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Current aspects of atrial fibrillation surgery

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Summary

Atrial fibrillation is the most common cardiac arrhythmia, with an increasing prevalence with rising age. Atrial fibrillation is associated with significant morbidity and mortality. Concomitant surgical ablation of atrial fibrillation in patients undergoing other cardiac surgical procedures is commonly practiced, with high success rates, short procedure times and a low additional operative risk profile. Minimally invasive stand-alone surgical procedures for atrial fibrillation are performed less frequently, even though they represent a valid therapeutic option for certain patients, with excellent results. Surgical procedures for atrial fibrillation treatment allow the excision or exclusion of the left atrial appendage, therefore eliminating a potential source of cerebral embolic events in the case of procedural failure to abolish atrial fibrillation. The hybrid approach, which combines the advantages of catheter and surgical ablation, is a promising approach for the future. This review focuses on surgical options in the curative treatment of atrial fibrillation.

Key words: Atrial fibrillation; surgical ablation; minimally invasive procedures; concomitant procedures

Introduction

Atrial fibrillation is the most common cardiac arrhythmia, with an increasing prevalence with increasing age. Atrial fibrillation increases the risk of stroke five-fold and leads to an enormous rise in healthcare costs [1]. James Cox and his coworkers did extensive research on the pathophysiology of atrial fibrillation, and based on their results they developed the Cox maze operation [2–4]. After two modifications the Cox maze III operation was introduced into clinical practice. This operation was a “cut-and-sew” technique with multiple incisions in the walls of both atria performed via median sternotomy with the help of extracorporeal circulation. Compared with the first and second procedures, the Cox maze III operation was associated with less arrhythmia recurrence, fewer pacemaker requirements and improved long-term sinus node and atrial transport function [5, 6]. Despite the fact that the Cox maze III procedure remains the gold standard in surgical treatment of atrial fibrillation, it never became widely used owing to long operative times and a challenging surgical technique [7, 8]. In 2002 the Cox maze IV operation was introduced. In this iteration most of the incisions were replaced with linear lines of bipolar radiofrequency ablation [9].

In 1998 Haissaguerre et al. published their results showing that in paroxysmal atrial fibrillation the arrhythmia is triggered by signals originating from the pulmonary veins. This finding led to the simplification of atrial fibrillation surgery by implementation of the pulmonary vein isolation approach [10]. Other studies showed the effectiveness of exclusively left atrial procedures in patients with chronic atrial fibrillation undergoing mitral valve surgery [11, 12]. The original “cut-and-sew” technique was further simplified by the introduction of cryo- and radiofrequency ablation instead of surgical incisions [13, 14]. Further alternative energy sources for surgical ablation were used to optimise and modify the surgical technique [15–18]. Because these modifications led to an easier procedure with shorter procedure times and less morbidity, the number of surgical ablation procedures, especially concomitant with other cardiac surgical operations, increased enormously [19]. Nowadays atrial fibrillation surgical ablation is mostly carried out as a concomitant procedure in patients who require other cardiac surgical procedures. In some cases surgical treatment of atrial fibrillation may also be performed in patients with lone atrial fibrillation (fig. 1).

Indications

According to the 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation, two indication classes have to be distinguished: concomitant or stand-alone atrial fibrillation surgery. Irrespective of the kind of atrial fibrillation (paroxysmal, persistent or long-standing...
persistent), symptomatic atrial fibrillation refractory to, or associated with intolerance of, at least one class 1 or 3 antiarrhythmic medication is a class IIa-indication (level of evidence [LOE] C) for concomitant surgical ablation of atrial fibrillation. Symptomatic atrial fibrillation prior to initiation of antiarrhythmic drug therapy with a class 1 or 3 antiarrhythmic agent is in the case of paroxysmal and persistent atrial fibrillation a class IIa-indication (LOE C) and in the case of long-standing persistent a class IIb-indication (LOE C) for concomitant atrial fibrillation surgery.

In the case of stand-alone atrial fibrillation surgery, symptomatic atrial fibrillation (paroxysmal, persistent and long-standing persistent) refractory to or with intolerance of at least one class 1 or 3 antiarrhythmic medication is a class IIb-indication (LOE C) in the case of patients who have not had a failed catheter ablation but prefer a surgical approach or in the case of patients who have had one or more concomitant attempts at catheter ablation.

In patients with symptomatic atrial fibrillation prior to initiation of antiarrhythmic drug therapy with a class 1 or 3 antiarrhythmic agent, stand alone atrial fibrillation surgery is not recommended (class III, LOE C) [20].

**Surgical approach**

In the case of concomitant atrial fibrillation surgery, the surgical approach depends on the cardiac surgical procedure performed besides the surgical ablation. In the case of mitral valve surgery an endocardial left atrial approach is possible since atriotomy is required for the valve procedure. Extracorporeal circulation and cardioplegic cardiac arrest are mandatory. Depending on the surgical approach for the mitral valve operation the ablation is either carried out through a right lateral mini-thoracotomy or a median sternotomy [7, 8, 18, 19].

If aortic valve surgery or coronary artery bypass grafting is performed, atriotomy is not carried out and therefore an epicardial ablation approach is preferable. In case of off-pump coronary artery bypass grafting the epicardial ablation is performed without the help of extracorporeal circulation [21, 22].

If the surgical ablation of atrial fibrillation is carried out as a stand-alone procedure, the surgical approach should nowadays be minimally invasive and video-assisted. It is performed on a beating heart without the use of the extracorporeal circulation. The minimally invasive approach may be uni- or bilateral, depending on the ablation system used, the performed lesion set and left atrial appendage management [16, 23, 24].

As another interesting approach, especially in cases of stand-alone procedures, the hybrid approach should be mentioned. This approach is a combination of a minimally invasive surgical approach combined with electrophysiological mapping and endocardial catheter ablation. With the combination of a minimally invasive surgical epicardial ablation and an endocardial catheter ablation the limitations of both techniques are supposed to be overcome [25–28].

**Lesion sets**

In order to understand lesion sets for surgical ablation, it is important to understand the underlying pathophysiology. Atrial fibrillation is initiated by an event (trigger) and the presence of a predisposing substrate is maintaining the arrhythmia. Additional factors may act as modulators in the initiation or continuation of atrial fibrillation. Triggers and substrates can be located in both atria. However they are usually found in the pulmonary veins and the left atrium [21]. Triggers in the pulmonary veins characterise paroxysmal atrial fibrillation, whereas in persistent atrial fibrillation multiple macro re-entry circuits and atrial remodelling is responsible for the maintenance of the arrhythmia [29, 30]. This fact shows that one aspect of the choice of a lesion set is the existing type of atrial fibrillation. In patients with paroxysmal atrial fibrillation, a pulmonary vein isolation procedure might be sufficient, since mainly ectopic...
foci in the pulmonary veins have to be addressed. In contrast, in cases of persistent atrial fibrillation a modification of the underlying substrates should be accomplished by linear ablation lines in the left atrium in addition to pulmonary veins isolation. Another additional factor that can be considered during surgical ablation procedures is the isolation of autonomic ganglia after electrophysiological testing for ganglionic plexi activity [24]. Scherlag et al. were able to show that the activation of autonomic ganglia may serve as a mediator upon which the pulmonary vein triggers act to induce atrial fibrillation [31].

Isolation of the pulmonary veins is the key concept of all surgical ablation procedures. It can be performed easily via an endocardial as well as an epicardial approach. It can either be performed as one large “box lesion” encircling all four pulmonary veins or as bilateral pulmonary veins isolation with a pairwise isolation of the right and the left pulmonary veins. Whether the box lesion or the bilateral pulmonary veins isolation is the better approach is still not finally clarified, but several studies were able to show better results with the box lesion [9, 32]. Pulmonary veins isolation techniques essentially lead to trigger elimination; however, in patients with persistent atrial fibrillation this might not be enough to treat atrial fibrillation successfully, since substrates outside the pulmonary veins will exist. Therefore, additional linear ablation lines in the left atrium will be required. Creation of linear ablation lines in the left atrium with an endocardial approach is feasible and reproducible. In the case of an epicardial, beating heart approach linear ablation may be technical challenging to achieve [22]. A linear ablation with a bipolar radiofrequency clamp towards the mitral annulus puts nearby anatomical structures (coronary sinus, circumflex artery) at risk of damage and fails to create an ablation line that reaches the mitral annulus [33].

In summary, in patients with persistent atrial fibrillation a left atrial lesion set consisting of pulmonary veins isolation and additional ablation lines towards the ostium of the left atrial appendage as well as the mitral valve annulus is reasonable (fig. 2). In selected patients, mainly with longstanding-persistent atrial fibrillation and right atrial triggers as well as substrate, lesions in the right atrium might be required to improve the outcome of the ablation procedure [21].

**Left atrial appendage management and postoperative anticoagulation**

One important advantage of surgical ablation procedures for atrial fibrillation over catheter ablation procedures is that the left atrial appendage can be easily excluded or excised (fig. 3). Therefore, even in case of procedural failure with the recurrence of atrial fibrillation, the left atrial appendage as a potential source of repeated cerebral embolic events is eliminated [24, 34]. Furthermore, it was shown that the left atrial appendage may be a site of atrial fibrillation initiation and that epicardial left atrial appendage clip occlusion leads to electrical isolation of the left atrial appendage, therefore eliminating this potential source of atrial fibrillation trigger signals [35, 36]. Several surgical techniques for left atrial appendage management exist. The left atrial appendage can either be excised or excluded. Excision means physical removal of the left atrial appendage with scissors or an amputating stapling device. In contrast, exclusion can be performed by closure of the orifice of the left atrial appendage with the appendage remaining attached. This can be achieved by suturing (running suture, pursestring or external ligation) or stapling [37]. Another way of exclusion is the application of an epicardial left atrial appendage clip [38]. Kanderian et al. examined the efficacy of surgical left atrial appendage closure by excision and exclusion. The overall rate of successful left atrial appendage closure was merely 40%, with excision showing
higher success rates (73%) than suture exclusion (23%) and stapler exclusion (0%) [37]. Emmert et al. were able to show a 100% success rate of left atrial appendage closure with the epicardial left atrial appendage clip occlusion after a mean follow-up of 3.5 years [38]. On the basis of these studies the epicardial clip occlusion of the left atrial appendage should be the preferred surgical approach, since complete closure of the left atrial appendage is of utmost importance.

With regard to the question of anticoagulation after successful complete left atrial appendage closure, it is important to mention that there exists no reliable data that supports the discontinuation of oral anticoagulation in the absence of an absolute contraindication for oral anticoagulation and postoperative persistence of atrial fibrillation.

Based on the 2012 HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation, oral anticoagulation should be continued for several months after surgical ablation of atrial fibrillation because of the relatively high incidence of early atrial tachyarrhythmias which occur after atrial fibrillation surgery. Anticoagulation may then be discontinued on a case-by-case basis after the documentation of the absence of symptomatic or asymptomatic atrial fibrillation episodes on follow-up electrocardiogram monitoring. Furthermore, a postoperative transthoracic echocardiogram should be obtained to rule out atrial stasis or thrombus prior to discontinuation of oral anticoagulation [20].

**Results**

Surgical ablation of atrial fibrillation, especially as a concomitant procedure to other cardiac surgical operations, is nowadays commonly performed. Reported success rates of both concomitant as well as stand-alone procedures mostly range between 60% and 80%, and the in-hospital mortality and periprocedural complication rate is low [8, 39–43]. Gillinov et al. studied the outcome of a surgical ablation procedure with bipolar radiofrequency as a concomitant procedure in 513 patients. They found that freedom from ablation failure was 72% at 12 months. An analysis of risk factors for ablation failure revealed three risk factors influencing ablation outcome: type and duration of atrial fibrillation, choice of lesion set in persistent atrial fibrillation and left atrial size [43].

Damiano et al. performed a prospective study on 282 patients who underwent the Cox maze IV procedure; 66% of the patients had a concomitant cardiac surgical procedure, the rest underwent a stand-alone atrial fibrillation operation. They were able to show rates of overall freedom from atrial fibrillation of 89%, 93% and 89% at 3, 6 and 12 months, respectively, postprocedure. There were no significant differences in success rates with regard to stand-alone versus concomitant maze operation (p = 0.361). They performed a multivariate logistic regression analysis of risk factors for failure after the Cox maze IV procedure and found left atrial diameter, early atrial tachycardias and failure to isolate the entire posterior left atrium to be predictors for procedural failure. Left atrial diameter was a significant predictor of failure with an odds ratio of 1.42 [9].

A meta-analysis of surgical ablation for atrial fibrillation during mitral valve surgery performed by Phan et al. revealed a significantly improved rate of sinus rhythm in the surgical ablation group. The meta-analysis, which included nine relevant randomised controlled trials comprising a total of 496 patients, showed, with regard to efficacy assessment, a sinus rhythm rate at 12 months of 75.5% in the surgical ablation group and 26% in the mitral valve only group (p < 0.00001). In patients with more than 12 months of follow-up success rates were 64.4% in the surgical ablation group versus 17.9% in the mitral valve only group (p < 0.00001). With regard to safety assessment 30-day all-cause mortality did not differ between the two groups (4.4% vs 2.2%; p = 0.46). The incidence of postoperative pacemaker implantations also did not reveal any significant differences (7.0% vs 7.5%; p = 1.00). The analysis of stroke rates revealed compa...
rable results between the groups (5.5% vs 3.9%; \( p = 0.45 \)) [7].

Boersma et al. published the results of a prospective randomised clinical trial comparing catheter ablation for atrial fibrillation with stand-alone surgical ablation (FAST trial); 124 patients with drug refractory atrial fibrillation with left atrial dilatation and hypertension or failed catheter ablation were randomised to either catheter or surgical ablation. The primary endpoint was defined as freedom from left atrial arrhythmia lasting more than 30 seconds without antiarrhythmic medication. After 12 months the primary endpoint was met in 36.5% of the catheter ablation group and in 65.6% of the surgical ablation group (\( p = 0.0022 \)). However the procedural adverse event rate was significantly higher in the surgical ablation group (23.0% vs 3.2%; \( p = 0.001 \)) as well as the overall serious adverse event rate at 12 months (34.4% vs 15.9%; \( p = 0.027 \)) [44].

Hybrid approach

As a result of suboptimal results with both catheter and surgical ablation a new concept of invasive treatment of atrial fibrillation was introduced in recent years – the hybrid approach. This approach is a combination of both surgical and catheter ablation of atrial fibrillation with the aim of optimising efficacy. [21, 26, 27]. La Meir et al. reported their experience with a hybrid procedure for atrial fibrillation treatment in 56 patients. They were able to show success rates of 92% at 2 years, 97% at 3 years and 95% at 4 years. These results are certainly promising and this approach needs to be further investigated in the future in order to evaluate whether the hybrid approach may become a standard treatment for lone atrial fibrillation.

Disclosures

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The importance of early referral for multidisciplinary management

Pulmonary valve stenosis due to undifferentiated pleomorphic sarcoma

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Summary

Pulmonary artery sarcomas are uncommon intracardiac tumours with poor prognosis. We report the case of a 69-year-old woman in good health presenting with rapidly progressive dyspnoea and an unfamiliar systolic murmur. Echocardiography revealed pulmonary valve stenosis due to an obstructing mobile mass. Imaging studies confirmed the presence of a contrast-enhancing lesion adherent to the valve, extending into the pulmonary trunk and right ventricular outflow tract, and suggestive of malignancy. Endovascular biopsy was attempted with no success. Surgical resection with autologous graft valve replacement and pulmonary artery reconstruction was performed. Postoperative histological examination confirmed the diagnosis of an undifferentiated pleomorphic sarcoma. Pulmonary artery sarcoma should be considered as a rare differential diagnosis in patients presenting with dyspnoea and a crescendo–decrescendo systolic murmur increasing with inspiration. Echocardiography is a useful first diagnostic approach but multi-imaging assessment is almost always necessary for definite diagnosis. Our case provides insights into the challenges met by cardiologists, radiologists and cardiac surgeons in the management of such cases.

Key words: pulmonary artery sarcoma; undifferentiated pleomorphic cardiac sarcoma; pulmonary valve stenosis; right ventricular outflow tract

Case description

A 69-year-old Caucasian woman presented to her general practitioner with new onset fatigue and rapidly progressive dyspnoea on exertion. Upon physical examination the patient was afibrile, blood pressure was 116/72 mm Hg, pulse rate 76/min and respiration rate 12/min. Heart sounds were normal, but a new, 3/6, crescendo–decrescendo systolic murmur, which increased during deep inspiration and was maximal along the left sternal border, was noted. Laboratory studies of electrolytes, renal function, liver and cardiac enzymes, acute phase reactants and complete blood count were within normal limits, as were a 12-lead ECG and a chest radiograph. Transthoracic echocardiography (TTE) revealed a moderate pulmonary valve stenosis caused by a globular mass protruding into the right ventricular outflow tract during diastole (fig. 1). The maximum pulmonary pressure gradient was measured at 42 mm Hg (mean 30 mm Hg). Left as well as right ventricle size and function were normal. The patient was promptly referred to our institution where cardiac computed tomography (CT) revealed a 25×35×24 mm mass attached to the RVOT and extending into the pulmonary trunk through the pulmonary valve (fig. 2). After intravenous injection of 60–80 ml iodinated contrast material, late image acquisition showed inhomogeneous enhancement of the mass and mural enhancement of the pulmonary trunk suggestive of infiltration. This technique in the context of cardiac CT consists of acquiring images 60 seconds after CT angiography in order to optimise tissue enhancement. Fluodeoxyglucose (18F) positron emission tomography-CT (18F-FDG PET-CT) revealed mild metabolic activity of the mass, with no signs of metastasis (fig. 3). Finally, cardiac magnetic resonance imaging (MRI) confirmed proximal extension of the valve mass, 4 cm towards the pulmonary trunk, without apparent invasion of the bifurcation (fig. 4). A hyperintense signal in T2-weighted images indicated oedema, whereas moderate signal enhancement during resting perfusion suggested rich vascularisation. Intense signals on gadolinium en-
Figure 1: (A) TTE parasternal long axis view of the pulmonary artery. The tumour is recognised as a large round mass invading the pulmonary valve. (B) Transoesophageal echocardiography (TOE) in transgastric inflow–outflow view of the right ventricle. The tricuspid valve and the right ventricular apex are seen at the top of the picture. The pulmonary valve is replaced by a ball-like tumour (*) obstructing the RVOT. Ao = aorta; PA = pulmonary artery; RV = right ventricle; RVOT = right ventricle outflow tract; TV = tricuspid valve.

Figure 2: Cardiac computed tomography, 2d multi-planar reformation showing the voluminous mass (*) attached to the pulmonary valve (A) and protruding in the pulmonary trunk during systole (B). Ao = aorta; LV = left ventricle; PA = pulmonary artery; RVOT = right ventricle outflow tract.

hancement sequences suggested rich extracellular content (fibrosis or extracellular matrix), but no significant necrosis (fig. 5).

A provisory diagnosis of intracardiac malignancy was made on clinical (rapid installation of symptoms) and imaging (vascular infiltrative mass) grounds, and endovascular biopsy was attempted. The procedure was uneventful, but yielded limited material for histology. The case was discussed by a multidisciplinary panel involving cardiologists, radiologists and a cardiothoracic surgeon, and surgery was decided on. The time delay from first diagnosis
on TTE to the operation was 22 days. During the procedure, a bulky, white mass, densely adherent and infiltrating the pulmonary valve and all of the pulmonary infundibulum was identified and removed, along with the deformed pulmonary valve, part of the RVOT, as well as most of the pulmonary trunk as proximally as possible to the bifurcation. An autologous graft was used to replace the valve; the right ventricle was then anastomosed to the distal part of the pulmonary artery. Postoperative histology revealed a mesenchymal tumour consisting of highly atypical spindle cells without specific differentiation (fig. 6) [1]. Given the retrograde extension of the mass and the possibility of residual fragments very low inside the RVOT, the patient was referred for adjuvant chemotherapy and radiotherapy after six weeks of convalescence in a cardiac rehabilitation centre.

**Discussion**

Our case highlights the major impediments encountered during management of these rare and often
fatal intracardiac tumours; from timely and accurate diagnosis to surgical resection and adjuvant care, pulmonary artery sarcomas (PAS) represent a diagnostic and therapeutic challenge for cardiologists, radiologists, cardiac surgeons and oncologists. Typical onset age is 45–55 years with a female to male ratio of 2:1. The tumour typically arises within the pulmonary trunk and spreads into the pulmonary arteries. Embryologically, it originates from the bulbus cordis, a structure that later gives birth to both the RVOT and pulmonary trunk, which could, in our case, explain valve involvement and RVOT extension. As with the majority of intracardiac tumours, presentation is non-specific, with dyspnoea on exertion, chest or back pain, cough and haemoptysis being common symptoms. In many cases, the clinical presentation mimics chronic thromboembolic pulmonary hypertension, and in advanced stages constitutional symptoms appear. Physical examination remains unremarkable until late. Our case is unique in the sense that it presented as a new unfamiliar murmur that alarmed the primary physician. Such a finding is extremely rare and highlights the paramount importance of good auscultation skills and a clinical acumen. A new harsh systolic murmur that increases with inspiration indicates pulmonary stenosis and warrants further investigation with echocardiography. TTE may show an increased gradient across a narrowed RVOT [2]. The presence of a pedunculated lesion arising from the RVOT, or pulmonary valve or trunk should always raise suspicion of PAS [3] and prompt a thoracic CT or a cardiac MRI.

Figure 5: (A) T1-weighted image; the mass (arrow) has a signal iso-intense to the myocardium. (B) T2-weighted image; the mass has a signal hyper-intense to the myocardium indicating presence of tissue oedema. (C) Late gadolinium enhancement sequence; the mass shows bright enhancement indicating rich vascularisation. (D to F) Perfusion imaging; (D) contrast in the right cardiac chambers; (E) contrast in the left cardiac chambers; (F) contrast enhancing the myocardium; the mass shows moderate first-pass enhancement with peak signal intensity lower than that of the myocardium.
CT findings can be misleading in over 50% of cases, as PAS is confused with the far more common condition of central pulmonary embolism. Both entities present with a filling defect in the pulmonary arteries. Suggestive of PAS are an inhomogeneous, soft tissue density, with the filling defect occupying the entire lumen of the pulmonary trunk, along with delayed contrast enhancement on angiography [4]. Once suspicion is raised, PET-CT and MRI are helpful to differentiate fully the mass. Preoperative histological diagnosis is generally not possible although it should be attempted through CT-guided transthoracic aspiration, transvenous catheter biopsy or transbronchial biopsy. In most cases definitive diagnosis is made only during surgery or at autopsy. Surgery remains the cornerstone of management, offering definite diagnosis, clinical improvement and the best prognosis [5]. Even if curative resection is not possible, palliative resection can provide symptom relief by restoring haemodynamics. Reported interventions range from bilateral pneumonectomy, pulmonary endarterectomy (PEA) with or without pneumonectomy and with or without reconstruction of the pulmonary artery to debulking and palliative stenting. Regarding adjuvant therapy, combined chemotheraphy should be adopted in nearly every case to extend palliation. Overall, PAS represents a fatal disease, with a mean survival time without surgical intervention of 1.5 months. Survival of 36.5 ± 20.2 months has been reported for patients benefiting from a “curative” resection while 11 ± 3 months for those undergoing “incomplete” resection [5].

In conclusion, it is important that physicians recognise the clinical features associated with PAS and include it as a differential diagnosis in the appropriate setting. Early referral for multidisciplinary management in a tertiary centre offers the only chance for prolonged survival and improved quality of life.

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A full list of references is available in the online version of this article.
Exercise-induced syncope – not always due to the worst cause

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\section*{Summary}

We report the case of a healthy 16-year-old ballet dancer with a history of three episodes of syncope during physical activity as the manifestation of an exercise-induced vasodepressor syncope. This diagnosis may be hard to demonstrate in clinical practice because of the potential poor reproducibility of the exercise test to provoke hypotension. Although most of the potentially fatal causes of exercise-induced syncope are usually well known and have a well-defined standard of care based on guidelines, vasodepressor type reflex syncope has only been described in a few case reports. The main mechanism seems to be an inappropriate peripheral vasodilation, which could be triggered by the activation of intraventricular mechanoreceptors sensitised by high circulating catecholamines in susceptible individuals.

Key words: exercise-induced vasodepressor syncope; exercise-related syncope

\section*{Case report}

A 16-year-old girl presented with a history of three episodes of syncope during physical activity. She was in full-time ballet training at a specialised ballet school and trained several hours per day. These three episodes of syncope happened around 15 to 20 minutes after starting medium to high-impact training, and were preceded by prodromal symptoms like fatigue, lightheadedness, blurred vision and nausea, without chest pain or palpitations. The loss of consciousness lasted less than 1 minute. She denied any injury as a consequence of syncope. No tonic clonic activity or postictal phase was observed.

The patient denied the use of drugs and was not taking any medication. She reported having had gastroenteritis 1 month before the episodes started. She had occasionally felt the same prodromal symptoms during training, with disappearance of these symptoms without interrupting her training. Although she reported a clearly small daily fluid intake (often as little as 1 L), she denied any past or present associated eating disorder, which was confirmed by her parents. Her weight was stable during the last year. She also denied amenorrhea, which should have raised the question of a “female athlete triad”. There was no past history of syncope. Her family history was negative for syncope or cardiovascular disease.

Physical examination was normal, except for a low Body Mass Index of 17.2 kg/m\textsuperscript{2}, with a height of 158 cm and weight of 43 kg. The ECG showed a normal sinus rhythm with a heart rate of 68 bpm (fig. 1). Thoracic echocardiography showed normal biventricular morphology and function, no valvular disease, and no signs of left ventricular outflow tract or supra-aortic obstruction. A treadmill test was performed (fig. 2), which showed a normal rise in heart rate and blood pressure during the first 8 minutes. After 12 minutes, at a velocity of 5.5 km/h and slope of 10\% (8.4 METs), the typical prodromal symptoms occurred. This was associated with a drop in blood pressure from 166/80 to 89/64 mm Hg and an increase of the heart rate to 166 bpm. Exercise was stopped after 13 minutes and during a recovery phase (velocity 2 km/h; no slope) with a duration of 5 minutes, her blood pressure gradually recovered with improvement of her symptoms. There were no significant electrocardiographic changes during treadmill testing.

Cardiac magnetic resonance imaging was then performed and confirmed the normal dimensions and function of the heart, including a normal coronary artery origin and course. There was no late gadolinium enhancement as a sign of a postmyocarditis or ischaemic scar. Head-up tilt-test (HUTT) was then performed (fig. 3). After 9 minutes in an upright position, a drop in blood pressure from 103/57 to 57/35 mm Hg and, after an increase in heart rate, sinus bradycardia (54 bpm) were observed, again provoking the prodromal symptoms of the patient, followed by a brief syncopal spell with rapid recovery after returning to a flat position. This pattern was consistent with a mixed vasodepressor and cardio-inhibitory vasovagal reaction.

Based on the abovementioned findings, exercise-induced vasodepressor syncope was diagnosed. An in-
crease was recommended in daily fluid and salt intake, which had certainly been inadequate considering her strenuous training programme. During follow-up, the patient still occasionally experienced her typical prodromal symptoms during exercise, but no more syncope episodes occurred.

Short review and discussion

This case illustrates a diagnosis that may be hard to demonstrate in clinical practice because of the potential poor reproducibility of the exercise test to provoke hypotension [5]. Although most of the potentially fatal causes of exercise-induced syncope are usually well known and have a well-defined standard of care based on guidelines, vasodepressor type reflex syncope has only been described in a few case reports since 1993 [5–9], and, maybe because of its good prognosis and low prevalence, no standard management recommendations can be found about this topic.

Definitions and prevalence

Exercise-related syncope is a general term used to describe post-exercise syncope as well as syncope occurring during exercise. Although the clinical distinction between these two forms of presentation is critical, they are often mixed in the medical literature (as in some references used for this manuscript). This can be explained by the fact that both forms may share some identical physiopathological mechanisms. Syncope occurring during exercise is and should always be feared by the clinician because it is known to be linked with significant and life-threatening pathology that should always be ruled out (table 1) [1].

After these serious causes have been ruled out, it should be considered that the syncope may be “neurally” mediated, or “neurocardiogenic”, in analogy to vasovagal syncope (fig. 4). Two different types have been described: a vasodepressor type and a cardioinhibitory type. Exercise-induced vasodepressor syncope (EIVS) has been described in case series published between 1993 and 2002 [4–9]. A cardioinhibitory type with prolonged asystole has also been reported, with a first case described as early as 1947 [12].

<table>
<thead>
<tr>
<th>Table 1: Exercise-related syncope: main causes to rule out.</th>
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<tbody>
<tr>
<td>Aortic stenosis</td>
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<tr>
<td>Hypertrophic cardiomyopathy (left ventricular outflow tract obstruction)</td>
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<tr>
<td>Bradycardia</td>
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<tr>
<td>Sick sinus syndrome</td>
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<tr>
<td>Atrioventricular block</td>
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<td>Tachyarrhythmia</td>
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<td>Ventricular tachycardia</td>
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<tr>
<td>(pre-excitation or long QT syndrome)</td>
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<tr>
<td>Catecholaminergic polymorphic ventricular tachycardia</td>
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<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy (ARVD)</td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
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<tr>
<td>Coronary anomalies</td>
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<tr>
<td>(not exhaustive)</td>
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</tbody>
</table>
A third entity named “exercise-associated collapse” is also found in the sport literature [2]. It occurs immediately after completing a race or stopping exercise and is commonly observed at endurance events. The athlete is unable to stand or walk as a result of lightheadedness, faintness, dizziness or syncope. The mechanism postulated is an abrupt decrease in venous return owing to the relaxation of the muscular pump when activity is stopped, with lower extremity pooling of blood and impairment of cardiac baroreflexes.

Training-related expansion of vascular volume is associated with a decreased heart rate response to baro-receptor stimulation and an exercise-induced change in cardiac filling volume and output may lead to a resetting of the cardiopulmonary baroreflex. For these reasons, trained individuals may depend more on maintenance of venous return to maintain upright body position [1, 2].

The exact prevalence of EIVS is unknown, but seems to be low, probably accounting for fewer than 2% of healthy young patients referred for unexplained syncope [3]. Most reported cases are seen in trained individuals (young athletes) and the typical patient is the young, female athlete.

Physiopathology

A few hypotheses have been described to account for EIVS, which are summarised in figure 5. These mechanisms have only been partially demonstrated [3] and there is no clear discriminator between exercise or post-exercise vasodepressor response.

The main mechanisms seem to be an inappropriate peripheral vasodilation (or lack of vasoconstriction), which could be triggered by the activation of intraventricular mechanoreceptors sensitised by high circulating catecholamines in susceptible individuals [3, 5, 6, 8, 9]. A blunted vasoconstriction during exercise has also been described and could be responsible for supplementary venous pooling in non-exercising regions [5].

The final result of these mechanisms is a profound hypotension with cerebral hypoperfusion, and, as a result, the occurrence of syncope.

In the post-exercise situation, the loss of the muscle pump with venous pooling implies also a sudden reduction in venous return to the heart, with forceful ventricular contraction and activation of c-fibre mechanoreceptors which trigger the vagal afferent pathway to the brainstem (this belongs to the ancient “Bezold-Jarisch reflex”), responsible for vasodilatation and the (sometimes) associated bradycardia [3, 5, 10].

A major part of the patients suffering from this condition seem also to demonstrate a positive head-up tilt response [4].

Investigations

Although no standard recommendations exist for this specific condition, the initial work-up should be the same as for any syncope. A cardiological work-up with a 12-lead ECG, echocardiography and a treadmill test should be performed to rule out structural heart disease responsible for a potentially life-threatening condition. The poor reproducibility of exercise-induced hypotension observed during a treadmill test may encourage repetition of the exercise test if initially normal [5]. A head-up tilt-test (HUTT) with pharmacological provocation should be considered to assess susceptibility to neurally mediated hypotension or bradycardia, which is a common finding in such patients. In our case, we decided to also perform
with reduction of syncope episodes in patients with documented vasodepressor response to HUTT [4]. However, no randomised study has been conducted to demonstrate this benefit. Because these same drugs have not been proven to have any beneficial effect in randomised controlled trials in vasovagal syncope, caution should be advised. For Swiss competition athletes, please refer to antidoping.ch.

The clinical course of patients suffering from exercise-induced vasodepressive syncope was favourable in the aforementioned case series, with symptom control most of the time. The only independent predictor of syncope recurrence was the number of exercise-related syncopal events in the clinical history [4].

Patients can safely continue to participate in recreational or competitive athletic activities, with the exception of athletes in whom hypotension or syncope could be life threatening for themselves or others (e.g., climbing, swimming, diving, etc.). The patient should be made aware of these dangers and possibly advised to take appropriate additional safety measures, or, if not feasible, stop the activity associated with syncope.

**Key points**

- Exercise-induced vasodepressor syncope is a relatively rare pathology among patients suffering from exercise-induced syncope.
- Exercise-induced vasodepressor syncope should always remain a diagnosis of exclusion.
- Basic noninvasive tests should be performed (ECG, echocardiography, treadmill test, head-up tilt-test, and possibly imaging of the coronary arteries) to rule out a structural heart disease responsible for a potentially life-threatening condition.
- The supposed physiopathological mechanism of EIVS is an inappropriate peripheral vasodilation from the vagal afferent pathway triggered by the activation of intraventricular mechanoreceptors in susceptible subjects.
- The prognosis of EIVS remains favourable and symptoms can be controlled most of the time, with no need of prolonged interruption of the activity associated with syncope. The exception is the cardioinhibitory form with prolonged asystole, in case of which pacemaker implantation should be considered.

**Disclosures**

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

**References**

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

A 76-year-old man was seen in the pacemaker clinic for routine follow-up. He had undergone implantation of a dual-chamber pacemaker (St. Jude Medical, Verity Adx XL, DR 5356) for bifascicular block and high-degree atrioventricular (AV) block on Holter-ECG after suffering syncope. After the implant, no more syncope occurred and the patient was feeling well. During the follow-up visit, recurrent runs of a tachycardia were observed (fig. 1), from which the patient was completely asymptomatic.

The following five questions need to be answered:

1. What is the tachycardia mechanism?
2. How does the tachycardia start?
3. Why does the AV delay prolong during tachycardia?
4. What is the mechanism of tachycardia termination?
5. What needs to be done to fix the problem?

What is the tachycardia mechanism?
The tracing in figure 1 shows a tachycardia with a CL of 500 ms and a 1:1 AV conduction. The tachycardia starts...
with a prematurely sensed atrial event (third atrial event on the tracing, marked * in figure 2) and the tachycardia is tracked to the ventricle with an AV delay of 240 ms. During tachycardia, there is double ventricular farfield oversensing in the atrium. The first additional signal on the atrial channel, marked with a blue up-arrow in figure 2, falls into the postventricular atrial blanking period (PVAB) and represents a farfield signal from the ventricular paced beat. The second oversensed signal (marked with a red down-arrow in figure 2) is probably oversensing of the end of the QRS complex; T-wave oversensing is unlikely because it occurred even before the T-wave. This atrial signal falls into the postventricular atrial refractory period (PVARP).

The differential diagnosis of the tachycardia at this point is an atrial tachycardia conducted to the ventricles by tracking of the pacemaker, or an endless-loop reciprocating pacemaker-mediated tachycardia (PMT) due to retrograde conduction of ventricular paced beats that are sensed in the atrium after the PVARP. The end of the tachycardia contains the solution to this: after seven beats, the tachycardia terminates with an atrial beat that is no longer tracked to the ventricle (marked § in figure 2). This makes atrial tachycardia highly unlikely, as one would have to assume simultaneous termination of an atrial burst and loss of ventricular tracking. The mechanism therefore is an endless-loop reciprocating pacemaker-mediated tachycardia.

How does the tachycardia start?

In this patient with higher degree AV-Block, there usually is concealed antegrade conduction into the AV-node preventing retrograde conduction of following ventricular paced beats back to the atrium. With the second atrial event in the tracing, however, there must have been block above the node or in the node, which then allowed retrograde conduction after the second ventricular paced (VP) event and initiation of the PMT (marked * in figure 2). Alternatively, this third atrial sensed (AS) event might also be a premature atrial beat, which blocks in the AV node and allows the next VP event to conduct back to the atrium and initiate the PMT. Given that the VA time is the same for all the tachycardia beats, this possibility appears less likely. Other precipitating factors capable of initiating a PMT could be ventricular extrasystoles, loss of atrial capture, atrial oversensing followed by atrial underpacing, atrial extrasystoles, or intermittent loss of atrial sensing.
Why is the atrioventricular delay prolonged during tachycardia?
The sensed and paced AV delays are programmed to 150 ms and 170 ms and are seen on the first and second beat on the tracing. With the third beat (first tachycardia beat), the AV delay is prolonged to 240 ms, which remains fixed throughout the tachycardia. The prolongation is due to upper-rate behaviour. Maximum tracking rate is 120/min, equalling a minimal ventricular interval of 500 ms. The interval between the two AS events initiating tachycardia, however, is 410 ms. Accordingly, the next VP can only be delivered after 500 ms, which results in a prolongation of the sensed AV delay (150 ms) by 90 ms to 240 ms.

What is the mechanism of tachycardia termination?
All contemporary dual-chamber pacemakers have algorithms to prevent endless-loop PMT. In the case of this patient’s SJM pacemaker, the PMT algorithm is active and would have intervened after 10 beats of PMT. However, the tachycardia already stopped after seven beats. On a closer look, the mode-switch was seen to be activated owing to inappropriate ventricular Far-Field oversensing in the atrium. With the inappropriate activation of the mode-switch and the associated change from the DDD-R into the DDI-R mode, the PMT terminated after seven beats even before the PMT algorithm was activated. The annotation of AMS (automode switch), however, only appears after the next VP event in older SJM devices.

What needs to be done to fix the problem?
The intrinsic VA time during V pacing was measured at 260 ms. After extending the PVARP from 250 ms to 275 ms, the PMT no longer occurred. Instead, the retrogradely conducted beats fell appropriately into the PVARP (fig. 3).

Summary
In summary, we report a pacemaker case, in which a common problem was resolved by the coincidental occurrence of a second common problem: a pacemaker-mediated tachycardia was terminated by inappropriate mode switch as a result of atrial oversensing. Even though the patient was asymptomatic in this case, the exact knowledge of the mechanisms leading to the occurrence of the two frequent problems as well as the appropriate steps to resolve them are important for the management of pacemaker patients.

Disclosures
No financial support and no other potential conflict of interest relevant to this article was reported.
Was fällt Ihnen auf diesem EKG auf?

Das paradoxe EKG

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Klinik für Kardiologie, Kantonsspital St. Gallen


Das EKG bei Aufnahme ist in Abb. 1 auf der linken Seitenhälfte zu sehen (Breitkomplextachykardie). Aufgrund der Befunde und der Anamnese wurde eine supraventrikuläre Tachykardie mit aberranter atrioventrikulärer Leitung angenommen und mittels intravenöser Adenosingabe ein Konversionsversuch unternommen. Abb. 1 zeigt das EKG während der Adenosingabe.

1. Welche Breitkomplextachykardien kommen bei Patienten ohne strukturelle Herzerkrankung vor?
2. Welche Diagnose können sie aufgrund des EKG nach Gabe von Adenosin (Abb. 1) stellen?


Zusammenfassend konnte anhand des EKG unter Adenosingabe eine orthodrome AV-Reentrytachykardie bei linksseitiger akzessorischer Bahn mit Linksschenkelblockaberration diagnostiziert werden, was in einer elektrophysiologischen Untersuchung verifiziert werden konnte.

Finanzierung/Interessenkonflikte

Die Autoren haben keine finanziellen oder persönlichen Verbindungen im Zusammenhang mit diesem Beitrag deklariert.

Literatur

Zum Gedenken an einen Pioni er der Angiologie

Zum Hinschied von Prof. Dr. med. Alfred Bollinger (1932–2015)

er die angiologische Dreiländertagung in Zürich durch
sowie verschiedenste mehrere nationale und lokale
Veranstaltungen. Zahlreiche nationale und internatio-
nale Preise und Ehrungen fielen ihm zu, natürlich auch
die Ehrenmitgliedschaften der Schweizerischen und
deutschen Gesellschaften für Angiologie sowie der
European und der World Society for Microcirculation.
Die Beschreibung dieser brillanten beruflichen Lauf-
bahn wird seiner Persönlichkeit aber nur zum Teil ge-
recht. Von seiner Herkunft her war ihm die Liebe zur
Literatur gegeben. Seine breiten Interessen umfassten
auch Natur und Kultur, die er beispielsweise beim
Bergsteigen (insbesondere auf Vulkan) und auf weiten
Reisen pflegte. Schon in Mexiko verfasste er eine Foto-
reportage über Tempel- und Ausgrabungen für die Kultur-
zeitschrift Du – und hatte natürlich auch den Popocate-
petl bestiegen. Von seiner Leichtigkeit des Schreibens
zeugen das Lehrbuch Funktionelle Angiologie, zahlrei-
che Buchbeiträge und Herausgeberschaften sowie an
die 500 Originalpublikationen aus der wissenschaft-
lchen Zeit. Unter anderem war er auch Herausgeber
der angiologischen Zeitschrift VASA.

Er zog sich 1995 von seiner Klinik vorzeitig zurück, um
sich fortan der Schriftstellerei und Fotografie zu
widmen. Er publizierte drei Romane, mehrere medi-
zengeschichtliche Artikel und Buchbeiträge. Am
schönsten wird seine vielseitige Begabung in den Foto-
büchern sichtbar, die er mit seiner Frau Verena mit
Aufnahmen aus seiner Wohnung hoch über dem
Zürichsee und von seiner Trauminsel Stromboli
verfasste.

Ein letztes Wort gilt seiner Fähigkeit zur Geselligkeit,
zum menschlichen Anteilnahme und langjährigen
Freundschaften. Es gibt kaum einen Mitarbeiter oder
eine Mitarbeiterin, ärztlich oder nichtärztlich, für
deren Leben und Fortkommen er sich nicht interesse-
sierte. Dazu gehörte auch «seine dritte Tochter, die
Angiologie», wie er es auszudrücken pflegte.

Für viele von uns ist er zu einer eigentlichen «Stan-
dard-Referenz» im Leben und Beruf geworden. Die ihn
persönlich kannten, werden ihn nie vergessen.

Felix Mahler
The New Postgraduate Course in Heart Failure (PCHF) – Update on the 1st and Announcement of the 2nd PCHF Course Edition

A project of the European Society of Cardiology European Heart Academy, the European Society of Cardiology Heart Failure Association (ESC HFA), the Zurich Heart House and the University of Zurich

Lydia Tchambaz, Ruth Amstein, Francesco Maisano, Thomas F. Lüscher and Frank Ruschitzka

Zurich Heart House Education Centre, University Heart Centre, Departments of Cardiology and Cardiovascular Surgery and Medical Faculty of the University of Zurich, Switzerland

Heart failure has become the major cause of hospitalisation in patients over 65 years and the new epidemic of the 21st century. New training programmes for cardiologists are needed in order to develop a professional profile of specialists competent in all aspects of the care of patients with acute and/or chronic heart failure.

Last year, the European Society of Cardiology Heart Failure Association (ESC HFA) published a core curriculum for heart failure physicians (1). The core curriculum, led by Theresa McDonagh, has become the reference for the first Postgraduate Course in Heart Failure. The Zurich Heart House (ZHH) in collaboration with the ESC HFA and the European Society of Cardiology (ESC) and its European Heart Academy (EHA) have developed a unique Postgraduate Course in Heart Failure (PCHF) leading to a Certificate of Advanced Studies (CAS) in Heart Failure Management delivered by the University of Zurich and accredited by the ESC.

The PCHF is a 2-year programme consisting of eight modules of 2.5 days each covering all relevant aspects of heart failure such as pharmacotherapy, devices and imaging, and containing live transmissions of procedures (i.e. cardiac catheterisation, device implantation, TAVI and MitraClip), patient visits, hands-on courses in device programming and cardiac imaging (in particular echocardiography, but also magnetic resonance imaging and nuclear tests), case-based learning and virtual interventions (i.e. cardiac resynchronisation therapy (CRT) and implantable cardioverter defibrillator implantation) with a total of 160 hours of lectures and 140 hours of self-study as well as an e-learning platform with webinars and webcasts of the lectures. The particular strength of PCHF is the provision of a platform for interactions and discussions between speakers and participants, which is highly appreciated. Considering the uniqueness of the course, PCHF is one of the most exciting current educational projects in cardiology, made possible thanks to a strategic collaboration between academia, professional organisations and industry partners.

The first PCHF – an update

Contrary to the original intention to accept only 40 participants and owing to the high number of applications, the organisers finally accepted 59 cardiologists coming from 31 different countries around the world (fig. 1). The first six modules have been held successfully and the feedback received so far is extremely positive and encouraging (fig. 2). The first module was held from 30 January to 1 February 2014, led by Theresa McDonagh from London. The highly motivated local team from the University Hospital Zurich, consisting of consultant and resident physicians under the leadership of Frank Ruschitzka and Thomas F. Lüscher, contributed excellent patient cases and the organisation of instructive clinical stations and rapid-fire sessions (fig. 3). The second module on different forms of heart failure was led by Karl Swedberg from Gothenburg and took place in April.

Figure 1: 59 course participants from 31 countries at the fourth module of PCHF, University Hospital Zurich, October 30th to November 1st 2014.
2014. The third module, also on different forms of heart failure, was held in June 2014 and co-chaired by Scott Solomon from Boston and Carolyn Lam from Singapore. The fourth module, co-chaired in October 2014 by John McMurray from Glasgow and Stephan Anker from Berlin, provided insight in the treatment and follow up of patients with heart failure. The fifth module, co-chaired by Jan Steffel from Zurich and Gerd Hindricks from Leipzig, provided a lively and interactive introduction to the topic of device therapy in heart failure, and the sixth module held in Brussels in March 2015 was entirely dedicated to practical training for implanters with live case transmissions from the University Heart Center of Leipzig (fig. 4). The distinguished faculty, the quality and content of the lectures, the lively interaction between participants and renowned experts, as well as the course location (University Hospital of Zurich) contribute to a high reputation of the programme.

The 2nd PCHF: ready for take-off in 2016
A 2nd edition of the PCHF was announced at the Heart Failure Congress in Seville. The course will again consist of eight modules over two years starting in January 2016 and lasting until October 2017. Applicants must be board-certified cardiologists or medical doctors (MDs) having completed at least three years of relevant clinical training in cardiology, have a strong interest in heart failure, preferentially also documented by publications in this specialty. Participants in the 1st PCHF who have passed the examination will be awarded a dual certification: a certificate for professional skills accredited by the ESC Heart Academy and an academic certificate from the Medical Faculty of the University of Zurich. The course has the status of postgraduate programme according to the Bologna system, leading to a CAS in Heart Failure Management equivalent to 10 ECTS. Interested cardiologists can apply online by September 15, 2015 at www.heartfailurecourse.ch.

Course directors
Frank Ruschitzka, Thomas F. Lüscher, Francesco Maisano, all Zurich, Stefan Anker, Berlin and Gerasimos Filippatos, Athens

Advisory board
Jeroen J. Bax, Leiden; Gerhard Hindricks, Leipzig; Michel Komajda, Paris; Aldo Maggioni, Milan; Theresa McDonagh, London; Burkert Pieske, Berlin; Fausto Pinto, Lisbon; Piotr Ponikowski, Wroclaw; Karl B. Swedberg, Gothenburg; Alec Vahanian, Paris; Panos Vardas, Heraklion.

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Figure 2: Topics of the eight course modules.

Figure 3: Course Directors and faculty members (from left): Frank Ruschitzka, Zurich, Thomas F. Lüscher, Zurich, Theresa McDonagh, London and John Cleland, London.

Figure 4: CRT implant hands-on session led by Christoph Leclercq from the University Hospital Pontchaillou, Rennes, France.
A PCI live course where it all began: The 4th European Live Summit on Retrograde Chronic Total Occlusion Revascularization at the Andreas Grüntzig Catheterization Laboratories on May 8–9, 2015

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When Andreas Grüntzig did his first procedures with his innovative balloon in 1977, he dared to treat only proximal type A lesions. With the introduction of guidewires more distal lesions became accessible. With stents also, more complex plaques were treatable, particularly with the availability of drug-eluting stents. Chronic total occlusions (CTOs) of a coronary artery, however, remain a challenge for most interventionists. With the initially available wires, the success rate was quite low, but thanks to the development of a large spectrum of innovative wires with different properties and microcatheters allowing wires to be changed even in already probed segments, the success rate could be markedly improved and in experienced hands is reaching 80% and more. Both the classical antegrade as well as the more challenging retrograde approach are currently used.

The practice of CTO interventions has been propagated in large part by the Euro CTO Club (www.eurocto.eu) which was founded in 2006 by the pioneers of the procedure Alfredo R. Galassi, Joachim Buttner, Carlo di Mario, Gerald S. Werner, Dariusz Dudek, George Sianos, Nicolaus Reifart and Hans Bonnier. Since its foundation the mission of the CTO Club was to teach the community of interested interventionists about the procedure and the use of novel materials developed by several companies on a yearly basis. To that end, yearly live courses have been organised and registries established. The First European Live Summit on Retrograde CTO Revascularisation took place in Catania in 2012, followed by the second meeting in 2013 and the third in Sofia, Bulgaria, in 2014.

The University Heart Centre is proud that the 4th European CTO Revascularisation Summit took place this May in Zurich, where it all started on September 16, 1977 with Andreas Grüntzig (fig. 1). The Zurich course took place on Friday and Saturday, May 8 and 9, 2015 with 12 live cases and live in-a-box case presentations by participants, as well as lectures by highly experienced interventional cardiologists in the Andreas Grüntzig Catheterisation Laboratories. With faculty members from 5 countries and 155 participants, the technique of antegrade and retrograde revascularisation of CTOs was demonstrated to participants in detail, with educational live cases (fig. 2). Each case was presented by a fellow of the Department of Cardiology and the strategy extensively discussed by the operators and the panel of experts, from the selection of the guiding catheter to the different guide wires, the handling of microcatheters, the use of intravascular ultrasound to the selection of balloons and stents (fig. 4). A session for nurses taken in the German language illustrated all tips and tricks for assistance with different devices in these particular procedures.

Figure 1: A. Galassi from Catania (Course Director) and O. Gaemperli from Zurich (Course Co-Director).

Figure 2: An enthusiastic audience followed the lecture by Masahisa Yamane, the pioneer of CTO procedures.
With the success of this year’s course, another session is already secured and awaits applications from colleagues interested in chronic total occlusions.

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