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Steam inhalation therapy

I think the conclusion of the article *Steam inhalation therapy: severe scalds as an adverse side effect*[1] is excessively restrictive.

I do not know how 'steam inhalation therapy' is administered in the Netherlands, but I know practice in Britain has changed in the last four decades. We no longer use Nelson inhalers.

Many patients inhale over a washing-up bowl of boiling water, which brings in risks of transporting water from kettle to bowl to accessible table. I recommend either the use of a mug-full of boiling water, or the less-risky 'hot beverage', that certainly appears to reduce the risks in handling and in the total quantity of thermal energy if there is a spill. Alternatively, I recommend 'steaming' in a bath or shower of normal bathing temperature, this is substantially less than boiling, and should not induce more scalds than the ordinary weekly ablutions.

I disagree with the article's conclusion that there is no evidence of therapeutic benefit. There is a huge amount of anecdotal evidence for its therapeutic efficacy, in ENT and chest medicine in hospital as well as in general practice. I have never seen a scald from steaming; I have seen many from hot drinks: should we ban drinking?

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Over-reliance of D-dimer in isolation to exclude venous thrombosis should be avoided

The shift to primary care expected in the initial 'diagnostic' management of cases of venous thromboembolism is indeed welcome. A recent article in the *BJGP* highlighted the role of D-dimer in reducing referrals for radiological imaging.[1] However, one of the messages that needs to be stressed in this context is the importance of clinical probability scoring system. It is important that the primary care physicians do not over-rely on the D-dimer, and clinical evaluation should be considered as the first step. Reliability on the D-dimer in isolation can have problems especially since there is evidence in the literature for thromboembolic episodes occurring in the context of normal D-dimer.[2,3]

There are several possible explanations for a normal D-dimer even in the presence of venous thromboembolism. The levels of D-dimer increase in the circulation due to the breakdown of the fibrin-bound clots. Very often, individuals present with symptoms of lower limb thrombosis many days after the onset of symptoms. The clot breakdown in these cases may have ceased by the time they arrive for medical attention and the result would be a normal D-dimer. Second, in the patients who receive anticoagulation treatment sometimes before the hospital assessment is undertaken (patients who have problems with transport, or from the hospice, started on anticoagulation empirically), inhibition of clot lysis can cause normal D-dimer. This phenomenon has been noted to occur within 24 hours after receiving heparin therapy.[4] It is also important to bear in mind that a normal cut-off of D-dimer is arbitrary and may not be applicable to every individual, since the clot-breakdown capacity varies between individuals. This is exemplified by the report in pregnancy of deep vein thrombosis and normal D-dimer.[3] Last, there is the issue of wide variability between many different D-dimer assays.[5] Each caregiver should take into consideration the appropriate cut-off suited for the assay and setting before they can attribute a level useful in exclusion of thrombosis.

In summary, there is no alternative to good clinical assessment in the exclusion of venous thromboembolism and D-dimer level is only a useful adjunct.

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DOI: 10.3399/bjgp12X658188

Eosinophilic oesophagitis: a clinical update

I would like to thank you for the recent clinical intelligence article on eosinophilic oesophagitis.[1] As a current GPVTS working in ENT I found this clinical update very informative and relevant to my work. Interestingly only a few days after reading this article we admitted a 17-year-old young man complaining of a food bolus sensation following eating chicken earlier in the day. He was normally fit and well, and of note did not suffer with any atopic conditions. He was managed initially with medical therapy, however, after some initial improvement his symptoms deteriorated and the time between consumption and regurgitation of water progressively shortened.

A gastrograffin swallow was arranged, by which stage the patient was struggling to swallow his saliva. Gastromiro was used and the procedure identified almost complete obstruction at approximately the distal one-third of the oesophagus. Gastromiro is a water-soluble contrast agent and was chosen because it is easier to swallow than barium, and is non-toxic. An urgent OGD identified retained food and fluid in the oesophagus. Linear furrows and concentric ring constrictions were noted in the oesophagus at OGD. Multiple random biopsies were taken and had been found to be diffusely infiltrated with eosinophils, consistent with a diagnosis of eosinophilic oesophagitis.

This article and subsequent case have significantly increased my awareness of this condition and brought it to the forefront of my mind when considering differential diagnoses of food boluses.

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Liquid nitrogen for cryotherapy

The West Lothian Community Health and Care Partnership along with the local pharmacy department have unilaterally decided that they will no longer supply practices with liquid nitrogen for cryotherapy. The liquid nitrogen had been supplied for over 20 years to our practice. They cite potential health and safety risks in the transport and use of liquid nitrogen. This seems another example of health and safety being used inappropriately.

They say 'there have been a number of recent incidents regarding the spillage and evaporation of liquid nitrogen during the transportation process and local storage at health centres,' but do not detail the incidents.

They allude to 'well documented hazards associated with liquid nitrogen including oxygen deficiency (spillages and venting/leaking dewars), asphyxia, damage to lungs, and cold burns, confined space exacerbation, oxygen enrichment, and ice plugs'.

Apparently 'The NHS Lothian policy prohibits transportation by car and states it is to be done only by a suitable transport vehicle. This is a vehicle with cab separate to the cab, that is, a box van. The storage facility for the flasks requires further upgrading to meet requirements of the policy and procedure — and as a result of a recent audit in relation to carriage of dangerous goods. Following the recent incidents, transport have reviewed their system and highlighted that local controls are not adequate'.

They also state 'the current facilities in our health centres and treatment rooms do not comply with the ventilation requirements — estimated cost of £1200 for ventilation to be installed for each room and appropriate alarm system. There is no standard operational procedure in place for reception of liquid nitrogen in the health centres'.

The volume that practices receive was around 500 ml, given the volume of a reception area or a consulting room is probably about 32 000 l (4m x 4m x 3m), then the volume of liquid nitrogen if a gas = 700 x 0.5 l = 350 l. Air is approximately 80% nitrogen therefore the volume of nitrogen in the room prior to any nitrogen spill 80% x 32 000 l = 25 600. After the spill of 500 ml of liquid nitrogen this increases the volume to 25 950 l, which is an increase in nitrogen concentration of less than 1%.

The whole scenario seems bizarre, liquid nitrogen historically is the chosen modality of most dermatologists for cryotherapy. Other freezing agents exist but may not be as efficient.

The main driver seems to be cost, however practices can obtain Histofreezer® 150 ml dimethyl ether/propane/iso butane aerosol on stock order in Scotland. This costs approximately £50 for 50 applications. Histofreezer reaches a temperature of –55°C within 15 seconds. There seem to be very few papers on its use, however one from Madrid[1] suggests it may be useful and I would be interested to hear of other GPs' experiences.

However I feel the withdrawal of liquid nitrogen will result in more referrals to dermatologists.

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DOI: 10.3399/bjgp12X658214

Confirming death in general practice

Kelso *et al* from Dumfries & Galloway raise the issue of 'confirming death in general practice'.[1] This is an area that is inadequately addressed in undergraduate and postgraduate training. Similarly the certification process following confirmation of death is similarly neglected. There is often a difference between confirming death at a patient's home and in a hospital as a hospital doctor is working in an acute setting where death may be sudden, with the need for a decision whether or not resuscitation is required and appropriate. Furthermore, in general practice, nurses are being trained in some parts of the UK to confirm death out-of-hours.

Every few months there is a global report in a newspaper of a patient 'waking up' in a mortuary where death has been mistakenly confirmed in circumstances of conditions that can induce coma. This led to my writing an editorial in the *BMJ* in 1996 on the subject of 'diagnosing death' and similarly concluded that this is a subject rarely mentioned in modern textbooks, although much is written about pronouncing brain death. In this editorial I provided guidelines for practitioners, as for many this process has fallen into a 'commonplace formality'.[2,3] In my days as a hospital doctor and a GP there was often considerable doubt about the actual moment of death, particularly for those witnessing the process of dying, as the warmth of the body and the long unnerving intervals between respiratory gasps can be misleading.

The authors ask how the process may be improved. Perhaps consideration could be made for this to be a mandatory Direct Observation of Procedural Skills (DOPS) for both Foundation Doctors and Associates in Training for General Practice based on the 2008 Academy of Royal Colleges code of practice for confirmation of death.[4]

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