure, urine flow or delta-creatinine rather than renal failure, cerebral blood flow rather than stroke, and so on. Such surrogate markers are of questionable

significance and often have no convincing relationships with patient outcome.

Surgical studies share some of the weakness of anaesthesia research, but there is a higher proportion of large case series which, in fact, do report on serious adverse outcomes. However these are nearly all non-randomized with many sources of bias. Small trials and (even) large observational studies can be misleading [4].

Small trials are prone to imbalances in prognostic factors that can have a potent effect on outcome: confounding [1,3]. A large randomized trial will equalize both known and unknown confounders between groups [1]. Large trials can provide a broad range of settings, in a variety of countries and regions, and offer an opportunity to identify other patient, clinician and institutional factors that may affect outcome.

Large trials with straightforward requirements reflecting standard practice are sometimes called *effectiveness, pragmatic, or practical* trials [3]. They thus optimize generalizability and so are clinically relevant. I will illustrate many of these issues when discussing the clinical background and study design of two of our current large trials investigating (i) nitrous oxide (www.enigma2.org.au) in 7,000 non-cardiac surgical patients, and (ii) aspirin and tranexamic acid – the ATACAS trial [6] – in 4,600 CABG patients (www.atacas.org.au). We are looking to include further study sites in the ATACAS trial.

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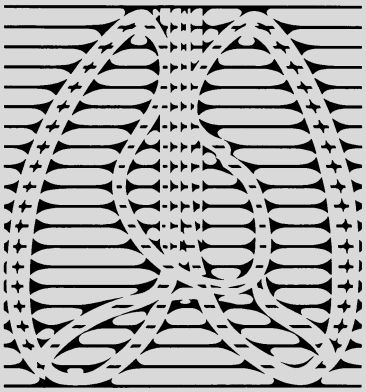

applied cardiopulmonary pathophysiology

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