A brief history of the European Society of Cardiology

A look back on the history of the European Society of Cardiology which officially came into existence in 1950

The European Society of Cardiology

In 1946 and 1948, during the Second and Third Inter-American Congresses of Cardiology held in Mexico and Chicago, several European Cardiologists discussed the possible creation of a European Society of Cardiology (ESC).

On 29 January 1949, a preliminary meeting was held in Brussels with representatives from 14 countries: Belgium, Denmark, Finland, France, Greece, Italy, The Netherlands, Norway, Portugal, Spain, Sweden, Switzerland, the UK, and Yugoslavia. A provisional Executive Committee was appointed that included C. Laubry (France) as Honorary President, G. Nylin (Sweden) as President, and D.E. Belford (the UK), E. Coelho (Portugal) and J. Lenegre (France) as Vice-Presidents.

The first Board prepared a draft constitution and, on 2 September 1950, prior to the First World Congress of Cardiology in Paris, the European Society of Cardiology officially came into existence.

The first European Congress of Cardiology was held in London in September 1952 under the Presidency of Sir John Parkinson. Subsequently, European Congresses were held every 4 years until 1988. After, the General Assembly overwhelmingly voted to hold the Congress annually, starting with the Vienna meeting in 1988.

The foundation stone of the European Heart House was laid on 27 November 1992 by the President of the ESC, Professor Michel E. Bertrand, FESC (Lille, France), who also inaugurated the Heart House on 31 August 1993, during the XV Congress of the European Society of Cardiology in Nice. Three hundred people, including the representatives of National Societies and Working Groups and 100 local dignitaries, attended the ceremony. The 1993 elected Fellows of the ESC had the unique opportunity to receive their FESC diploma within the context of the inauguration of the European Heart House.

Table 1

Past Presidents of the European Society of Cardiology

<table>
<thead>
<tr>
<th>President</th>
<th>Country of origin</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gustav Nylin</td>
<td>Sweden</td>
<td>1950–56</td>
</tr>
<tr>
<td>Evan Bedford</td>
<td>UK</td>
<td>1956–60</td>
</tr>
<tr>
<td>Jean Lenègre</td>
<td>France</td>
<td>1960–64</td>
</tr>
<tr>
<td>Luigi Condorelli</td>
<td>Italy</td>
<td>1964–68</td>
</tr>
<tr>
<td>Pavel Lukl</td>
<td>Czech Republic</td>
<td>1968–72</td>
</tr>
<tr>
<td>Herman Snellen</td>
<td>The Netherlands</td>
<td>1972–76</td>
</tr>
<tr>
<td>Henri Denoîn</td>
<td>Belgium</td>
<td>1976–80</td>
</tr>
<tr>
<td>Franz Loogen, EFESC</td>
<td>Germany</td>
<td>1980–84</td>
</tr>
<tr>
<td>Paul Hugenholtz, EFESC</td>
<td>The Netherlands</td>
<td>1984–88</td>
</tr>
<tr>
<td>Hans-Peter Krayenbühl</td>
<td>Switzerland</td>
<td>1988–90</td>
</tr>
<tr>
<td>Attilio Reale</td>
<td>Italy</td>
<td>1990–91</td>
</tr>
<tr>
<td>Michel Bertrand, FESC</td>
<td>France</td>
<td>1991–94</td>
</tr>
<tr>
<td>Philip Poole-Wilson</td>
<td>UK</td>
<td>1994–96</td>
</tr>
<tr>
<td>Gunter Breithardt, FESC</td>
<td>Germany</td>
<td>1996–98</td>
</tr>
<tr>
<td>Lars Rydén, FESC</td>
<td>Sweden</td>
<td>1998–2000</td>
</tr>
<tr>
<td>Maarten Simoons, FESC</td>
<td>The Netherlands</td>
<td>2000–02</td>
</tr>
<tr>
<td>Jean-Pierre Bassand, FESC</td>
<td>France</td>
<td>2002–04</td>
</tr>
<tr>
<td>Michal Tendera, FESC</td>
<td>Poland</td>
<td>2004–06</td>
</tr>
<tr>
<td>Kim Fox, FESC</td>
<td>UK</td>
<td>2006–08</td>
</tr>
<tr>
<td>Roberto Ferrari, FESC</td>
<td>Italy</td>
<td>2008–10</td>
</tr>
<tr>
<td>Michel Komajda, FESC</td>
<td>France</td>
<td>2010–12</td>
</tr>
<tr>
<td>Panos Vardas, FESC</td>
<td>Greece</td>
<td>2012–14</td>
</tr>
</tbody>
</table>

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The European Heart House represents a milestone in the long-standing commitment of the European Society of Cardiology to foster the development of cardiology and to further education in the field of cardiovascular disease. It actually meets the Society’s need for headquarters and for an Educational and Training Centre.

The surroundings of the European Heart House are not only pleasant. Sophia Antipolis (South of France) is in an area that has a unique concentration of technological-, research-, and development-related activities, including the fields of computer science, electronics, telecommunications, and health and pharmaceutical research.

Since the provisional Executive Committee of the ESC, 18 eminent cardiologists have been Presidents of the European Society of Cardiology (Table 1).

European Society of Cardiology

Environmental hazards, air pollution, and noise as novel cardiovascular risk factors

Introduction

The role of noise and air pollution as novel risk factors for cardiovascular disease is being increasingly recognized. Noise causes annoyance, disturbs sleep, and impairs cognitive performance. Furthermore, evidence from epidemiology studies demonstrates that environmental noise is associated with an increased incidence of arterial hypertension, myocardial infarction, and stroke. Both observational and experimental studies indicate that, in particular, night-time noise can cause disruptions of sleep pattern, vegetative arousals (e.g. increases of blood pressure and heart rate), increases in stress hormone levels and oxidative stress, which in turn may lead to vascular (endothelial) dysfunction and arterial hypertension in healthy subjects which is even more pronounced in patients with cardiovascular disease.

Results concerning air pollution research look very similar. There is now clear evidence that air pollution, in addition to its impact on lung disease, may cause cardiovascular disease. There is a clear positive association between short- and long-term exposure to air pollution and cardiovascular mortality as well as overall mortality.

Currently, the WHO estimates that—in western Europeans—45 000 years are lost annually due to noise-induced cognitive impairment in children, 903 000 years due to noise-induced sleep disturbance, 61 000 due to noise-induced cardiovascular disease, and 22 000 due to tinnitus. Additionally, while not being a disease per se, noise-induced annoyance decreases quality of life and thus also causes disability quantified in 587 000 disability-adjusted life years lost in the western European population.

Likewise, the joint effects of air pollution are also likely to be large. Household air pollution from solid fuels accounted for 3.5 million (2.7 million to 4.4 million) deaths and 4.5% (3.4–5.3) of global DALYs in 2010 and ambient particulate matter (PM) pollution accounted for 3.1 million (2.7 million to 3.5 million) deaths and 3.1% (2.7–3.4) of global DALYs.

Interestingly, there is a growing body of evidence that both, air pollution- and noise-triggered vascular dysfunction, may be caused by their capacity to induce inflammation and therefore increased oxidative stress within vascular tissue, ultimately leading to atherosclerosis, vascular (endothelial) dysfunction, myocardial infarction, heart failure, and arrhythmias (Figure 1). In our brief report, we focus on more recent evidence that air pollution and noise indeed represent important and novel cardiovascular risk factors.

Noise and cardiovascular disease, epidemiologic evidence

Aircraft noise causes arterial hypertension, stroke, and ischaemic heart disease

Already in 2001 a higher prevalence of arterial hypertension was demonstrated in the vicinity of airports. Likewise, a dose–response relationship has been shown in the HYpertension and Exposure to Noise near Airports (HYENA) study in 2008 with respect to night-time noise and development of hypertension. Children will also react to aircraft noise with the development of arterial hypertension as demonstrated by the results from the European Union-funded RANCH (Road Traffic and Aircraft Noise Exposure and Children’s Cognition and Health) study. Further metaanalysis revealed a relationship between air traffic noise in the range of 55–65 dB and the prevalence of arterial hypertension. Additionally, night-time aircraft
noise was associated with a higher rate of cardiovascular medicine prescriptions in residents exposed to high aircraft noise levels, particularly during the night and the early morning hours (3–5 am).

With respect to ischemic heart disease and stroke, earlier studies from the late 1970s carried out around Amsterdam’s Schiphol airport gave hints of a higher risk of cardiovascular disorders in subjects that lived closer to the airport. More recent studies also provided evidence of a higher cardiovascular risk for subjects who reside near airports. In an ecological study carried out around Heathrow airport, London, an increased risk of stroke and coronary heart disease was reported in relation to day- and night-time exposure to aircraft noise in people who were more exposed to aircraft noise. Another large cohort study in Switzerland reported an increased mortality due to myocardial infarction with increasing exposure levels and duration of aircraft noise. Finally, in the cross-sectional HYENA study, an association between (self-reported) heart disease or stroke (combined endpoint) and nocturnal aircraft noise was described. Taken together there is no doubt anymore that living closer to airports will cause more cardiovascular disease due to increased noise exposure.

**Road traffic noise, coronary heart disease, and stroke**

Road traffic noise has been demonstrated to be associated with myocardial infarction in case-control and cohort studies, a finding which was corroborated by the results of a more recent meta-analysis. Thus, the exposure to sounds in the range between 55 and 60 dB, which would include large fractions of the population, may also contribute to the burden of disease. Further, exposure to residential road traffic noise was associated with a higher risk for stroke among people older than 64.5 years of age.

In summary, given the ubiquitous exposure, road traffic noise should be considered a relevant risk factor for cardiovascular disease.

**Air pollution and cardiovascular disease**

**Air pollution causes arterial hypertension and coronary artery disease**

There is no doubt that PM air pollution can raise blood pressure. Acute exposure to PM causes a rapid increase in blood pressure and the blood pressure raising effect may even persist chronically when individuals reside in more polluted regions. In addition, more recent manuscripts provide evidence that long-term PM exposure can promote the development of overt hypertension. Less clear seems to be the situation concerning ozone (O₃) or NO₂.

These findings suggest that not only can short-term exposures possibly cause an acute increase in blood pressure, but chronically inhaling second-hand smoke can increase the risk for chronic hypertension.

Many cohort studies in diverse populations demonstrated an increased risk of incident fatal or non-fatal coronary artery disease in subjects exposed to air pollution. Since air pollution has also been shown to be associated with subclinical atherosclerosis such as intima media thickness of the carotid artery and of coronary artery or even aortic calcification, all these findings provide strong evidence that air pollution clearly adversely accelerates the development of atherosclerosis and therefore increases the risk for the development of coronary artery disease.

**Air pollution, heart failure, and stroke**

More than 100 billion euros are spent every year for direct and indirect costs for heart failure affecting more than 20 million people. This condition is associated with frequent hospital readmissions, which per se implicates a poor prognosis in these patients. There are several reports linking air pollution to a higher incidence of heart failure and a recent meta-analysis provided more evidence for an association between air pollution and heart failure and short-term increases in gaseous components and PM, with the risk of hospitalization or death from congestive heart failure.

There is also consistency concerning hospital admissions for stroke and air pollution and death from cerebrovascular disease.

**Mechanisms leading to vascular dysfunction: inflammation and oxidative stress**

**Noise**

Studies concerning the effects of noise on vascular function are scarce. Recently we performed two studies to address the effects of night-time noise on vascular (endothelial function), blood pressure, sleeping quality, and stress hormone levels in healthy subjects and in patients with established coronary artery disease. With these studies we found that nocturnal aircraft noise exposure played back with loudspeakers in the subjects’ bedrooms lead to a deterioration of endothelial function, which as expected was more pronounced in patients with established coronary artery disease compared with healthy subjects. Sleeping quality deteriorated in both studies and in healthy subjects we also established an increase in circulating catecholamine levels.

Importantly, endothelial function in healthy subjects exposed to 60 noise events per night was clearly improved with vitamin C, suggesting that night-time noise exposure may cause endothelial dysfunction primarily by inducing oxidative stress within the vasculature. We also observed that a previous exposure of the study subjects to 30 events per night led to a sensitization of the vessels to develop endothelial dysfunction. In addition, we found that deterioration of vascular function was occurring independently of annoyance, which means whether or not the noise makes a person angry or not, the vessel will respond with a deterioration of endothelial function.
Air pollution
Concentrated ambient PM2.5 potentiates plaque burden and vascular dysfunction in experimental models of atherosclerosis and increases oxidative stress within the vasculature.\textsuperscript{16} Oxidative stress per se causes inflammation, which will further increase the oxidative stress burden to the vasculature in a positive feedback fashion.\textsuperscript{28} Oxidative stress caused by PM can be further increased, e.g. by O\textsubscript{3}, which is highly oxidizing. Further, acute exposure to diesel exhaust gases causes impaired vasodilation to endothelium-dependent and -independent nitrovasodilators, pointing to a defect at the smooth muscle level (impairment of guanylate cyclase pathway),\textsuperscript{19} a phenomenon that persists for 24 h.\textsuperscript{24} Accordingly, results from a large population-based study suggest that long-term exposure to PM2.5 even at low concentrations may lead to persistent endothelial dysfunction.\textsuperscript{18}

Air pollution and noise can cause overweight and diabetes mellitus
There are more recent reports that chronic exposure to air pollution as well as to noise may lead to more overweight and diabetes mellitus. A recent systematic review and meta-analysis of published observational studies revealed an increase in risk of diabetes and susceptibility of people with diabetes to air pollution.\textsuperscript{14} These results were consistent between time-series, case-crossover, and cohort studies and between studies conducted in North America and Europe suggesting that exposure to air pollution may be a risk factor for diabetes.\textsuperscript{14}

Likewise a recently published meta-analysis revealed that people exposed at their homes to roughly $L_{DEN}$ (the 24-h LAeq with a 5-dB penalty for the evening (usually 6 p.m. to 10 p.m. or 7 p.m. to 11 p.m.) and a 10 dB penalty for the night (usually 10 p.m. to 6 a.m. or 11 p.m. to 7 a.m.)) $> 60$ dB had 22\% higher risk or type 2 diabetes in comparison to those exposed to $L_{DEN} < 64$ dB, indicating that noise pollution might bring about not only cardiovascular diseases but also endocrine dysfunction.\textsuperscript{9}

Summary and conclusion
The present article stresses the growing importance of environmental stressors in being risk factors for the development of cardiovascular disease or in exacerbating already existing cardiovascular disease. Based on the existing evidence, these environmental stressors should be included into the guidelines for the treatment of chronic stable angina and acute non-ST-segment elevation myocardial infarction (N-STEMI) and ST-segment elevation myocardial infarction (STEMI).

Reduce air pollution to reduce the burden of cardiovascular diseases!

The second highest environmental risk factor for years of lives lost annually

The European Society of Cardiology (ESC) pursues the mission to reduce the burden of cardiovascular disease (CVD) in Europe (http://www.escardio.org/ accessed 20 May 2015). The traditional view on CVDs and the prevention or delay of its development keeps a strong focus on personal attitudes, life styles, and medications.

However, this year’s theme of the ESC Congress ‘Environment and the Heart’ sends a different message: heart diseases—and their underlying pathologies such as atherosclerosis—are not just a matter of salt, sugar, lack of physical activity, smoking, or stress but indeed have an environmental dimension. This view is fully confirmed by assessments of the global burden of disease (GBD).

Among the 25 top-ranking risk factors, one-third are related to environmental factors (http://vizhub.healthdata.org/irank/heat.php, GBD heat map accessed on 19 May 2015). Indeed, with $\approx 174$ million years of lives lost (YLL) every year due to air pollution (household and ambient, including ozone), this environmental factor ranks second, after the 200 Mio YLL attributed to dietary risks. As the globally leading environmental risk factor, air pollution has an even stronger impact than high blood pressure (167 Mio YLL)—which
Air pollution policies are crucial

How can ESC fulfill the mission to reduce the air pollution-related burden of CVDs in Europe? The constituencies to approach are not cardiologists nor the pharma industry or patients but those responsible for the regulation of environmental conditions, namely Governments and policy-makers.

The science provides the evidence for what constitutes ‘healthy air’ and the WHO has summarized the evidence in the Guidelines for Clean Air, promoting values that Governments should adopt in their regulations to foster public health. However, there is large heterogeneity in how Governments deal with the WHO guidelines. A particularly bad example is the European Union (EU), sticking to annual mean ‘standards’ which, in case of PM10 and PM2.5, are set two times higher than what WHO recommends, namely at 40 instead of 20 μg/m³, and 20 instead of 10 μg/m³, respectively.

Recent reviews, conducted on behalf of the EU fully confirm air pollution to be a major public health burden in Europe and research funded by the EU to inform policy-makers reported long-term effects of air pollution on longevity among European populations living in neighbourhoods where air quality meets the WHO values, with no evidence for thresholds of no effect. However, the reduction in this preventable burden of disease is not on the agenda of the EU decision-makers. At times of economic crisis, this ignorance is disturbing. As shown, the costs of the air pollution-related burden of disease are many-fold higher than the costs of clean air strategies. The recent report of WHO Europe estimates the annual costs of air pollution-related morbidities and mortalities in the WHO European Region with its ~800 million inhabitants to be 1.575 trillion USD. As the less affluent countries of Central and Eastern Europe are more markedly affected by air pollution, the distribution of the disease burden and related costs amplify inequities across Europe.

Clean air policies result in better health

The success of setting ambitious science-based air quality standards can be witnessed in many regions of the world, be it in the middle of Europe or in the Los Angeles area with its smoggy history. Despite the 38% increase in motor vehicle activity, substantial economic growth, and a topography that favours the build-up of smog, ambient concentrations have drastically dropped over the last 20 years, falling on average some 3% every year in the communities that participated in the famous Children’s Health Studies. These air quality improvements had very direct benefits on the children’s lung development. The Californian air quality managers remain active and
Stress and cardiovascular mortality: an evident but underestimated connection

On 17 January 1994 at 4:31 a.m. an extremely strong earthquake struck the Los Angeles area. As a direct consequence, 57 people lost their lives and more than 5000 were injured. With an estimated property damage of more than 20 billion US dollars, it was one of the costliest natural disasters in North American history. The earthquake itself lasted for 1020 s. However, during the rest of this same day, 24 sudden cardiac deaths were recorded—a rate more than five times higher than the usual.1

This observation is consistent with other studies investigating the connection of emotional stress and cardiovascular events. These have revealed similar findings for different triggers such as missile attack2 or less severe conditions such as anger3 or even watching a football game.4 In line with this, the INTERHEART study which analysed the influence of different stressors in animal models and found a reproducible sequence of responses he named alarm, resistance, and exhaustion which he named general adaptation syndrome.5 While the alarm stage is congruent with Cannon’s fight–flight reaction, the resistance stage corresponds to an ongoing depletion of the animal’s resources. If the stressor persists remaining resources are depleted and a clearly pathological condition emerges, the exhaustion stage.

In 1974 Richard Lazarus introduced a more human focused and psychology related transactional model which emphasized the importance of individual properties.6 He claimed that the reaction to stressors is greatly influenced by personal attitude and experience. In 1988 Stevan Hobfoll introduced the terms investment and loss of resources.7 He assumed that stress is present as soon as the loss of the personal resources is imminent, apparent, or investment of resources does not lead to an adequate gain of additional resources (referred to as unprofitable investment).

Emotional stress: the historic view

In 1915 Walter Cannon described a particular behaviour of animals as the fight or flight reaction.6 Whenever a creature is faced with a potentially harmful or life threatening situation several adaptations occur, enabling the animal to react to this threat with better chances of success. The general activity status is raised to a higher level which allows faster and stronger reactions. At the same time, the accuracy of movements deteriorates since precise reactions would require complex operations in the cerebral cortex and thus be too slow.

It took another 21 years until Hans Selye first used the term stress in a biological context.7 He borrowed the word from physics where it describes pressure or traction affecting a solid material which consequently will either just bend or actually burst. Similarly, in a biological system a certain stressor may be tolerated by one creature, whereas the other is not able to cope with it. To provide for these two cases he coined the terms eustress and distress.

Eustress is a very important and effective tool to escape dangerous situations, whereas distress can lead to illness and death. Selye investigated the influence of different stressors in animal models and found a clear association between psychological stressors and acute myocardial infarction5 disclosed a clear association between psychological stressors and acute myocardial infarction with consistent results across regions, in different ethnic groups and sexes.

In light of this, it would be venturous to negate a strong impact of emotional stress on cardiovascular health. Nevertheless, mechanistic knowledge about the connection of stress and cardiovascular events is small and prophylactic strategies are rare.

Mechanisms linking stress and cardiovascular disease

Although many diseases have been shown to be stress-related, cardiovascular disease occupies an important position due to its high incidence and its clear correlation with stress.5 Several function levels and pathways are involved in the stress response (Figure 1). Emotional processing involves the cerebral cortex and the limbic system. Further processed in brain stem structures, these signals trigger (patho-) physiological responses. The hypothalamic–pituitary–adrenal axis is referred to as one classic stress axis.

Hypothalamic corticotrophin-releasing hormone leads to the release of adrenocorticotrophin from the anterior pituitary gland; the latter then triggers the production and release of glucocorticoids (in particular cortisol) from the adrenal cortex. Activation of the sympathetic nervous system leads to the release of epinephrine from

References

References are available as supplementary material at European Heart Journal online.
the adrenal medulla and norepinephrine from sympathetic nerve endings.\textsuperscript{11} In addition, several inflammatory cytokines, such as TNF–α, have been shown to be increased in stress models.\textsuperscript{12}

However, the multiple connections between these pathways and functional levels as well as the mechanisms linking these processes with the higher incidence of cardiovascular events are only partially understood. Our group observed, that in mice, stress can lead to accelerated arterial thrombus formation \textit{in vivo}\textsuperscript{13}; this effect was blunted by blockage of the sympathetic nervous system. Several potential mediators of stress-related arterial thrombosis, such as tissue factor expression\textsuperscript{12} or platelet activation,\textsuperscript{14} are being discussed. In addition, endothelial function has been observed to be impaired in stress models\textsuperscript{11} suggesting an influence of stress on coronary flow reserve.

Not only atherothrombotic events can be triggered by stress, but also Takotsubo cardiomyopathy is a severe condition with mortality rates similar to those of acute coronary syndromes.\textsuperscript{15} Also this fascinating clinical syndrome is not sufficiently understood yet.

**Stress and cardiovascular disease in this day and age**

In modern society people are faced with many demanding tasks, such as difficult social settings, job-related pressure, and the growing information intensity of our digitalized society. Depending on the individual resources, these circumstances can lead to stress with a massive impact on cardiovascular morbidity and mortality. In addition, stress leads to enormous generation of costs for the community. Even though estimations of the worldwide stress-related costs vary a lot, experts agree that it is matter of hundreds of billion U.S. dollars every year.\textsuperscript{16}

In regard of these facts, approaches to decrease stress levels and extend coping strategies are crucial and should be implemented in primary and secondary prevention of cardiovascular disease. However, it is neither easy to assess stress levels nor to reduce social stressors, making this task particularly challenging. Therefore, more effort is needed to understand the mechanisms of stress-induced cardiovascular events, thereby opening future perspectives and serving as a basis for clinical implications.

**References**

References are available as supplementary material at European Heart Journal online.
Stress and the heart: the role of type D personality in personalized care

Stress and the heart

Psychosocial stress is associated with biological and behavioural pathways of cardiovascular disease. For example, dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis may be involved as a biological mechanism. Elevated levels of the HPA stress hormone cortisol are related to altered immune activity as well as coronary calcification and cardiac cell damage.

People differ greatly in their sensitivity to psychosocial stress and these individual differences in perceived stress are related to personality traits that summarize the cognitive, affective, and behavioural tendencies of individuals. We will discuss here that taking these personality traits into account is critical for a better understanding of the link between stress and the heart.

Stress and type D personality

The Type D, or distressed, personality construct may help to explain the effects of stress and cortisol on the heart. Type D refers to a general propensity to stress that is characterized by the combination of two traits: the tendency to experience negative emotions (negative affectivity) and the tendency to inhibit expression of emotion/behaviour in social interaction (social inhibition). Negative emotions like depression, anxiety, or anger are well-known risk factors. Social inhibition causes high stress levels in non-human primates and children and this tendency to social stress has also been observed in adults. Below we will discuss some key domains relevant to Type D and the heart including biological pathways, cardiovascular outcomes, and clinical implications.

Biological plausibility of the Type D construct

Several lines of research support the biological plausibility of Type D as a risk marker (Table 1). Experimental stress research in healthy individuals shows that Type D is related to maladaptive HPA axis and autonomic responses that affect cardiovascular health. These include increased cortisol and cardiovascular reactivity to stress and decreased heart rate variability.

Clinical research in cardiac patients confirms the biological plausibility of the Type D construct. Type D is related to higher awakening cortisol levels in patients with an acute coronary syndrome, and decreased heart rate variability and heart rate recovery in patients with heart failure. Other biological correlates of Type D include increased tumour necrosis factor-α and oxidative stress levels, and a decrease in endothelial progenitor cell counts.

Epidemiological studies in the general population also hint at a possible link between Type D and cardiovascular disease, including population-based studies in Germany and Iceland reporting that Type D was associated with the prevalence of coronary artery disease. In people with no history of cardiovascular disease, Type D personality has been related to metabolic dysfunction, severity of coronary artery plaque, increased risk of ventricular arrhythmias, and incidence of Takot Subo cardiomyopathy due to emotion-triggered myocardial stunning.

Table 1 Biological pathways that have been associated with Type D

<table>
<thead>
<tr>
<th>Biological pathways that have been associated with Type D</th>
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<tbody>
<tr>
<td>Increased cardiovascular reactivity to laboratory-induced stress</td>
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<tr>
<td>Decreased heart rate variability and decreased heart rate recovery</td>
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<tr>
<td>Incidence of ventricular arrhythmias and acute myocardial stunning</td>
</tr>
<tr>
<td>Presence/Severity of coronary artery plaque in people with no CAD history</td>
</tr>
<tr>
<td>Heightened levels of the stress hormone cortisol (HPA-axis)</td>
</tr>
<tr>
<td>Higher plasma levels of tumour necrosis factor-α and its soluble receptors</td>
</tr>
<tr>
<td>More oxidative stress, decrease in endothelial progenitor cell counts</td>
</tr>
<tr>
<td>Metabolic pathways that contribute to cardiovascular disease risk</td>
</tr>
</tbody>
</table>

Type D and poor cardiovascular outcomes

Although biological findings suggest that Type D has an adverse effect on cardiovascular health, these findings should be cautiously interpreted. Follow-up studies in cardiovascular populations have produced inconsistent results. Meta-analyses showed that Type D predicted a 2-fold increased risk of adverse events but also revealed large heterogeneity between studies. This may relate to differences in samples and endpoints between negative and positive studies. A closer look at the individual Type D studies suggests several causes of heterogeneity.

Age and comorbidity

The mean age of patients included in negative studies was 65.2 years, which was almost 7 years older on average than the mean age of 58.4 years in the positive studies. Hence, it is possible that the effect of Type D recedes in older patients that already have a high mortality risk due to an ageing heart and comorbidities like diabetes or kidney disease.

Endpoints

There was large heterogeneity in endpoints. Negative studies used all-cause mortality as an endpoint but positive studies also included cardiac endpoints. In one negative study, Type D predicted myocardial infarction or death in previous analyses. Type D personality may be more related to fatal and non-fatal cardiovascular events than to all-cause mortality.
## Table 2 Prognostic effect of Type D in multivariable models of major adverse events

<table>
<thead>
<tr>
<th>First author (year) Journal</th>
<th>Mean age (years)</th>
<th>% Type D</th>
<th>CVD</th>
<th>FU (years)</th>
<th>Outcome</th>
<th>Type D effect</th>
<th>Depression or anxiety effect</th>
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</thead>
<tbody>
<tr>
<td><strong>Null studies</strong></td>
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</tr>
<tr>
<td>Coyne (2011) Psychosom Med, 73:557–562</td>
<td>M = 70.7</td>
<td>13%</td>
<td>CHF</td>
<td>1.5</td>
<td>All-cause mortality</td>
<td>NO</td>
<td>Depression: NO</td>
</tr>
<tr>
<td>Pelle (2010) Circ Heart Fail, 3:261–267</td>
<td>M = 66.6</td>
<td>20%</td>
<td>CHF</td>
<td>3.1</td>
<td>All-cause mortality</td>
<td>NO</td>
<td>Depression: NO or Anxiety</td>
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<tr>
<td>Meyer (2014) Ann Behav Med, 48:156–162</td>
<td>M = 63.5</td>
<td>31%</td>
<td>CAD</td>
<td>5</td>
<td>All-cause mortality and MACE</td>
<td>NO</td>
<td>Reversed effect: better survival with more anxiety</td>
</tr>
<tr>
<td>Grande (2011) Psychosom Med, 73:548–556</td>
<td>M = 63.3</td>
<td>24%</td>
<td>CVD</td>
<td>5.9</td>
<td>All-cause mortality</td>
<td>NO</td>
<td>Depression: NO</td>
</tr>
<tr>
<td>Damen (2013) Int J Cardiol, 167:2496–2501</td>
<td>M = 62.0</td>
<td>29%</td>
<td>CAD</td>
<td>7.0</td>
<td>All-cause mortality</td>
<td>NO</td>
<td>Anxiety: NO</td>
</tr>
<tr>
<td><strong>Positive studies</strong></td>
<td></td>
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<tr>
<td>Aquarius (2009) Arch Surg, 144:728–33</td>
<td>M = 64.8</td>
<td>35%</td>
<td>PAD</td>
<td>4</td>
<td>All-cause mortality</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>Denollet (2013) Int J Cardiol, 167:2705–2709</td>
<td>M = 62.6</td>
<td>23%</td>
<td>ICD</td>
<td>3.2</td>
<td>All-cause mortality</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>Martens (2010) J Clin Psychiatry, 71:778–783</td>
<td>M = 59.0</td>
<td>20%</td>
<td>MI</td>
<td>1.8</td>
<td>Cardiac death and nonfatal MI</td>
<td>YES</td>
<td>Depression: NO</td>
</tr>
<tr>
<td>Pedersen (2010) Europace, 12:1446–1452</td>
<td>M = 57.7</td>
<td>22%</td>
<td>ICD</td>
<td>1.7</td>
<td>All-cause mortality</td>
<td>YES</td>
<td></td>
</tr>
<tr>
<td>Denollet J. (2000) Circulation, 102:630–635</td>
<td>M = 56.7</td>
<td>31%</td>
<td>CAD</td>
<td>5</td>
<td>Cardiac death and nonfatal MI</td>
<td>YES</td>
<td>Depression: NO</td>
</tr>
<tr>
<td>Denollet (1994) Lancet, 347:417–421</td>
<td>M = 55.4</td>
<td>27%</td>
<td>CAD</td>
<td>7.9</td>
<td>All-cause mortality</td>
<td>YES</td>
<td>Depression: NO</td>
</tr>
<tr>
<td>Denollet (2007) J Heart Lung Trans 26:152–158</td>
<td>M = 54.1</td>
<td>29%</td>
<td>HTx</td>
<td>5.4</td>
<td>All-cause mortality and HTx rejection</td>
<td>YES</td>
<td></td>
</tr>
</tbody>
</table>

CABG, coronary artery bypass graft surgery; CAD, coronary artery disease; CHF, chronic heart failure; CVD, cardiovascular disease; FU, follow-up; HTx, heart transplantation; ICD, implantable cardioverter defibrillator; M, mean; MACE, major adverse cardiovascular event; MI, myocardial infarction; NR, not reported; PAD, peripheral arterial disease; PCI, percutaneous coronary intervention; VSUR, valve surgery.

*Significant in univariate analysis but not in a multivariable model including clinical variables and Type D.
Role of distress
Depression and anxiety were also largely unrelated to mortality in the negative studies of Type D (Table 2). In one negative study, the authors reported in a separate paper that anxiety predicted improved survival. In some studies, depression or anxiety were related to cardiovascular outcomes, but in multivariable analysis the effect of Type D prevailed.

Further studies of the biological and psychological features of Type D individuals need to improve our understanding of when and how Type D affects cardiovascular health. Recently, the ESC and AHA have advocated the integration of patient-reported outcomes (PROs) such as symptoms, health-related quality of life, and perceived health in clinical research and care. Type D is a vulnerability factor for poor PROs following myocardial infarction, coronary bypass surgery, ICD implantation, and heart transplantation. Meta-analysis confirms that Type D is an independent correlate of impaired patient-reported PROs in cardiovascular populations.

Translating the Type D perspective into personalized medicine
Patients have multiple characteristics that affect their risk profile or treatment outcome, and personalized medicine aims to individualize care according to these unique characteristics. Integrating information on the Type D phenotype into care may enhance (a) the prediction of outcomes, (b) the understanding of the patient’s behaviour, and (c) the adaptation of existing treatment to better fit with this phenotype. We propose a ‘psychological polypill’ approach that targets typical cognitive, affective, and behavioural features of Type D patients (Figure 1).

Perceived stress
Type D individuals frequently expect negative reactions from others and have an interpretation bias toward potential threat in social interaction. Accordingly, Type D is robustly associated with higher perceived stress levels and feelings of social isolation.

Emotion regulation
Type D individuals may have deficits in emotion regulation and coping skills that play an important role in subjective well-being. Type D is often associated with an increased risk for persistent elevated levels of depression, anxiety, and suppressed anger.

Lifestyle behaviour
We focused on biological correlates of Type D, but there are also several behavioural pathways. Type D is associated with unhealthy lifestyles such as physical inactivity, smoking, and unhealthy nutrition that may partly explain poor cardiovascular health.

Treatment adherence
Type D predicts poor medication compliance in patients with an acute coronary syndrome and heart failure. Type D patients may delay medical consultation and tend to use less healthcare.

Type D assessment
The ESC guidelines on prevention and the Cardiac Rehabilitation section of the ESC have indicated the importance of assessing psychosocial risk factors, including Type D personality. An obvious question is how to incorporate Type D assessment in clinical research and practice. Type D traits can be easily and reliably assessed with the brief DS14 scale, a copy of this scale is presented in the 2nd edition of The ESC Textbook of Cardiovascular Medicine (p. 1302). A large international study confirmed the validity of the DS14 in patients from 21 countries.

Given the large heterogeneity in study population and endpoints, evidence from prognostic studies is inconclusive and research needs to further explore moderating factors that may alter the link between Type D and cardiovascular health. Many other challenges remain, but this summary shows that Type D contributes to stress reactivity and may benefit from more personalized care.

References
References are available as supplementary material at European Heart Journal online.
Music is unique to man and its use to influence mood goes back to pre-history. Music is now widely used commercially (but uncritically) from medical applications to raising cattle. Rather little has been published in regular medical journals.

My interest arose in a roundabout way. I had had a longstanding interest in the arterial baroreflex. Rather surprisingly the exquisitely precise information sent to the brain by vascular receptors in, for example, the carotid sinus, evokes efferent responses within 1 beat down the myelinated vagus nerves, but delayed 2–3 beats down the unmyelinated sympathetic nerves. This imperfect control system leads to regular 10 s ‘hunting’ in BP and heart rate—the MAYER waves.

It has previously been found that, in general, there is a relation between better autonomic control and subsequent longevity. There is now good evidence that the more variability you have the longer you live: after a heart attack, with heart failure, or with hypertension. Heart rate variability depends on the baroreflex—which increases vagal tone. In 1992 I took a sabbatical to learn more about power spectral analysis of heart rate variability from Luciano Bernardi—a young researcher in the Medical Unit in Pavia, Italy, who was publishing on this new technique to investigate cardiovascular control in diabetic patients. But when I arrived I found that he did not fully understand the underlying physiology either!

We then decided to study the stress of mental arithmetic in young normal medical students (easier and they were more available), analysing the cardiovascular responses by power spectral analysis. As a non-stressful verbal control, we used the Ave Maria prayer. This prayer is familiar to all Italians and is repeated aloud about 50 times, in a Priest plus Congregation response—in a completely ritual and non-arousing way.

We were surprised to find that this repetitive Latin prayer had 10 s phrases which coincided exactly with (and so increased) the normal 10 s Mayer waves in blood pressure and pulse—resulting in reflex increased vagal tone. The Ave Maria did NOT have the same calming effect if said in Italian or other languages—these translations all exceeded 10 s!

Over the next 20 years (published in the BMJ, Heart, and Circulation) we found that this 10 s rhythm was also found in some music compositions, particularly by Verdi.

Studies of the comparative cardiovascular effects of different music styles are complex—because of the dominant effect of the order in which they are heard—the first style presented is always more influential. So, we presented different styles of music in random order through earphones (also with a randomly inserted silence) to 12 musically untrained Italian medical students and 12 conservatoire musicians. We found their cardiovascular responses to the six styles were very similar in both groups (and independent of their individual music preferences), although musically trained subjects’ responses were rather greater. The responses to calming (Indian rajas), or exciting music, (jazz or fast classical), were similar between individuals. So, these studies suggested that the therapeutic use of music to calm individuals could be relatively simple—not needing individually tailored music for each person.

Unfortunately, for commercial reasons, the use of music to calm people in therapeutic environments has happened without any critical controlled studies of its effectiveness. This commercial band wagon has held back proper evaluation, but more importantly, has led to new scepticism about whether there is any real therapeutic role for music therapy.

We desperately need some new properly controlled studies to evaluate the potential uses of music therapy.

References can be accessed via Bernardi et al., Circulation 2009;119:3171–3180.
The Asklepios project in perspective: the population on its way to ageing

Thierry Gillebert discusses an ambitious project that started in 2002 and is ongoing

At the time when he had just been appointed to full Professor of Cardiology at Ghent University and Chief of the Department, ‘a research fellow came to me and asked with uncertainty in his voice, if by chance I would be willing to support a major grant application. I was initially very sceptical and reluctant because of the ambition and scope of the project proposed by this young man. However, my concerns gradually vanished and I became quite enthusiastic when I learned that Ernst Rietzschel (the PI of the Asklepios study) had already at that time developed a close collaboration with a group of exceptionally motivated primary care physicians (GPs) of the twin cities Erpe-Mere and Nieuwerkerken. These communities are situated on both sides of the busy motorway from Brussels to Ghent. The GPs were highly committed to show that they were able to actively collaborate and to provide the necessary men and women for a top scientific project’ (Figures 1 and 2).

Serendipitously, a number of key necessary strengths were present at that moment and time at Ghent University. In the departments of cardiology and public health, there was an extensive expertise in cardiovascular epidemiology, hypertension, non-invasive haemodynamic, and cardiovascular function testing. Furthermore, the planned project provided an ideal population-based testing ground for a number of physiological questions that grew out of a long-standing collaboration with civil engineers (Patrick Segers and Pascal Verdonck), who had built up a leading expertise in biomedical and biomechanical modelling of the cardiovascular system. Furthermore, the inherent interest in ageing was the link to a new partnership with experts in telomere biology and epigenetics (Sofie Bekaert and Tim De Meyer). It is a source of particular pride that this initial team of collaborators/friends have remained at the core of the study for 12 years, now augmented with new ‘family members’.

‘Also personally, this was for me (says Gillebert) the unexpected possibility of extending concepts, which I had developed in the animal laboratory, to a carefully phenotyped human population. We would be able to test physiological hypotheses in an epidemiological dataset, all with non-invasive data. These hypotheses were derived from load clamp experiments in isolated cardiac muscle and in intact heart models.1,2 Non-invasive haemodynamics had been my dream since the early eighties. I developed this dream in 1984 when working with Liv Hatle and Björn Angelson in Trondheim, Norway, where I was being trained in cardiac Doppler haemodynamics’.

We then quickly established a consortium to write an application, which was fortunately granted by the Foundation of Research – Flanders. Very hard and often tedious work started for the general practitioners (GPs) organization Asklepios, for Ernst Rietzschel and for his small research team. Enrolment began in September 2002 and continued relentlessly week after week for more than 2 years. A random sample of participants from the Erpe-Mere and Nieuwerkerken communities were invited to participate, with support and active promotion of the study by the GPs. The participants were initially screened for exclusion criteria by the GPs. They were then referred to the cardiovascular examination centre, which was organized in a former city school (Figure 3).

The data obtained covered medical history and risk factor profiling, anthropomorphic data, a bio-bank of blood, serum, and DNA (and derived telomere length data). Co-designed with the engineers, key pressure, flow, and dimension data (all that is needed to haemodynamically characterize a system) were acquired using state-of-the-art cardiac and vascular echography and tonometric signals of all

Figure 1 Four of the key players in the general practitioners association Asklepios. From L to R: Piet Van Damme MD, Patrick De Coninck MD, Luc Cooman MD and Peter Cassiman MD. This instrumental association of GPs provided the name of the Asklepios study.
They developed sophisticated approaches for describing arterial development. 3 The team enrolled more than 2500 apparently healthy women and men, 35–55 years old, a representative sample of the Belgian population (and thus close to the average West-European population).

At that time Asklepios focused on novel biomarkers, lifestyle, and early cardiovascular damage in the general population. The initial research goal was to provide a population-based reliable testing ground for a broad cluster of questions on the interplay between haemodynamics, ageing, and the premature emergence of cardiovascular disease. The long-term goal was and remains, attempting to develop better strategies for the prevention of premature cardiovascular disease (CVD), i.e. more successful (cardiovascular) ageing through improved risk stratification and better understanding of early disease development. This first measuring round of the Asklepios cohort study led to almost 100 full publications in various areas, of which the largest majority were in the top quartile impact factor journals in their field. In broad brushstrokes, some of the major research efforts are listed below.

The team led by Bekaert and De Meyer published articles on telomere length of chromosomes. They showed that telomere length of peripheral blood leucocytes primarily reflected the burden of increased oxidative stress and inflammation, whether or not determined by an increasingly unhealthy lifestyle, while the association with classical cardiovascular risk factors is limited.4 Furthermore, our data strongly argued against one of the key telomere hypotheses, namely, that inherited shorter telomere lengths play a key role in atherosclerosis development. Neither the presence of early atherosclerosis, nor a family history of premature cardiovascular disease was associated with shorter telomeres. So, it seems that telomere length is a proxy for unhealthy lifestyle and exposure to oxidative stress, rather than a genetically transmitted risk factor for atherosclerosis. These first data on telomere length data in a large population helped alter the way scientists look at the relation between telomere length and CVD. The focus for the near future is on understanding telomere attrition rates and their relation to CVD development.

The team led by Segers investigated tonometry pressure and Doppler flow signals from carotid, brachial, radial, and femoral sites. They developed sophisticated approaches for describing arterial function with the combination of pressure and flow signals,5,6 and reported population data on wave reflection assessed by wave separation analysis. This key engineering research led to several doctoral theses and paved the way to the more applied haemodynamics developed thereafter.

These more applied haemodynamics focused on ventricular–arterial coupling by studying simultaneous pressure, flow, and dimensions as time-varying phenomena. Particularly fruitful has been the collaboration with Chirinos and St John Sutton from the University of Pennsylvania (Penn University). They showed in a large series of publications how pathological wave reflection in the arterial tree elevates time-varying pressure in late-systole and hence late-systolic wall stress. They elegantly showed that late-systolic wall stress is associated with population characteristics opposite of those observed with early-systolic wall stress.7 They provide an original, novel clue on distinct haemodynamic triggers of physiological and pathological hypertrophy.

Timing is crucial in molecular biology and adaptive mechanisms! Early-systolic load triggers physiological adaptations, while late-systolic load induces maladaptive changes. Chirinos et al. also investigated the predictive value of arterial wave reflection (linked to late-systolic load) on outcome in the US MESA cohort. Wave reflection appeared to be independently associated with diastolic dysfunction and elevated left ventricular mass. Clinically it proved to be a potent and independent predictor of cardiovascular events, specifically incident heart failure and mortality. The prognostic value of wave reflection is independent of and as potent as, e.g. systolic blood pressure.8,9

The Asklepios study collaborates in an expanding cluster of international collaborations such as the GRACE study,10 the Framingham and the AortaGen consortium,11 the MARE consortium,12 and the diabetes research initiated by Geneviève Derumeaux.13 It is generally accepted that Asklepios data, recorded by a single operator, with a single machine and software, are extremely reliable and reproducible. The data were repeatedly used as a source for normal cardiac and vascular measurements. They are included in the Reference Values for Arterial Stiffness’ Collaboration, in the EchoNormal database, in the recent EACVI-ASE Recommendations for Chamber Quantification,14 and in the upcoming EACVI-ASE Recommendations on the use of Echocardiography in Adult Hypertension.

In 2011, 10 years after the start of the study, a second measuring round was started and is scheduled until 2016. It had been planned from the start to schedule repeated measurements and to monitor outcomes.3 Technology has evolved in 10 years and the current measurements include state-of-the-art 3D echocardiography, optimized image acquisition for speckle tracking analysis, data acquisition at rest, and during isometric stress in the entire population (PhD programme of Caroline Van daele). The focus of the research has also changed. Asklepios broadened its focus on the way men and women evolve more or less successfully with ageing. Measurements were extended to urine analysis, pulmonary function analysis, home recording of breathing patterns, and oxygen saturation levels to assess sleep apnoea.

A long-standing research focus has been understanding the effects of obesity on the cardiovascular system. Earlier work studied the prognostic important subjects of indexation of echocardiographic parameters based on smaller and heterogeneous clinical sample populations. Asklepios provided the opportunity of evaluating a large and representative population sample. The appropriate cardiac mass to body ratio (referred to as allometric scaling) became
the study object and the investigators separated men and women in calculating the optimal scaling coefficient. This important effort results in improved indexation of left ventricular mass in men and women, and in persons of different ethnicities.\textsuperscript{15} The relevance of this research is of particular importance in obesity and in the presence of diabetes.

The relation between obesity and, for example, inflammation, the development of hypertension or maladaptive cardiac hypertrophy is obscured by what happens at night: sleep disordered breathing (SDB). Sleep apnoea is a huge potential confounder and the proverbial—in this case nocturnal—elephant in the room. Hence, we are actively performing home sleep apnoea testing in the entire Asklepios population cohort, and are intensively collaborating with the respiratory department (Guy Joos, Guy Brusselle, Fré Bauters). Initial data suggest that sleep apnoea (defined $>$15 apnoea-hypopnoea index (AHI), i.e. episodes per hour) will likely be present in $\sim$10\% of subjects. But of larger interest is that another 30–40\% of subjects have some degree of SDB below the level of true sleep apnoea, from 1–15 AHI. Currently we do not know what AHI threshold starts to impact the cardiovascular system (and it is exceedingly likely that thresholds might be phenotype-specific).

A rapid look at weight and waist circumference showed the Asklepios investigators that obesity rates are sharply increasing, particularly in women. The investigators were therefore not surprised that the recent obesity publication of the WHO proclaimed Belgium as the European champion of obesity in women, according to a projection in 2030. In the end and after many decades, the aged Asklepios investigators will presumably tell the cardiology community to eat less, to make healthy food choices, and to move, move, move...as so many have done before us.

Environmental influences on the cardiovascular system and on ageing in general are not neglected. The questionnaires include extensive interrogation on QOL, social support, depression, anxiety, and personality types.\textsuperscript{16} The participants to the cohort study live on both sides of a busy motorway. It therefore was obvious to register pollution by noise and small particles and relate these longitudinal exposure data over 10 years to the evolution we are observing in blood, heart, and vessels. Specifically this project will try to disentangle the contributions of background pollution (related to where you live) from exposure during our daily commute. The study of these environmental influences is in a preliminary phase so far, but look very promising indeed.

Inherently, our study design has always accepted that many interesting questions and insights will be provided, not by us, but by future collaborators with diverse backgrounds. It is often in the clash between distinct approaches and disciplines that the largest advances are generated. Therefore, it is only fitting that one of the study slogans has always been ‘Our door is always open, and the coffee is always hot’.

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References
References are available as supplementary material at European Heart Journal online.

European Heart for Children: a story of humanity and success

Since its foundation in 2009 and the last publications,\textsuperscript{1–4} European Heart for Children has grown substantially

The European Heart for Children (EHC) mission is to ‘promote knowledge and treatment of congenital heart disease (CHD) and related disorders in children and young adults in the European Society of Cardiology member countries and beyond’. To date, the EHC alone has accomplished:

- Children examined: 1873
- Children successfully operated on: 258
- Children haemodynamic procedures performed: 110
- Missions established: 22 in 8 countries
- Egypt, Haiti, Jordan, Kurdistan, Morocco, Romania, Syria, & Tunisia
- Scholarships awarded: 7

This is insufficient for the 2–6 million children affected with CHD, to which 800,000 to 1.5 million new cases are added each year.
The Global Forum operates with rigorous, transparent, solid, and impartial principles. Once an area or a country in need has been identified, it establishes contact with the local institutions (public hospitals, national cardiac societies, politicians, etc.) and professionals, with the aim of starting medical missions (to prove that ‘it can be done’) and to facilitate a training programme for doctors, nurses, technicians, etc. Thereafter, it explores the possibility of developing a permanent sustainable centre.

The infrastructure required often includes buildings, equipment, and human resources. The latter is the most lengthy and difficult process which requires a coordinated effort. Sustainability requires sufficient funds from the local government, insurance companies, members of the community, and of course the generous Global Forum benefactors, regardless of how small the contributions may be.

The EHC is now involved in two main projects.

The first is in Morocco. The mortality of children born with CHD in Morocco is 36%. Every year 5000 children are born with CHD, the majority of them could be saved if properly treated. At present, only two centres are able to perform simple interventions for CHD:

- in Rabat, the Centre/Polyclinique Universitaire Hospitalier de Rabat (public)
- in Casablanca, the Institut Humanitaire cardio-pédiatrique – Les bonnes oeuvres du Coeur (private).

The average cost for an intervention in the private centre is €12,000. A fee which the majority of the population cannot afford. Morocco is in urgent need to establish a public centre able to treat a large number of children born with CHD ‘free of charge’.

There is a long-standing relationship between EHC and the Centre Hospitalier Universitaire (CHU) Ibn Rochd Hospital in Casablanca. Therefore, EHC and Bambini Cardiopatici nel Mondo will develop a small fully equipped unit near the cardiac surgical department dedicated to the care of children with CHD. The existing department does not have the surgical or intensive care facilities needed to treat children with CHD and, more importantly, they do not have adequate medical equipment or personnel. The equipment for the unit will cost €1,636,000 and the cost to train the personnel will be €760,000.

Another project is in Senegal, Dakar where La Chaine de L’ Espoir is building a dedicated hospital. There, EHC will help in constructing a two-floor guest house with apartments for visiting doctors and relatives of the children being treated in the hospital.

The EHC has joined the Translational Giving European Network so that donations from any European country can be tax deductible.

Further information is available at http://www.europeanheartforchildren.org/donate.php or by direct contact: info@europeanheartforchildren.org

References

References are available as supplementary material at European Heart Journal online.
Announcement

European Heart for Children Global Forum Dinner

Roberto Ferrari MD announces an evening during the European Society of Cardiology Congress in London to benefit children with congenital heart disease at TROXY http://www.troxy.co.uk Sunday 30 August at 20:30

I am sure that by the time you read this announcement, you will already be in London for the European Society of Cardiology (ESC) Congress.

I would like to take a minute, if I may, to ask you to think about how lucky we are. We are all able to participate in a worldwide congress as speaker, chairman, or even participant among our colleagues and friends. As President of European Heart for Children (EHC), I also need to ask you to take a minute to think of those less fortunate than us.

I have an image in my mind of passing a lake and seeing a drowning child. He can be saved—but at a price... an expensive suit and shoes will be ruined. What would you do? The answer is clear but what is less obvious is that 9 million children under the age of 5 drown in the lake of poverty each year. 24 000 children each day! A full football stadium!! 1 million of these children have congenital heart disease (CHD) and only 200 000 have adequate healthcare. Today, over 2.4 million children are awaiting treatment and if they are treated, they can be saved. This is what EHC is all about.

This year, in London, you will not have to ruin your expensive suit and shoes. The EHC Global Forum annual fundraising dinner will be held at Troxy, a unique venue originally opened as a luxury cinema in 1933, combining vintage charm with modern facilities. Troxy is close to Excel, the Congress venue.

We will be honoured by the presence of HRH Prince Michael of Kent GCVO, one of the EHC Patrons.

A film relating to the situation of CHD in different parts of the world will be shown, and Prof. Hugo Saner, from the Prevention Association, and his jazz band will offer an unforgettable evening of entertainment.

This will be our 7th Annual dinner and to date we have raised sufficient funds to establish over 20 missions where we have examined more than 2000 children, operated on, and therefore saved the lives of 258 children and 9 children have been sent to Italy for more complex surgeries. We have also provided training grants for on-site medical staff. The funds that we are able to raise from the dinner this year will go towards several projects that are on-going in Morocco, where we will be able to develop a paediatric unit in Casablanca together with Bambini Cardiopatici nel Mondo and in Senegal, where we will participate in the construction of a Hospital in Dakar, together with La Chaine de l’ Espoir.

Tickets will be on sale at the EHC booth at Excel and can also be purchased on-line.

Figures quoted by European Heart for children

€ 10 can save a life
€ 100 diagnose and medically treat one child
€ 500 can treat one child by means of interventional cardiology and other follow up treatment for three months
€ 1500 can perform open heart surgery on one child

For further information, http://www.europeanheartforchildren.org/donate.php or contact: info@europeanheartforchildren.org

Together we can really make a difference! We hope to see you there!