Burns from a tumble drier

K Herbert, S K Varma

Burns Unit, Leicester Royal Infirmary, Leicester LE1 5WW
K Herbert, FRCS, *senior house officer*
S K Varma, FRCS, *registrar*

Correspondence to:
Mr Herbert.

Br Med J 1989;**298**:571

One third of accidents occur in the home, and the age group most at risk is the under 5s.[1] Home appliances play a part in many of these accidents. In 1986, 27 accidents in which a tumble drier was involved were reported to the home accident surveillance system. Most of these injuries resulted from falls off or against the appliance, and only one burn, to a person leaning against the door, was reported. We report what we think is probably the first case of burns sustained by someone trapped inside the drum of the drier.

Case report

A 5 year old girl was encouraged by two friends of the same age to climb into a tumble drier in the garage at her home. They switched on the machine and then left the scene in fear. The mother was upstairs putting another child to sleep and came downstairs to investigate the loud continuous banging noise. She rescued her daughter and then called an ambulance. The story was confirmed by social services staff after the two other children were interviewed.

The child arrived at the accident and emergency department of this hospital alert, oriented, and in no respiratory distress. She had sustained partial thickness burns to 8% of her body surface area over the arms and back. In addition, she had bruising over the eyelids and shoulders. She was treated as an inpatient. Her hospital stay was uneventful, and the burns healed spontaneously.

Comment

A third of households in Britain were estimated to have tumble driers in 1985, which is almost twice the 1979 figure.[2] These appliances run on mains electricity (220 volts) and can produce air temperatures within the drum of up to 65°C. The temperature of the drum itself may reach 53°C. The diameter of the door and capacity of the drum will easily admit a child up to 5 years old, but few manufacturers warn of this danger. We recommend that a childproof door mechanism be incorporated into these appliances.

We thank Mr T M Milward, consultant plastic surgeon, for allowing us to report on his patient.

1 Consumer Safety Unit. *Home accident surveillance system.* London: Department of Trade, 1986. (10th Annual report.)
2 Office of Population Censuses and Surveys. General household survey. Preliminary results for 1985. *OPCS Monitor* 1986 Sep 18. (GHS 86/1.)

(Accepted 22 November 1988)

Estimating alcohol content of drinks: common errors in applying the unit system

Tim Stockwell, Liz Stirling

Department of Psychology, University of Exeter, Exeter EX4 4LG
Tim Stockwell, PHD, *honorary research fellow*

Wonford House Hospital, Wonford, Exeter EX2 5AF
Liz Stirling, MRCGP, *senior house officer in psychiatry*

Correspondence to:
Associate Professor T Stockwell, National Centre for Research into the Prevention of Drug Abuse, Curtin University of Technology, GPO Box U 1987, Perth 6001, Western Australia.

Br Med J 1989;**298**:571-2

The unit system is widely recommended as a method of monitoring alcohol consumption.[1-3] A unit is the amount of alcohol (usually 8 g[4]) found in fixed amounts of different beverages that are of "standard" strength —for example, half a pint of beer (0·3 litre) or lager with 3·5% alcohol by volume, one glass of wine with 8%, or a single measure of spirits with 40%. Use of this system is complicated in that different brands of the same beverage vary considerably in strength and manufacturers give information about alcohol content in varying ways. We studied whether a cross section of the general public could use the system to assess the strengths of drinks with low, standard, and high alcohol contents.

Subjects, methods, and results

The subjects comprised 217 visitors to a road safety display at a county show aged from 16 to 73, with 80% being under 45; 117 were men. All were taught to use the unit system and were tested on it until they successfully answered three simple questions about its use. Altogether 150 had heard of units of alcohol before, and 63 had counted their drinks in units.

One hundred and four subjects were asked to examine the usual retail containers for three wines, three lagers, and three beers. We emphasised that the strengths of the drinks varied a great deal. In fact, the wines contained 0·05%, 7%, and 13% alcohol by volume; the lagers contained 0·9%, 3·4%, and 8·6%; and the beers contained 1%, 3·5%, and 10·9%. With one exception this was stated on the container, the strong lager being labelled only with its original gravity (1076-1082). Each subject was asked to estimate the number of units in one 150 ml glass of each wine and in one pint (0·57 litre) of each lager and beer. The remaining 113 subjects underwent an identical procedure with larger amounts of alcohol; the results (not detailed here) were comparable. Sample glasses were used to illustrate the amounts in both cases.

In the group of 104 subjects between 80 and 88 correctly estimated the number of units in the standard strength drinks. Fewer correctly estimated the strength of the low alcohol drinks. Fewer still correctly estimated the strength of the extra strong drinks: mostly this was greatly underestimated, with 53 underestimating the strength of the wine, 103 the lager, and 93 the beer (table). After being told of the relative strengths of the drinks 198 subjects agreed that it would be a good idea for bottles and cans of alcoholic drink to display their alcohol content in units.

Numbers of subjects underestimating, overestimating, and correctly estimating number of units in one pint or one wine glass of displayed drinks (n=104)

Drink	Response	Strength of drink: Standard	Low	High
Wine	Underestimate	1		53
	Correct	80	74	44
	Overestimate	23	30	7
Lager	Underestimate	11	20	103
	Correct	80	59	1
	Overestimate	13	25	
Beer	Underestimate	8	26	93
	Correct	88	65	9
	Overestimate	8	13	2

Comment

Our results suggest that most people can accurately apply the unit system to drinks of standard strength but that serious inaccuracies result when it is applied to either low or high strength drinks, even when subjects are instructed to make allowances for variations in alcohol content. Underestimating the strength of alcoholic drinks may have serious implications for

health: those needing to abstain may unwittingly drink if they believe low alcohol drinks to be effectively alcohol free. Others wanting to limit their drinking by counting units may drink far more than their chosen amounts if they fail to make adjustments for extra strong drinks.

There are two possible strategies for minimising these errors. Firstly, all drinks could have their alcohol content clearly labelled in units as well as in percentage alcohol by volume. This suggestion was endorsed by nearly all our subjects. Secondly, posters and leaflets providing current data on the strengths of commonly available drinks, similar to those provided by the Health Education Council for the tar and nicotine contents of cigarettes, could be widely distributed.

1 Royal College of Psychiatrists. *Alcohol—our favourite drug.* London: Tavistock, 1986.
2 Royal College of General Practitioners. *Alcohol—a balanced view.* London: RCGP, 1986.
3 Royal College of Physicians. *Alcohol—a great and growing evil.* London: Tavistock, 1987.
4 Robertson I, Heather N. *Let's drink to your health! A self-help guide to sensible drinking.* Leicester: British Psychological Society, 1986.

(Accepted 1 December 1988)

Digoxin toxicity due to interaction of digoxin with erythromycin

D L Maxwell, S K Gilmour-White, M R Hall

Department of Medicine, United Medical and Dental School of Guy's and St Thomas's Hospitals, Guy's Hospital, London SE1 9RT
D L Maxwell, MRCP, *senior registrar*
S K Gilmour-White, MRPHARMS, *principal pharmacist*
M R Hall, MB, *senior house officer*

Correspondence to:
Dr Maxwell, Chest Clinic, Guy's Hospital.

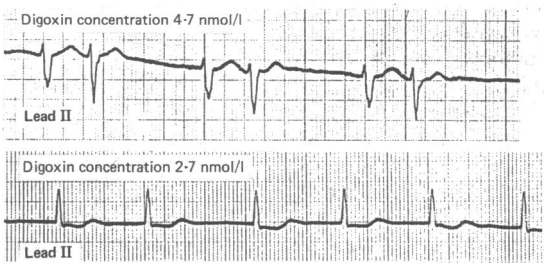
Br Med J 1989;**298**:572

Both erythromycin and digoxin cause gastrointestinal side effects. Erythromycin, however, may double digoxin concentrations in a minority of patients, and gastrointestinal side effects may mistakenly be attributed to it. We describe a patient in whom a course of erythromycin was associated with cardiac and gastrointestinal complications of digoxin toxicity.

Case report

A 59 year old woman (weight 50·1 kg) presented with bradycardia and a two day history of nausea and vomiting four days after starting erythromycin 500 mg three times a day for an upper respiratory tract infection. She had received maintenance treatment with digoxin (0·25 mg/day) and warfarin since 1972 after a mitral valve replacement. For three years she had received bumetanide (1 mg/day) and potassium chloride (Slow K; 16 mmol/day). Eighteen months previously her serum digoxin concentration had been 2·3 nmol/l (therapeutic range 1·0-2·8 nmol/l). On examination she had an irregular heart rate (40-50 beats/min) and cardiomegaly. An electrocardiogram confirmed slow atrial fibrillation (rate 40 beats/min), right bundle branch block with left anterior hemiblock, and ventricular bigeminy (figure (top)). Biochemical investigation showed normal plasma urea, sodium, and calcium concentrations and liver enzyme activities. Plasma potassium concentration was 3·6 mmol/l, serum digoxin concentration 4·7 nmol/l, and the international normalised ratio of the prothrombin time 2·4.

Twenty four hours after digoxin and erythromycin were stopped her symptoms and the arrhythmia resolved. Electrocardiography showed atrial fibrillation (rate 60 beats/min) without evidence of a ventricular conduction defect (figure (bottom)). Two weeks after she was discharged taking her usual treatment her serum digoxin concentration was 2·7 nmol/l. She recalled that one year earlier she had experienced similar symptoms during a course of erythromycin.



Electrocardiographic recordings obtained from lead II on day of admission (top) and next day (bottom)

Comment

For many years digoxin was generally considered to be excreted unchanged in the urine, but it is now clear that in about 10% of patients ("excretors") 30-40% of the drug is excreted in the urine as reduced metabolites.[1] Compared with digoxin, its principal metabolites are taken up less well by cardiac muscle and have less cardiac activity, and some are more rapidly excreted in the urine.[2] Some patients may therefore require daily doses as high as 1-2 mg.[1,3]

Urinary concentrations of these metabolites fall considerably and the serum digoxin concentration rises by as much as twofold after a change to a formulation of digoxin with greater bioavailability or during treatment with erythromycin or tetracycline.[4] These effects are due to reduction of digoxin by *Eubacterium lentum*, a common constituent of the normal gut flora. Other unknown factors, however, must also play a part as stool cultures from a fifth of "non-excretors" also grow this organism.[5] Whether other commonly used antibiotics cause a similar interaction is not known, but a preliminary report suggests that the β lactam antibiotics have little effect and that in vitro sensitivity of *E lentum* to an antibiotic does not mean that the formation of digoxin metabolites will be reduced.[2]

Suspicion of an interaction may be aroused by the need for high oral doses of digoxin. This was not, however, the case with our patient. Nevertheless, her gastrointestinal symptoms one year previously might have been due to digoxin toxicity and might have warned of future problems. Preparations of digoxin with greater bioavailability, such as encapsulated liquid digoxin or paediatric elixir, may reduce the risks of such interactions.[4] Alternatively, a temporary reduction in the dose of digoxin should be considered when erythromycin or tetracycline is required.

We thank Dr T J Gibson for allowing us to report on a patient under his care and for helpful comments on this paper.

1 Peters U, Falk LC, Kalman SM. Digoxin metabolism in patients. *Arch Intern Med* 1978;**138**:1074-6.
2 Lindenbaum J. The dihydrogenation of digoxin. In: Erdman E, Greef K, Skou JC, eds. *Cardiac glycosides 1785-1985.* New York: Springer-Verlag, 1985: 263-8.
3 Luchi RJ, Gruber JW. Unusually large digitalis requirements: a study of altered digoxin metabolism. *Am J Med* 1968;**45**:322-8.
4 Lindenbaum J, Rund DG, Butler VP, Tse-Tung D, Saha JR. Inactivation of digoxin by the gut flora: reversal by antibiotic therapy. *N Engl J Med* 1981;**305**:789-93.
5 Dobkin JF, Saha JR, Butler VP, Neu HC, Lindenbaum J. Digoxin-inactivating bacteria: identification in human gut flora. *Science* 1983;**220**:325-7.

(Accepted 14 December 1988)