arterial pressure is the primary cause.[6 17 18] We have suggested that increased arterial pressure acts by damaging blood vessels and promoting intravascular coagulation, which is responsible for the main features of the disease. Smoking could have its effect at this stage of the process, since it increases platelet stickiness,[19] the viscosity of blood,[20] and the likelihood of arterial thrombosis.[1]

In the group with malignant hypertension mortality was higher among smokers. Increased mortality would be expected in any group containing a high proportion of smokers, but a 36% mortality rate over four years in a group with a mean presenting age of 44 years is more than could be expected from this alone. The mortality rate among smokers with non-malignant hypertension was only 2·4% during the same period. Smoking was also related to renal failure, and, as would be expected,[6] renal failure was related to a fatal outcome. This raises the possibility that smoking predisposes to malignant hypertension and to a fatal outcome from renal failure. The two effects are not necessarily independent. The characteristic vascular abnormality of malignant-phase hypertension in the kidney is the same as that in other susceptible organs. The intravascular coagulation in the malignant phase, which may be produced by smoking, is particularly pronounced within the kidney.

People who stop smoking are less likely to develop vascular disease. Those with vascular disease decrease the chance of further complication when they stop smoking.[1 2 16] On the evidence presented here we believe that patients with hypertension should be advised to stop smoking. At worst their chance of developing cancer and several important vascular diseases will be reduced when they stop; at best there may be additional protection from malignant-phase hypertension, a particularly lethal form of vascular disease.

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Malignant hypertension and cigarette smoking

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Summary and conclusions

The smoking habits of 48 patients with malignant hypertension were compared with those of 92 consecutive patients with non-malignant hypertension. Thirty-three of the patients with malignant and 34 of the patients with non-malignant hypertension were smokers when first diagnosed. This difference was significant, and remained so when only men or black and white patients were considered separately.

Results suggest that malignant hypertension is yet another disease related to cigarette smoking.

Introduction

Epidemiological and clinical studies have consistently failed to show any relation between cigarette smoking and hypertension.[1-3] The present study was prompted by a clinical impression of a higher incidence of cigarette smoking among patients with malignant-phase hypertension when compared with those with non-malignant hypertension, and a report of an association between cigarette smoking and diabetic retinopathy.[4] We therefore analysed the smoking habits of all patients with malignant-phase hypertension admitted to this hospital over the past six years and compared them with 92 consecutive patients with non-malignant hypertension who were attending the blood-pressure clinic at this hospital. A similar and parallel study was conducted by colleagues in Glasgow.[5]

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Patients and methods

We obtained the case records of 48 consecutive cases of malignant-phase hypertension admitted to this hospital during 1972-8. The criterion for inclusion in this group was the presence of bilateral retinal haemorrhages or exudates with or without papilloedema. As this analysis was conducted retrospectively by examining the notes we could include only cases whose ICD diagnostic code was 400 (malignant or accelerated hypertension). Notes were checked to ensure that the retinal changes had been recorded. Patients with renal disease were not excluded; four patients had chronic pyelonephritis and one congenital hypoplastic kidneys. One patient had current phaeochromocytoma; the remainder had essential hypertension. Of the 48 patients, 16 had a serum urea concentration above 10 mmol/l (60 mg/100 ml). The series included six diabetics (fasting blood glucose >6·5 mmol/l (>117 mg/100 ml)), of whom four smoked;

these four patients, however, also had bilateral papilloedema, which is not normally a feature of diabetic retinopathy, and thus we thought that their inclusion would not invalidate our findings.

The control group of patients with non-malignant hypertension was taken from the first 92 cases attending the newly created blood-pressure clinic. All had had diastolic blood pressures greater than 100 mm Hg when first referred to the clinic. In both groups of patients we considered only the smoking habits at the time of diagnosis. In addition, data on the smoking habits of the general population[6] were compared with our own data.

Statistical analysis using the χ^2 test was carried out for the group as a whole and for subgroups by race (white, black, and Asian) and sex.

Results

Of the patients with malignant hypertension, 25 of the 33 men (76%) and eight of the 15 women (53%) were smokers. By contrast, of the patients with non-malignant hypertension, only 21 (44%) of the 48 men and 13 (30%) of the 44 women were smokers. Data from the *General Household Survey*[6] showed intermediate figures for cigarette smoking in comparable age groups in the general population (51% of men and 41% of women; table I). This excess of cigarette smoking among patients with malignant as compared with those with non-malignant hypertension was highly significant ($\chi^2 = 13{\cdot}84$, $P < 0{\cdot}001$). When the different sexes and racial groups were analysed separately (table II) the excess of smoking among patients with malignant hypertension was significant for all men ($\chi^2 = 6{\cdot}91$, $P < 0{\cdot}01$), all white patients ($\chi^2 = 5{\cdot}31$, $P < 0{\cdot}05$), and all black patients ($\chi^2 = 7{\cdot}83$, $P < 0{\cdot}01$). Data from women when racial groups were compared together or separately did not reach significance.

TABLE I—*Number (%) of male and female smokers in study group according to type of hypertension, and percentage in general population*

Hypertension	Men	Women
Malignant (n = 48)	25 (76)	8 (53)
Non-malignant (n = 92)	21 (44)	13 (30)
% in general population[6]	51	41

The average age of the white patients with malignant-phase hypertension was 46 years and that of the white patients with benign hypertension 57 years ($P < 0{\cdot}001$), but no significant difference was found in age between the two groups of black patients (average age 47 years). Further analysis including only the younger patients with non-malignant hypertension showed that age distribution was unlikely to account for the smoking association in the white population, which remained significant ($P < 0{\cdot}05$).

Discussion

Our results show that cigarette smoking is associated with the development of advanced retinopathy in hypertensive patients, although numbers were too small to determine whether any individual aspect—that is, haemorrhages, exudates, or papilloedema—was particularly prevalent. This contrasts sharply with the lack of association between cigarette smoking and non-malignant hypertension.[3] The cases of malignant hypertension in this study were obtained over six years, whereas those with non-malignant hypertension were all current attenders. While there had been some decline in national cigarette consumption this is not enough to account for the present observation. Most epidemiological studies have shown no relation between smoking and the height of the blood pressure; indeed, as in the present study, the prevalence of smoking among patients with non-malignant hypertension is lower than that in the general population. It is unlikely that the presence of haemorrhagic retinopathy can be accounted for by the height of the blood pressure alone, and possibly cigarette smoking is a factor that makes patients with hypertension develop the malignant phase. The untreated blood-pressure levels among the attenders at the hypertension clinic were usually not available, as patients were referred to the clinic while receiving drug treatment, but in at least five cases pressures were considerably higher than those seen in the malignant group. An association between cigarette smoking and the malignant phase of hypertension is, because of its rarity, unlikely to be detected in population studies or analyses of outpatient blood-pressure clinics.

Paetkau *et al*[4] recently reported an association of smoking with the proliferative type of diabetic retinopathy, although this observation was later disputed.[7] In considering a possible causative relation they noted the reported effects of smoking on blood carboxyhaemoglobin concentrations and the evidence that carbon monoxide may affect the separation of endothelial cells.[8] Hawkins[9] found that smoking increased platelet stickiness, a factor associated with the pathogenesis of diabetic retinopathy. All these mechanisms may be relevant to our findings, but in addition Pichon *et al*[10] showed that inhaling cigarette smoke caused ophthalmic artery tension to rise in 60% of their subjects. They attributed this rise to catecholamines released by nicotine, but it may reflect the rise in systemic blood pressure noted in acute experiments on cigarette smoking.[11]

Fundal exudates or haemorrhages are a characteristic of malignant hypertension[12] with its attendant high mortality and morbidity rates from renal failure, heart failure, heart attack, and stroke. The identification of factors promoting their formation is valuable. As the risk of coronary heart disease and cerebrovascular accidents is much increased in smokers with non-malignant hypertension compared with non-smokers with hypertension of comparable severity,[2] the added risk of developing the malignant phase makes the case for encouraging abstention from smoking even stronger. Our results suggest that at least this particularly severe form of hypertension may be added to the list of diseases related to cigarette smoking.

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Requests for reprints should be addressed to DGB.

TABLE II—*Numbers of smokers and non-smokers with benign and malignant-phase hypertension according to racial group*

Type of hypertension	Racial group					
	White		Black		Asian	
	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers
			Women			
Benign	8	15	5	14		2
Malignant	7	5	1	1		1
			Men			
Benign	14	15	2	5	5	7
Malignant	15	4	10	2		2

Significance of difference: all white patients $\chi^2 = 5{\cdot}31$, $P < 0{\cdot}05$; all black patients $\chi^2 = 7{\cdot}83$, $P < 0{\cdot}01$; all men $\chi^2 = 6{\cdot}91$, $P < 0{\cdot}01$; all women $\chi^2 = 1{\cdot}82$, $P < 0{\cdot}5$.

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Study of special-care baby services in North-west Thames region

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Summary and conclusions

During three months in 1975 admissions to 17 of the 21 special-care baby units in the North-west Thames region were analysed by birth weight and category of care. Of the 1718 babies admitted, one-third needed only observation. Neonatal intensive care formed only a small proportion of the work load in most units. Considerable variation in the pattern of admissions was found. There was some evidence of concentration, about 100 babies being transferred for urgent medical or surgical reasons, and the work load of one unit suggested that it was serving as a referral centre.

It is concluded that the quality of care given to some infants needing intensive care might be improved by greater concentration, and that some units should review their admission policies in order to prevent unnecessary postpartum separation of mother and baby.

Introduction

There is good evidence in Britain[1-3] of the beneficial effect of modern intensive neonatal care in improving the prognosis of infants of very low birth weight and those suffering from other serious conditions, but little information is available on the overall work load in special-care baby units (SCBUs). In 1974 the provision of special-care baby cots in England was 36/100 000 population aged 0-14 years, but this ranged between areas from seven to 99.[4] Nationally[4 5] about 15% of all live-born infants are admitted to SCBUs, but little is known about the reasons for admission or the type of care given. Alberman *et al*[6] reported the results of one-day censuses in three Thames health regions and showed that although there appeared to be some overall overprovision of cots, many SCBUs were smaller than recommended by the Expert Group on Special Care for Babies[7] and had deficiencies of equipment and staffing. Alberman *et al* acknowledged the limitations of the "cross-sectional" approach in estimating work load, and we have attempted to overcome these by surveying all admissions to SCBUs in one Thames health region over three months. Since the one-day census reported was performed while our study was in progress, the data presented here are additional and complementary to those given for the North-west Thames health authority by Alberman *et al*.

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Method

Each of the 21 SCBUs in the North-west Thames region was asked to complete a form on every baby admitted during January to March 1975. Information included birth weight, length of stay, reason for admission, reason for transfer (if applicable), investigations performed, and treatment given. The number of forms returned each month was compared with the number of recorded admissions. Twenty units agreed to participate, and 17 returned completed forms on 89% or more of admissions. We classified infants by birth weight—namely, normal (>2500 g), low (1501-2500 g), and very low (≤1500 g) and type of care given—namely, transitional, special, and intensive.

Transitional care—We included in this category babies who fulfilled all the following criteria: (*a*) were admitted at birth for observation; (*b*) had a birth weight exceeding 2500 g; (*c*) stayed in the unit for three days or less; and (*d*) had no special investigations or treatment.

Special care—This group comprised babies not included in the transitional or intensive care category.

Intensive care—Babies in this category fulfilled one or more of the following criteria: (*a*) had a birth weight of 1250 g or less; (*b*) were transferred to another SCBU or hospital for medical or surgical treatment; (*c*) had continuous positive airway pressure (CPAP), mechanical ventilation (IPPV), exchange transfusion, or surgery; or (*d*) died.

Results

There were 1718 admissions (including transfers from other SCBUs) to the 17 units. One unit had 36 admissions, 14 had between 50 and 150 admissions, and two had 230 and 280 admissions respectively. The mean proportion of babies admitted to the SCBUs as indicated by the total number of admissions, excluding babies transferred in, related to the total number of live births in the associated maternity departments was 24·7%, ranging from 14% to 47% in individual hospitals. Six units admitted 10-20% of such infants, five units admitted 20-30%, and four units 30-40%; one unit admitted 47% of its maternity department's live-born infants, and one SCBU had no associated maternity unit, so all of its admissions were "transfers in."

Forms were returned on 1654 (96·3%) of the 1718 babies admitted, and we estimate that this sample constituted 77% of all admissions to the 21 units. Over two-thirds of the infants were of normal birth weight (table I).