enzyme; Dr A Holmgren, department of clinical physiology, Karolinska Hospital, for measurements of lung volume; and Mrs Margit Tjernberg and Ms Karin Lilja for skilful technical help. This study was supported by grants from the Swedish Medical Research Council, the Gustaf V 80 year fund, and the Swedish Association against Chest and Lung Diseases.

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(*Accepted 14 March 1985*)

SHORT REPORTS

Failure of random zero sphygmomanometer in general practice

The main cause of inaccuracy in measuring blood pressure is systematic bias between observers,[1] a major factor being preference for a particular final digit.[2] Use of a random zero sphygmomanometer (Gelman Hawksley Ltd) can reduce this inaccuracy.[3] The device has a random zero position (between 0 and 60 mm Hg), which is changed blind by the observer before each measurement. The true blood pressure can thus be determined only by means of subtraction once the column has come to rest. I assessed terminal digit preference when this instrument was used.

Patients, methods, and results

Doctors from four general practices participated in a screening programme for hypertension. The technique for measuring blood pressure was standardised, each doctor being provided with a random zero sphygmomanometer and instructed in its use. Blood pressure was measured after a rest of five minutes. The cuff was inflated above the systolic pressure estimated from palpation of the radial artery and the constant relief valve set at 2 mm Hg/s. The level of the column was read to the nearest 2 mm Hg at Korotkoff sounds I and V and at the resting zero point. The systolic and diastolic pressures were then recorded. The pressures recorded in 2096 patients over three years from 1 January 1980 were used to assess terminal digit preference and to examine differences both between practices and over time.

Terminal digit preference was evident with both systolic and diastolic recordings. Fifty four per cent of the values given for systolic pressure and 49% of those for diastolic pressure ended in zero, with a random distribution between the four other even digits (table). Despite instructions to record to the nearest 2 mm Hg, 12% of systolic and 15% of diastolic readings ended in an odd number, principally 5. All four practices showed the preference for zero, though the proportion of readings ending in zero ranged from 44% to 75% for systolic and 40% to 67% for diastolic pressures. In the practice with the greatest bias (C) a subsequent review of 200 random blood pressure readings taken before the study showed a distribution of final digits that was not significantly different from that recorded during the study with the random zero sphygmomanometer. In the other practices 36% of systolic and 38% of diastolic pressures ended in an even digit other than zero compared with less than 5% of the pressures measured before the study.

Terminal digit preference in each practice (figures are numbers (%) of readings)

Practice	No of readings	Final digit						
		0	2	4	6	8	5	Other odd
				Systolic				
A	781	480 (61·5)	51 (6·5)	43 (5·5)	45 (5·8)	45 (5·7)	87 (11·2)	29 (3·7)
B	842	370 (44·0)	105 (12·5)	114 (13·5)	71 (8·4)	66 (7·8)	66 (7·8)	51 (6·1)
C	171	128 (74·9)	5 (2·8)	6 (3·4)	7 (3·9)	10 (5·6)	15 (8·9)	1 (0·6)
D	302	150 (49·6)	50 (16·7)	38 (12·7)	30 (9·8)	27 (9·1)	7 (2·2)	
				Diastolic				
A	781	437 (55·9)	52 (6·6)	40 (5·1)	52 (6·6)	60 (7·7)	112 (14·4)	28 (3·6)
B	842	340 (40·4)	104 (12·4)	107 (12·7)	77 (9·1)	86 (10·2)	73 (8·7)	56 (6·7)
C	171	115 (67·0)	7 (3·9)	4 (2·2)	4 (2·2)	7 (3·9)	32 (19·0)	3 (1·7)
D	302	129 (42·8)	53 (17·4)	31 (10·1)	40 (13·4)	35 (11·6)	11 (3·6)	3 (1·1)

In the first year of the study 55% of systolic values ended in zero, in the second year 55%, and in the third year 51%; the corresponding figures for diastolic pressures were 51%, 48%, and 52%. Thus the bias was consistent throughout the study.

Comment

Using the random zero sphygmomanometer did not abolish terminal digit preference, though it probably reduced its magnitude in three of the four practices. This degree of bias is unacceptable

for epidemiological studies of blood pressure and undesirable in good clinical practice. Why the bias persisted is not clear as some reduction was observed, but it probably resulted from some doctors reading not only both the systolic and diastolic sounds but also the zero resting point to the nearest zero. The random zero device often takes 15-20 seconds to reach its nadir. Failure to allow for this delay could have prevented an accurate reading of the zero resting point and led to the biased results obtained. A recently designed modification to the device—a digital display—might reduce the bias in reading the zero point,[4] but this would be insufficient to give accurate readings as biased readings were recorded at the relevant Korotkoff sounds.

The random zero sphygmomanometer is an accurate device,[5] and it is perhaps unreasonable to expect a precision instrument intended for research to be similarly precise for normal medical care. Incorrect use of this instrument will result in incorrect readings.

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(*Accepted 19 February 1985*)

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Use of urea for treatment of water retention in hyponatraemic cirrhosis with ascites resistant to diuretics

Patients with cirrhosis and ascites who are resistant to normal treatment with diuretics (spironolactone with or without long loop diuretics) tend to develop hyponatraemia, which is aggravated by the hormone with urea[2] or long loop diuretics,[3] we felt it was appropriate to use urea as a complementary treatment to diuretics in patients with cirrhosis and ascites. We studied a hyponatraemic patient with resistant ascites for whom the oral administration of urea was not contraindicated. Intermittent treatment with urea induced a significant increase in diuresis that was associated with weight loss. Moreover, the hyponatraemia was corrected after treatment with urea despite the continued administration of diuretics and unrestricted water intake.

Case report

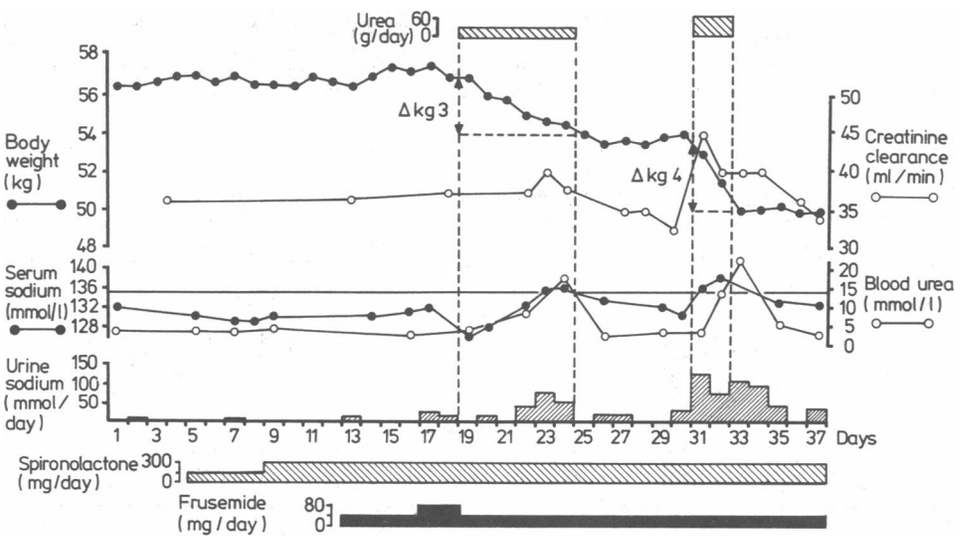
A 45 year old woman presented with alcoholic cirrhosis in icteroascitic decompensation (serum bilirubin 54 μmol/l (3 mg/100 ml) on admission) with moderate peripheral oedema. The diagnosis of cirrhosis was histologically confirmed after the removal of fluid. The patient was put on a low salt diet (10 mmol/day) and was initially given 150 mg and then 300 mg spironolactone without any change in weight, although liver function improved: serum bilirubin value decreased to 23 μmol/l (1·3 mg/100 ml). The addition of 40 mg frusemide did not increase the urine sodium output and 80 mg taken once a day exacerbated the tendency towards hyponatraemia (125 mmol(mEq)/l) (see figure). Before treatment with urea, the creatinine clearance was 30 ml/min (or 40 ml/min/1·73 m²) and serum albumin concentration was normal (34 g/l).

A daily urea intake of 30 g over six days induced a weight loss of 3 kg, an increase in serum sodium concentration (136 mmol/l) and in urinary sodium output, without any change in creatinine clearance (35-40 ml/min). When urea was stopped weight loss halted and hyponatraemia recurred (129 mmol/l). A further course of urea (60 g/day for two days) induced a weight loss of 4 kg in three days, the disappearance of any clinical features of salt retention, and an increase in serum sodium concentration (138 mmol/l) was again observed without any impairment of glomerular filtration. Arterial ammonium concentration did not change and urine volume increased from 0·6-0·8 l/day to 1·0-1·5 l/day during treatment.

Comment

Hyponatraemia associated with cirrhosis arises from renal dysfunction in water excretion. The condition can be so severe as to produce neurological symptoms, and diuretics given to treat the ascites can worsen it.

The management of hyponatraemia in an oedematous state, which has or which has not developed as a result of treatment with diuretics, is usually the restriction of water. In our patient the administration of urea induced an increased diuresis, a correction of hyponatraemia



Evolution of creatinine clearance, serum sodium concentration, blood urea concentration, urine sodium excretion, and body weight during intermittent treatment with urea.
Conversion: SI to traditional units—Serum sodium: 1 mmol/l = 1 mEq/l. Blood urea: 1 mmol/l ≈ 6 mg/100 ml.

administration of diuretics. Restricted water intake corrects the hyponatraemia, but is slow to take effect. Water retention in patients with hyponatraemia and cirrhosis has been treated with demeclocycline, in the same way as in patients suffering from inappropriate secretion of antidiuretic hormone. This treatment greatly increases the risk of renal dysfunction, however.[1] Having already treated water retention in patients with inappropriate secretion of antidiuretic without water restriction, weight loss, and no impairment of renal function. She had a creatinine clearance of 35 ml/min, and the surgical placement of a Le Veen shunt[4] seemed appropriate but was finally not required. Treatment with demeclocycline induces an increase in urinary sodium output by an unknown mechanism.[1] Our patient who was treated with urea also presented higher losses of urinary sodium. This phenomenon can result either from the effect of urea,