objections to the previous research that looked at stress and the development of cancer. In particular, we rigorously controlled for the main physical and pathological factors that are known to influence prognosis in breast cancer. A measure of life stressors was used that accurately dated events and difficulties to ensure that they preceded the clinical onset of progression of the disease. This measure also attempted to assess the objective threat of life events and difficulties independently of both the subject's emotional reaction and investigator bias. This is important as neither the patient nor the investigator could be blinded to the woman's disease state. Unconsciously motivated differences in the subject's recall and interviewer's techniques remain a possible source of bias. If these were operating, however, a more systematic excess of both non-severe and severe stressors among the women with a relapse would be expected.

The small numbers of women who participated in this study must be borne in mind when interpreting the results, a factor that is reflected in the wide confidence intervals associated with the estimates of relative risk. The findings of this study now need to be corroborated in a large prospective investigation.

The mechanism whereby stress might affect the relapse of breast cancer is unknown. Suggested intermediaries include the neuroendocrine⁹ and immune systems,^{10,11} which could promote growth of previously dormant or subclinical metastases. Investigations of this are complex and difficult.¹²⁻¹⁴ Modifications in behaviour leading to direct exposure to carcinogens must also be considered as a possible mediating process.

The impact of severe life stressors on the recurrence of breast cancer may be modified by other psychosocial factors. Further analysis is required to explore the interaction between severe life stressors and variables such as coping behaviour and social support, both of which have been suggested as prognostic factors in themselves.¹⁵⁻¹⁷ Understanding the nature of such

interactions may have important implications for managing patients and for the development of cognitive^{18,19} and other psychological treatments aimed at helping patients with cancer adjust to the impact of their disease and cope with the consequences of subsequent severe life stressors.

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Smoking in hospitals: a measure of improvement

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The argument for restrictions on smoking in health service premises is clear. Most smokers and non-smokers support restrictions in hospitals,¹ and the Department of Health and Social Security set out firmly the exemplary role that health authorities should have in the campaign to stop smoking.²

A survey of all health service premises in the Wessex region was undertaken in 1981, and the information obtained was used to set goals for reducing smoking in hospitals and health centres by 1985.^{3,4} We carried out a study in 1985 to monitor the progress towards these goals and to identify improvements that might be made.

Methods and results

We asked administrators of all health service premises in Wessex to complete a postal questionnaire identical with that used in the survey in 1981.³ We requested information on the extent of restrictions on smoking in their premises; how restrictions on smoking were identified and monitored; and sales of cigarettes. We inspected a sample of the premises to

validate the results. Replies were received from 246 of the 250 premises.

RESTRICTIONS ON SMOKING IN PUBLIC AREAS

Short stay hospitals (n=61)—The proportion of hospitals achieving the recommended goals in wards and outpatient and other public areas had increased since 1981 (table). Only 23 of the hospitals, however, had achieved the desired goal for day rooms. Fifty three offered day rooms where smoking was permitted, while only 26 provided day rooms that were always smoke free.

Maternity hospitals (n=7)—The high level of restrictions found in 1981 was maintained, with no smoking in any of the wards. Restrictions in day rooms were less satisfactory.

Psychiatric hospitals (n=32)—Low levels of restrictions were found in all areas.

Long stay and geriatric hospitals (n=45)—The proportion of premises with satisfactory restrictions had increased since 1981.

Health centres (n=44)—Forty two of the health centres had a total ban on smoking in public areas.

The task of monitoring restrictions was undertaken by nurses and administrators, while doctors had a minor role.

RESTRICTIONS ON SMOKING IN STAFF AREAS

Canteen and restaurant facilities—Restrictions had increased since 1981, but one third of long stay and

Area of premises	Goal (% of floorspace designated a no smoking area)	Type of hospital							
		Short stay		Maternity		Psychiatric		Long stay and geriatric	
		1981 (n=60)	1985 (n=61)	1981 (n=9)	1985 (n=7)	1981 (n=16)	1985 (n=32)	1981 (n=29)	1985 (n=45)
Wards	100	42 (70)	51 (83)	9 (100)	7 (100)	5 (33)	11 (35)	16 (54)	32 (70)
Day rooms	60	10 (16)	23 (38)	1 (13)	1 (14)	1 (8)	9 (27)	3 (11)	20 (45)
Outpatient department	100	48 (80)	60 (98)	9 (100)	5 (67)	8 (47)	22 (68)	21 (71)	39 (87)
Public waiting areas	100	35 (58)	52 (86)	9 (100)	6 (80)	2 (14)	11 (33)	19 (65)	31 (68)

*Numbers of premises in 1985 vary from those in 1981 according to available facilities.

psychiatric hospitals did not have smoke free eating areas.
Coffee lounges and rest rooms—Less than one third of the premises had achieved their goal.

SALE OF CIGARETTES

Seven (11%) of the short stay hospitals and 12 (37%) of the psychiatric hospitals sold cigarettes to patients; none of the maternity hospitals did so. This represented a decrease since 1981. The proportion of long stay units selling cigarettes, however, had increased. Some short stay, psychiatric, and long stay hospitals also sold cigarettes to visitors and staff.

Comment

Considerable progress was made in restricting smoking in health service premises in Wessex after 1981. In some key areas, however, progress was

disappointing. Most people in all social groups are now non-smokers,⁵ yet most hospital day rooms allow smoking. The need to provide smoke free day rooms should be considered when new buildings are being planned, especially long stay and psychiatric premises. Sales of cigarettes to patients should be restricted to long stay hospitals, and low tar brands should be offered. One district in Wessex already does this, suggesting that it is a realistic goal.

Most NHS staff are non-smokers, yet many more premises offer rest areas for staff where smoking is permitted than offer smoke free areas. The NHS, as an important employer, has a considerable opportunity to promote non-smoking by providing smoke free working environments and advice and help to employees who wish to give up smoking. Handbooks and induction courses for staff are underused as opportunities to communicate policies on smoking to staff. The adoption of such policies in health service premises by health authorities will lead to change only if clear goals are set and progress towards these is monitored. A regional review can promote change by providing useful information for districts.

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Recurrent cardiovascular collapse due to surreptitious ingestion of propranolol

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Self poisoning can usually be diagnosed without difficulty, but sometimes patients deliberately conceal the ingestion of drugs and present with a confusing clinical picture.¹ Intensive investigation may then be needed and inappropriate treatment given. We present a case of repeated surreptitious self poisoning with propranolol.

Case report

In January 1985 a 28 year old woman presented with "dizziness." She was hypotensive (blood pressure 70/30 mm Hg) with a regular bradycardia. An electrocardiogram showed a junctional bradycardia (45 beats/minute), absent P waves, and a broadened QRS complex. Her condition improved after she received intravenous atropine: the junctional rate

increased to 65 beats/minute and the QRS complex narrowed (figure). She recovered completely in the next 24 hours without further specific treatment.

Several investigations were performed. Baseline haematological and biochemical values, including activity of cardiac enzymes, were normal. No sequential ST or T wave changes were seen on electrocardiography, and a chest x ray film and echocardiogram were both normal. Exercise testing was limited by leg fatigue at a low workload. Electrophysiological studies performed eight days after her admission showed minor dysfunction of the sinus node and isolated impairment of atrioventricular nodal conduction. Her serum was positive for antinuclear factor (1/256), but results of a DNA binding assay were in the normal range. No clear diagnosis was made. She was discharged for follow up as an outpatient.

Two months later she was again admitted with a symptomatic junctional bradycardia that resolved spontaneously within hours. Because of these repeated episodes a dual chamber sequential permanent pacemaker was inserted. A further brief admission with hypotension occurred in November 1985, at which time the pacemaker was functioning satisfactorily.

Early in 1986 she was admitted with profound hypotension, convulsions, and vomiting. Her heart rate was 72 beats/minute, and an electrocardiogram confirmed sequential atrioventricular pacing. Despite volume expansion guided by central venous pressure monitoring and inotropic support she remained hypotensive and oliguric for eight hours, but she eventually made a full recovery.

An interview with her partner elicited that he regularly attended the hospital with ischaemic heart disease. Despite recent coronary artery surgery he had had frequent admissions with chest pain and was being treated with propranolol and verapamil. Both he and

Electrocardiograms (a) before and (b) after intravenous atropine was given