In one of the preceding contributions to Heart News and Views, "Molecular Biology: The Beginnings" reference was made to Max Delbruck as a driving force in the new science of Molecular Biology [1998; 6 (1): 1-2]. Delbruck’s life spanned two continents in a critical portion of the 20th Century, when physics was changing our view of the infinitesimal small and the infinitesimal large. It was thought then that concepts of the new atomic physics could be adopted to biology, providing it with a fundamental basis.

It is not the prime goal of this communication to recite all the facts of Delbruck’s life. But, the fate of Delbruck’s family caught in the turmoil of the middle of the 20th Century is mentioned because it is an indication of how the intellectual elite of Germany was exterminated by the Nazis. Max Ludwig Henning Delbruck was born in Berlin in 1906. He grew up in a suburb of Berlin, the Grunewald, which for many years was the center of Berlin’s intelligentsia. His father was professor of history at Berlin University. Three of his father’s cousins were respectively a professor of German literature, Chief Justice of the Supreme Court, and Minister of State. His mother’s brother-in-law was professor of Theology at Berlin University. The circle around young Delbruck included the Bonhoeffers and the Von Harnacks. Karl Bonhoeffer was professor of Psychiatry. The Bonhoeffers played a tragic role: Hitler executed two Bonhoeffer brothers, Klaus and Dietrich, and two Von Harnacks cousins, Ernst and Arvid, together with the latter’s American wife, who were leading members of the resistance. Delbruck’s brother Justus was later arrested by the Russians, and died in a Russian camp.

Delbruck’s scientific career was determined by his relationship to Niels Bohr in Copenhagen, at a time when Bohr formulated his concept in which he attempted to apply laws of atomic physics to biological phenomena. Delbruck fervently embraced this concept and pursued it for many years, until overwhelming evidence convinced him that his approach had failed to solve the secret of the gene. Biology, unlike theoretical physics, does not reveal its secrets through formulae alone. And yet, as Newton has stated, the real basis of science is the conviction that Nature, under the same conditions, will always exhibit the same regularities. This fact certainly applies to Physics as well as to Biology, although in Biology, many different forces determine the final single vector.

Bohr formulated his concepts in an article published in Nature in 1933, “Light and Life”. As he wrote, “The unexpected discovery of an essential limitation of the mechanical description of natural phenomena, revealed by the recent development of natural phenomena of the atomic theory, has lent new interest in the old problem”. Bohr believed that irregularities peculiar to atomic processes, which find their place only within the complementary mode of description, are as important for the account of the behavior of living organisms as they are for the specific properties of inorganic matter. In other words, biological processes are subject to physical laws. As an example he cites the carbon assimilation of plants, or the stability of atomic structures exhibited in the properties of chlorophyll or hemoglobin. Bohr wrote that the existence of life must be thought of as an elementary fact that cannot be explained, but which must be taken as a starting point in Biology, “in a similar way as the quantum of action” which forms the foundation of atomic physics.

Bohr’s work, his personality and his approach greatly influenced Delbruck. As a result, Delbruck attempted to approach the subject of x-ray induced mutagenesis by physical laws together with Zimmer, a physicist, and Timofeeff, a biologist. The question was, what causes mutagenesis after radiation? Delbruck asked how physics...
can account for changes and for consistence in mutation, the hallmarks of mutation. He believed that biological phenomena can be explained by quantum mechanics, the quantum mechanical model of the gene. Delbruck wanted to demonstrate facts of genetics by fundamental or physical theory, which he called, “atomic association”—the stability of the gene was due to the strength of interatomic forces, and its mutations were the result of a quantum jump from the stable configuration of the energy hump which separated one configuration from another. Thus, Delbruck actually depreciated biochemistry, and according to Watson, opted for a combination of genetics and physics.

But after 1949, Delbruck had ceased to trust in a combination of genetics and physics. He realized that experimental methods could be used to furnish viable models. He and Luria sent Watson to Europe to learn nucleic acid chemistry.

The answer to the nature of the gene came, to a large extent, from x-ray diffraction discovered in 1912 in Munich’s Institute for Theoretical Physics, a method later extended by Bragg in Cambridge, England. The first successful picture of DNA was obtained in 1938.

What shall we make of Delbruck’s failed initial approach to solve the riddle of the gene? Should we look at it as an insignificant mistake, which does not deserve to be recorded in the fast progressing history of science? By no means! Delbruck’s failure was caused by the overwhelming impact of new physics of the early and middle 20th Century, which, with its unrelenting advance swept biology into its sphere. The approach was wrong, but it was an error of heroic proportions. Heroic mistakes in science deserve as much attention as the truly successful discoveries.

Delbruck was honored with the Nobel Prize in 1969.

References


Richard J. Bing, M.D.
Why do the birds keep on singing?

What a difference a couple of months makes! After September 11th America was nothing less than under siege. First, the terrorist attacks in New York and Washington. Then there was a week of stranded travelers and eerily empty skies except for the occasional prowling fighter jet. The entire country came to a halt and it was just as well because we were all in a state of shock and mourning. We literally could not think of anything else but our sorrow. Just when we thought things couldn’t get any worse the anthrax deaths started. It felt like we were in a hole we could never get out of. Despite our despair, however, the sun did continue to rise and birds did continue to sing. Gradually life began to take on a sense of routine once again and we adapted to the tighter security and the new rules. There was a rather disquieting announcement from the government encouraging us to travel again but they also stated that if you happened to be on a hijacked airliner it would be shot down immediately. Not exactly a winning marketing campaign for the travel industry. Nevertheless, little by little Americans returned to their business.

The American Heart Association Scientific Sessions in Anaheim last November had only about 2/3 the normal number of delegates. Actually if you were able to get Anaheim the meeting was very nice. Usually the AHA is chaos with wall-to-wall people. But this year you could get a hotel room within walking distance of the convention center and there was never a wait for a lunch table. I haven’t seen an AHA like that in decades. Amazingly most of the basic science presenters did show up for their talks. Bob Mentzer, Masafumi Kitakaze, Masatsugu Hori and myself organize a small satellite symposium at the AHA meetings every year. Understandably our advance registration was miniscule and on October 1st we had to make a go or no-go decision with the Queen Mary where we were contracted to meet. After much debate among the four of us we canceled. In actuality we probably should have gone through with the meeting as people had started to venture out of their bunkers by November.

On the second day of the AHA conference I woke up to the morning news cameras trained on a column of smoke rising from a downed airliner in a New York suburb. It seemed like déjà vu all over again. Everyone had one question on his or her mind: was it another terrorist attack? Had the security failed? Soon the word came that it appeared to be mechanical failure and we all breathed a sigh of relief. Somehow it just feels better if you crash because the tail fell off the plane rather than a hijacking although I guess you would be just as dead. Actually much of the security turned out to be more window dressing than real. We had soldiers wearing camouflage crawling all over the air terminals but it seems that due to limited resources no one was checking the baggage for explosives (is this an oxymoron? “The checked baggage was unchecked”). Apparently the fates must have decided that we had suffered enough, and as a result, and no suicide bomber capitalized on that gaping security hole before it was finally closed this January.

Then we turned our attention to the Stans: Afghanistan, Pakistan, Uzbekistan. My secretary even started worrying about her ex-husband Stan. The military action in Afghanistan so far has gone amazingly well (all of our Taliban members might want to disregard that last point). America started out to get some good old-fashioned revenge and as of this writing it looks like we ended up building a nation. I think the world is now optimistic that Afghanistan can get finally back on its feet again. Despite an economic recession our mood in America is good. Actually, after last fall our mood had nowhere to go except up. Most importantly, though, the thoughts of my colleagues and myself are once more focused on science. I have even been able to turn my wrath away from Osama Bin-What’s-His-Name and back to where it’s really needed, those pesky journal referees. The last few flights I was on were sold out so even the most faint of heart seems to be flying the friendly skies again. Yes, the birds do keep on
singing! All of this bodes well for a good turnout for the upcoming ISHR meetings this summer and fall and I hope to see each and every one of you there as we try to get back to some sense of normality.

James M. Downey

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**Troponin and its Role in the Altered Contractile Function of the Failing Human Heart**

My introduction to scientific research came in the final year of my Bachelor of Science honours degree at the University of Liverpool. My degree was in pharmacology and the honours year project that I undertook was to study the effects of estrogens on the relaxation/contraction of vascular smooth muscle and determine their mechanism of action. This was a three-month project and gave me a valuable insight into the important and exciting world of cardiovascular research.

**From B.Sc. to PhD**

After graduating from the University of Liverpool in the summer of 1998 I then decided study for a PhD. This came in the form of a PhD project provided by the National Heart and Lung Institute, part of Imperial College, London, which I began in December of 1998. The project involved working in Professor Steven Marston’s laboratory investigating the structural and functional properties of troponin, which is the regulatory protein of cardiac muscle.

Human heart muscle is made up of myofibrils, which are in turn made up of myofilaments. These consist of interdigitating thick and thin filaments. The contraction of the muscle occurs by sliding of these filaments across one another in a process known as the cross-bridge cycle. Myocardial thick filaments are made up of myosin. Thin filaments are made up of three components, actin, tropomyosin and troponin. Actin has sites which bind myosin. Tropomyosin is a long molecule, which spans seven actin molecules, and the position of the tropomyosin molecule on the actin determines whether cross-bridges between actin and myosin can form.

Troponin is the regulatory component of the myocardial thin filament and is made up of three subunits, troponin T, troponin I and troponin C, each of which has a specific function. Troponin T is the tropomyosin binding subunit of the complex and this plays an essential role in anchoring the troponin to the thin filament. Troponin I is the inhibitory subunit of the complex and has important interactions with both troponin T and troponin C and also actin. Troponin I has several phosphorylation sites, the level of which are reported to be altered in the failing heart. In addition, troponin I is susceptible to degradation at both its N and C-termini. Troponin C is the Ca²⁺ binding subunit and too has important interactions with troponin T and troponin I. Upon Ca²⁺ binding to the C-terminal domain of the molecule, a conformational change in the troponin complex occurs such that the inhibitory properties of the troponin complex on the thin filament are alleviated.

The major technique used to study the functional properties of troponin was the *in vitro* motility assay. This technique was devised by Kron and Spudich in 1986 [1] and has been adapted in our laboratory to optimise our studies. This technique allows us to study the functional properties of reconstituted thin filaments using very small amounts of protein. From the *in vitro* motility assay two main parameters are obtained, these are the fraction of filaments motile, which corresponds to the on/off state of the thin filament, and filament velocity, which corresponds to the cross-bridge cycling rate (speed of muscle contraction/relaxation).

**Does Troponin Differ between the Non-failing and Failing Heart**

The early stages of my PhD involved trying to successfully isolate fully functional troponin from human heart muscle. Once functional troponin was consistently isolated, troponin extracted from non-failing and failing human hearts were analysed. From my studies it was found that thin filaments...
containing troponin from failing human hearts had a slower maximum filament velocity compared to non-failing heart troponin. This suggests that the failing heart has a reduced cross-bridge cycling rate and hence a slower contraction and relaxation. It was also found that thin filaments reconstituted with failing heart troponin were more Ca²⁺-sensitive with an EC₅₀ approximately 2-fold greater than thin filaments containing non-failing heart troponin. These findings were consistently found comparing a range of non-failing and failing hearts of different aetiologies.

Since consistent functional differences were detected between troponin from non-failing and failing hearts, it was then my aim to assign a structural difference to account for this. From the literature there were several popular theories that were studied. These included altered expression of troponin T isoforms, with reports suggesting a reversion towards the foetal isoform expression in the failing heart, troponin I phosphorylation, where a reduction in troponin I phosphorylation has been reported in the failing heart and finally degradation of the troponin in heart failure.

In my studies, no difference in troponin T isoform expression was detected between non-failing and failing heart troponin samples that had been shown to be functionally different. There was no detectable difference in degradation of the troponin either. With regards to troponin I phosphorylation, there was no difference in the level of protein kinase A (PKA) phosphorylation between failing and non-failing hearts. However in an experiment where the troponin complex was non-specifically dephosphorylated, the difference in Ca²⁺-sensitivity between thin filaments containing non-failing and failing heart troponin disappeared and the difference in maximum filament velocity was also reduced. This suggests that the phosphorylation of troponin by kinases other than PKA may be involved in heart failure. The kinase that is receiving significant attention at present is protein kinase C (PKC) with some fascinating results.

Future work will involve experiments where subunits of the troponin complex will be substituted with known subunits to try and identify the subunit that is the major player in causing the differences in function between non-failing and failing heart troponin. An important question, which as yet has not been conclusively answered, is: are the alterations to the myocardial thin filament primary events in heart failure or is it alterations in Ca²⁺-handling? Recent papers by Perez et al [2] and Ruf et al [3] have suggested that it in fact the alterations to the myocardial thin filament which is the primary event and that the changes in Ca²⁺ handling are compensatory. Further work addressing this issue will further help our understanding of the sequence of events leading to heart failure and thus lead to improvements in the treatment of the disease.

### The Perfect Ending to an Intriguing PhD

In July 2001, I had the honour of being short-listed as one of the finalists for the ISHR-European Section Young Investigator Award. This involved me presenting my work at the XVIII World congress in Winnipeg. Based on my submitted manuscript and presentation at the meeting, I was selected as the winner of this prestigious award and it is with tremendous pride that I accept it and in doing so thank my fiancée and family for their support and encouragement throughout my PhD.

### References


The ISHR - European Section / SERVIER Research Fellowship

THE EUROPEAN SECTION of the International Society for Heart Research and SERVIER invite submissions for the second ISHR-ES / SERVIER Research Fellowship. The purpose of this Fellowship is to support the initiation and development of scientific collaborations between outstanding groups in the field of cardiovascular biology by providing a young investigator from a European laboratory with one-year post-doctoral support allowing him/her to carry on a research program in another European country. The term European refers not only to the countries of the European Community but also to all countries belonging to the European Section of ISHR.

Details of the competition are as follows:

1. Candidates must be members (or have applied for membership) of the ISHR-ES (membership application forms are available at the ISHR-ES web site www.biomed.cas.cz/fgu/ishr_es/ or can be obtained from Dr Frantisek Kolar, Secretary of the ISHR-ES, Institute of Physiology, Academy of Sciences of the Czech Republic, Videnska 1083, 142 20 Prague 4, Czech Republic. Tel. +420 2 475 2559; Fax +420 2 475 2125; E-mail kolar@biomed.cas.cz).
2. Candidates must have defended their Ph.D. thesis not earlier than January 1, 2000 and be less than 35 years of age on July 1, 2002.
3. Applications must include the following:
   - Curriculum vitae (family name, first name, date of birth, current employment and position, summary of previous positions, degrees, special area of interest and expertise, other activities, publications);
   - Research program of a maximum of 10 pages (including one page summary and references) detailing the research program (title, aims, rationale, working hypothesis, scientific expertise of each group, preliminary results if any, plan of investigation detailing the scientific procedures and role of each investigator of each group and the precise role of the candidate in the proposed program and funding);
   - Letters of the candidate’s current immediate supervisor and future immediate supervisor (Division Heads, Department Chairmen, or Institute Directors) detailing why the collaboration between the two research groups is essential for the success of the research program and why, among all other potential applicants, the applicant is the most appropriate candidate for the Fellowship, and offer rationale for their opinion.
4. Eight copies of the application should be received by Dr Jean-Jacques Mercadier, President of the ISHR-ES, Departments of Physiology and Cardiology and INSERM U460, Groupe Hospitalier Bichat – Claude Bernard, 46 rue Henri Huchard, 75877 Paris cedex 18, France, no later than April 30, 2002. Applications received after this deadline will not be considered.
5. The two collaborating research groups can submit only one application.
6. The applications will be reviewed in Paris in May 2002 by a committee composed of five members of the ISHR-ES Council and one representative of SERVIER. The three best applications will be classified. The second and the third will receive one year free electronic subscription to the Journal of Molecular and Cellular Cardiology.
7. The winner of the Fellowship will receive a travel grant to cover economy airfare and other travel costs up to 1000 Euro towards his/her attendance at the next annual Congress of the ISHR-ES in Szeged, Hungary, July 3-6, 2002. At the Congress, the winner will present his/her research program to the Society. He/she will receive a plaque and check of 20,000 Euro as a personal support. Any winner who, for any reason, cannot personally present his or her research program at the Congress must withdraw from the competition. Substitute presenters are not allowed.
8. It is expected that the results of the investigation will be presented by the recipient at the annual ISHR-ES Congress in Dresden, Germany, in 2004.
9. Applications will not be returned.

Jean-Jacques Mercadier, M.D., Ph.D.
President, ISHR European Section
ONE OF THE KEY MISSIONS of the ISHR is to recognize outstanding work by cardiovascular scientists. Since the inception of my tenure as Secretary General, I have regarded this area as one that needed to be developed in our Society. In 1998 I proposed the Research Achievement Award, which was launched in 2001 at the XVIIth World Congress in Winnipeg with great success. However, I felt that this Award is insufficient to fulfill our mission because it is bestowed only once every three years. To fill this “gap”, last July I proposed to Council the establishment of another award, the Outstanding Investigator Prize of the ISHR, which will be bestowed yearly except on the years when the ISHR World Congress convenes. The proposal was unanimously approved by Council and is now being implemented.

The purpose of the Outstanding Investigator Prize is to recognize scientists who have already made major and independent contributions to the advancement of cardiovascular science and are likely to further develop their research in the future. Thus, the main criteria for selecting the awardees will be scientific excellence, independence, and potential for future scientific growth. As is the case for the Research Achievement Award, the Outstanding Investigator Prize is therefore targeted at investigators who are in the “intermediate” phase of their academic career; the major difference between the two is that the Research Achievement Award is presented during the ISHR World Congress while the Outstanding Investigator Prize will be given at ISHR Section meetings. There is no age limit for the Outstanding Investigator Prize. To avoid overlap with the Research Achievement Award, the Outstanding Investigator Prize will not be given in the years when the World Congress convenes. By alternating the triennial Research Achievement Award with the Outstanding Investigator Prize, the ISHR will be able to bestow these recognitions on a yearly basis, thereby establishing a tradition of continuity that will enhance its profile in the scientific community.

The first Outstanding Investigator Prize will be presented in 2002 during the meeting of one of the ISHR Sections. The winner will receive $3,000, a plaque, and travel reimbursement. He/she will be announced in the Journal of Molecular and Cellular Cardiology and in HEART NEWS AND VIEWS as well as in the ISHR website. Nominations will be evaluated by a multidisciplinary Selection Committee composed of at least ten individuals, representing at least four Sections of the Society, which will include the President of the ISHR, the Editor of JMCC, and the Secretary General and at least three members of the International Council.

I am pleased to report that Aventis has agreed to support a $75,000 endowment which will fund the Outstanding Investigator Prize in perpetuity. This endowment will be accomplished in $25,000/year installments over three years, with the first installment already paid for 2001. We are indebted to Aventis for their generous support of the scholarly activities of our Society.

The Outstanding Investigator Prize is an important addition to the Award portfolio of the ISHR. It will play an important role in the career of many scientists in years to come and it will also enhance the visibility of the ISHR worldwide. As always, I welcome your comments regarding this initiative. Please email me at rbolli@louisville.edu or fax at +1 502-852-6474.

Roberto Bolli, M.D.
Secretary General and Treasurer, ISHR
In a splendid setting and a milieu of warm hospitality the XVII World Congress of the International Society for Heart Research was held in Winnipeg, Manitoba, Canada from July 6 - 11, 2001. Concurrently with these sessions the European and American Sections of ISHR held their annual meetings too. The St. Boniface General Hospital’s Institute of Cardiovascular Sciences and the University of Manitoba, Faculty of Medicine were honored to host these events. Dr Naranjan Dhalla, Director of the Institute of Cardiovascular Sciences at the St. Boniface General Hospital Research Centre and an internationally recognized scientist for his excellent contributions to the advancement of cardiovascular science served as the Meeting Chairman. Because of his well known reputation as a gracious host and as a tribute to Dr Dhalla’s international recognition more than 1,800 delegates, including cardiologists, cardiac surgeons, basic medical scientists and other allied health care professionals from 70 countries, participated in the Meeting. Strong support for this event also came from government officials of local, provincial and national rank.

Dr Dhalla’s Planning Team of more than 200 global experts created the most important scientific convention ever held in Canada, in terms of numbers of visitors and the exceptional quality of the science and the people attending. Over 600 of the world’s leading cardiologists, surgeons and scientists accepted invitations to share their expertise in the exceptional Professional Program consisting of 20 Landmark Lectures, Awards Competitions, 72 Symposia, an Exhibition, and three sessions, each presenting 200 Posters. All speakers and chairmen received the Institute of Cardiovascular Sciences “Medal of Merit”. The local volunteer Organizing Committee performed in an extraordinary manner to guarantee that visitors enjoyed every minute of the sessions, superb networking opportunities and hospitality that only Manitoba can provide.

A series of Landmark Lecturers included such prominent cardiovascular researchers as Dr Eugene Braunwald, Boston, USA; Sir Magdi Yacoub, London, England; Dr Yoshio Yazaki, Tokyo, Japan; Dr Claude Lenfant, Bethesda, USA; Dr Lionel Opie, Cape Town, South Africa; Dr Eric Olson, Dallas, USA; Dr Shigetake Sasayama, Kyoto, Japan; and Dr Rodolfo Paoletti, Milan, Italy. In recognition of their contributions to cardiovascular science over the years, the International Academy of Cardiovascular Sciences presented each lecturer with an individual award.

During the proceedings prestigious international awards were presented to the following scientists: Dr Robert Lefkowitz, Duke University, Durham, USA – Peter Harris Award from the ISHR; Dr Eduardo Marban, Baltimore, USA – ISHR Research Achievement Award sponsored by Chugai Pharmaceutical Company; Dr Hilchen Sommerschild, Oslo, Norway – ISHR European Section / SERVIER Research Fellowship; Dr Jacques Genest, Montreal, Canada – International Academy of Cardiovascular Sciences; Dr Ruth Collins-Nakai, President, Canadian Cardiovascular Society, Edmonton, Canada - International Academy of Cardiovascular Sciences Award; and Dr Eugene Braunwald, Harvard Medical School, Boston, USA – 2001 International Humanitarian Award of the St. Boniface Hospital & Research Foundation.

This World Congress, called Frontiers in Cardiovascular Health, featured very prominent cardiovascular researchers from around the world, including Dr...
Eugene Braunwald of Harvard University, who wrote the textbook on modern heart treatment, and Sir Magdi Yacoub from London, England, who is credited with having performed more heart transplants than any other surgeon in the world. Conference organizers at the St. Boniface General Hospital Institute of Cardiovascular Sciences had spent long hours over previous years preparing for the event.

The International Academy of Cardiovascular Sciences sponsored an extraordinary Symposium: “Global Pandemic of Cardiovascular Diseases” featuring world-renowned experts from Egypt, Iran, Italy, Croatia and Health Canada’s Dr Arun Chockalingham who had edited the World Heart Federation “White Paper” on the subject “Deaths from Heart Attack, Stroke Expected to Double”.

In recognition of the high quality of poster presentations, the Heart & Stroke Foundation of Manitoba presented 20 awards for outstanding Posters. Deputy Mayor Lillian Thomas awarded 12 visitors Honorary Citizenships of the City of Winnipeg.

To encourage increased participation of young investigators, many from Mongolia, China, Israel, Cuba, India, Czech Republic, Nigeria, Rumania, Hungary, Slovak Republic, Tanzania and Jordan were extended Travel Awards with assistance including organizations from Germany and United Kingdom.

Significant sponsorships for the World Congress were identified from a global network, including all three levels of Government, Mitsubishi-Tokyo, Merck-Frosst, Kowa, Medicure, Aventis, EliLilly, Kaito, Pfizer, Bayer, World Heart Corp., University of Western Ontario, Kowa, ATL, Manitoba Liquor Control Commission, CanWestGlobal, Pulsus Group, Manitoba Hydro, Wawanesa Insurance, DeFehr Foundation, National Research Council, Myles Robinson Memorial Heart Fund, GreatWest/London Life, Canada Safeway, Dairy Farmers of Canada and Mars/M&M.

Media interest was exceptional – locally, nationally and internationally (even CNN and the BBC aired a report from the Congress). In particular, the Winnipeg Free Press published an 8-page section and more than 30 articles. Planning continues for nine publications of the work presented at the Congress, including material adapted for the public.

A major highlight of this World Congress, that resulted in the dissemination of new information, was a public forum dedicated to education in advances and treatment of cardiac disease. Dr Arun Chockalingham from Health Canada, the governmental agency responsible for legislating medical treatments in Canada, gave a landmark lecture on the “Global Pandemic of Cardiovascular Diseases”. Dr Chockalingham identified that the incidence of heart attack and stroke are expected to double globally and reach pandemic proportions by the year 2020. With this in mind, Dr Chockalingham suggested that cardiovascular medicine must unify, such that different countries become linked globally to bridge gaps between westernized medicine and that currently available in third world and developing countries. Toward this goal, Dr Chockalingham, along with other health experts, established in 1998 the World Health Organization and the Global Forum on Health Research on Cardiovascular Disease Initiative in Developing Countries. The headquarter for this is located in New Delhi, India.
The primary goal of Dr. Chockalingham’s work is to develop new innovative treatment strategies to reduce the morbidity and mortality of cardiovascular disease in third world countries that have limited access to conventional medical interventions.

In addition, the public forum included a lecture on “Diet and Lifestyle for Cardiovascular Health” presented by Dr. Subhash Manchanda, Chief of Cardiology at the Cardio Thoracic Sciences Centre in New Delhi, India. This innovative lecture illustrated how yoga can reduce risk factors leading to cardiovascular disease. For example, Dr. Manchanda’s research study which included 42 men with advanced coronary artery disease, demonstrated that the implementation of a daily routine consisting of meditation had beneficial effects for cardiovascular health, including a reduction in the incidence of angina, lower serum cholesterol and triglycerides, weight loss as well as increased physical endurance. These exciting new findings highlight how a better and improved life-style, with simple yoga exercises and meditation, can significantly improve patient outcome and reduce incidence of cardiac morbidity.

Research presented by Dr. Bruce Holub, an expert in the field of nutrition and cardiovascular health, illustrated how consuming foods rich in omega-3 fatty acids can reduce disease and improve cardiovascular health. Dr. Holub’s research suggested that the consumption of 900 milligrams of fish oil containing omega-3 fatty acids was sufficient to reduce the number of cardiac related deaths by as much as 30% and could further reduce the incidence of fatal heart attacks by approximately 45%. The fact that the cardiac disease can be reduced by the simple consumption of omega-3 fatty acids, commonly found in fish and fish products, has profound implications for patients with preexisting heart disease.

Importantly, Dr. Carl Keen, head of the Department of Nutrition at the University of California, presented new exciting research on the cardioprotective effects of food rich in flavonoids. Dr. Keen’s findings revealed that chocolate that is rich in flavonoid containing substances could be cardioprotective. Dr. Keen’s preliminary research studies indicate that certain chocolate, that is rich in flavonoid containing antioxidants, can exert a cardioprotective effect by reducing platelet aggregation, leading to a reduction in clot formation. Foods rich in these antioxidants can be seen to improve cardiovascular health by reducing the incidence of blood vessel diseases leading to heart attack or stroke.

Other research touched on benefits of moderate consumption of red wine, the advantages of so-called functional foods, such as fish-oil supplements rich in omega-3 fatty acids, and a smoke-free environment. Healthy eating and moderation are the best weapons against cardiovascular disease according to most experts.

Overall, the World Congress Meeting of the International Society for Heart Research held in Winnipeg, was considered to be a major success and the largest cardiovascular meeting held in Canada. This was attributed to excellent contributions made by Dr. Dhalla and his organizing team, that resulted in the dissemination of new information and innovative research strategies to treat cardiovascular disease in the new millennium. The meeting further highlighted the importance of international exchange of information and the establishment and strengthening of network ties among the different countries around the world, with the common goal of improving the quality of patient life and reducing cardiac morbidity and mortality through improved education and research.

Lorrie A. Kirshenbaum, Ph.D.
Winnipeg, Canada

Book Review


Heart failure is the final common pathway to death in the cardiovascular continuum. In the last two decades, the decline in mortality as a result of remarkable progress in the treatment and management of conditions like myocardial infarction and hypertension, has ironically been responsible for the marked increase in the incidence and
prevalence of heart failure. In the elderly population of the United States, it is today the most common cause of hospital admission with a dismal prognosis. Until not long ago our treatment options for heart failure were, to a large extent, empirical. However, the advancement in the field of molecular biology has enabled greater comprehension of some of the mechanisms of maladaptive processes that lead to the progression of heart failure. Consequently, treatment strategies are being designed and tested to target the molecular abnormalities. The ever increasing literature on heart failure: disparate and often contradictory in content, makes a discerning understanding of its varied aspects a formidable challenge at the very least. In Heart Failure, Arnie Katz brings to us a remarkable synthesis of the current knowledge in the field of heart failure. Working through the myriad of complexities he presents, through time, a lucid account of the concepts, pathophysiology and the clinical management of this syndrome.

He starts by demarcating six paradigms in the evolution of our understanding of heart failure since antiquity. From simple clinical observations and anatomic correlations to description of circulatory physiology before the 20th century, attention shifted to the role of hemodynamic abnormalities, and subsequently to alterations in excitation-contraction coupling and cell biochemistry. The most recent developments relate to our understanding of the role of altered gene expression in abnormal cellular growth. With beautiful illustrations and insightful analogies, each of these paradigms is elucidated in detail in successive chapters.

Great emphasis is laid on the crucial role that maladaptive hypertrophy of the terminally differentiated myocytes play in the progression of the disease. The processes involved in the hypertrophic response such as the changes in signal transduction, cell cycle and apoptosis are explained in the most intelligible manner.

Heart Failure is a book of great worth and comes at the most opportune time. With characteristic adept elegance, reminiscent of his earlier works, Dr Katz has admirably succeeded in unraveling the intricacies of heart failure. This book is a must for every health care provider, clinical scientist and basic research worker.

Inder S. Anand, M.D.
Minneapolis, MN, USA
HEART NEWS AND VIEWS
is published thanks to an educational grant from Servier

a private French pharmaceutical company committed to therapeutic advances in cardiovascular medicine as well as other key therapeutic areas. We have successfully developed products in the field of cardiovascular diseases (ischemic heart disease, hypertension, and heart failure), as well as in other major therapeutic fields. A number of landmark studies like PROGRESS, EUROPA, PREAMI, PEP, and HYVET are being conducted with our support.

The dynamism of our research is ensured by consistent allocation of as much as over 25% of the annual turnover of the Group to search for new molecules and develop their therapeutic applications.

Servier supports a number of important projects in the field of cardiology, such as the Education and Training Programs of the European Society of Cardiology.

Servier is also the founding father of The European Cardiologist Journal by Fax and Dialogues in Cardiovascular Medicine, a quarterly publication with a worldwide circulation edited by Roberto FERRARI and David J. HEARSE. Dialogues discusses in a comprehensive way issues from the cutting edge of basic research and clinical cardiology.

The forthcoming issue, devoted to KININ RECEPTORS AND ENDOTHELIUM-DEPENDENT RESPONSES will feature articles by:

P. M. Vanhoutte, H. Shimokawa, L. Raij, T. Lüscher

For further information on Dialogues in Cardiovascular Medicine please contact:
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