Percutaneous coronary intervention, not all roads lead to Rome

The weak heart: perioperative management

Timely diagnosis of congenital heart disease – did we improve?

Regelmässige Breitkomplextachykardie bei dekrementaler Präexzitation
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Regelmässige Breitkomplextachykardie bei dekrementaler Präexzitation

Ein Patient mit wöchentlich au/f tretenden Palpitationen ...
Andreas Grüntzig Lecture at the Swiss Society of Cardiology Congress 2015 in Zurich

Percutaneous coronary intervention, not all roads lead to Rome

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Summary
Percutaneous coronary intervention (PCI) started with a first patient in Zurich, Switzerland, treated by Andreas Grüntzig on September 16, 1977. Having been part of that intervention, I enjoy the privilege of taking care of this patient since. He is still enjoying excellent health and needed only two additional percutaneous interventions in his coronary arteries after 23 and 37 years, respectively. PCI saw an unprecedented evolution to today’s role as the most common major medical intervention around the globe. As is typical for a success story, many people have been co-builders. Even more people have tried to contribute to PCI or even replace it with modifications or alternatives that do not benefit patients. This was not always recognised immediately but the only real breakthrough, the coronary stent, was finally recognised by all as the only necessary adjunct to the initial balloon. The achieved degree of perfection of PCI will make it hard, if not impossible, to improve upon it by a change of paradigm, while small adaptations will continue to be introduced because they do not need randomised trials for approval.

Key words: percutaneous coronary intervention; coronary artery disease

How it all began
The first diagnostic arterial procedure goes back to Stephen Hales. In 1726 he measured live arterial blood pressure in a supine horse using a brass glass tube assembly held erect to assess the maximum height of the systolic blood column. It was 241 cm, corresponding to 186 mm Hg. The introduction of X-ray by the Zurich University graduate Wilhelm Conrad Röntgen in 1895 allowed the first contrast angiogram, performed in 1896 in a human cadaver hand by Victor Teichmann with a mercury containing contrast medium. Werner Forssmann introduced human cardiac catheterisation by conducting a self-experiment in 1899. He inserted a thin rubber catheter, used at the urology department he was working for, from a cut-down in his left cubital vein into his right atrium. This was not only the first invasive procedure in cardiology but also an ambulatory one as he walked to the X-ray department to shoot what became a historical picture: the catheter with its tip residing in his heart. Diagnostic cardiac catheterisation was born and was first used for pressure measurements by André Cournand. They were endowed jointly with the Nobel prize in 1956.

Percutaneous coronary interventions (PCIs) were a logical extension of selective diagnostic coronary angiography, first performed by Mason Sones in 1958. Although this ground-breaking diagnostic study occurred inadvertently when an end-hole catheter used for a contrast aortogram slid into the ostium of the right coronary artery, and in spite of the fact that prolonged but self-limiting asystole occurred in the patient, it laid ground for what we know today as coronary angiography. Active coughing by the patients initially bridged asystolic phases after each coronary injection until the advent of modern contrast media no longer inducing bradycardia. It took seasoned angiographers years to break the habit of having the patient cough after each injection. More recent endeavours to replace classical coronary angiography by contrast examinations with computed tomography (CT) or magnetic resonance imaging (MRI) have created significant industrial volume without, however, advancing the field. The unsurpassed ticket conventional coronary angiography with ad-hoc PCI prevails and is performed in millions of patients around the globe every year.

PCI, the most significant Swiss contribution to medicine of all time
Based on Mason Sones’ coronary angiography technique, the possibility to access the systemic circulation with larger catheters through the femoral arteries introduced by Melvin Judkins, and a catheter-based technique of improving arterial vessel stenosis by dilating them with catheters of increasing diameters put forward by Charles Dotter, Andreas Grüntzig developed balloon angioplasty which was initially called percutaneous transluminal angioplasty (PTA). In contrast to Werner Porstmann, who was also working on this idea in Berlin, East Germany, Grüntzig found a solution to render balloons form-constant and pressure-resistant. Latex balloons of the time could not withstand pressure. They rather grew in size. Porstmann tried to con-
that by longitudinally splitting a plastic catheter and inflating the balloon inside it. While this did resist pressure in a usable oval shape, balloon deflation resulted in catching the dissected arterial wall while the slits were closing. Making the slits larger resulted in protrusion of balloon segments. Grüntzig found help from a retired plastics expert at the Technical University just across the street from the University Hospital of Zurich where Grüntzig was working in the Division of Angiology. Polyvinyl chloride could be heat treated to meet the requirements for balloon angioplasty. There was no industrial production site at hand, so Grüntzig used his kitchen stove to produce prototypes which then were directly used in humans. This shortcut approach was still possible at that time and certainly to the benefit of the method and the patients. PTA appeared less traumatic for the patients than the Dotter technique, and it was. Grüntzig had also tried a stiff wire with an oval bend close to the tip. He connected it to a household drill for rapid revolution and pulled it backwards through a narrowing, executing some thrombectomy or atherectomy with the thrashed material being embolised to the periphery. This was a dog experiment and never made it to human use. Being promoted to a staff position in cardiology in what appears to be the world’s speediest career of any cardiologist, i.e., virtually no training in cardiology, miniaturisation of this equipment and application to the coronary arteries became first priority. This required industrial machines and the Schneider company took over the balloon catheter production. The first coronary balloon for what was initially going to be called percutaneous transluminal coronary angioplasty (PTCA) and subsequently percutaneous coronary intervention (PCI) was not fed over a guide wire like the peripheral dilatation balloons. As fluoroscopy resolution was poor at that time and still frames or even replay were not a feature yet, Grüntzig needed distal pressure measurements for immediate assessment of the effect of PCI. Only the processed 35 mm cinefilm had adequate resolution to see the lumen and dissection flaps. However, it was highly unpractical to repeatedly stop the procedure for processing the cinefilm, which took about 20 minutes. In addition to the lumen for pressure measurement, a lumen for balloon filling was required and there was no room for feeding a wire through the shaft on top of that. This balloon was not steerable, but other than that it could still be used nowadays, at least for a proximal PCI. While the balloon was tested in dogs and ready in early 1976 for a first human case, it was not until September 16, 1977 that PCI was finally performed in a patient for the first time worldwide. Even several trips to the United States (USA) to find a first patient had been futile. At the time, both in the USA and, much more, in Europe, indications for coronary angiography were very restrictive. Patients usually had to have experienced long periods of angina pectoris refractory to multiple drugs, which usually included a history of one or more myocardial infarctions, before they were considered for invasive work-up. Just about never did this then reveal single-vessel disease, let alone a proximal discrete stenosis such as Grüntzig was looking for. Grüntzig was just returning from another trip to San Francisco, California, USA, again quite frustrated at not finding a suitable patient, when I presented him with a 38-year-old man (the same age as Grüntzig’s), who had been suffering from daily angina attacks for several weeks, was put on a bicycle ergometer (not quite what would be done today), and was found to have severe ST-segment elevation, ventricular tachycardia, and chest pain during exercise. An exception was made and he was subjected to coronary angiography revealing a single proximal stenosis of the left anterior descending coronary artery (LAD). Coronary artery bypass grafting (CABG) was scheduled, but I was the resident responsible for him and I had a better idea. Grüntzig was utterly pleased with the case and took me to the patient on the spot to obtain oral consent. Quite bluntly he told the patient that he was going to try something that had never been done in humans but worked in leg arteries and had also worked in dogs, and that there was a risk that immediate CABG would become necessary if it did not work out. The patient still recalls this conversation and how he immediately trusted Grüntzig and found nothing wrong with at least a reasonable chance to avoid open chest surgery. The procedure was performed the next day, as Grüntzig had obtained the necessary nods from the head of his department and the head of the Department of Cardiac Surgery long ago. The crowd attending was not really a crowd but rather a handful. Surgical stand-by (drop-by actually) was inaugurated with that case as two cardiac surgeons dropped in and out during the procedure. The patient tolerated the balloon inflation well and the lesion was remedied, yielding what would nowadays be called a stent-like result. The patient left the catheterisation laboratory with a transient right bundle branch block which recurred during a thallium exercise stress test two days later, which also revealed some, but improved, ST-segment elevation and reduced thallium uptake. It was thought at the time that this was a normal finding after PCI. I was never to see anything like that again and I doubt that Grüntzig was. The patient is still alive and well, now 76 years old. He had two additional episodes.
of chest pain, one 23 years after the procedure requiring first a bare metal stent (BMS) close to the initial lesion and a few months later a re-dilatation for in-stent restenosis, and one at 37 years requiring a drug-eluting stent (DES) again in the vicinity of the initial lesion plus one in the proximal part of the right coronary artery. Figure 1 shows the milestone angiograms of this historical patient. Exercise stress tests the day after this last procedure and 6 months later were normal and the patient still enjoys life without physical restrictions. While he had not taken any drugs for decades after his first procedure, he now accepted the idea of a lifelong single antiplatelet compound and a statin, in spite of his conviction that drugs taken chronically lost their effect.

The indications encompassed from the beginning primarily single vessel disease, more rarely double vessel disease, and only exceptionally triple vessel disease with a maximum of three to four discrete proximal coronary lesions. The left main stem was initially included but then banned for many years for all the wrong reasons. Two of the initial left main stem patients died within a few months of follow-up. Although their lesions were found patent at autopsy and the reasons for their demises remained unclear, it was assumed that PCI for the left main stem was not safe. Now with stents, in particular the refined DESs, left main stem indications have returned for good. Randomised trials between PCI and CABG have confirmed iteratively that indications were appropriate from the

Figure 1

PCI number 1 in the world (a man, 38-year-old at the initial and 75-year-old at the latest procedure). The pictures have to be read in chronological order first left top to bottom, then centre to bottom, and finally right top to bottom. The lesion shown on April 10, 2000 in an angiography indicated by angina would normally have been a perfect indication for PCI to me. In this particular case I preferred to refrain from angioplasty, not to spoil the so far perfect story, albeit the lesion was slightly more proximal than the original one. The patient kept his symptoms and insisted on having treatment. Thus the angiogram was repeated on September 9, 2000. To find an excuse again not to dilate, I performed the only clinical fractional flow reserve (FFR) measurement in my career and used the normal result to convince the patient that he needed no treatment. However, he insisted and got a reasonably good balloon angioplasty result which I would have accepted had he not insisted on a stent. It took only a few weeks for an in-stent restenosis to develop (drug-eluting stents [DESs] were not yet available) which had to be re-dilated. Another 14 years later a new lesion somewhere in-between this stent and the original site caused angina again and was stented, this time with a DES. At the same time the right coronary artery also received a DES (not shown).
beginning and that pushing the envelope to advanced and highly complex multivessel disease was beyond the scope of PCI and still is [1, 2]. On the other hand, reports advocating a conservative attitude in early coronary artery disease [3, 4] are misleading, as exemplified by figure 2. In particular, the use of fractional flow reserve (FFR) for clinical indication may not be best for the patient. With a pathological FFR, PCI will ensue and the FFR measurement could have been spared. With a normal FFR annulling the indication for PCI, a chance for at least symptomatic if not prognostic improvement may get missed (fig. 3) [5].

Alternatives and complements to PCI

Table 1 depicts what has emerged, has been tested, and has at least temporarily been adopted as replacement for or complement to the balloon catheter since the inception of PCI. In the 1980s, almost simultaneously coronary stents were introduced mainly in Europe and atherectomy devices mainly in the USA. It was immediately apparent to the critical user of both so-called new devices that the stent had tremendous potential. It remedied the worst flaw of PCI, namely obstructive coronary dissection with subsequent abrupt vessel

<table>
<thead>
<tr>
<th>Table 1: Equipment for percutaneous coronary intervention.</th>
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<tr>
<td><strong>Standard</strong></td>
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<tr>
<td>Balloons</td>
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<tr>
<td>Guide wires</td>
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<td>Guiding catheters</td>
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<tr>
<td>Stents (drug eluting)</td>
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<tr>
<td>X-ray</td>
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<tr>
<td>Contrast medium</td>
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<tr>
<td>Heparin</td>
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<tr>
<td>ADP (and GP IIb/IIIa) antagonists</td>
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<tr>
<td>Aspiration catheters</td>
</tr>
<tr>
<td>Femoral plugs or sutures</td>
</tr>
<tr>
<td>Percutaneous ventricular assist devices</td>
</tr>
<tr>
<td><strong>Optional</strong></td>
</tr>
<tr>
<td>Distal protection devices</td>
</tr>
<tr>
<td>Rotablation</td>
</tr>
<tr>
<td>Drug eluting balloon</td>
</tr>
<tr>
<td>Absorbable stents</td>
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<tr>
<td>Optical coherence tomography</td>
</tr>
<tr>
<td>Intravascular ultrasound (2 or 3 dimensional, Doppler)</td>
</tr>
<tr>
<td>Fractional flow reserve</td>
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<tr>
<td>Direct thrombin inhibitors</td>
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<tr>
<td><strong>Obsolete</strong></td>
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<tr>
<td>Directional atherectomy</td>
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<tr>
<td>Brachytherapy</td>
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<tr>
<td>Drugs against restenosis</td>
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<tr>
<td>Angioscopy</td>
</tr>
<tr>
<td><strong>Not recommended</strong></td>
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<tr>
<td>Laser</td>
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<td>Stem cell therapy</td>
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Sequence within category as per importance. ADP = adenosine diphosphate; GP = glycoprotein.
Closure. Atherectomy remedied nothing but promised a better long-term result. The promise did not materialise. An exception is rotablation, which to the present day solves problems where the balloon cannot pass or fails to crack a circumferentially calcified lesion. These cases, however, have become exceedingly rare with the ever lower profiles of balloon catheters and their ever growing pressure resistances (currently more than 40 bar).

The reason why it took a while until the superiority of the stent over other new devices was also recognised in the USA lay in one of the many misinterpretations of data in the history of PCI. As stents were initially used exclusively as bailout devices, the outcome of stented patients was significantly worse than the outcome of elective PCI patients without stents [6]. Laser and atherectomy on the other hand were used electively and preferably in patients with large coronary vessels, which were less prone to abrupt closure. Although their outcome was not better than balloon-alone treatment, it was superior to the stent results of the bail-out patients. Looking at when stents and other new devices were used during live-cases and what their results were [7] and a publication promoting rational behaviour [8] helped to eventually set things straight.

Irrational use of drug-eluting stents

When DESs were introduced in the first years of this millennium, another misinterpretation of data led to irrational behaviour for many years [6]. Initially, things were alright when the drastic reduction of restenosis was highlighted and only the stiff price prevented people from using DESs exclusively. Then a bane of the early DESs that had been predicted [9] was picked up and reported out of context, and its proportion and importance were tremendously blown up. The early DESs were apparently somewhat overdosed and only permitted a very thin tissue coating of the stent struts over time. This then allowed erosion of the coating after the first year of follow-up leading to a stent thrombosis rate over the subsequent years at around 1% per year which was significantly higher than that of BMSs. While it was correct to point that out and caution about intensifying long-term antiplatelet treatment in patients with DESs, it was erroneously assumed that this also pertained to the early period. In fact, during the early period even the first generation DESs had a significantly lower stent thrombosis rate than BMSs, irrespective of the intensity of antiplatelet therapy. So for the past almost 10 years, patients with a high risk of stent thrombosis (poor compliance with antiplatelet drugs, need for interruption of antiplatelet therapy for planned surgery soon after PCI, etc.) typically received a BMS when they should have received a DES. Even the lower stent thrombosis rate of BMSs after the first year did not compensate for the higher stent thrombosis rate with BMSs during the first year for many years of follow-up. Moreover, current generations of DESs have an even lower stent thrombosis rate during the first year, again irrespective of the intensity of antiplatelet therapy, and even increase that advantage over BMSs during long-term follow-up (fig. 4) [10].

The current use of modern DESs in all indications on the basis of a flurry of data comparing DESs to BMSs in various settings, affords a demonstrable survival benefit with PCI compared with medical treatment for the first time in the history of PCI [11]. The initial PCI before the introduction of the BMS had been close to that goal but then the use of BMSs temporarily worsened outcome owing to the new problem of stent thrombosis. Initial DESs improved it somewhat but only current DESs achieved a significantly reduced mortality.

Future modifications of PCI

It is uncontested that more stringent control of risk factors by behavioural measures and statins reduces the age-corrected prevalence of coronary artery disease. Nonetheless, with the ever increasing average age of western populations, demand for coronary revascularisation will stay high. Early coronary angiography is already a standard. Alternative imaging techniques

![Figure 4](https://example.com/figure4.png)
such as CT or MRI can easily be skipped, as coronary angiography is nowadays available instantly virtually everywhere and its risk, amount of contrast medium bearing some renal toxicity, X-ray exposure, and overall inconvenience are comparable to if not lower than that of CT. This is independent of whether the femoral or the radial approach is selected. In fact, the femoral approach should remain the standard, as the radial approach is fraught with a chronic occlusion rate of the used radial artery of about 5–10% [12]. Smaller catheters would improve on that but they make the radial technique even more intricate and the learning curve for operators even flatter. It does not appear justified to impose the significantly longer learning phase, the need for crossovers, and the risk of permanently occluding the radial artery on patients unless they demand it fully cognizant of these downsides.

Another myth is that absorbable stents, currently referred to as bioabsorbable vascular scaffolds (BVSs), improve the late outcome in terms of reduction of late stent thrombosis, resumed normal vasoactivity of the coronary artery, and normalization of future access points for CABG. First, the current models are far from the ideal absorbable stent that would disappear within a few weeks without any local inflammation and without having chunks not opposed to the wall (e.g., overstented side-branches), embolising distally during the resorption process. They are also far from the ease of use, visibility, hoop strength, and economical production costs of current DESs. Using a current BVS implies struggling during implantation like in the early BMS years, risking a higher early complication and restenosis rate, and hoping for a completely normalised vessel in the future. The latter is as unlikely to happen as the reconstruction of necrotic myocardium by injection of stem cells meant to not only reconstitute muscle that align perfectly but also to rebuild their vascular support and electrical connectivity. Why not admit that we have reached a standard of PCI that may still be several percentage points below perfection, but that is hard to improve upon. Things that might look promising while being on the horizon are unlikely to prevail, be that because of lack of superiority or just because of the lack of proof of it. Small as any further advantage can only be, randomised trials with thousands of patients and prolonged follow-up will be required to prove them and paying for that may not be attractive and not even make sense. Once arrived in Rome you can easily cope with the fact that not all roads lead to Rome.

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References
The weak heart: perioperative management

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Summary

Heart failure (HF) is known to be a major risk factor in perioperative care. It should be subdivided into systolic or diastolic dysfunction as well as left or right ventricular failure. The perioperative management of HF patients is complex, consisting of prevention, diagnosis and therapy. The adequacy of the perioperative management determines the late postoperative outcome and will be presented in this review.

Key words: heart failure; perioperative management; anaesthesia

Introduction

Epidemiology

Heart failure (HF) has an incidence of 1 to 2% in the general population [1]. Its prevalence increases further with age, rising from approximately 2% in individuals 65–69 years old to >8% in those ≥85 years [2]. The mortality rate of systolic HF is 14% at 1 year and up to 50% at 5 years, when symptomatic [2–4]. The average duration of survival after one hospitalisation is 2.4 years and drops to 0.6 year after the fourth hospitalisation [5]. About 25–40% of patients with congestive HF have a preserved ejection fraction and suffer from diastolic failure; their mortality is 50% lower than in the case of reduced ejection fraction [4].

Heart failure as a perioperative risk factor

HF is known to be a major risk factor in perioperative care and is found in 2.5 to 10% of noncardiac surgical patients [6–8]. The incidence of severe cardiac events after major noncardiac surgery is between 2 and 8% [6, 9, 10]. In cardiac surgery, the incidence rises to over 20% [11]. In vascular surgery, it is 18% in the case of isolated diastolic failure, 23% in the case of asymptomatic systolic insufficiency and 49% in the case of congestive systolic failure [12]. In a retrospective study including 1532 HF patients and 1757 coronary artery disease (CAD) patients undergoing major noncardiac surgery, the risk-adjusted mortality within 30 days was 11.7% in HF, 6.6% in CAD and 6.2% in control patients. The risk-adjusted 30-day readmission rate was 20% for HF, 14.2% for CAD and 11% for control patients [13]. HF patients undergoing major noncardiac surgery suffered substantial morbidity and mortality despite advances in perioperative care, whereas patients with CAD without HF had similar mortality to a more general population [10, 13, 14]. It is not only the perioperative cardiac risk which is high in HF patients but also the noncardiac complication incidence is higher than in patients without CAD or HF, as shown recently [14]. In this retrospective series, a near doubling of postoperative death was reported in HF patients, as well as a 40 to 69% increased risk of sepsis, and pulmonary and renal complications, but not of myocardial infarction. Perioperative management may have been focused on preventing myocardial ischaemia at the expense of other organ systems.

Interestingly, the newly published European Society of Cardiology (ESC) / European Surgical Association (ESA) guidelines on noncardiac surgery define the perioperative cardiac risk as follows: “cardiac complications can arise in patients with documented or asymptomatic ischaemic heart disease, left ventricular dysfunction, valvular heart disease and arrhythmias, who undergo surgical procedures that are associated with prolonged haemodynamic and cardiac stress” [15]. We may wonder why the role of right ventricular failure (RVF) in perioperative cardiac risk is so neglected. There is a plethora of literature and guidelines on the perioperative management of patients with coronary artery disease and left ventricular failure (LVF), but until now, there is a lack of guidelines on the perioperative management of the patient with RVF. However, RVF has been clearly associated with increased mortality among cardiac surgical patients as well as in the non-cardiac setting and in the intensive care unit (ICU) [16]; it is present in approximately 40% of postcardiotomy cardiogenic shock [15, 17, 18].

Risk assessment

Preoperative risk assessment is essential, considering the risks of surgery (emergency, type, invasiveness, duration and potential blood loss), and the risks of the patient, considering their personal history (history of
ischaemic cardiac disease, HF, cerebrovascular disease, insulin-dependent diabetes mellitus and impaired renal function) and their functional capacity (in metabolic equivalents, METs) [19]. Many different indices have been designed since the Revised Cardiac Risk Index of Lee [20], but their accuracy and concordance are limited [21]. According to the 2014 ESC/ESA guidelines, patients with established or suspected HF scheduled for noncardiac intermediate or high-risk surgery should undergo transthoracic echocardiography and/or assessment of natriuretic peptides, and should be therapeutically optimised as necessary, using beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, mineralocorticoid antagonists and diuretics [15]. In cardiac surgery, indicators of major clinical risk in the perioperative period are: unstable coronary syndromes, decompensated HF, significant arrhythmias and severe valvular disease [19]. The EuroSCORE II is currently used for perioperative risk assessment in cardiac surgery [22].

**Definition and diagnosis of heart failure**

**Definition**

HF is a functional and structural impairment of ventricular filling and/or ejection, leading to a failure of oxygen and nutrient delivery at a rate in accordance with the requirements of the tissues. It should be subdivided into left and right ventricular failure as well as systolic or diastolic dysfunction. Diastolic dysfunction resulting in depressed filling of the ventricle is mostly a disease of the left ventricle. Systolic dysfunction results in poor ventricular ejection and affects both ventricles. Whereas diastolic failure may occur without overt systolic failure, the latter is always accompanied by diastolic failure. Basically, five myocardial mechanisms lead to HF: arrhythmias, pressure overload, volume overload, coronary disease and primary myocardial disease (cardiomyopathy). Clinically, acute HF presents frequently as pulmonary oedema, left/right congestive HF or cardiogenic shock with progressive end-organ malperfusion and possible failure [23]. Both the American College of Cardiology Foundation (ACCF) / American Heart Association (AHA) stages of HF and the New York Heart Association (NYHA) functional classification are useful complementary information about the presence and the severity of HF (table I) [2]. HF patients commonly suffer from comorbidities among which the 10 most frequent are: systemic hypertension (84%), ischaemic heart disease (72%), hyperlipidaemia (60%), anaemia (50%), diabetes (46%), arthritis (43.5%), chronic kidney disease (42%), chronic obstructive pulmonary disease (COPD) (30%), atrial fibrillation (28.5%) and Alzheimer’s disease / dementia (28%) [19].

**Diagnosis**

The diagnosis of acute HF is based on clinical signs, echocardiography and cardiac biomarkers. Among the clinical signs are: orthopnoea, rales, abdominal discomfort, peripheral oedema, hypotension, tachycardia, oliguria, cyanosis, mottling and disorder of consciousness. Perioperative echocardiography should be performed as early as possible and will quickly provide information on regional and global ventricular function, right or left ventricular dysfunction, valvular dysfunction, tamponade and volume status. Whereas an increase in troponin level is highly correlated with postoperative major adverse cardiac events (MACE), the diagnostic role of natriuretic peptides (BNP, NT-pro-BNP) in the perioperative period remains to be demonstrated [24].

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**Table 1: Comparison of ACCF/AHA stages of HF and NYHA functional classification.**

<table>
<thead>
<tr>
<th>ACCF/AHA stages of HF</th>
<th>NYHA functional classification</th>
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<tr>
<td>A At high risk for HF but without structural heart disease or symptoms of HF</td>
<td>0 No limitation of physical activity, ordinary physical activity does not cause symptoms of HF</td>
</tr>
<tr>
<td>B Structural heart disease but without signs or symptoms of HF</td>
<td>I No limitation of physical activity, ordinary physical activity does not cause symptoms of HF</td>
</tr>
<tr>
<td>C Structural heart disease with prior or current symptoms of HF</td>
<td>I No limitation of physical activity, ordinary physical activity does not cause symptoms of HF</td>
</tr>
<tr>
<td>D Refractory HF requiring specialised interventions</td>
<td>IV Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest</td>
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ACCF = American College of Cardiology Foundation; AHA = American Heart Association; HF = Heart failure; NYHA = New York Heart Association.
Perioperative triggers of acute cardiac decompensation

In the perioperative period, HF patients face numerous triggers of acute cardiac decompensation including hypertension, tachyarrhythmias, anaemia, hypercoagulability, inappropriate fluid management, pain, surgical stress, pulmonary hypertension (PHT), pulmonary or fat emboli and myocardial ischaemia [19]. Cardiac surgery may be complicated by coronary bypass occlusion, intracoronary air embolism, paravalvular or residual valvular regurgitation, PHT, tamponade, haemo- or pneumothorax. Patients with endstage or advanced HF who are symptomatic at rest despite maximal medical therapy (stage IV) present a unique challenge for perioperative management, and are at high risk for perioperative mortality (10–30%); however, their mortality drops to 6% when they are in a compensated stage (I–II) [10]. If not an emergency, surgery should be postponed in patients with decompensated, new onset or untreated HF [13]. Anaesthetising patients with ongoing therapy for acute HF requires a good knowledge of the disease and its treatments, as well as possible haemodynamic consequences of anaesthesia [25]. An assessment of a patient’s prognosis linked to his or her cardiac disease can help to determine the role of interventions for noncardiac diseases, meaning that patients with a very poor cardiac prognosis may not survive long enough to benefit from some noncardiac procedures [26]. The optimal perioperative course of high-risk cardiovascular patients should be based on close cooperation between cardiologists, surgeons, anaesthesiologists and intensivists. Any patients with RVF or LVF poorly tolerate circulatory overload; overzealous fluid infusion could explain some forms of iatrogenic perioperative HF. If LVF patients nicely tolerate the vasodilating effects of anaesthetic agents and LV unloading with mechanical ventilation, the most vulnerable time is the period of weaning from the ventilator and extubation. In contrast, RVF patients poorly tolerate the negative inotropic effect of anaesthetic agents and the increase in right ventricular afterload resulting from mechanical ventilation; any significant drop in systemic pressure (right ventricular perfusion) or hypoxic event (intraoperative mishaps or postoperative pulmonary complications) could precipitate or aggravate ongoing right ventricular decompensation. Since right and left ventricular failure represent different entities with different management, they will be discussed separately.

Specific perioperative considerations of left ventricular failure

Premedication

In the absence of evidence-based studies, similar perioperative management can be recommended in patients with LVF with preserved ejection fraction as in patients with LVF and reduced ejection fraction. The perioperative management of LVF starts with the preoperative visit. The first step will be to identify pre-ex-
ing systolic or diastolic abnormalities of the left ventricular function and establish the diagnosis of the underlying cardiac disease (table 2). Clinical signs of left ventricular dysfunction, although nonspecific, (tachypnoea, orthopnoea, rales, legs oedema, hypotension, tachycardia, oliguria, cyanosis, mottling and somnolence), and the electrocardiogram (ECG; arrhythmias, tachycardia, signs of old or recent myocardial infarction, strain pattern, left bundle-branch block), chest X ray (cardiomegaly, pulmonary oedema), transthoracic echocardiography (TTE), coronary angiogram if relevant, as well as laboratory data (troponin, BNP or proNT-BNP, kidney and liver parameters, haematology values) will all be studied carefully. TTE is the screening investigation of choice. Individualised premedication and anxiolytic therapy is then administered, avoiding stress, but also respiratory depression and hypotension. Preoperative HF therapy other than diuretics (beta-blockers, angiotensin converting-enzyme inhibitors, angiotensin receptor blockers) should be further administered over the perioperative phase. When angiotensin converting-enzyme inhibitors and angiotensin receptor blockers are prescribed for systolic ventricular failure, the risk of hypotension during anaesthesia is much less than when they are prescribed for systemic hypertension; in this latter case, their transient discontinuation 24 hours before surgery should be considered [15].

**Intraoperative prevention**

Intraoperatively, the next step will be to prevent any worsening of left ventricular function. The haemodynamic goals in the case of left ventricular failure are to optimise left ventricular preload and contractility, to reduce afterload, and to avoid tachycardia, bradycardia or arrhythmias. Simultaneously, hypoperfusion of major organs and reduction of coronary blood flow must be prevented. Particular attention on avoiding overdose of drugs should be kept in mind, particularly during induction, as the patient’s sensitivity is high and circulation time is slow. Frail patients maintain an acceptable cardiac output only within very restricted limits at rest, but have lost all physiological reserve in case of increased demand such as during surgery. Therefore, it is of the utmost importance to maintain rigorously a stable haemodynamic status, to immediately correct any significant deviation, and to equilibrate cardiac output with metabolic requirements. Major surgery can be successfully undertaken in patients with a depressed haemodynamic condition as long as no intercurrent complication supervenes. Oxygen debt, acidosis and hypoperfusion intraoperatively will lead to multiple organ failure appearing later in the postoperative course. Fatal issue usually happens after a few days of intensive care, which may leave the worst impression that anaesthesia management is not involved in this dismal outlook.

A recent meta-analysis showed that goal-directed therapy (GDT) in high-risk surgery is beneficial in reducing cardiovascular events (odds ratio 0.54), irrespective of the choice of monitored physiological parameter or haemodynamic monitor in use [27]. The benefit was most pronounced in patients receiving fluid and inotropic therapy to achieve a supranormal oxygen delivery target, with the use of minimally invasive cardiac output monitoring. However, a Cochrane review found no differences in the rate of arrhythmias, myocardial infarction, congestive HF or pulmonary oedema between patients treated with perioperative GDT and control patients [28]. These inconsistent results regarding clinical outcomes may be explained by poor adherence by clinicians to the protocol or the inappropriateness of the proposed algorithm in selected high-risk patients. The current evidence does not support widespread implementation of GDT to reduce mortality but does suggest that complications and duration of hospital stay are reduced [28]. Large randomised studies are needed to solve this question definitely. Until then, individualised haemodynamic goals should be defined, depending upon the patient’s characteristics and institutional preferences, considering optimisation of ventilous saturation (central or mixed), blood lactate, stroke volume index and/or cardiac index.

**Monitoring**

The choice of monitoring for these patients should be discussed. In addition to the standard ASA surveillance, extended haemodynamic monitoring must be adapted to the LVF patient and to the type of surgery. An arterial catheter and a multilumen central catheter will be necessary in most cases. The current transoesophageal echocardiography (TEE) guidelines recommend the use of TEE in noncardiac surgery if severe haemodynamic, pulmonary and neurological complications are anticipated, or in the case of life-threatening circulatory instability unresponsive to conventional interventions [9, 29]. It is recommended in any open-heart surgery and may be considered for coronary artery bypass surgery. The authors believe that TEE is mandatory in any perioperative case of LVF, except in the case of absolute contraindications. Details on the TEE assessment of left ventricular function can be found elsewhere [30, 31]. There is no convincing evidence for the use of a pulmonary artery catheter (PAC) in perioperative patients during noncardiac surgery. In a case-control analysis,
the perioperative use of a PAC was associated with a higher incidence of postoperative HF and noncardiac events than in a matched control group [32], and a meta-analysis of 13 randomised studies showed a null effect of PAC on outcome, when used in a random fashion during major surgery among critically ill patients [33]. However, these negative results do not preclude the use of PAC in selected cases. The use of less invasive perioperative cardiac output monitoring techniques (PiCCO™, Oesophageal Doppler, Flow-Track™ Vigileo) could be associated with a reduction in length of stay and complications [34], but large randomised studies are still lacking [15]. It should be reminded that the impact of a monitoring device on clinical outcome relies entirely on the interpretation of the data made by the physician and on the therapeutic algorithm implanted in the institution, but not on the technique itself.

Choice of anaesthesia

In the general population, the choice of the anaesthetic agent has been considered to be of little importance in terms of patient outcome, provided that vital functions are adequately supported. In compromised patients, however, induction agents are best chosen among the substances with the least haemodynamic effects. Most volatile and intravenous anaesthetic agents reduce preload, afterload and contractility, and require proper management to ensure maintenance of organ flow and perfusion pressure. Induction agents may be classified by increasing order of negative inotropic action as follows: etomidate, midazolam, propofol, ketamine, and thiopental. The key conditions for a stable induction are: a reduction of the dose according to the degree of ventricular dysfunction, and a slowdown in the rate of drug administration, completing very progressively the dose required for intubation. Opioids have no known adverse effects on the left ventricular function. In patients suffering from ischaemia-induced ventricular failure, use of halogenated gases may be advised because of their preconditioning effect [35]. In coronary artery bypass graft (CABG) surgery, they tend to improve myocardial performance recovery [36]; whether this can be extrapolated to noncardiac surgery is still debated. Intermittent positive pressure ventilation (IPPV) is well tolerated in left ventricular failure because the increase in intrathoracic pressure corresponds to a decrease in left ventricular afterload. If a neuraxial technique is chosen, the local anaesthetic agent should be introduced slowly to avoid systemic vasodilation. When the blockade reaches the fourth thoracic dermatome, a reduction in cardiac sympathetic tone may occur with a decrease in myocardial contractility, heart rate, and change in loading conditions. The ESC/ESA guidelines have estimated, however, that neuraxial anaesthesia and analgesia may be considered for the management of patients with cardiovascular risk factors or diseases [15].

Perioperative LVF management

Early identification of LVF and the underlying cardiac disease, as well as prompt and aggressive management, decrease postoperative morbidity and mortality. The search for reversible conditions is essential: myocardial ischaemia or infarction, acute valvular dysfunction, left ventricular tract obstruction with systolic anterior motion of the mitral valve, septic shock,

Table 3: Management of perioperative heart failure.

<table>
<thead>
<tr>
<th>Left ventricular failure</th>
<th>Right ventricular failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoidance of drug-induced myocardial depression</td>
<td>Avoidance of drug-induced myocardial depression</td>
</tr>
<tr>
<td>Preservation of ventricular interaction</td>
<td>Preservation of ventricular interaction</td>
</tr>
<tr>
<td>Optimisation of preload</td>
<td>Optimisation of preload</td>
</tr>
<tr>
<td>Maintenance of SR and A–V synchrony</td>
<td>Maintenance of SR and A–V synchrony</td>
</tr>
<tr>
<td>Heart rate control</td>
<td>Heart rate control</td>
</tr>
<tr>
<td>Reduction of left ventricular afterload</td>
<td>Avoidance of PHT exacerbation: hypoxemia, hypercarbia, hypothermia, acidosis, stress and pain</td>
</tr>
<tr>
<td>Maintenance of adequate systemic perfusion pressure for organ perfusion</td>
<td>Optimization of ventilator settings</td>
</tr>
<tr>
<td>Avoidance of nephrotoxic and hepatotoxic drugs</td>
<td>Maintenance of systemic perfusion pressure while minimising right ventricular dilatation</td>
</tr>
<tr>
<td>Minimisation of blood transfusion, especially of old blood</td>
<td>Minimisation of blood transfusion, especially of old blood</td>
</tr>
<tr>
<td>Tailoring of therapy to the specific aetiology of the LVF</td>
<td>Tailoring of therapy to the specific aetiology of the RVF</td>
</tr>
<tr>
<td>Reduction of right ventricular afterload, preferentially with inhalative therapy</td>
<td>Reduction of right ventricular afterload, preferentially with inhalative therapy</td>
</tr>
<tr>
<td>Inotropic support</td>
<td>Inotropic support</td>
</tr>
<tr>
<td>Mechanical assist devices</td>
<td>Mechanical assist devices</td>
</tr>
</tbody>
</table>

A–V synchrony = atrioventricular synchrony; LVF = left ventricular failure; RVF = right ventricular failure; PHT = pulmonary hypertension.
aortic dissection type I or postcardiotomy in the case of cardiac surgery must be treated appropriately. Re-suscitation measures must be undertaken immediately. Similarly to sepsis therapy, the concept of the “golden hours” for acute HF management is essential [19]. Oxygenation and ventilation should be immediately maximised and acid-base as well as electrolyte abnormalities should be corrected. Optimisation of preload, afterload and contractility (table 3), ideally under echocardiographic monitoring, and control of the heart rate and rhythm should stabilise the left ventricle. Positive inotropic agents must be used with caution, with careful consideration of their risk–benefit ratio; several studies indicate an association between prescription of inotropes and poor clinical outcome [37]. It remains of great use in patients with acute systolic dysfunction and low cardiac output, and evidence of systemic hypoperfusion or congestion. The dosage should be kept as low as possible and the possibility of weaning should be regularly assessed (table 4). Until now, no catecholamines have been shown to improve outcome of patients, except levosimendan [38, 39]. In a recent large meta-analysis considering 45 randomised controlled trials and analysing 5480 patients, levosimendan was shown to reduce mortality of adult patients in cardiology and cardiac surgery settings [40]. In a consensus of experts, levosimendan was included among eight nonsurgical ancillary drugs, techniques or strategies that might decrease mortality in cardiac surgery [41]. It has the great advantage of acting as both a positive inotrope and an afterload reduction agent, without increasing myocardial oxygen consumption. If LVF persists, mechanical assistance should be started as soon as possible and preferentially before organ dysfunction. Intra-aortic balloon counterpulsation (IABP) improves coronary perfusion through augmentation of diastolic pressure, decreases afterload and, consequently, reduces myocardial oxygen consumption and increases cardiac output. It has been the most widely used mechanical circulatory support device for nearly five decades, particularly during and after cardiac surgery. Following the results of the first large randomised, open-label trial on the use of IABP in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II), the indications for IABP have been greatly restricted [42, 43]. In the current guidelines, IABP is indicated only in cardiogenic shock complicating myocardial infarction [15]. The use of IABP in noncardiac surgery is founded on case reports and small case series [44, 45], with good results in the acute perioperative time. In cases of refractory low cardiac output syndrome, extracorporeal life support (ECLS) might become an option. It should be inserted before irreversible organ dysfunction develops, as a bridge to decision, to recovery, to ventricular assist device (LVAD) or to transplantation. The early postoperative care of LVF patients should be conducted in a high-acuity nursing environment, with invasive monitoring and requisite assessment of cardiac biomarkers (e.g., troponin and brain natriuretic peptides) [7]. Recent meta-analyses demonstrated that increased postoperative troponin and BNP concentrations after noncardiac surgery were associated with a significantly increased risk of mortality [15, 46, 47]. Preoperatively and postoperatively, patients who could most benefit from BNP or troponin measurements are those with METs ≤4 or with a revised cardiac risk index value >1 for vascular surgery and >2 for nonvascular surgery [15].

Diastolic dysfunction and failure are initially characterised by a maintained cardiac output, but by restrictive filling conditions (lack of relaxation, stiff ventricle). Elevated filling pressures, intolerance to tachycardia or bradycardia, and extreme dependence of stroke volume from preload (intolerance to hypovolaemia, large variations of arterial pressure with posi-

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**Table 4: Dose-related haemodynamic effects of intravenous inotropic agents in heart failure (modified from [2, 72]).**

<table>
<thead>
<tr>
<th>Inotropic agent</th>
<th>Dose mcg/kg</th>
<th>Dose mcg/kg/min</th>
<th>Effects</th>
<th>Adverse effects</th>
</tr>
</thead>
</table>
| Dobutamine      | N/A         | 2.5 to 5        | ↑↑↑     | ↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑→
Noncardiac surgery

- PHT: hypoxia, hypoventilation, atelectasis, high ventilation pressures, acute pulmonary embolism (orthopaedic surgery)
- Myocardial ischaemia: coronary artery disease
- Elevated LAP: mitral valve disease, systolic or diastolic LVF
- LVAD
- Lung transplantation
- GUCH
- Cardiac surgery
- Myocardial ischaemia or infarction
- Inadequate myocardial protection, intracoronary air embolism
- RV diastolic dysfunction associated with abnormal interventricular septal motion
- PHT: CPB, protamine reaction, acute on chronic PHT
- LVAD
- HTPL
- GUCH

CPB = cardio-pulmonary bypass; GUCH = grown-up congenital heart disease; HTPL = heart transplantation or LVAD implantation are at higher risk.

Specific perioperative considerations of right ventricular failure

The anaesthesiologist will be confronted with patients with right ventricular failure in various circumstances (tables 2 and 5):

- In noncardiac surgery, perioperative right ventricular failure is most often, although not exclusively, secondary to acute PHT; a normal right ventricle can cope for only 1–2 hours with a mean positive airway pressure of ≥40 mm Hg [49].
- In cardiac surgery, right ventricular failure may be secondary to acute PHT, but also frequently to volume overload, myocardial ischaemia, preexisting right ventricular dysfunction or arrhythmias. Among cardiac surgery patients, those undergoing cardiac transplantation or LVAD implantation are at higher risk.
- Grown-up congenital heart disease (GUCH) patients, for cardiac or noncardiac surgery.

Premedication

The perioperative management of right ventricular failure, like the management of left ventricular failure, consists of several successive steps, starting with a preoperative visit. The first step is to identify preexisting abnormalities of the right ventricular function and of the pulmonary vasculature, knowing that perioperative risk factors for right ventricular decompensation include a preexisting right ventricular dysfunction, or severe PHT without right ventricular dysfunction. A thorough history and clinical examination is required. Clinical signs of right ventricular dysfunction, although nonspecific (dyspnoea, hypotension, right upper quadrant discomfort, ascites, jugular vein distension), and ECG (situs tachycardia, T-wave inversion in III and aVF or V1 to V4, right bundle-branch block, rightward axis), chest X-ray (pulmonary artery, right atrium and right ventricular dilation), transthoracic echocardiography (TTE), right heart catheterisation in cases of moderate to severe right ventricular dysfunction combined with severe PHT [50] and laboratory values, must all be studied carefully. TTE is the screening investigation of choice. Individualised premedication and anxiolytic therapy is then administered, avoiding stress, but also respiratory depression with the risk of secondary PHT. Preoperative chronic PHT therapy should be further administered over the perioperative period.

Intraoperative prevention

Intraoperatively, the next step will be to prevent any aggravation of possible pre-existing RV dysfunction. In this view, any increase in pulmonary vascular resistance (PVR) and right ventricular myocardial ischaemia, which exacerbate each other, should be avoided. The haemodynamic goals are to maintain right ventricular preload and contractility, minimising the PVR and avoiding right ventricular coronary hypoperfusion.

Monitoring

As for the patients in left ventricular failure, an arterial catheter and a multilumen central venous catheter will be useful, particularly in the case of IPPV. Intraoperative TEE is mandatory in all patients with RVF, except in the case of absolute contraindication [51]. Details on the TEE assessment of right ventricular function can be found in previous publications [52–54] as well as on the ASE website (www.asecho.org).

A PAC may be indicated in case of severe PHT and RVF, but should be used with caution considering the risk for arrhythmias and catheter-induced pulmonary artery rupture [55]. The measurement of cardiac output may be altered in the presence of tricuspid regurgitation. The use of continuous right ventricular pressure waveform monitoring might be helpful, as described by Denault et al. [16] (fig. 1). In cases of right ventricular dysfunction, a progressive change in the diastolic pressure slope from horizontal to obliquely ascending will...
be observed. As right ventricular function deteriorates, the slope will change to a square root shape, and finally right ventricular and pulmonary diastolic pressures will equalise [66]. Combining right ventricular pressure waveform and TEE monitoring allows rapid determination of the cause of right ventricular systolic and diastolic dysfunction.

**Choice of anaesthesia**

The choice of anaesthetic is equally relevant in the prevention of RVF. The depth of anaesthesia and postoperative analgesia should be sufficient to avoid large sympathetic haemodynamic responses to pain and surgery. Volatile anaesthetics may all worsen right ventricular dysfunction by reducing preload, afterload and contractility. The PVR is increased by both desflurane and nitrous oxide [56, 57]. Ketamine seems to increase PVR in adults but not in children [58, 59]. Although the potential deleterious effect on PVR remains a concern, it must be balanced against the potential benefits of combined analgesia and hypnosis and its absence of significant myocardial depression and vasodilation [60]. Etomidate has been advocated as the induction agent of choice although there is no comparative data [61, 62]. Opioids have no known adverse effects on the right ventricular function. If a neuraxial technique is chosen, a local anaesthetic agent should be introduced slowly to avoid inconsiderate systemic vasodilation.

**Perioperative RVF management**

The third step consists of early identification of RVF. The functional state of the right ventricle cannot be determined from the severity of PHT alone. In a patient with known PHT developing RVF, the PAP will pseudo-normalise as right ventricular function fails. In other words, while a falling PAP may be due to a reduction in PVR or left atrial pressure (LAP), it might also be a sign of a failing right ventricle that cannot build up the pressure any more. Conversely, an increase in cardiac output in the face of a high, relatively fixed PVR will increase the PAP. In cases of progressive RVF, PAP may appear normal but right atrial pressure (RAP) will be elevated. Both systemic and pulmonary artery pressures will be reduced to a similar degree; the ratio of systemic to pulmonary mean arterial pressures may better reflect the severity of PHT. In cardiac surgery, this ratio has been shown to be a better predictor of postoperative complications than the absolute values [63]. A search for possible underlying reversible conditions is essential: pulmonary embolism, lung infection, bronchial asthma, COPD, ventilation/perfusion mismatch, valvulopathy, myocardial ischaemia, intracardiac shunt and left ventricular diastolic or systolic dysfunction can all contribute to RVF.

Finally, in presence of progressive RVF, standard therapy is initiated (table 3), keeping in mind that one of the main goals of management is the maintenance of the ventricular interaction. Right ventricular function is significantly reduced if the septum is dysfunctional; the maximum right ventricular developed pressure is reduced by 30% when the septum is inactivated [64]. Conditions that reduce the left ventricular systolic pressure or increase the right ventricular systolic pressure reverse the trans-septal gradient and lead to further RVF [65, 66]. Although the right ventricle is highly preload-dependent, overfilling can cause right ventricular dilation and secondary tricuspid regurgitation with a resulting increase in right ventricular wall stress, decrease in left ventricular compliance and progressive reduction of cardiac output, leading to further right ventricular dilatation (fig. 2). Preload is optimised, observing the effect of a volume tolerance test on central venous pressure, PAP and right ventricular filling (TEE/TTE). However, pulse pressure variation cannot be used for fluid assessment in patients with RVF, as pulsus paradoxus might result from RVF and ventricular interdependence [67, 68]. Sinus rhythm is maintained as well as a heart rate of at least 90/min. The right ventricular afterload is decreased, initially using

![Figure 1: Zoomed right ventricular pressure (P_{RV}) and pulmonary arterial pressure (P_{PA}) with their corresponding Doppler hepatic venous flow (HVF) before (a, b) and after cardio-pulmonary bypass. Note the change in the diastolic slope of the P_{RV} waveform and the corresponding change in the systolic (S) to diastolic (D) ratio of the HVF. From: Denault AY, Haddad F, Jacobsohn E, Deschamps A. Perioperative right ventricular dysfunction. Current opinion in anaesthesiology. 2013;26(1):71–81, reprinted with permission.](image-url)
REVIEW ARTICLE

FiO², mild hyperventilation, alkalinisation, and adaptation of ventilation settings; the relationship between tidal volume and PVR has a unique "U" shape, PVR being minimal at functional residual capacity and maximal in the event of hypoventilation or hyperinflation [52, 53]. Any sympathetic stimulation such as stress, pain, hypothermia and shivering is suppressed, and anaesthetics potentially associated with augmentation of PVR are avoided. If these diverse measures do not allow a stabilisation of the right ventricular function, pulmonary vasodilatation is then started, preferentially using inhalational substances such as nitric oxide (NO), iloprost or milrinone, to avoid systemic vasodilation. Because of their different modes of action, combination of inhalational vasodilators might be synergistic [50, 62]. Keep in mind that the key haemodynamic sign of a therapeutic response to inhaled pulmonary vasodilators is not a reduction in pulmonary artery pressure but a decrease in CVP and an increase in cardiac output [62]. Considering further ventricular interdependence [69, 70] as well as the coronary perfusion of the right ventricular myocardium, the systemic arterial pressure is maintained elevated to avoid right ventricular myocardial ischaemia, using vasopressors as needed (noradrenaline, vasopressin). The benefit of systemic vasoconstriction has to be balanced with the risk of pulmonary vasoconstriction; fortunately, the pulmonary arterial tree has fewer alpha-receptors than the systemic arteries and is devoid of receptors for vasopressin [50]. Finally, inotropic agents are deployed as needed: dobutamine, adrenaline, phosphodiesterases III inhibitors (milrinone) or levosimendan have all been shown to be effective in case of acute RVF. However, as levosimendan and milrinone both produce systemic vasodilation, vasopressors may need to be coadministered to prevent reduced right coronary blood flow [62]. Increased left ventricular contractility can also result in increased right ventricular systolic function through ventricular interdependence.

In cases of persistent refractory RVF, mechanical support with a ventricular assist device should be initiated, preferentially before the appearance of irreversible organ dysfunction.

Conclusion

In conclusion, periooperative heart failure is the result of inadequate contractility, arrhythmia, volume or pressure overload and is associated with worse outcomes in noncardiac and cardiac surgery. Particular care has to be taken of the right ventricle. Prevention, diagnosis and therapy are difficult tasks for the team managing the patient, including anaesthesiologists, cardiologists, surgeons and intensivists. The adequacy of the periooperative management determines the late postoperative outcome.

Disclosure statement

No financial support and no other potential conflict of interest relevant to this article were reported.

References

The full list of references is included in the online version of the article at www.cardovasmed.ch.

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Figure 2: Right ventricular dysfunction after tricuspid valve replacement with obvious septum shift toward the left ventricle.

100% FiO₂, mild hyperventilation, alkalinisation, and adaptation of ventilation settings; the relationship between tidal volume and PVR has a unique "U" shape, PVR being minimal at functional residual capacity and maximal in the event of hypoventilation or hyperinflation [52, 53]. Any sympathetic stimulation such as stress, pain, hypothermia and shivering is suppressed, and anaesthetics potentially associated with augmentation of PVR are avoided. If these diverse measures do not allow a stabilisation of the right ventricular function, pulmonary vasodilatation is then started, preferentially using inhalational substances such as nitric oxide (NO), iloprost or milrinone, to avoid systemic vasodilation. Because of their different modes of action, combination of inhalational vasodilators might be synergistic [50, 62]. Keep in mind that the key haemodynamic sign of a therapeutic response to inhaled pulmonary vasodilators is not a reduction in pulmonary artery pressure but a decrease in CVP and an increase in cardiac output [62]. Considering further ventricular interdependence [69, 70] as well as the coronary perfusion of the right ventricular myocardium, the systemic arterial pressure is maintained elevated to avoid right ventricular myocardial ischaemia, using vasopressors as needed (noradrenaline, vasopressin). The benefit of systemic vasoconstriction has to be balanced with the risk of pulmonary vasoconstriction; fortunately, the pulmonary arterial tree has fewer alpha-receptors than the systemic arteries and is devoid of receptors for vasopressin [50]. Finally, inotropic agents are deployed as needed: dobutamine, adrenaline, phosphodiesterases III inhibitors (milrinone) or levosimendan have all been shown to be effective in case of acute RVF. However, as levosimendan and milrinone both produce systemic vasodilation, vasopressors may need to be coadministered to prevent reduced right coronary blood flow [62]. Increased left ventricular contractility can also result in increased right ventricular systolic function through ventricular interdependence.

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Results for the period 2008–2011 compared with 1996–1999

Timely diagnosis of congenital heart disease – did we improve?

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Center for Congenital Heart Disease, University Hospital Berne, Switzerland

Summary

Introduction: Delayed recognition of congenital heart disease (CHD) is not infrequent and may have a negative impact on the child’s prognosis. A study from our institution published in 2001 showed a rate of late diagnosis as high as 10% of all relevant CHD requiring therapy, and this rate was equal in cyanotic and acyanotic CHD.

Methods: A study identical to the one 12 years ago was performed with prospective evaluation of the time of first diagnosis of CHD during a 3-year period ending in June 2011. Only CHD that required surgical or catheter-based treatment was included. Late diagnosis was defined as diagnosis of CHD after discharge from the birth hospital for cyanotic CHD; for acyanotic CHD it was defined as first diagnosis at a time when therapy should already have taken place in accordance with recommended standards or if immediate treatment was necessary at the time of CHD recognition. In between the two studies came the recommendation of a nationwide neonatal pulse oximetry screening, starting in 2006.

Results: A total of 209 patients were included, 41% of these had cyanotic and 59% had acyanotic CHD. According to the entry criteria, late diagnosis was observed in 21 patients (10% of total population); 6% of cyanotic CHD patients (5 of 85) and 13% of acyanotic CHD patients (16 of 124). The two most frequently missed CHDs were atrial septal defect and coarctation (seven and six patients, respectively). Delayed diagnosis resulted in one death (unrecognised interrupted aortic arch in one neonate). Compared with the historical study in our referral population the main finding was that the total number of late diagnoses remained stable at 10%, with a slight decrease only in the rate of late diagnosed cyanotic CHD (from 10 to 6%) but with a rise in late diagnosed acyanotic CHD from 10 to 13% of all patients observed.

Conclusions: After 12 years of referring physician education and the implementation of a nationwide neonatal pulse oximetry screening, the rate of late diagnosis of CHD remained unchanged at 10% of all patients with only the rate of cyanotic CHD showing a slight decline.

Key words: congenital heart disease; late diagnosis; prognosis

Introduction

For most congenital heart disease (CHD) there is a consensus as to what would be the optimal timing for corrective intervention, allowing for the best possible prognosis of the affected children. For a long time literature has dealt with delayed diagnosis of even critical CHD [1, 2], and in recent years there was still substantial published evidence of the negative impact on outcome due to late diagnosis of relevant CHD [3–6].

In our referral population a study was conducted and published in 2001, showing the rate of late diagnosis of CHD to be 10% without any difference between cyanotic and acyanotic CHD in the rate of late detection [7]. These results led to intensified education of referring physicians (focused clinical workshops for the referring paediatricians every 2 years since publication of the first study), and in 2006 a nationwide recommendation for neonatal pulse oximetry was implemented in our country [8]. The current study evaluated whether these measures led to a decline in the rate of children with CHD being treated late because of missed diagnosis.

Methods

The study was identical to that done in our historical cohort [7], with a prospective evaluation covering a 3-year period ending in June 2011. The timing of first diagnosis of all CHD patients was registered. Only patients with haemodynamically relevant CHD, with necessity for either surgical or catheter-based therapy were included. Children with CHD but without an indication for therapy and solely on observation on an outpatient basis were excluded. Patients admitted for reoperation or reintervention were excluded, as were patients referred from other institutions for treatment.

Patients with late diagnosis in cyanotic CHD were defined as newborns discharged from their birth clinic without a diagnosis of heart disease. For acyanotic CHD, late diagnosis was defined as first diagnosis of the defect at a time when, according to recommended standards [9], treatment should already have taken place, or if at the time of diagnosis immediate therapeutic action was required. Echocardiographic screening in foetal life in our country is done by the caring obstetrician twice in pregnancy. Pregnant women are referred to a tertiary...
centre for further evaluation by a specialised foetal cardiologist only if primary screening revealed suspect findings.

Results

During the timespan covered by the evaluation, a total of 209 patients with newly diagnosed relevant CHD were prospectively included in this study. Eighty-five of the patients had cyanotic CHD and 124 children acyanotic CHD. About half of the patients were diagnosed in the early neonatal period (57%) while 51 of the patients (24%) were diagnosed at an age later than the neonatal period extending up to the age of 15 years. Especially pathologies of the aorta and atrial septal defect had a trend towards higher age at diagnosis of CHD. For a contemporary study there was an astonishingly low rate of in-utero diagnosis of only 19% (39 children).

Table 1 gives an overview of the individual diagnoses encountered and the respective proportion of late diagnosis for each entity.

Late diagnosis of CHD was observed for the group of cyanotic patients in 5 of 85 patients (5.9%) and in 16 of 124 acyanotic patients (12.9%, table 2). For the entire study population the rate of late diagnosis was 10%, the same level as in our historical patient cohort, where it was 9.9%. As shown in table 2, a mild decline in the rate of late diagnosis was seen for the cyanotic patients (without statistical significance owing to the low number of index patients with late diagnosis), as was to be expected because of the introduction of the neonatal pulse oxymetry screening in Switzerland in 2006. Out of the five infants with late diagnosis in cyanotic CHD, two born in private clinics did not have neonatal pulse oxymetry screening, two had it correctly done and in the last patient it was incorrectly done on the right arm instead of the leg.

For the patients with acyanotic CHD there was even a mild rise in the rate of late diagnosis from 10% in the first study period to currently 12.9%, and this was especially due to underdetection of atrial septal defects and coarctation.

A few typical clinical findings that were not detected adequately were seen in the patients who were referred late: fixed split heart sounds in atrial septal defect were clinically present in seven patients. Absent or weak

Table 1: Spectrum of cardiac malformations and proportion of patients with late diagnosis.

<table>
<thead>
<tr>
<th>Cardiac diagnosis</th>
<th>Total cases</th>
<th>Late diagnosis number of patients</th>
<th>Age at late diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyanotic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>25</td>
<td>1</td>
<td>4 m</td>
</tr>
<tr>
<td>Transposition of great arteries (simple)</td>
<td>17</td>
<td>1</td>
<td>4 m</td>
</tr>
<tr>
<td>Atrioventricular canal</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single ventricle</td>
<td>11</td>
<td>1</td>
<td>6 m</td>
</tr>
<tr>
<td>Interrupted aortic arch</td>
<td>5</td>
<td>1</td>
<td>At death</td>
</tr>
<tr>
<td>Total anomalous pulmonary venous connection</td>
<td>4</td>
<td>1</td>
<td>6 m</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Various</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acyanotic</td>
<td>124</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>25</td>
<td>7</td>
<td>9–16 y</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>30</td>
<td>1</td>
<td>4 m</td>
</tr>
<tr>
<td>Coarctation</td>
<td>22</td>
<td>7</td>
<td>1 m–10 y</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>6</td>
<td>1</td>
<td>6 m</td>
</tr>
<tr>
<td>Double outlet right ventricle</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Various</td>
<td>21</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

m = months; y = years.

Table 2: Proportion of patients with late diagnosis.

<table>
<thead>
<tr>
<th>Late diagnosis</th>
<th>Study period I 1996–1999</th>
<th>Study period II 2008–2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients with CHD</td>
<td>32 / 323 (9.9%)</td>
<td>21 / 209 (10%)</td>
</tr>
<tr>
<td>Cyanotic CHD</td>
<td>7 / 72 (9.7%)</td>
<td>5 / 85 (5.9%)</td>
</tr>
<tr>
<td>Acyanotic CHD</td>
<td>25 / 251 (10%)</td>
<td>16 / 124 (12.9%)</td>
</tr>
</tbody>
</table>

Comparison between the current and the historical study.
femoral pulses were the leading clinical sign in seven patients but were not detected until late diagnosis was made. Clearly present increased precordial pulsation on palpation of the thorax was noticed in four infants but was not reported before.

Only a few patients had clinical consequences as a result of late referral: there was one mortality due to late diagnosis of interrupted aortic arch with intractable cardiogenic shock at referral to the intensive care unit at 7 days of life. Two teenagers with an atrial septal defect (13 and 15 years old at referral and at device closure with an Amplatzer) did not normalise the size of their right heart structures during a 2-year follow-up.

Discussion
The main finding of this study was that in a contemporary cohort of children with newly diagnosed relevant CHD there was still a significant proportion of patients with late referral and late diagnosis of CHD. In comparison with an identical historical study published in 2001 [7], the overall rate of late diagnosis remained stable at 10% of patients with relevant CHD. The only difference was that there was a mild (although not significant) decline in late diagnosis of cyanotic CHD, whereas a slightly larger proportion of patients with a cyanotic CHD were diagnosed late. The rise in the number of acyanotic patients diagnosed late was largely due to patients with coarctation and atrial septal defect. Late diagnosis of atrial septal defect even in the adult age is a constant finding in large populations undergoing echocardiographic studies [10] and thus not surprising. The mild decline in late referrals of cyanotic CHD patients is attributable to neonatal pulse oximetry screening (on the first day of life), although the remaining few patients not detected by screening make clear that in addition to pulse oximetry screening, a clinical examination still plays an important part as outlined in the recommendations accompanying the original screening guidelines [8].

The aim of a timely diagnosis of CHD is to minimise morbidity and mortality that may be associated with late referral [3, 5] and which may be relevant to the child’s prognosis. Morbidity was low in the current study, but the case of a newborn who died because of a missed interruption of the aortic arch is alarming, this condition being amenable to surgical treatment with good long-term results.

Another astonishing result of the current evaluation is the very low rate of antenatal echocardiographic diagnosis of relevant CHD, the detection rate of 19% being clearly below target standards in the current era [11], given the proven benefit of antenatal diagnosis in the management of critical CHD [12].

In summary, because during the past decade the rate of late detection of CHD did not show any decline, efforts to improve this performance have to continue on all levels, a higher detection rate of critical CHD already in utero as well as better recognition neonatally must be aimed for. Clinical recognition of atrial septal defects and coarctation must also be improved in order not to compromise the individual patient’s prognosis.

Disclosure statement
No financial support and no other potential conflict of interest relevant to this article was reported.

References
A very unusual observation

Prolonged complete AV block after balloon aortic valvuloplasty in a child

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Introduction

Balloon aortic valvuloplasty (BAV) is the first-line treatment option in congenital valvular aortic stenosis in the absence of relevant valvular regurgitation. BAV is palliative in nature but can delay further intervention for several years. Especially in growing children this is a major advantage. It is a well established, and in general quite safe, method with few complications, especially in older children, with aortic regurgitation being the most frequent complication after BAV. There are only few reports of rhythm problems after such an intervention. We report a case of a prolonged and haemodynamically relevant complete atrioventricular (AV) block after standard BAV in a 10-year-old boy.

Case report

A 10-year-old boy was followed up in our outpatient clinic for congenital valvular aortic stenosis. The stenosis was at the valvular level, with thickened, dysplastic leaflets in an anatomically tricuspid valve (Fig. 1). After years of stable findings without symptoms, with moderate stenosis but no aortic regurgitation, he presented with increasing stenosis, with a peak gradient of 90 mm Hg (mean 53 mm Hg) and signs of left ventricular hypertrophy in transthoracic echocardiography. On the basis of these findings, the decision to perform a balloon valvuloplasty was made. The preinterventional electrocardiogram (ECG) did not show any conduction abnormality (sinus rhythm, heart rate 73 bpm, PQ 110 ms, QRS 80 ms, QT 370 ms, normal repolarisation).

Catheterisation was performed under general anaesthesia. The stenotic aortic valve could easily be passed with a 5-French pigtail catheter. Invasive peak gradient was 46 mm Hg. The valve annulus was 16.7 mm (measured in an aortography-stillframe in systole between the valve hinge points). The stenosis was dilated using left ventricular rapid pacing (220 bpm) with a 16 mm Tyshak-balloon. During the first two dilatation attempts, the balloon slipped proximally, the third dilatation being successful (Fig. 2), with rapid disappearance of the central waist. Peak gradient was reduced from 46 to 6 mm Hg. Better valve opening was noted but also moderate aortic regurgitation. After the suc-
cessful dilatation, complete atrioventricular block with a wide QRS escape rhythm of 80 bpm was seen on ECG. Because of persistence of the complete heart block the patient was transferred to the intensive care unit for better surveillance. In the following hours he presented with desaturation, headache, abdominal pain, agitation and lactic acidosis. Therapy with isoprenaline did not improve symptoms. Finally he had to be supported with intravenous inotropes and diuretics because of clinical signs of low cardiac output with severe arterial hypotension and dyspnoea associated with bradycardia. Brain natriuretic peptide (BNP) rose to a maximum of 537 pg/ml at that time (normal value <100 pg/ml). In a transthoracic echocardiogram we could confirm moderate aortic regurgitation with good systolic function and a normal dimension of the left ventricle. Complete AV block persisted after 24 hours (fig. 3). Because of poor clinical tolerance of the new aortic regurgitation with persistent bradycardia (70–80 bpm) a transient intravenous (transjugular) Swan Ganz bipolar pacing catheter was placed 30 hours after BAV. The patient was paced at 100 bpm. Under this therapy the evolution was positive. The clinical symptoms of heart failure rapidly diminished. For afterload reduction therapy with an angiotensin converting-enzyme (ACE) inhibitor was started. Under constant pacing, the underlying rhythm was tested every 12 hours, but only at 72 hours after BAV a normofrequent sinus rhythm reappeared (fig. 4). After stabilisation and recompensation the pacing catheter could be removed 5 days after the intervention and the patient was transferred to the paediatric ward. He left hospital 7 days after BAV under diuretic and ACE inhibitor therapy. A Holter monitor confirmed a stable sinus rhythm at around 90 bpm at rest. Transthoracic echocardiography before discharge showed a mild residual aortic stenosis with residual peak gradient of 28 mm Hg (mean 11 mm Hg), and moderate aortic regurgitation with a calculated regurgitant fraction of 46%. BNP normalised to 44 pg/ml. Follow-up at 1 month showed stable sinus rhythm on Holter monitor and identical findings on echo with well tolerated moderate aortic regurgitation without left ventricular dilatation.

**Discussion**

Aortic regurgitation is the most common complication after balloon valvuloplasty. BAV is an established method, but especially in newborns the complication rate can be as high as 15% [1]. Short-term and transient arrhythmias are common in paediatric catheter interventions, but we were not able to find reports on long-lasting arrhythmia after simple BAV in children. Such a prolonged occurrence of complete AV block was very surprising, and it was because we counted on the reappearance of sinus rhythm at any moment that implantation of a transvenous pacing catheter was deferred initially despite poor haemodynamics. More data exist about important, persistent arrhythmia and need of permanent pacemaker (PPM) implantation after transcatheter aortic valve replacement.

![Figure 2: Balloon position during dilatation.](image)

![Figure 3: ECG at 48 hours after dilatation with complete AV dissociation; sinus rate of 108/min and narrow complex escape rhythm of 64/min.](image)
(TAVR) in the elderly. A meta-analysis [2] showed male sex, baseline conduction disturbances, especially right bundle branch block, and intraprocedural AV block to be predictors of PPM implantation. One prospective study in adult patients undergoing balloon aortic valvuloplasty before TAVR analysed the frequency of cardiac conduction disturbances [3]. In this study 1.5% needed permanent pacemaker implantation. It was shown that the ratio of balloon size to left ventricular outflow obstruction (LVOT) diameter was an important factor, smaller LVOT being associated with more damage to the conduction system.

In our patient the reason for the prolonged complete AV block remains unclear. The preinterventional ECG did not show any conduction abnormality and the intervention itself was a standard procedure without major problems. It is known that trauma to the LVOT has the potential to damage the conduction system [4]. Balloon size was a proper choice for the diameter of the aortic annulus (16 mm balloon for an aortic annulus of 16.7 mm, ratio balloon/annulus = 0.96). Clinical observations support the concept of damage to the conductive system from mechanical manipulation in the area of the aortic valve. One might also speculate on an anatomic variant of the conduction system with a more subendocardial position on the left ventricular aspect.

In conclusion, this case of prolonged complete AV block after BAV is a very unusual observation which does not allow for advice on what should be done differently in future cases.

Disclosure statement
No financial support and no other potential conflict of interest relevant to this article was reported.

References
Ein Patient mit wöchentlich auftretenden Palpitationen

Regelmäßige Breitkomplextachykardie bei dekrementaler Präexzitation

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Fallbeschreibung


Abbildung 1A: 12-Kanal-Ruhe-EKG (50 mm/s; 10 mm/mV) des Patienten während der klinischen Breitkomplextachykardie mit LSB-Morphologie (roter Pfeil zeigt retrograde P-Welle bei 1:1-VA-Leitung; das Intervall vom Beginn des QRS-Komplexes bis zum Nadir der S-Zacke in den Brustwandableitungen ist rot eingezeichnet und misst 80 ms, was gemäß Brugada-Kriterien gegen eine VT spricht).

Abbildung 1B: 12-Kanal-Ruhe-EKG (25 mm/s; 10 mm/mV) des Patienten im Sinushynchron zeigt eine Präexzitation (PQ 115 ms, Deltawelle positiv in I und aVL, negativ in III). Die EKG-Kriterien sprechen gegen eine linkslaterale akzessorische Bahn [3].
Während der Palpitationen gelang keine EKG-Dokumentation. Daher wurde die Indikation zu einer elektrophysiologischen Untersuchung (EPU) gestellt. Bereits beim Einführen der Katheter wurde eine regelmäßige Breitkomplex-tachykardie (Abb. 1A: 12-Kanal-Ruhe-EKG, 50 mm/s, 10 mm/mV) mit einer Zykluslänge von 316 ms (entspricht einer Herzfrequenz von 190/min), Linksschenkelblock-(LSB)-Morphologie mit einer QRS-Dauer von 150 ms, spätem R/S Umschlag (Transition) in V6 sowie superiorer Achse (II, III und aVF negativ) induziert, die spontan terminierte.

**Fragen**

*Handelt es sich bei dieser regelmäßigen Breitkomplex-tachykardie um eine supraventrikuläre (SVT) oder ventrikuläre Tachykardie (VT)?*


*Kommt bei dieser SVT der breite QR-Komplex mit LSB-Morphologie durch eine aberration (also Blockierung des linken Tawara-Schenkels während der Tachykardie) oder durch eine akzessorische Leitungsbahn (Präexzitation) zustande?*


![Abbildung 2A: Die intrakardialen Ableitungen bestätigen eine Präexzitation mit einer verkürzten Dauer vom His-Bündel zur frühesten Ventrikelregression (HV-Zeit) von 22 ms. CS 1–2: Katheter im Koronarsinus, distale Koronarinselselektroden (bipolar); CS 7–8: proximale Koronarinselselektroden am Ostium des Koronarsinus (bipolar); CS 3–4 und CS 5–6: zwischen proximalen und distalen Koronarinselselektroden liegende Elektroden (bipolar); HBE 1–2: distale Elektroden des His-Katheters (bipolar); HBE 3–4: mittlere Elektroden des His-Katheters (bipolar); HABE 5/6: proximale Elektroden des His-Katheters (bipolar); RVA 1–2: Elektroden am RV-Apex (bipolar). Katheterposition siehe Abbildung 3C.](image-url)

Abbildung 3A: Die dreidimensionale elektroanatomische Rekonstruktion des rechten Vorhofs (LAO links, AP rechts) zeigt die proximale Insertionsstelle (blauer Punkt) des akzessorischen Mahaim-Bündels im Bereich der lateralen Wand der Trikuspidalklappe (rosa Punkte). Das zugehörige intrakardiale Elektrogramm (weisser Pfeil) zeigt das typische Mahaim-Potential im Bereich des blauen Punkts. Der gelbe Punkt markiert das His-Bündel. Die roten Punkte markieren die Ablationspunkte, die an dieser Stelle zur erfolgreichen Ablation führten.
Abbildung 3B: Separate Darstellung der intrakardialen Elektrogramme; die distalen Elektroden des Ablationskatheters (Map dis) befinden sich im Bereich des Mahaim-Bündels und zeigen ein typisches Mahaim-Potential (rote Pfeile) an der lateralen Trikuspidalklappe.


Disclosure statement

Literatur
Mitteilungen

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Wir gratulieren!

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