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JRCALC
JOINT ROYAL COLLEGES AMBULANCE LIAISON COMMITTEE

ASA

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Introductory message from the NCEP Manager

AMBEX is fast approaching and much preparation is being made to ensure that the Clinical Effectiveness Committee is represented. This year there will be an exhibition stand providing an opportunity to speak about the work undertaken by the Committee and also to speak about the future work plan.

The Committee are looking forward to this event and I am hoping that myself and other members will have the opportunity to meet with you, so do take a few moments to seek us out for an informal chat. Tea/Coffee and some light refreshments will be available.

We will be located at stand Q51 in the main exhibition hall.

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The Cochrane Collaboration – A Pre-hospital field?

The Cochrane Collaboration, developed by Archie Cochrane receives world-wide recognition for the high quality systematic reviews that it conducts to provide users with the best available research evidence.

Melbourne, Australia, recently saw a meeting take place to examine the feasibility of developing a pre-hospital field to the Cochrane Collaboration. Representatives from Scandinavia, UK, America, Australia and Asia were present to discuss the benefits of developing such a field and to agree a proposal to commence the formal process of applying to Cochrane to develop this field. Cochrane representatives were also there and spoke about the processes involved in developing such a field.

So what does this mean to pre-hospital care?

We are all probably aware of the dearth of pre-hospital research currently available and subsequent lack of systematic reviews of such evidence. If the application to develop a pre-hospital field is successful, then a dedicated facility will be available to help address these concerns and hopefully generate research interest into pre-hospital care.

A pre-hospital Cochrane field will have the skills and ability to systematically review all of the available evidence around any particular heath care

area/intervention associated with pre-hospital care in order that users are provided with the highest quality and best available evidence.

Further details will be appearing on the ASA Clinical Effectiveness website shortly so visit www.asancep.org.uk for further information.

Thrombolysis

The introduction of paramedic led pre-hospital thrombolysis has been making steady progress and in excess of 200 patients have now received pre-hospital thrombolysis.

Below are detailed the five different models approved by JRCALC for the provision of pre-hospital thrombolysis and also details about which Ambulance Trusts are currently providing thrombolysis and which thrombolytic agent is used. This information was obtained as a result of a survey conducted in March 2003. A regular feature of Candour will now be a bi-monthly update of progress made.

The Five Models that may be adopted by Ambulance Trusts for thrombolysis are:-

1.	Recognition of eligibility for thrombolysis by paramedics (based on clinical features and the ECG). No transmission of data but the A/E department or CCU will be alerted.
2.	As above (Model 1), but in addition with transmission of clinical information and the ECG so that the hospital has all relevant details for clinical decision before patient's arrival.
3.	As above (Model 2), but with direct contact with a physician who might then authorise the prehospital administration of a thrombolytic drug on a named patient basis.
4.	Co-operation in rural areas with primary care physicians who will administer the agents in the patient's home, within the ambulance, or in local community hospitals before transfer to a DGH.
5.	Administration of thrombolytics by trained paramedics acting autonomously, either using streptokinase (available under the PMO arrangements) or reteplase using Patient Group Directions.

Thrombolysis Update

There are currently 10 trusts in the UK that are trained and equipped to provide pre-hospital thrombolysis. These are as follows:

Ambulance Service	Number of Patients Thrombolysed	Agent Used
Cumbria	0	TNK
Dorset	21	TNK
East Midlands	33	rPA
Essex	20	TNK
Hereford and Worcester	22	TNK
Lincolnshire	4	rPA
Scotland	90 (GP + Para schemes)	TNK
Staffordshire	44	rPA
Warwickshire	1	TNK
Westcountry	8 (not autonomous)	TNK

Total 243

If any Ambulance Trust is considering providing pre-hospital thrombolysis and would like any additional information regarding evidence/education or any other issues please don't hesitate to contact the NCEP Manager mark@bizuk.com

Use of Blue Calls for Ambulance Service Patients with Acute Coronary Syndromes

In April 2001, the London Ambulance Service NHS Trust's (LAS) treatment protocol for chest pain was revised to advise crews to place a blue call to inform the receiving A&E department of estimated time of arrival when conveying patients with a suspected Acute Coronary Syndrome (ACS). A snapshot audit of crews' compliance with submitting these blue calls was conducted.

All Patient Report Forms (PRFs) that were completed during October 2001 and reported chest pain as the chief complaint were clinically reviewed by a LAS paramedic. A sample of 100 consecutive PRFs that identified suspected cardiac chest pain were selected and retrospectively analysed to determine whether a blue call had been placed. The target compliance level was 100%, with no exceptions.

When the blue call was used, the average time taken to convey the patient to hospital was 6.4 minutes, as opposed to 13.5 minutes when no blue call was submitted. The use of the blue call was not determined by the patient's age, gender, time of day or geographical location within Greater London. Unfortunately there was only a compliance rate of 48%.

The use of the blue call resulted in patients arriving at hospital on average seven minutes earlier than if no blue call was placed. It is well known that early treatment and a shorter time to definitive care for patients with an Acute Myocardial Infarction is associated with an increased chance of survival and more favourable prognosis. As such, the blue call should, be expected to be associated with a more positive outcome. Although only 48% of PRFs reported blue call compliance, this does not necessarily mean that a blue call was not placed – it may be a reflection of poor PRF documentation. Future audit needs to incorporate other means of assessing whether a blue call was made, and compliance with the protocol must be improved using means of communication and training.

Further details regarding this study available from Dr R Donohoe, London Ambulance Service.

Frusemide and Suspected Pulmonary Oedema

Pulmonary oedema caused by left ventricular failure e.g. in acute myocardial infarction or as part of the picture of congestive cardiac failure is distressing and dangerous. Emergency treatment is required and should begin if at all possible in the pre-hospital period. Successful treatment saves lives and is one of the most rewarding situations in modern cardiology as the patient's condition may improve rapidly. Whatever the cause of pulmonary oedema an abnormally high left atrial pressure is the principal problem, whether or not accompanied by congestion (the evidence of high central venous pressure and peripheral oedema). It follows that treatment aimed at reducing left atrial pressure can be very valuable in addition to supportive treatment which may reduce the degree of congestion. In the pre-hospital setting this is the most important goal.

Frusemide is a powerful loop diuretic which produces an increase in urine output. However in the pre-hospital setting it is other effects that which may be most valuable to the patient. Intravenous frusemide also causes a rapid increase in venous capacitance and a decline cardiac filling pressures[1]. The effect is dependant on having functioning kidneys and is believed to be mediated by Renin release in response to the intravenous frusemide bolus. The beneficial effect on venous capacitance is then caused Renin dependent angiotensin II actions. The same renin angiotensin II can cause arterial vasoconstriction and increase afterload which is potentially harmful to cardiac function. Afterload is generally already increased in patients with pulmonary oedema so the beneficial effect on preload with its subsequent lowering of pulmonary artery and left atrial pressure is the dominant effect in the pre-hospital setting. Importantly the improvements in cardiac haemodynamics follow from small doses intravenously and a 20mg intravenous frusemide dose[2] will achieve much of the benefit although the diuretic effect is dose dependent and will be helped by larger doses. Other specific effects on the lungs are also claimed from experimental models.

It follows from the above that there is much that can be done in the pre-hospital setting to treat pulmonary oedema.

Treatments with direct effect upon pulmonary oedema:

- Buccal GTN has a powerful effect on reducing preload and left atrial pressure as well as reducing afterload (particularly valuable in the hypertensive patient).
- Frusemide IV has a beneficial effect on preload and left atrial pressure.
- Diamorphine (when available) reduces pulmonary artery and left atrial pressure.

Treatments with benefit in pulmonary oedema not directly related to preload or left atrial pressure:

- Oxygen to improve tissue oxygenation, cardiac function.
- Analgesia reducing catecholamine release.
- Relief of cardiac ischaemia e.g. buccal GTN.

It follows that intravenous frusemide is of value in the pre-hospital treatment of suspected left ventricular failure but should be seen as one of several interventions that will benefit the patient. It should be used alongside the other treatments above and not in place of e.g. buccal GTN which will add to the power of your treatment.

References:

1. Dikshit K, Vyden J.K, Forester J.S. et al. Renal and extrarenal haemodynamics effects of furosemide in congestive cardiac failure after acute myocardial infarction. New England Journal of Medicine. 288: 1073,1973.
2. Johnston G.D, Nicholls D.P, Leahey W.J, et al. The dose response characteristics of furosemide in normal subjects. British Journal of Clinical Pharmacology. 18:75,1984.

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