From the Chair
Incidental finding of right atrial pacemaker lead thrombus
Scottish Meeting
A case of unexplained chest discomfort and ST elevation
Arrhythmia Challenge
Improving practice for Cardiac Physiologists with the integration of contemporary research
Situations Vacant
From the Chair

Modernising Scientific Careers
A meeting regarding the Modernising Scientific Careers (MSC) scientific training programme (STP) curriculum took place on the 29th July where the outline structure was agreed. A further meeting on curriculum took place on the 16th September where the content of the STP was discussed and preliminary content agreed, although there is still a lot of work to do. The practitioner training programme (PTP) training manual was also discussed and the importance of adhering to tight timelines emphasised, with a number of universities keen to implement both PTP and STP next year. Further meetings will take place in October and November of this year.

SCST Scientific Meeting returns to the BCS Conference for 2011
The SCST “physiologist day” is set to return to the British Cardiovascular Society (BCS) Conference for June 2011. A programme meeting relating to next year’s conference is to take place on the 30th September. It has been agreed that SCST will have autonomy in relation to the programme content for the “physiologist day” and to this end I ask for any last minute ideas regarding the programme to be forwarded through SCST administration (details on the website). We do need to know soon as the programme should be finalised in early Autumn.

Physiological Measurement Accreditation
The physiological measurement accreditation development process co-ordinated by the Department of Health is continuing. The drafting of the framework on the clinical domain began in earnest at the meeting on the 19th July 2010. The day proved to be effective and work continues on this section. A workshop to move towards finalising the non-clinical domains is to take place on the 28th and 29th September.

Editorial
This October edition of the Journal brings you a range of features with case studies from the world of pacing and electrocardiography, opportunity to test your ECG interpretation skills in the arrhythmia challenge and a review of current research from the Cardiac Physiologist’s perspective.

All written by Cardiac Physiologists for Cardiac Physiologists – send us your contributions before the 17th October via editor@scst.org.uk
A 73 year old male was admitted to the Emergency Assessment Unit due to jaundice and progressive worsening of shortness of breath. His history was complicated and included LV dysfunction with an ejection fraction of less than 15% due to myocardial infarction. He had an ICD in-situ, implanted in 2006. He also had chronic obstructive pulmonary disease and in 2009 had anterior resection for sigmoid carcinoma.

An urgent echocardiogram was arranged which showed a severely dilated heart with bi-ventricular failure. There was a large echogenic mass-like structure within the right atrium attached to the pacing lead (figure 1 and 2). The mass was noted to cause tricuspid inflow obstruction (mean tricuspid valve gradient recorded at 5mmHg) and severe bi-directional tricuspid regurgitation (figure 3).

The main problem was to find the cause of the mass: - was it due to thrombus formation or endocarditis? Echocardiography alone cannot differentiate between these two diagnostic alternatives. However the gentleman did not exhibit any symptoms of endocarditis and it was found at a later date that thrombus formation had been discovered on his pacing lead at a local tertiary centre during transoesophageal echocardiography back in 2007.

Central venous catheters and pacemaker electrodes are known to be predisposing factors for thrombus formation. Pacemaker lead thrombosis within the right atrium is a rare condition that is generally diagnosed as an incidental echocardiographic finding or by symptoms of right sided heart failure and obstruction or embolisation of the pulmonary artery.

Several mechanisms have been postulated in the pathogenesis of pacemaker induced thrombosis. First, the long-term residence of a permanent pacing lead may lead to endothelial injury and act as a thrombogenic nidus for the formation of thrombus. Pacing leads may produce a foreign-body-type reaction and subsequent inflammation and fibrosis along the wire which may lead to thrombus formation. Finally, congestive heart failure, hypercoagulable states such as antithrombin III, protein C and S deficiencies, thrombocytosis and malignancies may also lead to thrombus formation.

The treatment of right atrial thrombosis of pacing leads is controversial. The size and site of thrombosis and the duration of symptoms are the main determinants of treatment strategy. The options include medical therapy (antiplatelet therapy, anticoagulation and/or thrombolysis), surgical extraction, and percutaneous intervention.

References
1 Tuggu A et al. Right atrial pacemaker lead thrombosis causing tricuspid inflow obstruction. PACE 2009;32:262-4.
Registration and Lunch- 12.00-13.00

13.00 - The Repatriation of ICD patients to their local hospital - Alison Gilliland, Highly specialist Physiologist Devices - Ayr Hospital.

13.30 - Ambulatory 12 Lead ECG - Martin Hayes, Western General Edinburgh.

14.00 - Presentation on Grown Up Congenital Heart disease (GUCH) - Dr Niki Walker, Consultant cardiologist - Adult Congenital.

14.30 - ANNUAL GENERAL MEETING

15.15 - Clinical Physiology Degree – update – Catriona MacGregor, Clinical Coordinator CP degree, Glasgow Caledonian University.

15.30 - Head Up Tilt Testing - Dr Lara Mitchell-Cons Physician & Senior Lecturer, Southern General Hospital, Glasgow.

16.00 - Case studies

16.30 – 18.00 - Wine and Canapés and catch up with friends old and new.

Registration fee: £35 – Please make cheques payable to SCST Scottish Branch and forward to Joanne Cusack, Cardiac Dept, Stirling, Livilands, Stirling, FK8 2AU.

CLOSING DATE 10TH November 2010

Registration details

NAME:---------------------------------------------

Hospital Name:---------------------------------

Address---------------------------------------

Email address---------------------------------

Please include email address for correspondence
A 48 year old female was admitted to Accident and Emergency with episodes of central chest heaviness at rest. On admission, her ECG showed T wave inversion in leads V4 – V6 (Fig 1) and a 12 hour Troponin was normal.

The patient had recently been diagnosed with colorectal cancer which required surgery a few months previously. Prior to her admission to Accident and Emergency she had commenced her chemotherapy. Once she was admitted her chemotherapy was stopped. The doctor in Accident and Emergency then referred her for an exercise stress test, which showed ST elevation in the inferolateral leads. The patient requested to stop the treadmill after 4 minutes and 6 seconds because of chest discomfort and fatigue. At this time there was slight ST elevation in the inferolateral leads. (Fig 2). During recovery, ST elevation increased. Maximum elevation, 5 minutes into recovery, occurred in leads II, III, avF and V4 to V6 (Fig 3). The patient’s chest discomfort eased and was totally gone at 8 minutes. After 12 minutes the ST segments had returned to baseline. Her maximum heart rate was 146bpm (102% predicted maximum) and her maximum blood pressure was 140/80mm Hg.

The patient was then admitted to CCU to await urgent coronary angiography and further testing.

(continues on page 6)
A Case of Unexplained Chest Discomfort and ST Elevation

The patient underwent echocardiography to determine whether the ST elevation was associated with any wall motion abnormalities. The test showed inferior hypokinesis and a left ventricular ejection fraction of 55%.

A coronary angiogram was then performed in order to seek an explanation for the ST elevation. This showed normal coronary vessels and normal LV function (Fig 4a & b).

The investigations failed to identify a cardiac cause for the chest pain and the patient was referred back to the Oncology team to make a decision regarding ongoing chemotherapy.
A Case of Unexplained Chest Discomfort and ST Elevation

Figure 4a angiogram of the right coronary artery

Figure 4b angiogram of the left coronary arteries

Conclusion

This case illustrates that not all ST changes can easily be explained by conventional testing. Particularly surprising in this case is that there was no evidence on angiography of fixed coronary artery disease. The patient remains well.

Gerardin MacKin
Student Cardiac Physiologist
Belfast City Hospital

Arrhythmia Challenge – the solution

The ECG demonstrates sinus rhythm with 3:2 Wenckebach AV block and rate-dependent bundle branch block. Although the rhythm may look superficially like ventricular bigeminy, the typical Wenckebach sequence is evident from the relationship between the P waves (arrowed) and the QRS complexes. The reason that there are alternating QRS morphologies is that the longer RR interval, containing the non-conducted P wave, provides enough time for the bundle branch system to recover completely and conduct normally with a narrow QRS. The shorter of the two RR intervals leaves one of the bundle branches - probably the left – still refractory, hence the broad QRS.

I wish to thank Lyndsey Watson, senior cardiac physiologist at the University Hospital of North Tees, for providing me with this ECG.

Dave Richley
The North of England Cardiovascular Network
Improving Practice for Cardiac Physiologists with the Integration of Contemporary Research

‘Evidence based practice’ is the gold standard when considering the integration of new concepts and procedures into clinical practice. This is true for all healthcare professions including cardiac physiology. Carefully designed clinical trials inform of how a new technique or procedure may benefit current practice. The genesis of such trails is to be found in the primary clinical studies.

The focus of this and subsequent articles is to suggest how contemporary clinical research may be considered by cardiac physiologists in the improvement of their practice. In this article a range of current procedures employed by cardiac physiologists will be discussed with reference to contemporary publications.

- Surface electrocardiography and the ‘detection of postural long QT’
- Ambulatory blood pressure monitoring and ‘BP limits in type 2 diabetics’
- Cardiac rhythm device management and ‘remote monitoring in ICDs’
- Care pathways for the cardiac physiologist and ‘syncope’

Surface electrocardiography

It cannot be understated that electrocardiography in its many forms is the mainstay investigation for cardiac physiologists. The techniques of surface electrocardiography are well established in practice and it is relevantly rare for a new technique to be introduced. A recent report by Viskin\(^1\) may qualify as a rare occurrence. Viskin and his team have suggested that postural changes on the ECG may indicate long QT syndrome. Long QT syndrome is linked to sudden cardiac death and any technique which reliably improves its detection is worthy of mention.

Viskin and his team suggest that acquiring an ECG rhythm while a patient moves rapidly (from supine to a standing position) can demonstrate a blunted effect on the QT interval. The technique relies upon rapid heart rate acceleration or reflex tachycardia normally associated with this maneuver. With an increase in heart rate the QT interval should become shorter in respect to the shortening ‘R to R’ interval. However, this response is blunted in patients with suspected or established QT syndrome. The QTc becomes relatively longer, potentially indicating long QT syndrome.

The technique involves recording a rhythm strip and instructing the patient to remain in a supine position for 10 minutes; followed by a rapid movement to standing. The rhythm strip is then recorded for a further 5 minutes in the standing position. Typically this technique is included in Head-up Tilt Table Tests (HUTT). The team noted that in the long QT group the QT interval lengthens by 4ms and QTc by 50ms, compared to the normal decreased QT of 21ms and QTc of 89ms.

It is worth mentioning that the lack of a reflex tachycardia has been linked to disorders of the autonomic nervous system.


Ambulatory blood pressure

One of the most useful techniques for the directing the management of cardiac risk factors in hypertension is the 24 hour ambulatory blood pressure monitor (ABPM). The ABPM allows healthcare professionals to effectively utilise anti-hypertension therapies. The 24 hour ABPM has been reported as being superior to other blood pressure measurement methods for detecting and managing hypertension.

In the effective management of hypertensive patients and those with comorbidities, various professional organisations have stated the optimum blood pressure values to be achieved. Debate continues on the optimum blood pressure value for type II diabetic patients. Previous practice considered that to improve risk management the ‘lower the blood pressure the better’. However the message from the ‘ACCORD BP’ (Action to Control Cardiovascular Risk in Diabetes Blood Pressure) trial is that there is no evidence to support that lowering blood pressure below 130mmHg\(^3\) will have long term benefits; and to avoid excessive anti-hypertensive treatment of type II diabetics\(^{1,2}\).

From the physiologist’s perspective the interpretation of an ABPM from a type II diabetic should consider if the patient’s treatment is optimal. The physiologist should not underestimate the important of the accurate acquisition and reporting of a 24 hour ABPM. These skills are vital to the management of the patient. All reports should be tailored to the patient’s pathology and management.

3. Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure, December 2003
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Cardiac rhythm device management

The decision between the utilisation of remote monitoring or outpatient clinic follow-up for our cardiac rhythm device patients rests upon the patient’s pathology and the need for intensive management. The benefits that remote monitoring brings to reducing outpatient visits have been described extensively in the appropriate literature. Under general debate is whether remote monitoring is more suited to cases with potentially no urgent management issues or conversely whether it could be used to effectively manage complex cases.

From the physiologist’s perspective, is there evidence to demonstrate that remote monitoring can be used safely in the latter, and also allow the physiologist to quickly act in the event of an emergency?

The CONNECT (Clinical Evaluation of Remote Notification to Reduce Time to Clinical Decision) trial demonstrates that remote monitoring offers a significant improvement on current ‘times to act’ in the United States. The study assessed the time taken from the occurrence of an adverse event (such as arrhythmia and potential device malfunction) to a decision on the patient’s management. The technology utilised in this study was the Medtronic ICD with the Conexus telemetry system and the Carelink network.

The average ‘time to act’ was reported as 29.5 days in the remote monitoring group. The ‘event-to-decision’ time was 22 days in the outpatient to 4.6 days in the remote group. Although for ICD shocks there were generally no differences between the groups.

This trail clearly adds weight to the discussion to use remote monitoring in our clinics. It would be interesting to compare the stated outpatient follow-up times to UK follow-up times. How quickly do you see patients following adverse events?


Care pathways

The relationship between the cardiac physiologist and syncope is as synonymous as fish and chips. The cardiac physiologist is typically one of the first healthcare professionals to assess the syncopal patient on first presentation and the subsequent initiation of a care pathway. The involvement of the cardiac physiologist will continue through to invention and post-intervention care. The initial steps of any care pathway require the accurate diagnosis of the pathology.

It is common sense for all cardiac physiologists to have working knowledge of ‘how to identify syncope’ and to avoid inappropriate referrals or a missed opportunity for diagnosis. The patient and referring healthcare professional may be unaware that syncope may be a potential diagnosis. The cardiac physiologist has an opportunity to clarify the clinical picture with a series of brief questions and investigations.

A recent article by Fay published in the British Journal of Cardiology outlines simple but effective steps in the initial diagnosis of the syncopal patient. All cardiac physiologists should be mindful of the first seven steps.

Step 1 – During consultation with the patient the cardiac physiologist may be informed of terms such as fainted or fallen. These terms may be heard during any investigation from routine 12 lead ECG to echocardiography. Do not assume that because the patient has been referred for an investigation that the diagnosis is complete. The cardiac physiologist should be suspicious of these terms, suspect syncope and consider exploring the patient’s history further. Ask the patient to describe the event fully from the point just before and after when fully recovered.

Step 2 – Does the event description sound syncopal? The difference between syncope and non-syncope is difficult. As a guide a fall due to epilepsy will demonstrate tonic-clonic movements for >15 seconds, cerebral hypoxia demonstrates short duration irregular movements of distal limbs, etc. Some falls will not cause the patient to lose consciousness, e.g. vertigo. Syncope generally does not display these characteristics.

Step 3 – If possible ask a witness to describe the event. Ask the same situational questions as the patient received.

Step 4 – Ask for a brief clinical history to put the event into context. Has the event happened before? For instance valvular heart disease can cause syncope.

Step 5 – Ask for a family history to put the event into context. Is there a family history of syncope, cardiac pathology, or unexplained deaths?

Step 6 – Undertake a clinical examination (CE) within the remit of a cardiac physiologist. CEs are the remit of a physician but the cardiac physiologist may undertake some investigations to clarify the clinical picture. Consider the following:

1. Take a pulse and examine for rhythm and rate (this will provide additional information when combined with an ECG)

2. Record a 12 lead ECG to assess for all rhythm and structural abnormalities

3. Record a non-invasive blood pressure in supine and standing position, ideally measure after 10 minutes of supine, then on immediate standing and 2 mins after. Look for a systolic of <90mmHg or a fall of 20mmHg, or a diastolic fall of 10mmHg. This is strongly suggestive of orthostatic hypotension. Also consider the QT interval.

4. The physician would continue with a more detailed clinical examination

Step 7 – Consider further investigations within the remit of a cardiac physiologist. Consider referral for; Echocardiogram for the assessment of ECG structural abnormalities or a positive family history; Ambulatory ECG monitoring for assessment of short term or medium term event frequencies; or HUTT. The physician would also consider referral for any investigations, e.g. blood profiles

If the cardiac physiologist suspects that a patient may have undocumented or undiagnosed syncope, referral to a physician is recommended. The acquisition of the 12 lead ECG is the one of the most effective investigations that can be performed prior to referral. Close communication between the physiologist and the physician is essential for the management of the patient.


For further discussion please contact the author at pmlewis@glam.ac.uk. All enquires welcome, particularly from students or registered cardiac physiologists considering evidence based practice research.

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For an informal discussion please contact Christine Pope, Senior Chief Cardiac Physiologist on 01271 322475.

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