**Viewpoint: Pericardiocentesis — Is it Undervalued?**

The only authoritative guidelines for the diagnosis and management of pericarditis are from the European Society of Cardiology. They were produced by a task force chaired by Bernhard Maisch, MD, FESC, FACC, who spoke to Barry Shurlock, MA, PhD, about better aetiological diagnosis based on pericardiocentesis and new treatment modalities.

Diagnostic skills in pericarditis are not tested by the large effusions, but by the smaller ones, which are often overlooked, according to Dr Bernhard Maisch, director, Department of Internal Medicine and Cardiology, University Hospital, Marburg, Germany. He commented, “It is important not to be content with the commonly held idea that every case of pericarditis is idiopathic. If we see pericarditis at echocardiography we think we have a diagnosis, but we don’t have an aetiopathological diagnosis. With respect to treatment, it’s most important to classify the disease properly, but of course the aetiological diagnosis is also very important.”

In his department, in appropriate cases, the presentation of pericarditis is followed by pericardiocentesis, even if there are no signs of cardiac tamponade. At the same time, if indicated, pericardioscopy and pericardial biopsy are performed, thereby providing the material for a full diagnostic workup, using polymerase chain reaction (PCR) techniques, cytology, and histology. The same access site can then be used for placing intrapericardial forms of treatment, using a pigtail catheter.

An experienced operator is required, as well as special percutaneous puncture devices, either a Tuohy mandrel or a PerDUCER (Comedicus Inc, Columbia Heights, Minn), developed by Dr Maisch and his group. By means of a vacuum, the PerDUCER captures the pericardium, which is then punctured tangentially by an introducer needle. The process is carried out under visualisation with pericardioscopy.

When guided by echocardiography, pericardiocentesis is a satisfactory technique for large effusions, but with smaller ones, fluoroscopy is required. Dr Maisch advocates the use of the subxiphoid approach to the puncture site, which requires fluoroscopy in a cardiac catheterisation suite and involves inserting a needle subxiphoidly via the left side of the scapula, at a 30° angle to the skin. During pericardiocentesis, the operator intermittently aspirates fluid and injects contrast medium. After aspiration, the needle is quickly replaced with a soft J-tip guidewire. After dilation the guidewire is replaced in turn by a pigtail catheter.

With echocardiographic guidance, access is usually made intercostally. Dr Maisch said, “The subxiphoid approach allows a needle to be inserted between the pericardium and the epicardium, so a catheter can be placed there for access. Then you can first make a cytological diagnosis, then a histological diagnosis, and finally, with a pigtail catheter, you can treat the disease by introducing the appropriate agent into the pericardial sac.”

Pericardiocentesis is relatively straightforward with large effusions, but considerably trickier for those with smaller volumes of fluid. Among the most serious risks are laceration and perforation of the myocardium and coronary arteries. According to published series, all from institutions with a good deal of experience, major complications with echocardiography occurred in 1.3% to 1.6% of cases. With fluoroscopy, arterial bleeding occurred in 1.1% of cases, cardiac perforation in 0.9%, and other complications less frequently.

Pericardiocentesis is most frequently used to drain off fluid, rather than to seek a full aetiological diagnosis, and many less severe forms of pericarditis are missed, often with serious long-term consequences, said Dr Maisch. He remarked, “Very few institutions will establish the aetiology, except in patients with neoplastic disease, when you might explore the pericardium and surroundings of the heart with pericardiocentesis as well as computed tomography.”

Dr Maisch continued, “For pericarditis in general, if you do not have an early aetiological diagnosis, you will probably have recurrent or persistent effusions. This may be a silent process for the patient at first, but may end up with cardiac tamponade weeks or months later. With pericarditis, you may also have perimyocarditis, and then cardiac function will deteriorate. You may then end up with end-stage cardiomyopathy if the aetiology is not established.”

As proof, he cites his department’s patient registry.
Dr. Michel Haïssaguerre, MD

Dr Michel Haïssaguerre is professor of cardiology at the Hôpital Cardiologique du Haut-Lévêque, University of Bordeaux, France. He proved in 1998 that atrial fibrillation originates mainly in the pulmonary veins and pioneered catheter ablation for this disorder. He spoke to Robert Short, BSc, about his life and work.

How did you come to specialise in electrophysiology?
As a child I wanted to be an archaeologist. Indeed, I used my first earnings from holiday work to buy a metal detector. In my late teens, I inclined towards psychology and so entered medical school with that interest in mind.

However, in 1979 as a second-year intern in cardiology I discovered my passion: electrophysiology. I owe that meeting with my specialty to Jean François Warin, MD, then professor and head of the rhythmology service at the Hôpital St Andre in Bordeaux.

How did Dr Warin help shape your career?
I learned about the world of electrophysiology in his department. I became fascinated that, from the examination of a simple 12-lead ECG trace, it was possible to deduce an invisible electrical mechanism operating within a heart. Then later you could reveal that mechanism and prove its existence by electrophysiological mapping. Also, Dr Warin recommended me to Philippe Coumel, MD, in Paris. Dr Coumel was professor of cardiology at the Hôpital Lariboisière and, together with Guy Fontaine, MD, (also in Paris, at the Hôpital La Pitié-Salpêtrière) they were the leading lights in cardiac electrophysiology in France at this time.

I went to work with them in 1980. This was an immensely exciting move and happened at just the right time for me. Melvin Scheinman, MD, and John Gallagher, MD, had recently reported their initial experience of fulguration of the
bundle of His by intracardiac application of ablative energy. Their results seemed like magic to me. Not only could we describe and debate a putative mechanism, but we could now describe the correct one, and also treat the condition.

In 1982, I returned to Bordeaux to work with Dr Warin as chef de clinic (senior resident). Three weeks later, we saw a young man with ventricular fibrillation and an accessory pathway. After 9 hours of mapping, we successfully fulgurated his accessory pathway. This began our series of fulguration of accessory pathways, based on the recording of precise electrophysiological parameters, pathway activity, unipoles, etc. Later on, we had the opportunity to perform the initial intracardiac treatment of nodal tachycardias using the same energy source.

What do you consider your main contribution to the field?
My main interest is to distill the mass of information available within the intracardiac electrogram to its essence in particular, and to pinpoint the components and characteristics of an electrogram that identify the source of (or any critical components of) an arrhythmia.

By 1992, accessory pathways and atrioventricular nodal reentry tachycardias were on the way to becoming an endangered species, as Douglas Zipes, MD, a professor at Indiana University, put it. The final frontier was seen as atrial fibrillation, and so we sought to tackle this arrhythmia. After Dr Warin died, I moved to the Hôpital Cardiologique du Haut-Lévêque under the kind direction of Jacques Clementy, MD, who gave me total freedom to pursue this complex and developing area of electrophysiology.

Maze surgery (a technique that creates a maze of new electrical pathways so that the electrical impulse can travel more effectively through the heart) for atrial fibrillation was the gold standard treatment at that time. Although effective, it was extremely aggressive. We tried to emulate the Maze procedure by creating linear lesions. The group at the Hôpital Cardiologique du Haut-Lévêque at the time included Pierre Jais, MD, Dipen Shah, MD, and Meleze Hocini, MD. But there were also many bright and passionate colleagues from around the world working with us.

With the catheters in place during these procedures, we were able to observe spontaneous initiation of atrial fibrillation and isolated ectopy with the same morphology as the initiating beat of atrial fibrillation on the 12-lead ECG. This led us to discover that the source of ectopy was the pulmonary veins, at least in a group of patients with short, repetitive episodes of atrial fibrillation. Once this was recognised, we placed the catheters in the pulmonary veins and awaited spontaneous or pharmacologically induced atrial fibrillation, sometimes for many hours. We were able to confirm the importance of the pulmonary veins in the initiation of atrial fibrillation (see Figure).

So, contrary to the general belief that any atrial site could generate atrial fibrillation, we found that in most cases the sources of ectopy were clustered around venous tissue. Surprisingly, the electrical impulse came from a simple venous conduit that was believed to be electrically inert and of no possible interest to an electrophysiologist.

From these beginnings, atrial fibrillation ablation has grown rapidly, and its various mechanisms are now the focus of research and targets for therapy worldwide. In chronic atrial fibrillation, the contribution of the pulmonary veins is less important, and the role of other cardiac structures, such as the coronary sinus and the left atrial appendage, is being increasingly recognised. Interestingly, the observation of a trigger initiating arrhythmia can be applied not only to the atrium, but to the whole fibrillating heart. We reported that ventricular fibrillation in normal hearts, as well as in hearts with ischaemic disease, is driven by ectopy from the Purkinje network — the specialised conducting system. Again, ablation of these sources is very effective in eliminating ventricular fibrillation in those patients with frequent episodes.

What has given you most satisfaction in your career?
The effect of catheter ablation on patients. These techniques cure the arrhythmia and have replaced treatment by drugs that was only palliative therapy. It is gratifying to hear patients who have had a dangerous arrhythmia or disabling atrial fibrillation tell me, “I have started a new life” or “I feel like a new person.” We treat about 1000 patients a year, but I heard that, worldwide in 2005, about 50 000 people were treated in a single year and that this figure is increasing by 20% annually. All this is tremendously satisfying.

What do you enjoy in your leisure time?
I like to restore ancient houses and buildings. For example, I spent 3 years restoring a 16th-century convent. I try to find original materials to replace features that have to be replaced, so that the building is as original as possible. Electrophysiology for the working week, and family and old buildings at the weekend. This is my life.

What are the intellectual challenges for you now?
There is a need to create better whole-chamber and even whole-heart mapping techniques, rather than discrete point mapping, and to optimise ablation tools, which can then be applied to the more extensive procedures needed for patients with chronic atrial fibrillation or ventricular tachycardia. Current techniques need to be improved and extended to address the massive problem of patients with heart failure and sudden death. We must aspire to meet all these challenges.
The Future of Cardiology Research in the United Kingdom

Michael Rees, PhD, FRCP, FACA, is chairman of the British Medical Association’s Medical Academic Staff Committee and professor of vascular studies at the University of Wales, Bangor. He discusses the state of clinical cardiology research with Jennifer Taylor, BSc.

Clinical cardiology research, along with research in many other clinical subjects, has been lagging behind in the United Kingdom for the past 10 years compared with the rest of Europe, and there is an urgent need to close the gap, says Dr Michael Rees. “Cardiology is one area where the UK was a world leader. Britain was, with the US, a leading exponent of clinical techniques,” he observes. “But while the US remains the most powerful country in cardiac research, Germany, Japan, France, and the Netherlands are now ahead of the UK.” Dr Rees says the governments of these countries pay more attention to developing and supporting structures for research.

“Clinical cardiology research is much less well organised and structured in the UK than it could be,” he says. “With fragmentation of the National Health Service (NHS) and lack of infrastructure investment from the universities, it has become less cutting-edge over the last 10 years. This is not due to a lack of bright people.” The fragmentation of the NHS has also decreased the ability of centres to coordinate and do clinical research.

Dr Rees says that electrophysiology research, basic science, and laboratory-based research continue to be excellent, but clinical research is falling behind, and a lot of developmental work in clinical cardiology techniques is now done outside the United Kingdom. The problem is being compounded by an apparent £300 million cut to health research funding.

The UK treasury has plans to create a new single fund of at least £1 billion for health research. But currently, health research is jointly funded by the Medical Research Council and the NHS Research and Development programme, which have an expected combined budget of £1.3 billion for 2007–2008.

“Paradoxically, the result of creating a single, if even smaller fund could be an increase in the amount of money that can be applied for,” says Dr Rees. “This result can be explained by the current lack of transparency in how research and development money is spent. The funding goes to hospitals and trusts, but it is unclear what proportion is actually spent on research. The new combined fund will be spent solely on research.”

In cardiology there is an urgent need to get research back on track. There has been a loss of 1000 medical academics over the past 5 years, with a particularly steep drop off in some specialities, including cardiology. As a result, a new scheme has been started to train medical academics. The Walport Training Initiative, undertaken by the government and the British Medical Association, aims to create 2000 posts during the next 5 years. These will include posts for juniors, lecturers, and senior lecturers, and in whole-time equivalents will amount to around 1000 posts.

“However, in the first round of applications there was a disappointingly low number of applications for research training posts in cardiology,” Dr Rees says. “One reason for this is that universities in general terms have not regarded clinical cardiac research as an area to invest in, in terms of infrastructure.” Over the last decade, investment in clinical, observational, and translational research has declined, and money has been spent on basic science and molecular research. A lack of coordination between universities and clinical training schemes, which were supposed to form joint applications, has resulted in the paucity of interest.

On top of this, fewer doctors are earning clinically related higher degrees, so there are fewer doctors, including cardiologists, interested in applied clinical and translational research. “Given the potential strength of research in cardiology, and the importance of the topic, I would have expected more applications,” says Dr Rees. “I hope a second round of applications, with a deadline of 12 October 2006, will have generated more responses.”

It is a national problem that the United Kingdom has not invested in or targeted enough money toward clinical and translational research. And, in addition, many advances in basic laboratory science are not translated to clinical work, but are sent to other countries. Major UK charities like the Wellcome Trust, based in London, could spend more on cardiology research, argues Dr Rees, rather than leaving the British Heart Foundation (also based in London) to shoulder the responsibility.

Another problem is that academic careers are unstable and less well-paid than clinical ones. Both of these factors are disincentives for doctors to take up cardiac research. Cardiologists can earn more as NHS consultants doing a bit of private practice than they can in research. But, Dr Rees says, “I think people would be happier earning a bit less if they had a more stable job. Life as a researcher abroad is much more stable.”

In mainland Europe, academic hospitals employ doctors to do both clinical work and research. By contrast, in the United Kingdom, the NHS has separated itself from the university structure, and doctors pursuing academic work have 2 contracts of employment. Dr Rees says there have been some moves to form academic hospitals along the continental model.

In another attempt to rectify the situation in England, the Department of Health has formed the National Institute for Health Research to gather researchers together in a more organised way. This involves identifying doctors in the NHS, then coordinating research between the NHS and the universities, and supporting doctors with time away from their clinical responsibilities.

Dr Rees predicts that, if the new structures being implemented in England are successful, the country’s clinical research, including cardiology, will return to world-leading status. “But while the future of cardiology research may look brighter for England, the devolved countries of Wales, Scotland, and Northern Ireland may have no additional funding in the new arrangements,” he says, “and there is no cardiac research network in Wales.”

Jennifer Taylor is a freelance medical writer.
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