Kardiologisk vårmøte i Bergen, 29.-31. mai 2008

VESTNORSK CARDIOLOGISK FORENING

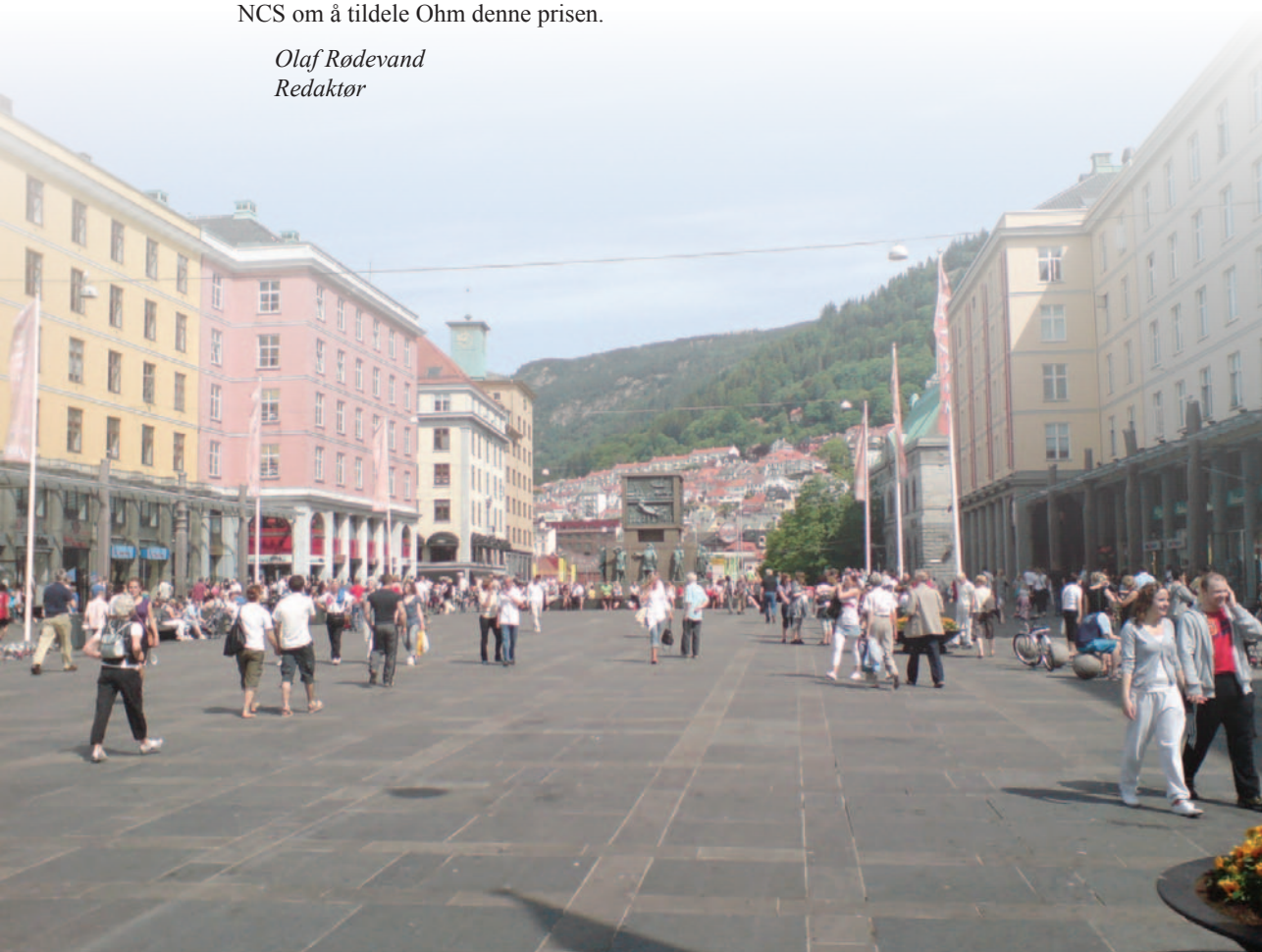
Et usedvanlig flott vær og mange internasjonale foredragsholdere preget årets NCS-vårmøte som ble avholdt i Bergen. Selv om deltakerantallet på 233 lå noe under fjorårets Oslo-tall på 330, må man anse at møtet var godt besøkt. For de tallinteresserte kan vi supplere med at deltakerne besto av 179 leger (inkludert foredragsholderne), 15 sykepleiere og 39 ustillere.

Det var et variert program med 8 sesjoner: hjertesvikt, helsepolitikk, preventiv kardiologi, arytmier, posterpresentasjon, medfødte hjertefeil og hjertekirurgi, noninvasiv diagnostikk og endelig invasiv kardiologi. Bak bringer vi referater fra en del av innleggene.

Prisen for beste posterpresentasjon gikk til Vegard Thuseth for presentasjonen: Percutaneous Left Ventricular Assist Device in Cardiac Arrest.

Professor Ole-Jørgen Ohm ble tildelt NCS' hederspris for sitt langvarige og banebrytende arbeid for kardiologisk forskning innen pacemakerutvikling og elektrofysiologi. Bak møtereferatene har vi tatt med den skriftelig begrunnelsen forslagstillerne la frem når de oppfordret styret i NCS om å tildele Ohm denne prisen.

Olaf Rødevand
Redaktør



The link between exercise intolerance, myocardial dysfunction and heart failure

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Heart failure is a complex syndrome characterized by a series of maladaptive responses in several districts to left ventricular dysfunction, generating a vicious cycle of progressive deterioration. The most common abnormalities are sympathetic hyperactivity and fluid retention which both contribute to the clinical picture and outcome. Exercise intolerance is a hallmark symptom very popular to stratify the severity of chronic heart failure and the prognosis. It may be defined as a reduced capacity to perform physical activity as compared to normal individuals matched for age, sex, height, weight and race.

From a database of 5400 consecutive cardiopulmonary exercise stress tests from our laboratory at the Lancisi Heart Institute, chronic heart failure patients had a peak VO2 of 15.8±5.3 ml/kg/min corresponding to 48±14% of predicted VO2max, while normal subjects had a peak VO2 of 28.1±7.8 ml/kg/min corresponding to 83±23% of VO2max ($p<0.001$). Oxygen uptake is probably the most accurate measure of functional capacity because it reflects the product of cardiac output by a-v O2 difference at any given workload. Peak VO2 gives important clinical information to grade the severity of heart failure (Weber classification) and to assess the prognosis of heart failure patients. The combination of peak VO2 with other parameters derived from analysis of ventilation and gas exchange, such as ventilation versus VCO2 (VE/VCO2 slope) or anaerobic threshold significantly improves the prognostic assessment and clinical decision making process of heart failure patients. A decreased functional capacity may depend on several factors along the "O2 cascade". Each of the above mentioned parameter may contribute to exercise intolerance and amplify the effect of others.

The majority of studies published before the '80s emphasizes the role of central hemodynamics as major limitation to exercise capacity. Although peak VO2 is correlated with peak exercise blood flow and cardiac output, central factors are not the only one playing a role in this setting. For instance, peak exercise pulmonary wedge pressure is not correlated to peak VO2 or symptoms of exercise limitation like breathlessness or fatigue. Acute increase in skeletal muscle blood flow with vasodilators or inotropic agents does not increase exercise capacity or decrease lactate production in CHF despite increased cardiac output. Peak VO2 is decreased as compared to normal subjects. To compensate a decreased peak VO2 and increased vascular resistances a-vO2 difference is increased at submaximal levels as well as O2 venous content and saturation. However, at peak exercise all three parameters are not different from normal individuals, suggesting that O2 delivery is not the prevalent limiting factor. In support of this interpretation there is evidence that some CHF patients have a normal cardiac output/work rate slope and normal leg blood flow during exercise and yet early lactate production and decreased peak VO2. In another study, reductions in phosphocreatine and pH during exercise were associated with normal blood flow of the forearm. By converse, early anaerobic metabolism has been observed in CHF even after occlusion of skeletal muscle blood flow. All this evidence suggests that, in addition to O2 delivery, histological and biochemical changes in skeletal muscle may play an important role in determining skeletal muscle

alterations contributing to early acidosis and fatigue.

Chronic heart failure is characterized by an impaired arterial BR and augmented sympathetic nerve activity which may contribute to exercise intolerance and the progression of the disease. A variety of factors are involved in the genesis of reflex abnormalities. These include changes in the responsiveness and end organs to autonomic stimulation, alterations in autonomic neurotransmission, changes in synthesis, storage and release of autonomic neurotransmitters, alterations in the central integration of information from the periphery, the influence of elevated levels of regulatory hormones on reflex function, and changes in the sensory elements as well. As a result, muscle sympathetic nerve activity (MSNA) is increased independent of age or resting heart rate, while it is correlated with the severity of cardiac dysfunction. Muscle metaboreflex is also activated by early anaerobic metabolism and contribute to MSNA and exercise intolerance.

Patients with CHF have a high oxidative stress. Our group recently found a decreased activity of extracellular superoxide dismutase (ecSOD) in plasma of patients with chronic heart failure that was partially reversed by the oral administration of coenzyme Q10 at doses of 300 mg t.i.d. The decreased levels of ecSOD may explain the increased activity of superoxide anion that inactivates nitric oxide by producing peroxynitrite. In fact, we found a correlation between ecSOD and the endothelium-dependent dilation measured at the brachial artery. In conditions of high oxidative stress, such as chronic heart failure and multiple coronary risk factors, the rate of inactivation of nitric oxide to peroxynitrite by superoxide anions may be reduced by CoQ10, which can also protect against nitrosative damage. An increased oxidative stress has been also documented in the brain. Recent studies by Zucker et al showed that oxidative stress plays an important role in the sympathoexcitation in CHF. An upregulation of NADPH oxidase expression and activity has been found in the rostral ventrolateral medulla (RVLM) that represents the primary central site for the maintenance of sympathetic nerve activity (SNA). A decrease of central superoxide anion by tempol, a SOD mimetic, reduced sympathetic outflow in CHF rabbits. In contrast, an increase in central O_2- induced by a SOD inhibitor (diethyldithiocarbamic acid (DETC)) significantly augmented sympathetic outflow in normal and CHF rabbits.

Angiotensin II is implicated in this process. Increases in central angiotensin II have been shown to increase sympathetic outflow and blunt arterial baroreflex responses, while administration of losartan has been shown to reduced sympathetic outflow and enhance baroreflex sensitivity in rats with CHF. Angiotensin II augments ROS formation and increases oxidase activity by upregulation of NADPH oxidase. Since ROS are linked to regulation of sympathetic nerve activity, a reduction in ROS should decrease SNA and improve ex tolerance.

Angiotensin II acts via an AT1 receptor mechanism to activate sympathetic outflow and impair arterial BR function by stimulation of NADPH oxidase and ROS in the RVLM. Angiotensin II increases mRNA and protein levels of NADPH oxidase components with concomitant increases in superoxide anions in the RVLM, elevations in RSNA and impaired arterial baroreflex function. Losartan- a specific AT1 receptor antagonist- significantly decreases basal RSNA and improves arterial baroreflex sensitivity with no change in NADPH levels and local superoxide production in RVLM, which suggests a non-ROS pathway.

In summary, exercise intolerance is a typical symptom of heart failure. It depends on a complex interplay between central and peripheral factors. Many parameters have been associated with a decreased functional capacity in heart failure. A “muscle” hypothesis emphasizes the main role of peripheral abnormalities in the genesis of symptoms. However, the fact that central factors such as the amount of viable myocardium, a restrictive pattern of left ventricular filling and a decreased slope of cardiac output related to work rate may predict the response to exercise training in CHF patients suggests that peripheral changes are not the main determinants of exercise intolerance. A reduction of sympathetic hyperactivity should be attained in order to improve functional capacity and patient survival.

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Medisin eller økonomi: Hva skal styre pasientbehandlingen?

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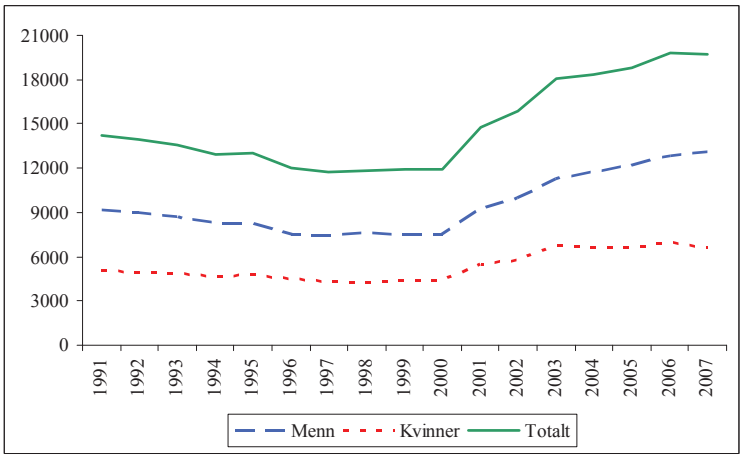
Svaret er begge deler. Samfunnsfagene, og i særdeleshet økonomifaget, rykker imidlertid nærmere klinikken. Samfunnsviterne og økonomene vil gjerne være med på å prege medisinerens beslutninger og av og til overstyre dem. Det kan det bli god medisin av. Jeg skal gi tre eksempler på hvordan økonomer og samfunnsvitere indirekte og direkte påvirker medisinske beslutninger og deretter litt provokatorisk spørre hva kardiologer kan lære av dette.

Mitt første eksempel gjelder fordelingsmodeller mellom regioner. Magnussenutvalget har nettopp framlagt sin innstilling og foreslår at ressursene fordeles etter demografiske, sosioøkonomiske og klimatiske kriterier. Klimakriteriene, inklusive breddegrader, har med rette skapt debatt, men virker i dag å være aksepterte. En fordelingsmodell basert på kriteriene gir en viss omfordeling fra Helse Sør-Øst til de andre RHFene. Målet er at tilbudet skal reflektere behovet for tjenester – en skal gi likt tilbud for like behov. Hvordan står det til med tilbudet hos norske kardiologer? Jeg har gått gjennom bruk av PCI hos pasienter som er over 90 år og som er innlagt for hjerteinfarkt. Dette er naturligvis en marginal gruppe. Likevel – forskjellene er store. I Oslo, Akershus og Hordaland fikk mellom 4-6 % av denne gruppen utført PCI i 2007. I Midt-Norge ingen. Gir det grunn til refleksjon?

Mitt andre eksempel gjelder finansieringssystemene. De siste årene har finansieringssystemene for somatiske helsetjenester vært sammensatt av en ramme og komponent av aktivitetsbasert finansiering (ABF). Intensjonen med ABF har vært å øke aktiviteten for å redusere ventetidene. En har et stykke på vei lykkes. Men det har hele tiden vært en uklarhet i system knyttet til hvor stor aktivitetsveksten skal være. ABF-komponenten har vært relativt lav, men vært gitt uavkortet og har dermed gitt signaler om sterk aktivitetsvekst. Samtidig har rammen vært justert lite. Sykehusene har respondert ved sterk aktivitetsvekst. Siden den marginale inntekten har vært lavere enn de marginale kostnadene ved denne aktivitetsveksten, har en endt med store underskudd i sykehusene. Samfunnsviternes råd har hele tiden vært klare. Vi har sagt at en må etablere klarere koplinger mellom aktivitet og finansi-

ering, enten ved å velge en høyere aktivitetsbasert komponent eller ved å gjøre rammen styrende for aktiviteten. Medisinerne har heller ropt på tilleggsbevilgninger. Hvorfor tror dere at tilleggsbevilgninger er en god måte å finansiere sykehus på?

Mitt tredje eksempel gjelder effekter av medisinske intervensjoner. Økonomer stiller spørsmål ved om nytten av en intervensjon står i forhold til kostnaden. Nytten av en intervensjon måles gjerne som QALYs, der QALYs er produktet av økt forventet levealder og økt forventet helsetilstand. Kostnadene måles i penger og omfatter ressurser forbundet samfunnets samlede resursbruk. Bør en utføre PCI på 90-åringer? Vi vet ikke. Per mai 2008 finner jeg ingen publiserte kostnads-nytte-analyser av denne intervensjonen for denne selekterte gruppen. Men jeg fant en studie som indikerer relativt høy overlevelse etter PCI. Det er et viktig funn, men vi må gå dypere. Her kan samfunnsvitere og medisinere gå sammen.



Figur: Antall infarkt 1991-2007. Antall innlagte med AMI (ICD9 410 eller ICD10 I21og I22) som hoveddiagnose.
Kilde: Reikvam & Hagen (2002) Scandinavian Cardiovascular Journal, samt oppdateringer

Til slutt, en bonusplansje. I og med at jeg talte opp alle PCIer gjennomført blant 90-åringene så tok jeg også og talte opp antall innleggelser for hjerteinfarkt de siste årene. For øvrig håper jeg å ha provosert til debatt ….

ECG in screening of young athletes - the Swedish recommendations

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Sudden cardiac death (SCD) in athletes is an uncommon but tragic event in sports that gives rise to large media coverage. Several highly exposed cases of SCD in recent years have highlighted the question if these cases could have been prevented, or the risk for events reduced, by the use of cardiac screening of athletes.

Competitive athletes - risk group?

Elite sports activity puts great pressure on the cardiovascular system to perform at maximal function. In fact, ultra-endurance exercise has been associated with myocardial damage as expressed by cTnI as well as a self-resolving reduction in right ventricular function (La Gerche A, Heart 2008; 94: 860-6). Several aspects related to sports, including catecholamine release, platelet adhesion/activation, electrolyte disturbances, heat, cold, altitude, medications, (doping), dehydration and nutrition could increase the risk in vulnerable individuals.

As shown by Corrado in 2003 (JACC 2003;42:1959-63) competitive athletes indeed have a higher risk for SCD (relative risk 2.8) compared to non-athletes.

The reasons for SCD in young athletes (<35 years old) are mainly congenital/inherited heart diseases such as hypertrophic obstructive cardiomyopathy (HCM) or arrhythmogenic right ventricular cardiomyopathy, coronary artery anomalies as well as long QT-syndrome or Brugada Syndrome. Aortic stenosis, myocarditis and complications related to Marfans syndrome are other reasons for SCD in young athletes. However, coronary artery disease is a very uncommon cause of SCD in young athletes, but the most common reason in athletes (and non-athletes) >35 years of age. Around 1/300 young athletes do have an (unknown) underlying cardiac abnormality that increases the risk for SCD during athletic activity.

Cardiac screening

In 2005 the European Society of Cardiology published recommendations on cardiac screening of competitive athletes (Corrado D, Eur Heart J. 2005: 26: 516-24), including personal history, physical investigation and 12-lead resting ECG. The aim was to produce common European recommendations to identify athletes with underlying heart disease and a higher risk for SCD and thereby to reduce the incidence of SCD related to sports. In Italy, cardiac screening had been mandatory in all athletes since 1971, while in several other countries including the Nordic countries no formal screening existed.

Automated defibrillators- a complement

If an acute event of sudden cardiac arrest does occur in a sports setting (or elsewhere) the key to survival is the ”time to defibrillation”. The goal for a good medical action plan is to apply ”the chain of survival” with acute resuscitation, early defibrillation (within 3-5 minutes) and cooperation with the local emergency system, which has been shown to considerably increase the survival rate of sudden cardiac arrest. Thus, the automated external defibrillator is an important part in increasing the survival in sudden cardiac arrest. However, recently some doubts have been aired, concerning the efficacy of automated external defibrillators for sinus conversion in athletes with underlying myocardial structural disease as compared to the non-athletic sudden cardiac arrest cases with coronary artery disease. But, the use of automated external defibrillators may complement cardiac screening to decrease death in sports.

The Swedish experience

In Sweden, several highly exposed cases of SCD in 2004 and 2005 prompted the minister of Sports and minister of Health to initiate two separate expert groups to produce Swedish recommendations on the role of screening in SCD. The first group headed by the Swedish National Federation of Sports declared in September 2005 that cardiac screening (comprising physical history, investigation and resting ECG) was recommended in Sweden for elite athletes only, from the age of around 16. It was the responsibility of the sports, i.e. the team doctor or elite sporting schools physician that the screening was performed. The Swedish National Board of Health and welfare included the expert groups recommendations in the ”National guidelines for cardiovascular health care (Socialstyrelsens riktlinjer för hjärtsjukvård) in December 2006, stating that cardiac screening was recommended in risk groups only. These risk groups include persons with alarming symptoms (syncope during exercise etc), positive family history (case of SCD or inherited cardiac disease in first line relatives), in those having an abnormal ECG as well as elite athletes. These recommendations confirmed the earlier recommendations by the National Federation of Sports and also confirmed that abnormal findings in the initial screening by the sports community should be further evaluated within the Health Care system (by physicians with a special interest in sports cardiology).

Does the screening save lives?

Since the step-wise introduction of cardiac screening in northern Italy in 1979, the incidence of SCD has decreased by 89% and is now lower in athletes compared to the non-screened non-athletic population (Corrado D, JAMA 2006; 1593-601), where the SCD-incidence is unchanged. The lower death rates are due to a decrease in deaths related to HCM. At the same time the disqualifications due to HCM has increased, indicating that the cardiac screening

has been effective in reducing the incidence of SCD in athletes.

Sensitivity and specificity of ECGs in screening

The ECG is recommended in cardiac screening both by the ESC and in Sweden, in contrast to the US where only personal history and physical examination is recommended. The ECG is practical, easy to obtain, relatively cheap and is also important for reference. What about sensitivity and specificity?

The sensitivity of the resting ECG for the most common cause of SCD, HCM, is high. Up to 90% or more of HCM-patients show ECG-abnormalities, often in the absence of symptoms. However, the sensitivity is considered lower in children. Also, the sensitivity for arrhythmogenic right ventricular cardiomyopathy is fairly high, up to 88% in an Italian study. The sensitivity for coronary artery anomalies is low, but other arrhythmia related diseases such as long QT-syndrome, WPW and Brugada syndrome may often be detected. Most SCD cases in a Swedish study by Westén (J Int Med 2004; 255: 213-20) showed previous ECG abnormalities (82%) in addition to a positive family history (18%) and symptoms (76%).

Regarding specificity, we now know that although 15% of athletes may show abnormalities on ECG, these changes mostly reflects ”innocent” ECG-changes, and only 2-5% show changes that necessitate further cardiovascular investigations. The presence of severe ECG-abnormalities may signal the initial expression of an underlying cardiomyopathy that may be visible only on repeated follw-up (Pelliccia N Engl J Med 2008; 358: 152-61). In contrast, a normal ECG at cardiac screening has a very favourable prognosis, indicating a very low risk of underlying cardiac disease. Only 2.2 % of healthy athletes in the 1994 study by Björnstad (Cardiology 1994; 84: 51-60) showed T-negatives beyond 2 leads. The ESC is currently working on developing tools to help the clinician define what are acceptable ECG-changes due to physiological adaptation to training, and what constitutes an abnormal ECG that necessitates further cardiovascular examinations.

Implementation

Cardiac screening of athletes is now also mandatory in FIFA organized tournaments as well as in UEFA-tournaments, while the IOC in 2006 published their Lausanne protocol that recommends cardiac screening with personal history, physical examination and ECG. For the Nordic countries we recently proposed (Hernelahti M, Scand J Med Sci Sports 2008; 18: 132-9) a screening programme of the elite athletes only (partly for logistical reasons). Denmark has started cardiac screening on elite athletes in 2008. In Norway, no formal recommendations exist at present.

Summary

Directed cardiovascular examination of elite athletes makes common sense. We do need personal history to find individuals with a positive family history and/or severe symptoms, and if we do not ask we will not get an answer. Resting ECG adds valuable information for future reference and has a high sensitivity for the most common cause of SCD in athletes, hypertrophic cardiomyopathy.

Prevention of events in hypertrophic cardiomyopathy: the Italian registry

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With an estimated prevalence of 1:500, hypertrophic cardiomyopathy (HCM) is no more considered a rare disease and affects a fair number of people in the world. In the last 50 years the understanding of this fascinating disease and optimal care of HCM patients has greatly improved in most areas of the world, with reduced HCM-related events and death. Medical treatment is generally used on an empirical basis in order to control symptoms, slow disease progression, control arrhythmias and prevent heart failure and sudden death (1).

The classification of cardiomyopathies was recently updated by the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology (2). It emphasized a practical approach based on different phenotypes, and familial disease transmission (3). The identification of the genetic or metabolic background, as lysosomal storage or mitochondrial diseases (sarcomeric versus non sarcomeric mutations), when available, is of great importance for precise characterization of the HCM subtype and specific treatment options. In clinical practice differential diagnosis with other causes of LVH, including supravalvular or fixed subaortic stenosis is necessary. Amyloidosis and LV non compaction should also be carefully excluded.

The Italian HCM registry collected 1677 patients from 40 centres, with on average 9.7 years follow-up (figure 1) (4). Atrial fibrillation and disease progression with increased levels of functional limitation to the end stage phase and refractory heart failure are expected in a substantial proportion of patients with HCM. Cardiovascular mortality was 1% per year, while sudden and unexpected death (SD) was rare, with an incidence of 0.3% per year. Atrial dilatation with a cut off point of 48 mm (by M-mode echocardiography) emerged as a major risk factor both for atrial fibrillation and stroke, for HCM related death and even sudden death (5).

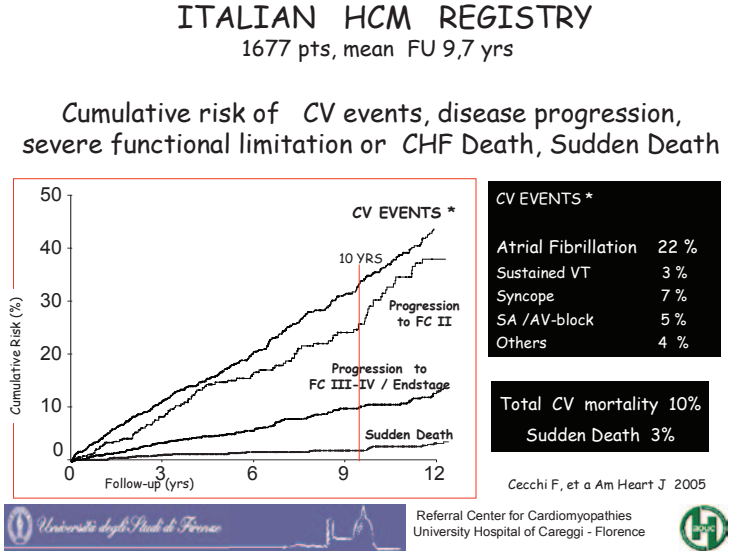

Figure 1. The Italian HCM registry

In order to prevent events and start appropriate treatment, old and new risk factors should be taken in account. Careful assessment of family history, functional limitation, symptoms and risk factors, including site and extent of hypertrophy, left atrial size, diastolic and systolic dysfunction, microvascular dysfunction, basal or inducible intraventricular obstruction and mitral regurgitation, atrial and ventricular arrhythmias, is mandatory (6-9). Competitive sport and intensive physical activity should be discouraged and avoided.

Therapeutic strategy should no longer be limited to control symptoms, but also to reduce the risk of future complications, including disease progression, atrial fibrillation, stroke and heart failure. Relief of intraventricular obstruction and mitral regurgitation by cardiac surgery or other interventional approaches may be greatly beneficial for symptom control. However, evidence for increased survival has only been demonstrated for patients who had extended myectomy (10).

Medical treatment is anyway necessary, and usually patients refer symptom improvement with β-blockers when mild symptoms and functional limitation are present. In more advanced disease stages, usual heart failure treatment is the treatment of choice, including furosemide, ARB, oral anticoagulation, and finally CRT or heart transplant in the end-stage phase. β-blockers and amiodarone are often beneficial when supraventricular arrhythmias occur, both for treatment and prevention. Careful drug titration is necessary in order to avoid chronotropic incompetence and iatrogenic symptoms (Table 1) (11). Atrial fibrillation can be successfully treated with pulmonary veins ablation and additional linear incision even when left atrium is enlarged and persistent atrial fibrillation is present (12).

Table 1. Medical therapy

Intraventricular obstruction
- ß-blockers
- Disopyramide (250-750 mg)
- ß-blockers + Disopyramide

Arrhythmias (AF; EB; nsVT)
- Amiodarone (low-dose)
- ß-blockers + amiodarone (low dose)

Angina and Microvascular dysfunction
- ß-blockers + Cc-blockers (e.g. Felodipine)

Heart Failure
- ß-blockers (Carvedilol, Bisoprolol),
- Amiodarone (low dose),
- Furosemide
- ACE-I, ARB, Nitrates
- Oral anticoagulation
- Heart transplant

In addition: Pacing, CRT - when needed (SA or AV block)

Sudden and unexpected death is difficult to predict. ICD may prevent SD with an appropriate annual discharge rate of 10% in secondary and 4% in primary prevention. However, about 25% of patients also experience inappropriate discharges, mostly due to atrial fibrillation (13). Together with depression, sepsis and lead rupture, they constitute the price patients have to pay after an ICD is implanted. Careful patient selection and information of the consequences of ICD is mandatory.

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Early Experience with a New Magnetic Navigation System for Catheter Manipulation

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The availability of remote navigation by mean of two permanent magnets, the positions of which are computer controlled, has started a new era in cardiac electrophysiology and radiofrequency catheter ablation of cardiac arrhythmias. When located close to the thorax (in navigate position), both magnets create a relatively uniform magnetic field (0.08 Tesla) inside the chest of the patient. The distal tip of the mapping/ablation catheter is loaded with three small permanent magnets. These will align with the direction of the externally controlled magnetic field, thus steering it effectively. The advancement and retraction depend on a small system (cardiodrive) that is sticked to the operating field and connected to a computer controlled rotating wire. One major advantage is the particularly smooth catheter tip, which results in an increase of the safety of the procedure by preventing any possibility of heart perforation, while providing a remarkable accuracy and reproducibility of catheter movements. Simultaneously, a growing number of ablation are being performed using a special version of a commercially available computerized electro anatomic systems (CARTO RMT).

The real time position of the catheter tip is permanently calculated and displayed on the computer screen, while the catheter is remotely manipulated, and the radiofrequency current delivered from the control room. Examples of successful ablation are shown. Similarly cardiac chamber voltage mapping using the system has been performed in the diagnosis of suspected arrhythmogenic ventricular dysplasia and has been compared with conventional (manual) mapping. The former seems to be more precise and generates non distorted yet larger volume right ventricular silhouettes.

The combination of the accurate real-time location of the catheter tip with the enhanced magnetic navigation capabilities allows remote mapping and ablation of various supraventricular and ventricular tachycardia.

Pharmacogenomics of Hypertension

Sandosh Padmanabhan, BHF Glasgow Cardiovascular Research Centre, University of Glasgow

Optimal blood pressure control is achieved in only one quarter of hypertensives, despite >100 antihypertensive drugs available, leaving most with suboptimal BP control and an increased risk of cardiovascular sequelae. This can be attributed partly to the heterogeneity of the response to antihypertensive therapy, to non-compliance or to side effects that contribute to withdrawal of treatment. Obvious candidate genes that influence drug responses are those that code for components of a system targeted by the drug or components of the counter-regulatory systems opposing the drug-induced fall in BP. Many pharmacogenetic studies have been conducted using a priori selected candidate genes in hypertension.

An alternative to gene-specific testing uses panels of SNPs or microsatellites to search the entire genome for "linked" regions likely to harbour such genes. Because no prior knowledge or assumptions are required about gene function, one attractive feature of this approach is the possibility of identifying new genes previously unsuspected to influence the trait.

The Medical Research Council funded British Genetics of Hypertension (MRC BRIGHT) study[1], attempted to increase detectance (the probability of carrying any particular susceptibility genotype given that the individual has a particular phenotype) of the disease locus by ascertaining for disease severity and including only non-diabetic, non-obese hypertensives whose blood pressure was in the top 5% of the blood pressure distribution in the UK. However this study was able to identify only a single locus suggestive of linkage on chromosome 5q[2]. Subsequently, we demonstrated a successful attempt at reducing heterogeneity by using antihypertensive drug response to partition different pathways of hypertension[3]. In the BRIGHT population, hypertensive sib-pairs who were non-responsive to ACE inhibitors, ARBs or beta-blockers showed significant linkage on chromosome 2p (LOD = 4.84 at 90.68 Kosambi cM)[3]. This susceptibility locus co-localises to a region found in African-American hypertensives in the Family Blood Pressure Program who showed evidence of linkage with hypertension status at 93 cM with a LOD score of 2.84[4]. Thus the chromosomal 2p locus independently identified in different populations may contain a gene or genes for the salt-sensitive form of hypertension which is common among Africans, and the same mechanism may be operative in a subset of white European hypertensives identified by unresponsiveness to β blockers and ACE inhibitors.

Preliminary results from from genome wide association testing using data from the Wellcome Trust Case Control Consortium will be presented but this can be validated only after replication which is currently ongoing.

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Percutaneous mechanical assist for acute STEMI.

José P.S.Henriques, MD, PhD

Despite considerable improvements in treatment of acute ST elevation myocardial infarction (STEMI), outcomes have predominantly improved in STEMI patients without cardiogenic shock. Nevertheless, cardiogenic shock occurs in approximately 7-10% of STEMI patients and is the leading cause of death for hospitalized patients. In-hospital mortality rates of STEMI complicated by cardiogenic shock are around 50%, despite reperfusion by primary percutaneous coronary intervention (PCI), the current standard of treatment (1). Currently, two therapeutic approaches can be adopted for STEMI patients presenting with cardiogenic shock or cardiogenic pre-shock to support the endangered circulation and the failing myocardium:

A. ***Pharmacological inotropic support.*** There is a variety of inotropic and vasopressor agents enabling quick improvement of hemodynamic parameters in cardiogenic shock. However, these agents failed to demonstrate improved survival in randomized studies. Currently, pharmacological circulatory support is listed as a class IIA recommendation.(2)

B. ***Mechanical left ventricular support.*** This modality of support has been made possible in humans first and foremost by the introduction of intra-aortic balloon counterpulsation about four decades ago. Currently, mechanical left ventricular support with an intra-aortic balloon pump (IABP) is listed as a class IB recommendation.(2)

IABP

The IABP was first introduced in the setting of cardiogenic shock in 1968 (3). Ever since, and especially after the development of a percutaneous insertion technique, IABP therapy has been used increasingly for several clinical conditions requiring mechanical LV support. In current practice, it is still the most frequently used method of mechanical cardiac assistance in the catheterization laboratory.

Currently, the main indication for IABP therapy in STEMI, as adjunctive therapy to revascularization, is cardiogenic shock not quickly reversed by pharmacologic therapy. This indication is listed in the ACC/AHA guidelines as a class IB recommendation, although no randomized controlled trials have been performed in cardiogenic shock. In our recently conducted, simultaneously performed meta-analysis of observational studies in STEMI patients with cardiogenic shock, data were importantly affected by confounders (4).

Notwithstanding the lack of evidence to support the use of IABP therapy, either in STEMI patients or in STEMI patients presenting with cardiogenic shock, it is still a popular treatment strategy. Moreover, it is the only method for mechanical cardiac assistance which is widely available and easily applicable in current practice.

Percutaneous left ventricular assist devices

Surgically implantable left ventricular assist devices (LVADs) have been shown to provide more effective circulatory support. However, in the setting of STEMI complicated by cardiogenic shock, the applicability of this therapy is limited. Therefore, the development of percutaneous LVADs has been of great interest. More recently, the TandemHeart and the Impella 2.5LP and the Impella 5.0LP have been introduced (5).

TandemHeart

The TandemHeart percutaneous ventricular assist device (VAD) is an extracorporeal, dual-chambered, centrifugal continuous-flow pump. It is a left-atrial to femoral artery bypass system, designed for short-term mechanical LV support (figure 1). At a maximum rotational speed of 7500 rpm, the TandemHeart pVAD can deliver a maximum output of 4.0 L/min.

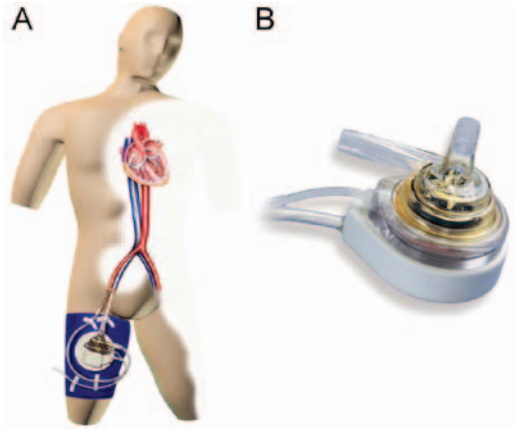

Figure 1.
Panel A. The TandemHeart ventricular assist device (VAD) (CardiacAssist, Pittsburgh, PA), including a left atrial transseptal inflow cannula, an arterial return cannula, a centrifugal blood pump and an external controller.
Panel B. The centrifugal blood pump, with the upper housing accommodating blood and the lower housing which provides connection to the infusion line and the external controller.

The device can be inserted in the catheterization laboratory, under fluoroscopy. The 21F transseptal inflow cannula is first inserted through the femoral vein and positioned in the left atrium, guided by fluoroscopy. The outflow cannula (15 to 17 Fr) is inserted through the femoral artery and positioned at the level of the aortic bifurcation. The implantation procedure takes around 30-45 minutes, but requires an important learning curve.

Two randomized trials comparing IABP versus TandemHeart have been conducted in STEMI patients with cardiogenic shock. In both of these trials, hemodynamic parameters improved significantly in patients who were supported by the TandemHeart VAD. However, both small studies revealed a high complication rate in the TandemHeart-supported patients. Complications observed included tamponade, major bleeding, critical limb ischemia, sepsis and arrhythmias. The most important factors contributing to these complications are likely to be the highly invasive and complex insertion procedure and the extracorporeal support method, combined with full high anticoagulation. In a recent review article we performed a meta-analysis, including only 74 patients (6), revealing a slight trend towards an odds ratio in favour of IABP therapy (odds ratio [OR] 1.17, 95% confidence interval [CI], 0.47-2.96, p = 0.73).

In conclusion, although the TandemHeart device is capable of delivering effective mechanical LV and circulatory support, the complexity of the device and the high complication rate may impede its widespread use. Nevertheless, it may be useful in specific circumstances. The device may be specifically useful if a left atrial approach is required, for instance in the case of aortic valve disease or as a means of percutaneous mechanical circulatory support during aortic valve interventions. Also, it may be a useful treatment option when complexity is less of an issue, for instance in the post-cardiotomy setting.

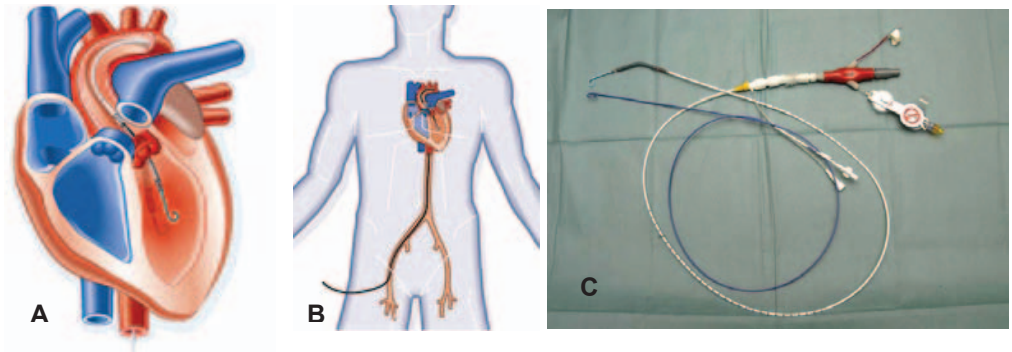

Figure 2 The Impella device.
Panel A and B. Schematic overview displaying the Impella LP2.5 pump, which is inserted percutaneously and positioned across the aortic valve in the left ventricle
Panel C. The Impella LP2.5 pump, next to a 6 Fr diagnostic catheter

Impella

The Impella LP2.5 and LP5.0 are catheter-mounted micro-axial blood pumps, designed for short-term mechanical LV and circulatory support. Both of these pumps are inserted through the femoral artery and subsequently positioned across the aortic valve into the left ventricle using fluoroscopy (figure 2). The Impella LP2.5 can be introduced percutaneously, whereas the larger Impella LP5.0 still requires a surgical cut down of the femoral artery. At a maximum rotational speed of 33.000 and 50.000 rpm, they produce a maximum output of 2.5 L/min and 5.0 L/min, respectively, by expelling aspirated blood from the left ventricle into the ascending aorta.

In the setting of mechanical LV support during elective high-risk PCI (7), we have previously reported on safety and feasibility of Impella LP2.5 support.

The safety, feasibility and efficacy of Impella LP2.5 support was studied in patients with large anterior STEMI in the MACH 2 trial (8). In this non-randomized study, prolonged Impella 2.5LP support was evaluated as adjunctive therapy to primary PCI (n=10), compared to routine care (n=10). Besides demonstrating safety and feasibility of Impella LP2.5 support, the study revealed an improvement in mean left ventricular ejection fraction (LVEF) from 28% at baseline to 41% after 4 months in the Impella-supported patients. In the control group, mean LVEF improved from 40% to 45%. These data suggest a beneficial effect of LV unloading on post-infarct LV remodelling and therefore, a beneficial effect on LV function.

In the setting of cardiogenic shock, several studies have been performed as well. In the ISAR-SHOCK trial, in which 26 STEMI patients with cardiogenic shock were randomized to concomitant Impella LP2.5 support or IABP therapy, Impella LP2.5 support resulted in reduced blood lactate levels. However, no difference in mortality rate was observed (9). Preliminary data from our database of cardiogenic shock patients treated with either Impella LP2.5 or Impella LP5.0 shows that 2.5 L/min of support in most cases is insufficient. The Impella LP5.0 was more effective in LV unloading and reversal of cardiogenic shock. A percutaneous method for the insertion of the Impella LP5.0 without the need for a surgical cut-down is currently being developed and eagerly awaited.

In conclusion, Impella technology offers the opportunity for mechanical LV support in many clinical settings. Both the Impella LP2.5 and the percutaneously implantable Impella LP5.0 may be a preliminary answer to the need for a minimally invasive and easy deployable mechanical assist device that provides superior hemodynamic support compared to IABP.

Future perspectives

In STEMI patients without cardiogenic shock, outcomes have improved considerably. Nevertheless, long-term outcome is strongly affected by LV remodelling, especially in the case of a large myocardial infarction. Unloading of the left ventricle may beneficially affect the remodelling process. The efficacy of pharmacological LV unloading has been demonstrated. The recently conducted MACH 2 trial suggests the efficacy of mechanical unloading in a clinical setting. To further elaborate on the efficacy of LV unloading and the beneficial effect on LV remodelling, we have recently initiated the IMPRESS in STEMI trial, comparing mechanical support by IABP versus Impella LP2.5 in STEMI patients with signs of pre-shock (10). The primary endpoint will be LVEF after 4 months, as assessed by MRI.

In STEMI complicated by cardiogenic shock we will initiate the IMPRESS in severe shock trial. This trial will compare the effects of mechanical support by IABP versus Impella LP5.0 in STEMI patients presenting with deep cardiogenic shock. The primary endpoint will be mortality after 30 days.

Conclusion

Many left ventricular support devices have been developed but only a few have reached a more widespread usage in the catheterization laboratory. A suitable percutaneous left ventricular support device should be ease to use and powerful in its circulatory support.

Mechanical left ventricular support could be beneficial in a variety of clinical settings, but especially in the setting of STEMI. In STEMI patients without cardiogenic shock, the primary purpose of mechanical LV support is myocardial recovery. In STEMI with cardiogenic

shock, mechanical LV support is directed at both myocardial and organ recovery.

The IABP is currently the most widely available and most easily applicable method of mechanical left ventricular support. However, IABP therapy has failed to improve clinical outcome in randomized trials. Surgical LVADs provide superior hemodynamic support. However, many issues withhold their widespread application in the setting of cardiogenic shock.

Therefore, after four decades of IABP support, the development and increasing clinical experience with percutaneous left ventricular assist devices heralds the dawn of a new era of superior hemodynamic support as an additional treatment besides primary PCI, to eventually improve outcome in STEMI patients with and without cardiogenic shock.

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www.Heartfailurematters.org

Kenneth Dickstein, Stavanger

Kenneth Dickstein presenterte nettstedet, www.Heartfailurematters.org i sitt innlegg. Nettstedet er utviklet under styring av the Heart Failure Association of the European Society of Cardiology. Målet er å gi god informasjon og praktiske råd til pasienter med hjertesvikt, til deres familie og til andre som bistår dem. Dickstein har oversendt informasjonsposteren under som orientering til Hjerteforums lesere.

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▪ Anna, an **animated guide**, helps users navigate the site

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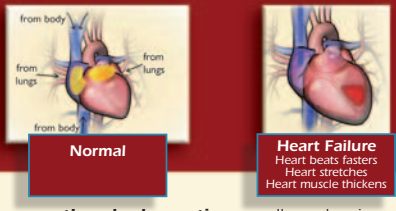

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Ole-Jørgen Ohm er tildelt NCS' hederspris for 2008

Ole-Jørgen Ohm er tildelt NCS hederspris for 2008 for hans langvarige og banebrytende arbeid for kardiologisk forskning innen pacemakerutvikling og elektrofysiologi.

Ole-Jørgen Ohm ble født i Odda 3. mars 1938. Han ble cand. med i Bergen 1963, spesialist i indremedisin 1976 og hjertesykdommer 1976. Han disputerte fra Chr. Michelsens Institutt, Avd. for Naturvitenskap og Teknikk ved Universitetet i Bergen 1979 med avhandlingen "Electrophysiological aspects related to artificial heart stimulation. A study of mycardial treshold, pacemaker electrode tissue interface impedance and electrogram characteristics in man, compared with in vitro investigations".

Han har vært ansatt ved Indremedisinsk avdeling, senere Hjertemedisinsk avdeling ved Haukeland sykehus siden 1971, bortsett fra stipendiatårene ved Chr. Michelsens Institutt. Han ble dosent i indremedisin i mars 1982 og professor i januar 1985.

Ole-Jørgen Ohm bygget opp et forskningsmiljø som har vært stabilt og produktivt gjennom 35 år. Den elektrofysiogiske forskningen har vært på høyt internasjonalt nivå med et stort antall publikasjoner og 6 doktorgrader. De første årene var aktiviteten sentrert rundt pacemaker/elektrodeteknologi og filterkarakteristikker svært viktig i forbindelse med myopotensialers hemming av pacemakere. Senere har gruppen forsket på nye algoritmer, sensorteknologi og anvendelse av ekkokardiografi for å studere hemodynamiske forhold ved pacemakerbehandling. De senere årene har pacemakergruppen særlig jobbet med biventrikulær pacing.

Over de siste 10 år har Ole-Jørgen Ohms gruppe drevet avansert forskning på behandling av atrieflutter og -flimmer. De har bygget opp et avansert laboratorium for ablasjonsbehandling og har utgitt en rekke publikasjoner som har oppnådd internasjonal oppmerksomhet.

Ole-Jørgen Ohm var medlem i Det norske råd for hjerte- og karsykdommer fra 1984, Hjerterådets arbeidsutvalg 1987-88, nestleder 1988-91 og leder fra 1991, medlem i Sentralstyret i Nasjonalforeningen for folkehelsen fra 1991, og Rådet for Chr. Michelsens Inst. fra 1987. Medlem i nucleus i ESC' arbeidsgruppe for Cardiac Pacing 1985, Fellow of American College of Cardiology 1982.

Priser:

Stimarec-prisen, 2. europeiske pacemakersymposium Firenze Italia 1981.

Prisbelønning for Med Videnskap 1981 (Søren og Sigurd Falchs Fond)

Ole Storsteins pris for fremragende studier i kardiologi 1985.

Thalen-prisen 3. europeiske pacemaker-symposium Torremolinos Spania 1985.