

functioning, underlining the independence of clinical and social outcome in schizophrenia. High rates of unemployment and inactivity persisted, and the numbers in specialised accommodation did not change. *The Health of the Nation* emphasises that improving services can do much to reduce the harm that mental illnesses can cause and outlines targets for improving both health and social functioning of those suffering from a mental illness.²¹ These targets will not be achieved with patterns of community care such as those found in this study, in which very few patients are receiving coordinated packages of social care to address poor social functioning. There is a need for comprehensive treatment programmes involving long term social activity as well as assertively delivered medical care.

We are grateful to the patients and their relatives who volunteered to be interviewed and to Professor T Craig for his advice.

- Lamb HR. Lessons learned from deinstitutionalisation in the US. *Br J Psychiatry* 1993;162:587-92.
- Shanks J. Mental illness services in Britain: counting the costs, weighing the benefits. *Hosp Community Psychiatry* 1989;40:878-9.
- Craig T, Timms PW. Out of the wards and on to the streets? Deinstitutionalisation and the homeless in Britain. *J Ment Health* 1992;1:265-75.
- Johnstone EC. Disabilities and circumstances of schizophrenic patients—a follow-up study. *Br J Psychiatry* 1991;159(suppl 13):13-20.
- Melzer D, Hale AS, Malik SJ, Hogman GA, Wood S. Community care for patients with schizophrenia one year after hospital discharge. *BMJ* 1991;303:1023-6.
- Intagliata J. Improving the quality of community care for the chronically mentally disturbed: the role of case management. *Schizophr Bull* 1982;8:655-74.
- Shepherd G. Case management. *Health Trends* 1990;2:59-61.
- Olfson M. Assertive community treatment: an evaluation of the experimental evidence. *Hosp Community Psychiatry* 1990;41:634-41.
- Hirsch S. *Psychiatric beds and resources: factors influencing bed use and service planning*. London: Gaskell/Royal College of Psychiatrists, 1988.
- Thornicroft G. Social deprivation and rates of treated mental disorder. Developing statistical models to predict psychiatric service utilisation. *Br J Psychiatry* 1991;158:475-84.
- Spitzer RL, Endicott J, Robins E. *Research diagnostic criteria instrument No 58*. New York: New York State Psychiatric Institute, 1975.
- Wing JK, Cooper J, Sartorius N. *The measurement and classification of psychiatric symptoms*. Cambridge: Cambridge University Press, 1974.

- World Health Organisation. *WHO psychiatric disability assessment schedule*. Geneva: WHO, 1988.
- Ellis RE, Wilson NZ, Foster MF. Statewide treatment outcome assessment in Colorado: the Colorado client assessment record. *Community Ment Health J* 1984;20:72-89.
- Norusis MJ. *Statistical package for the social sciences PC+*. Chicago: SPSS, 1986.
- Ciompi L. Is there really a schizophrenia? The long-term course of psychotic phenomena. *Br J Psychiatry* 1984;145:636-40.
- Harding CM, McCormick RV, Strauss JS, Ashikaga T, Brooks GW. Computerised life chart methods to map domains of function and illustrate patterns of interactions in the long-term course trajectories of patients who once met the criteria for DSM-III schizophrenia. *Br J Psychiatry* 1989;155(Suppl 5):100-6.
- Watt DC, Katz K, Shepherd M. The natural history of schizophrenia: a five year prospective follow up of a representative sample of schizophrenics by means of a standardized clinical and social assessment. *Psychol Med* 1983;13:663-70.
- Johnstone EC, Owens DGC, Gold A, Crow TJ, MacMillan JF. Schizophrenic patients discharged from hospital—a follow-up study. *Br J Psychiatry* 1984;145:586-90.
- Addington DE, Addington JM. Attempted suicide and depression in schizophrenia. *Acta Psychiatr Scand* 1992;85:288-91.
- Department of Health. *The health of the nation: a strategy for health in England*. London: HMSO, 1992.
- Lee PWH, Lieh-Mak F, Yu KK, Spinks JA. Patterns of outcome of schizophrenia in Hong Kong. *Acta Psychiatr Scand* 1991;84:346-52.
- Breier A, Schreiber JL, Dyer J, Pickar D. National Institute of Mental Health longitudinal study of chronic schizophrenia. Prognosis and predictors of outcome. *Arch Gen Psychiatry* 1991;48:239-46.
- Carone BJ, Harrow M, Westermeyer JF. Posthospital course and outcome in schizophrenia. *Arch Gen Psychiatry* 1991;48:247-53.
- Shepherd G. The management of schizophrenia in the community: what services do we need? In: Jenkins R, Field V, Young R, eds. *The primary care of schizophrenia*. London: HMSO, 1992.
- Thornicroft G, Breakey WR. The COSTAR programme. 1: Improving social networks of the long-term mentally ill. *Br J Psychiatry* 1991;159:245-9.
- Lawrence RE. The impact of community care on the course of schizophrenia in Kidderminster. In: Hall P, Brockington IF, eds. *The closure of mental hospitals*. London: Gaskell/Royal College of Psychiatrists, 1991.
- Rice P, Irving D, Davies G. *Information about district health authorities in England from the 1981 census*. London: King's Fund, 1984.
- Wing JK. Meeting the needs of people with psychiatric disorders. *Soc Psychiatry Psychiatr Epidemiol* 1990;25:2-8.
- Scottish Schizophrenia Research Group. The Scottish first episode schizophrenia study. VII. Two-year follow-up. *Acta Psychiatr Scand* 1989;80:597-602.
- Bhugra TS, Wing JK, Smith BL. Physical health of the long term mentally ill in the community. Is there unmet need? *Br J Psychiatry* 1988;155:777-81.
- King MB. Management of patients with schizophrenia in general practice. *Br J Gen Pract* 1992;42:310-1.
- Kendrick T, Sibbald B, Burns T, Freeling P. Role of general practitioners in care of long term mentally ill patients. *BMJ* 1991;302:508-10.

(Accepted 1 November 1993)

Nocturnal blood pressure in normotensive subjects and those with white coat, primary, and secondary hypertension

Martin Middeke, Joachim Schrader

Abstract

Objective—To compare the mean nocturnal blood pressure of patients with various forms of renal and endocrine hypertension with that in patients with primary and white coat hypertension, and normal blood pressure.

Design—Ambulatory monitoring of blood pressure over 24 hours in a prospective study.

Setting—Two German centres for outpatients with hypertension and kidney diseases.

Subjects—176 normotensive subjects, 490 patients with primary hypertension including mild and severe forms, 42 with white coat hypertension, 208 patients with renal and renovascular hypertension, 43 with hypertension and endocrine disorders, and three with coarctation of the aorta.

Main outcome measures—Fall in nocturnal blood pressure.

Results—Blood pressure in normotensive subjects fell by a mean of 14 mm Hg (11%) systolic and 13 mm Hg (17%) diastolic overnight (2200 to 0600). The falls in patients with primary and white coat hypertension were not significantly different. In all patients with renal and renovascular hypertension, however, the fall was significantly reduced

(range of fall from 3/3 mm Hg to 7/9 mm Hg). In patients with hypertension and endocrine disorders the pattern of night time blood pressure was not uniform: patients with hyperthyroidism, primary hyperaldosteronism, and Cushing's syndrome had significantly smaller reductions in blood pressure (6/8, 4/7, 3/6 mm Hg, respectively). In patients with pheochromocytoma the mean night time blood pressure increased by 4/2 mm Hg. In patients with hypertension, primary hyperparathyroidism, and unoperated coarctation of the aorta the falls in blood pressure were normal.

Conclusions—In normotensive subjects and those with primary hypertension there is usually a reduction in blood pressure at night. In all renal forms of secondary hypertension and in most endocrine forms the reduction in blood pressure is only a third to a half of normal. Patients with primary hyperparathyroidism and unoperated coarctation of the aorta show a normal reduction.

Introduction

After the description by Zadek in 1881 of a diurnal variation in blood pressure¹ Howell later observed the

Reha-Zentrum Spreewald,
D-03096 Burg/Spreewald,
Germany
Martin Middeke, chief doctor

St Josefs-Hospital,
D-49661 Cloppenburg,
Germany
Joachim Schrader, chief
doctor

Correspondence to:
Professor Middeke.

BMJ 1994;308:630-2

nocturnal fall of blood pressure.² In 1922 Katsch and Pansdorf were the first to describe an abnormal rhythm in blood pressure in five uraemic patients.³

In recent years several groups have described a disturbed circadian rhythm in blood pressure in some secondary forms of hypertension by using indirect ambulatory measurements of blood pressure.⁴⁻⁶ We investigated profiles of blood pressure in normotensive subjects, in patients with white coat and primary hypertension, and in patients with various forms of secondary hypertension.

Subjects and methods

All patients with established secondary hypertension of renal origin and with endocrine disorders associated with hypertension who underwent ambulatory measurements of blood pressure in two outpatient units for hypertension in 1989-92 were included in the study. They were compared with a group of normotensive subjects, a group with white coat hypertension, and a group with varying severities of primary hypertension. Secondary hypertension was diagnosed from clinical features; routine laboratory tests; hormone analysis (suppressed activity of thyroid stimulating hormone in hyperthyroidism; increased excretion of aldosterone over 24 hours, and suppressed renin activity in primary hyperaldosteronism; increased plasma calcium concentration and increased intact parathormone concentration in primary hyperparathyroidism; increased plasma concentrations of catecholamines and increased excretion of metabolites in phaeochromocytoma; positive result of dexamethasone suppression test in Cushing's syndrome); abdominal ultrasonography, computed tomography, and digital subtraction angiography of the kidneys (renal artery stenosis); and aortography (coarctation of the aorta).

Ambulatory blood pressure was measured over 24 hours with a fully automatic recorder (SpaceLabs ABP 90202 and 90207, SpaceLabs, Redmont, Washington). The blood pressure of each patient was measured every 15-20 minutes between 0600 and midnight and every 30-40 minutes between midnight and 0559. All patients had a normal activity-sleep rhythm. The study was prospective.

The mean (SD) systolic and diastolic blood pressures over 24 hours, between 0601-2159 and 2200-0600 were calculated in the various groups. The fall in nocturnal blood pressure (daytime blood pressure minus nocturnal blood pressure) was calculated by taking into account all raw data from each patient.

Normal or log normal distributed variables (in normotensive patients and those with primary hyper-

tension) were analysed with Student's *t* test and mean values were compared by analysis of variance (Scheffé multiple range test). For variables that were not normally distributed (subjects with white coat and secondary hypertension) the U test was performed to compare two independent samples (Wilcoxon and Mann-Whitney) or the H test to test several independent samples (Kruskal-Wallis).

Results

The different groups consisted of 176 normotensive subjects, 42 patients with white coat hypertension, 490 with primary hypertension, and 254 with secondary hypertension (table). A mean (SD) of 54 (7) valid blood pressure measurements were recorded for each patient over 24 hours.

In normotensive subjects blood pressure fell over night by a mean of 14 mm Hg (11%) systolic and 13 mm Hg (17%) diastolic. The fall in blood pressure was not significantly different between normotensive subjects and those with white coat and primary hypertension (table). The fall in blood pressure in patients with renoparenchymal and renovascular hypertension was significantly reduced, ranging from 3/3 mm Hg (kidney transplantation) to 7/9 mm Hg (renal artery stenosis). In hypertensive patients with endocrine disorders the fall in blood pressure was either significantly reduced, ranging from 3/6 mm Hg (Cushing's syndrome) to 6/8 mm Hg (hyperthyroidism), or even increased (by 4/2 mm Hg in phaeochromocytoma). In hypertensive patients with primary hyperparathyroidism, however, the circadian modulation in blood pressure was no different from that seen in normotensive subjects or in those with primary hypertension. Three patients with non-operated coarctation of the aorta had high blood pressure with a normal circadian pattern.

Discussion

The main characteristics of the circadian rhythm in blood pressure were described at the end of the last century.^{1,2} By using automated indirect ambulatory measurement of blood pressure the circadian rhythm in many normotensive subjects and in patients with primary hypertension has been well documented in recent years.

We studied patients with different forms of secondary hypertension and compared them with a group of normotensive subjects and patients with white coat and primary hypertension. The data showed a uniform pattern of a blunted circadian curve in blood pressure in patients with renal hypertension—for example,

Details of fall in nocturnal blood pressure in subjects with normal blood pressure and those with different forms of hypertension

Type of hypertension	No of subjects	Mean (SD) age (years)	Mean (SD) systolic/diastolic blood pressure (mm Hg)		Mean (SD) change in nocturnal systolic/diastolic blood pressure	% Change in systolic/diastolic blood pressure	P value
			Day	Night			
Normotension	176	41 (12)	126/78 (16/13)	112/65 (14/11)	-14/13 (9/8)	11/17	
White coat	42	44 (14)	126/79 (15/14)	111/65 (13/11)	-15/14 (10/11)	12/18	>0.1*
Primary:							
Mild-moderate	460	47 (14)	142/89 (15/13)	127/75 (14/11)	-15/14 (9/7)	11/16	>0.1*
Moderate-severe	30	52 (17)	154/10 (21/16)	138/87 (21/15)	-16/13 (12/10)	10/13	>0.1*
Renal:							
Chronic glomerulonephritis	58	43 (11)	154/96 (25/19)	150/88 (28/19)	-4/8 (10/14)	3/8	<0.01
Renal artery stenosis	52	54 (8)	155/94 (23/19)	148/85 (22/17)	-7/9 (12/9)	5/10	<0.05
Kidney transplantation	44	43 (12)	135/87 (20/13)	132/84 (20/15)	-3/3 (11/8)	2/3	<0.01
Haemodialysis	30	49 (14)	134/86 (23/15)	129/83 (22/17)	-5/3 (12/8)	4/5	<0.01
Diabetic nephropathy	24	59 (8)	149/86 (22/17)	146/82 (27/18)	-3/4 (10/8)	2/5	<0.01
Endocrine:							
Hyperthyroidism	14	55 (17)	141/85 (21/17)	135/77 (21/15)	-6/8 (10/9)	4/9	<0.05
Primary hyperaldosteronism	12	54 (22)	160/107 (23/18)	156/100 (22/17)	-4/7 (9/10)	3/7	<0.05
Primary hyperparathyroidism	9	61 (13)	139/86 (19/18)	123/70 (19/16)	-16/16 (8/9)	12/19	>0.1
Phaeochromocytoma	6	53 (18)	162/82 (27/16)	168/85 (32/14)	+4/2 (16/17)	2/2	<0.001
Cushing's syndrome	2	46 (19)	146/95 (20/12)	143/89 (17/12)	-3/6 (12/14)	2/6	<0.01
Coarctation of aorta (not operated)	3	21 (15)	147/93 (21/20)	127/76 (20/18)	-20/17 (6/5)	14/18	>0.1

*White coat and primary hypertension v normotension; all other P values are related to normotension and primary hypertension.

Clinical implications

- Systolic and diastolic blood pressure follow a circadian rhythm in normotensive people and those with hypertension, values being lower at night than during the day
- An inverse circadian rhythm in blood pressure has been reported in some forms of secondary hypertension
- This study found a blunted circadian blood pressure curve in all renal forms of hypertension and in all endocrine forms except primary hyperparathyroidism, but the circadian rhythm was normal in patients with unoperated coarctation of the aorta
- The fall in nocturnal blood pressure was about 15 mm Hg in systolic and diastolic readings (by 11% and 17% respectively) in normotensive subjects and those with primary hypertension
- The change in nocturnal blood pressure could aid the differential diagnosis of secondary hypertension

renoparenchymal and renovascular hypertension. In some hypertensive subjects with endocrine disorders nocturnal blood pressure even increased (phaeochromocytoma), whereas others had a significantly smaller fall in blood pressure (hyperthyroidism, primary hyperaldosteronism, and Cushing's syndrome). In patients with hypertension and primary hyperparathyroidism we surprisingly found a normal fall in nocturnal blood pressure. We also found a normal fall in three patients with unoperated coarctation of the aorta. This may indicate a purely mechanical mechanism for this form of hypertension. In endocrine

forms of secondary hypertension, however, as in primary aldosteronism, the blunted blood pressure curve could be due to increased hormonal activity over 24 hours.⁹

A lack of a fall in nocturnal blood pressure is not specific for secondary hypertension. If blood pressure does not sufficiently decrease during the night or even increases, however, further investigation may be warranted to diagnose secondary hypertension. A normal decrease in nocturnal blood pressure does not exclude secondary hypertension. The absence of an adequate fall in nocturnal blood pressure has important consequences for end organ damage and therefore for treatment.^{10 11}

We thank Bernard Doran for his linguistic advice.

- 1 Zadek I. Die Messung des Blutdrucks am Menschen mittels des Basch' schen Apparates. *Zeitschrift für Klinische Medizin* 1881;2:509-51.
- 2 Howell WHA. Contribution to the physiology of sleep, based on plethysmographic experiments. *J Exp Med* 1897;2:313.
- 3 Katsch G, Pansdorf H. Die Schlafbewegung des Blutdrucks. *Münch med Wschr* 1992;69:1715-8.
- 4 Hany S, Baumgart P, Frielingsdorf J, Vetter H, Vetter W. Circadian blood pressure variability in secondary and essential hypertension. *J Hypertens* 1987;5(suppl 5):487-9S.
- 5 Imai Y, Abe K, Sasaki S, Minami N, Nihei M, Munakata M. Altered circadian blood pressure rhythm in patients with Cushing's syndrome. *Hypertension* 1988;12:11.
- 6 Middeke M, Mika E, Schreiber MA, Beck B, Wächter B, Holzgreve H. Ambulante indirekte Blutdrucklangzeitmessung bei primärer und sekundärer Hypertonie. *Klin Wochenschr* 1989;67:713-6.
- 7 Middeke M, Klüglich M, Holzgreve H. Circadian blood pressure rhythm in primary and secondary hypertension. *Chronobiol Int* 1991;8:451-9.
- 8 Schrader J, Person C, Pferner U, Buhr-Schinner H, Schoel G, Warneke G, et al. Fehlender nächtlicher Blutdruckabfall in der 24-Stunden Blutdruckmessung: Hinweis auf eine sekundäre Hypertonie. *Klin Wochenschr* 1989; 67:659-65.
- 9 Kern DC, Weinberger MH, Gomez-Sanchez C. Circadian rhythm of plasma aldosterone concentration in patients with primary aldosteronism. *J Clin Invest* 1973;52:2272-7.
- 10 O'Brien E, Sheridan J, O'Malley K, Dippers and non-dippers. *Lancet* 1988;i:397.
- 11 Schrader J, Schoel G. Benefits of noninvasive ambulatory blood pressure monitoring during antihypertensive therapy. *Journal of Ambulatory Monitoring* 1990;3:203-14.

(Accepted 2 November 1993)

Effect of advertising on awareness of symptoms of diabetes among the general public: the British Diabetic Association Study

Baldev M Singh, Jeremy J W Prescott, Roland Guy, Simon Walford, Moira Murphy, Peter H Wise

Abstract

Objective—To determine the impact of posters advertising symptoms of diabetes on public knowledge of these symptoms.

Design—Structured street interviews of members of the general public before, at the end of, and 10 weeks after a campaign advertising the main symptoms of diabetes.

Setting—Basingstoke and Wolverhampton.

Subjects—Three samples of 1000 members of the general public were interviewed. Samples were selected randomly but stratified to match the local population's age (20-75), sex, social class, and racial characteristics.

Main outcome measures—Knowledge of symptoms of diabetes; perceived seriousness of diabetes; and induction of anxiety about symptoms in the target population.

Results—Advertising significantly raised knowledge (without prompting) of symptoms: thirst, 245 before v 411 at end of campaign ($P < 0.0001$) v 341 after ($P = 0.0012$ v before); polyuria, 72 v 101 ($P = 0.0211$) v 92 ($P = 0.5169$); lethargy, 180 v 373 ($P < 0.0001$) v 298 ($P < 0.0001$); knowledge of weight loss and visual disturbance was unaffected. The number of subjects lacking knowledge of any symp-

oms was reduced from 550 to 388 ($P < 0.0001$). The perceived seriousness of diabetes was unaffected (mean 7.6 in each phase on a scale of 1 (not) to 10 (very). Before advertising, 449 (45%) claimed to have one or more symptoms of diabetes, but this number fell at the end of the campaign (403; $P = 0.0419$) and 10 weeks afterwards (278; $P < 0.0001$).

Conclusions—An advertising campaign raised public knowledge of diabetes symptoms without inducing fear of diabetes or anxiety about symptoms. Its potential for achieving earlier detection of non-insulin dependent diabetes should be evaluated.

Introduction

In patients with non-insulin dependent diabetes mellitus, microvascular disease at diagnosis is attributed to prolonged asymptomatic hyperglycaemia.¹⁻³ We have recently shown, however, that non-insulin dependent diabetes is infrequently asymptomatic. When systematically questioned, 93% of newly diagnosed patients reported classic symptoms, often previously ignored; 40% had had these symptoms for 12 months or more.⁴ This failure to recognise symptoms may reflect the general public's lack of knowledge of the symptoms of diabetes—half are unable to name any

Department of
Endocrinology, Charing
Cross Hospital, London
W6 8RF
Baldev M Singh, senior
registrar
Peter H Wise, consultant

Kilmartin-Baker Ltd,
London WC2E 9OS
Jeremy J W Prescott,
managing director

Basingstoke District
Hospital, Basingstoke
RG24 9NA
Roland Guy, consultant
physician

New Cross Hospital,
Wolverhampton
WV10 0QP
Simon Walford, consultant
physician

British Diabetic
Association, London
W1M 0BD
Moira Murphy, research
director

Correspondence to:
Dr Singh.

BMJ 1994;308:632-6