Abstracts: 46th St Andrew’s Day Festival symposium: updates on acute and internal medicine

**ABBREVIATIONS** Computerised tomography (CT), intensive therapy unit (ITU), sub-arachnoid haemorrhage (SAH), cerebrospinal fluid (CSF), British Thoracic Society (BTS), electrocardiogram (ECG), Faculty Research Interests Science Comparator (FRISC)

**DAY I**

**SESSION 1**

Chair: Dr M Strachan, Consultant Physician, Western General Hospital, Edinburgh

**Syncope, seizures and pseudoseizures**

Dr R Duncan, Consultant Neurologist, Institute of Neurological Sciences, Southern General Hospital, Glasgow

**Email** r.duncan@clinmed.gla.ac.uk

**Abstract**

**Background** The differential diagnosis of epileptic seizure is wide, but when a patient presents with an attack disorder, the most common distinctions that need to be made are between vasovagal syncope and seizures, and between epileptic seizures and pseudoseizures.

**Methods or Theme** Factors in the background of the patient can help determine why a patient has a particular disorder: for example, a past head injury may be a cause for epilepsy. However, they are less useful in the diagnosis itself, and may even mislead. Eliciting a history of triggers for attacks is crucial in making a diagnosis of vasovagal syncope, as is eliciting a history of a typical syncope prodrome. Cardiac syncope uncommonly presents as epilepsy, but attacks triggered by exercise should ring cardiac alarm bells. In the distinction between seizure and pseudoseizure, accurate history from the patient of their experience of the attack, and from the eyewitness are crucial, and in particular, accurate description of movements during the attacks (tremor vs jerks).

A standard EEG is of some diagnostic value when the history indicates a high probability, but not certainty, of epilepsy. Where the history is unlike epilepsy, positive predictive value is low, and false positives will result. Techniques that record attacks, such as ambulatory EEG monitoring, short outpatient video EEG, and inpatient vide EEG, are usually diagnostic if attacks can be recorded.

**Conclusions** The diagnosis of attack disorders is based on clinical history. Most oddities are unusual presentations of common conditions.

**Sponsors** None.

**Declaration** No conflict of interest declared.

**Acute headache: who needs investigation, which ones, and when?**

Dr RJ Davenport, Consultant Neurologist, Department of Clinical Neurosciences, Western General Hospital, Edinburgh

**Email** rjd@skull.dcn.ed.ac.uk

**Abstract**

**Background** Most patients presenting with sudden, severe headache will have a benign primary headache diagnosis such as migraine or thunderclap, but about 10–25% will have serious underlying pathology (secondary headache syndrome), most commonly SAH. In an ideal world, all such patients would be assessed by neurologists, but, in the UK at least, this is not currently feasible. Thus general physicians need to know who to investigate, how, and when.

**Methods or Theme** Diagnosis begins with an accurate history, and it is essential to differentiate truly abrupt onset headache (maximal immediately or within minutes at the most), from an evolving headache, which is rarely, if ever, due to SAH. Associated symptoms, such as vomiting, neck stiffness, transient disturbance of consciousness/seizures, and focal neurology, are insufficient to accurately differentiate primary from secondary syndromes. All patients with a truly abrupt onset headache therefore require investigation.

Unenhanced CT brain scanning is the investigation of choice, and performed promptly (within 48 hours of ictus), and interpreted correctly, is very accurate for SAH, with a false negative rate of less than 5%. The longer the delay from ictus to scan, the greater the false negative rate. All patients with an abrupt onset headache and a ‘negative’ CT require a lumbar puncture, which should ideally be postponed for at least nine
hours after the ictus, and performed by an experienced operator. Correct interpretation is vital. Confirmed SAH patients should then proceed to vascular imaging (CT angiography). Other serious intracranial pathology (e.g. cerebral venous thrombosis, pituitary apoplexy, arterial dissection) may present with abrupt headache, mimicking SAH.

Conclusions Regarding sudden severe headache:

- History allows identification of who requires investigation and who does not, but does not allow the distinction of primary from secondary headache syndromes.
- A normal CT brain scan does not exclude SAH (and other serious pathology).
- CSF examination is still required in ‘negative’ CT scan cases, and interpretation may not be straightforward.
- Sudden severe headache may be due to serious intracranial pathology other than SAH.

References

Key words Subarachnoid haemorrhage, sudden headache.

Sponsors None.

Declaration No conflict of interest declared.

SESSION 2

Chair: Dr A Howie, Consultant Physician, Stirling Royal Infirmary

Investigation and management of neutropenia

Dr PRE Johnson, Department of Haematology, Western General Hospital, Edinburgh

Email Peter.johnson@luht.scot.nhs.uk

Abstract

Background Neutropenia is a frequently encountered blood abnormality in clinical practice. The bone marrow produces one million neutrophils every second, and the process of neutrophil production including cell division and differentiation was presented. The definition of a normal range encompassing two standard deviations either side of the median was discussed along with the importance of ethnic variations affecting normal range. The principal causes of neutropenia include drug effects, immune mechanisms and bone marrow disorders. The principles of managing neutropenia include assessment of severity, precipitating factors, general measures and specific measures. A special strategy is required for the management of severe neutropenia in patients receiving chemotherapy.

Key words Neutropenia, normal range, drug effects, immune mechanisms, bone marrow disorders, chemotherapy.

Sponsors None.

Declaration No conflict of interest declared.

AL-HAMMADI LECTURE

Chair: Professor N Douglas, President, Royal College of Physicians of Edinburgh

Epilepsy in acute medicine - acute asthma

Professor MR Partridge, Professor of Respiratory Medicine, NHLI Division, Imperial College London

Email m.partridge@imperial.ac.uk

Abstract

Background Approximately half of those with out-of-control asthma attending Emergency Departments in the UK are adults, and half are children. Many who are admitted to hospital have clear evidence of severe asthma, but it is not necessarily acute. Lesser exacerbations of asthma are similarly more often of gradual onset, and for many, time is available for the patient, or the patient with health professional help, to alter therapy to prevent themselves deteriorating to the point that they need urgent medical care. A quarter of those who require mechanical ventilation for severe asthma have been shown to be deteriorating for more than three weeks before admission to hospital.

Self management education is therefore of vital and proven importance, and is the approach recommended in the BTS/SIGN British Asthma Guidelines. For those who do nevertheless require hospital care it is important that treatment is prompt, its effect monitored, and that, upon recovery the opportunity is taken to explore the circumstances surrounding the deterioration in an attempt to prevent recurrence.

The Emergency Management of Asthma.

A checklist for use after an Emergency attendance or admission because of asthma:

(Note that every episode of severe asthma represents a potential failure of our previous management).
• Was the patient’s inhaler technique satisfactory?
• Prior to the attack were they on, and were they taking, sufficient preventive therapy?
• Was there an avoidable precipitating cause, e.g., aspirin use, alcohol, allergen exposure or occupational cause?
• Was this a genuine, sudden, severe (brittle) attack and do they need to be taught the special first aid measures needed by this group?
• Is the patient a poor perceiver of severity?
• Did the patient react appropriately to the impending attack, and did they have a written personal asthma action plan?

References

Key words Acute asthma, British Asthma Guidelines.

Sponsors None.

Declaration No conflict of interest declared.

SESSION 3

Chair: Professor T MacDonald, Professor of Pharmacology, University of Dundee

Lifestyle drugs: the final frontier of therapeutics?

Dr S Maxwell, Clinical Pharmacology Unit, University of Edinburgh, Queen’s Medical Research Institute, Royal Infirmary of Edinburgh, Edinburgh

Email s.maxwell@ed.ac.uk

Abstract

Background The exact definition of a ‘lifestyle’ drug is unclear, but the term is usually applied to an expanding number of drugs that are used to achieve non-health-related goals. However, there remains considerable debate about what constitutes merely a lifestyle goal rather than a true health gain. This new area of drug usage is estimated to be worth around US$30 billion annually. Examples of agents that are often considered as lifestyle drugs include sildenafil (impotence), orlistat and sibutramine (weight loss), bupropion (smoking cessation), minoxidil (hair loss) and oral contraceptives. Further agents are likely to be targeted at enhancing our mental capacities, mood and social interactions. A closely related phenomenon is ‘disease-mongering’, which involves redefining life experiences as abnormal and unhealthy, with the aim of expanding indications and use of lifestyle drugs. The rapidly increasing availability of lifestyle drugs presents a major challenge to the utilisation of health-related resources and traditional routes to accessing medicines. Opinions about whether this is a positive trend and how best to manage these challenges vary and depend upon our definition of disease, our view concerning the role of the National Health Service, and concerns about a significant expansion of drug use in society.

Key Words ‘Lifestyle’ drug, sildenafil (impotence), orlistat and sibutramine (weight loss), bupropion (smoking cessation), minoxidil (hair loss), oral contraceptives.

Sponsors None.

Declaration No conflict of interest declared.
**CME**

References


**Abstract**

**Background** Management varies widely, in the UK and elsewhere, among patients presenting to hospital with suspected acute coronary syndrome and a modest troponin elevation. Based on post hoc data from randomised studies, the benefits of intervention were seen among higher risk patients, including those with troponin elevation. However, newer higher sensitivity troponin assays are now available, and have been widely implemented. These can detect very minor troponin elevations (e.g. 0.03ng/ml of troponin I). Is this sufficient to identify higher risk patients for more aggressive pharmacological and interventional therapy?

**Methods or Theme** Prospective registries have been conducted (GRACE, CRUSADE and others) and these have demonstrated the importance of a more comprehensive risk assessment beyond that based on troponin alone.

21,688 patients from the GRACE registry were used to derive a prospective risk score for the whole spectrum of acute coronary syndrome. The score was validated in the subsequent 22,122 patients and also validated externally in the GUSTO IIb dataset of 12,142 patients. The C-statistic for death was 0.81 and for death or myocardial infarction was 0.73. Independent studies have demonstrated the risk prediction of the GRACE score to be superior to that of the TIMI risk score or the PURSUIT score (Goncalves, European Heart Journal 2005; 26:865–72).

The five year results of interventional studies have demonstrated that patients in the moderate or higher risk categories have the most to gain at 1 year, and at 5 years (RITA 3 study, FRISC II study and ICTUS study).

**Conclusions** Binary decisions based on troponin alone are inadequate. A more systematic approach to assessing risk (using a simple risk prediction tool) provides more robust identification of patients for interventional treatment and for more aggressive pharmacological treatment.

**References**


when a cerebral artery is occluded by clot, the blood flow to the brain supplied by that artery may drop to a critical level so that neuronal function ceases and the patient develops the symptoms of a stroke. If the artery can be re-opened within a few hours. then brain tissue which would otherwise have died may be salvaged, resulting in a less severe stroke deficit and improved functional outcome. Thrombolysis with tissue plasminogen activator given within three hours of stroke onset improves stroke outcomes. It is less clear whether thrombolysis is effective if given later after the stroke onset and whether other methods of re-opening vessels are effective. Ongoing studies aim to refine the decision making about who should be thrombolysed, when and how.

Conclusions Few patients with stroke in the UK currently receive thrombolyis. The challenge for the NHS is to make patients aware of the importance of presenting early with the symptoms of stroke and then to provide services to deliver thrombolytic therapy. This has to involve collaboration between primary and secondary care, stroke specialists and other specialists working in emergency medicine services.

Key Words Stroke, thrombolysis tissue plasminogen activator.

Sponsors None.

Declaration No conflict of interest to declare.

SESSION 4

Acute liver failure — alcoholic hepatitis: a challenge for UK medicine in the 21st century

Professor JP Iredale, The MRC Centre for Inflammation Research, The Queen’s Medical Research Institute, Edinburgh

Email John.Iredale@ed.ac.uk

Abstract

Background Whilst there are many causes of acute liver failure, changes in the pattern of drinking, increased availability of cheap alcohol and an apparent absence of cultural constraints on excessive consumption means that the predominant form of acute liver disease seen by general physicians is, and will be, alcoholic hepatitis. Alcoholic hepatitis is a challenge to manage because it is, in effect, an acute-on-chronic liver injury. Alcoholic hepatitis is a severe form of liver disease with in hospital mortalities in excess of 50% in some series. With careful attention to detail and scrupulous management with respect to the presence of infection and treatment of complications, this mortality can be brought down, although it may still be as high as 20%.

Specific treatments for alcoholic hepatitis include immunosuppression and TNFα antagonists. These approaches will be reviewed, as will specific aspects of management of the acute features of alcoholic hepatitis.

Key Words Acute liver failure, alcoholic hepatitis, immunosuppression, TNFα antagonists.

Sponsors None.

Declaration Professor Iredale has worked as a Consultant for GEC Healthcare on imaging strategies for hepatic fibrosis.