"Drink plenty of fluids"

A systematic review of evidence for this recommendation in acute respiratory infections

Guppy, Michelle P B; Mickan, Sharon M; Del Mar, Chris B

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Michelle P. B. Guppy
University of Queensland

Sharon M. Mickan
University of Queensland

Chris Del Mar
Bond University, chris_del_mar@bond.edu.au

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Michelle P B Guppy, Sharon M Mickan and Chris B Del Mar

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Michelle P B Guppy, Sharon M Mickan, Chris B Del Mar

Doctors often recommend drinking extra fluids to patients with respiratory infections. Theoretical benefits for this advice are replacing insensible fluid losses from fever and respiratory tract evaporation, correcting dehydration from reduced intake, and reducing the viscosity of mucus.[1 2] To many this advice is self evident and justified on the basis that even if the benefit is uncertain, or at best small, at least it is harmless.

However, there are theoretical reasons for increased fluid intake to cause harm. Antidiuretic hormone conserves fluid by stimulating water reabsorption from the renal collecting ducts. Increased antidiuretic hormone secretion has been reported in adults and children with lower respiratory tract infections of bronchitis, bronchiolitis, and pneumonia of viral and bacterial aetiology.[3 4] It is uncertain if this also occurs in upper respiratory tract infections.

Several mechanisms have been proposed for this increased hormone secretion, acting through fever, hypoxia, hypercarbia, pain, emotion, or nausea. Secretion may be stimulated by a resetting of osmostat receptors to lower levels.[3] Also, lung hyperinflation and pulmonary infiltrates may stimulate hormone secretion by causing a false perception of hypovolaemia by intrathoracic receptors.[4] This would be in keeping with findings that antidiuretic hormone secretion in pneumonia increases proportionally with the extent of lung parenchymal involvement.[3]

Giving extra fluids while antidiuretic hormone secretion is increased may theoretically lead to hyponatraemia and fluid overload. Clinical symptoms of hyponatraemia are irritability, confusion, lethargy, coma, and convulsions. Fluid restriction may be appropriate management to prevent this.

Methods and results

To determine whether recommending increased fluids was beneficial or harmful, we undertook a systematic review and posed three questions:
Does recommending increased fluid intake for acute respiratory infections improve duration and severity of symptoms?
Are there adverse effects from this recommendation?
Are any benefits or harm related to site (upper or lower respiratory tract) or severity of illness?

Using the Cochrane Acute Respiratory Infections Group search strategy, together with additional terms (see bmj.com for details), we did a conventional search of the Cochrane Central Register of Controlled Trials, Medline (1966-2003), Embase (1974-2003), and Current Contents (1966-2003). We examined references of relevant papers and contacted experts in the subject.

We found no randomised controlled trials comparing increased and restricted fluid regimens in patients with respiratory infections. Two prospective prevalence studies reported hyponatraemia at rates of 31% and 45% for children with moderate to severe pneumonia

Extra details about the search strategy used and tables detailing results from other studies appear on bmj.com

Centre for General Practice, Medical School, University of Queensland, Herston, 4006, Queensland, Australia

Michelle P B Guppy *academic general practice registrar*

Sharon M Mickan *senior research fellow*

Chris B Del Mar *professor of general practice*

Correspondence to: C B Del Mar c.delmar@cgp.uq.edu.au

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Studies showing hyponatraemia in non-dehydrated patients with respiratory infections

Study	No of patients	Age	Diagnosis	Exclusions	Serum sodium level (mmol/l)	Serum or urine osmolality	Symptoms associated with low sodium level
Dhawan et al 1992[2]	100	1 month-12 years	Pneumonia	Clinical dehydration	31 patients had Na <130	Serum dilute in 29, concentrated in 2 (probable dehydration)	4 died, Na <125 mmol/l
Shann et al 1985[1]	73	≥1 month	Pneumonia	Clinical dehydration	33 patients had Na <134	—	Hyponatraemia 2-3 times more frequent with severe pneumonia
Rivers et al 1981[5]	4	6-8 weeks and 6 months	3 bronchiolitis, 1 pneumonia	—	3 patients had Na 114-124	Concentrated urine, or not maximally dilute urine	Seizures in one patient with bronchiolitis

(see table).[1 2] None of these children showed clinical signs of dehydration. Symptoms associated with hyponatraemia were not reported, but four children with a serum sodium below 125 mmol/l died during one study.

We also found several case series in which patients with respiratory infections developed hyponatraemia, of which some were symptomatic (table).[5] These patients were all successfully treated with fluid restriction.

Comment

We found data to suggest that giving increased fluids to patients with respiratory infections may cause harm. To date there are no randomised controlled trials to provide definitive evidence, and these need to be done. Until we have this evidence, we should be cautious about universally recommending increased fluids to patients, especially those with infections of the lower respiratory tract.

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Effect of statin treatment for familial hypercholesterolaemia on life assurance: results of consecutive surveys in 1990 and 2002

H A W Neil, T Hammond, D Mant, S E Humphries Topic: 183;163

Oxford Centre for Diabetes, Endocrinology and Metabolism, Churchill Hospital, Oxford OX3 7HJ
H A W Neil *honorary consultant physician*

Division of Public Health and Primary Health Care, Institute of Health Sciences, University of Oxford, Oxford OX3 7LF
T Hammond *research assistant*
D Mant *professor of general practice*

Centre for Cardiovascular Genetics, British Heart Foundation Laboratories, Royal Free and University College London Medical School, London WC1E 6JJ
S E Humphries *professor of cardiovascular genetics*

Correspondence to:
H A W Neil
andrew.neil@wolfson.ox.ac.uk

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One of the concerns often raised about genetic testing is the possibility that a positive result (or even disclosing that the test has been taken) may result in difficulty in obtaining life assurance. Currently the UK insurance industry has declared a moratorium on requiring genetic tests from applicants,[1] but since DNA based tests offer a definitive, highly specific diagnosis they are likely eventually to replace less specific clinical diagnostic criteria for many inherited disorders. Early, presymptomatic treatment may increase life expectancy by preventing or reducing the risk of developing the disease or associated complications. However, if life assurance policy premiums do not adequately reflect the reduction in mortality with treatment relatives of affected probands may be deterred from being tested. We examined how life assurance companies have responded to the improvement in the prognosis of heterozygous familial hypercholesterolaemia with statin treatment.[2]

Familial hypercholesterolaemia is an autosomal dominant disorder, inherited on average by one in two children of an affected parent. The prevalence in the United Kingdom is about 1:500, but at least three quarters of cases remain undiagnosed.[3] It is usually caused by mutations in the gene for the low density lipoprotein (LDL) receptor that result in accumulation of plasma cholesterol and substantially increased coronary mortality. Without effective treatment the cumulative risk of a coronary event is at least 50% in men and 30% in women by the age of 60 years.[4] With statin treatment, however, the standardised mortality ratio for coronary heart disease for such patients aged 20-59 years has more than halved over the past decade.[2]

Participants, methods, and results

We conducted a survey of 41 life assurance companies in 1990[5] and repeated it in 2002 by sending the same questionnaire to 26 companies still underwriting term life assurance. They were asked to assess a fictional proposal for a 20 year policy (paying benefit only on death) by applying an excess mortality rating defined as the percentage increase over the assumed rate of mortality. In the follow up survey companies were asked to assess the rating before and after statin treatment. The applicant was a normotensive (120/80 mm Hg), non-smoking, 30 year old man taking no medication, with a body mass index of 22.1 kg/m^2 and a normal resting electrocardiogram, whose father had had a non-fatal myocardial infarction aged 45 years. He had no other family history of heart disease, other medical history of note, or tendon xanthomata on examination. The results of the lipid profile (mmol/l) were total cholesterol 11.4, high density lipoprotein (HDL) cholesterol 1.7, triglycerides 1.3, and calculated LDL cholesterol 9.1; and the presumptive diagnosis was familial hypercholesterolaemia. On treatment with atorvastatin 80 mg daily his total cholesterol concentration was 6.2 mmol/l (compared with 10.7 mmol/l on cholestyramine prescribed in 1990).

The figure shows the excess mortality ratings applied. Because two companies would assess proposals only from patients receiving statin treatment statistical comparisons were restricted to 24 companies. The