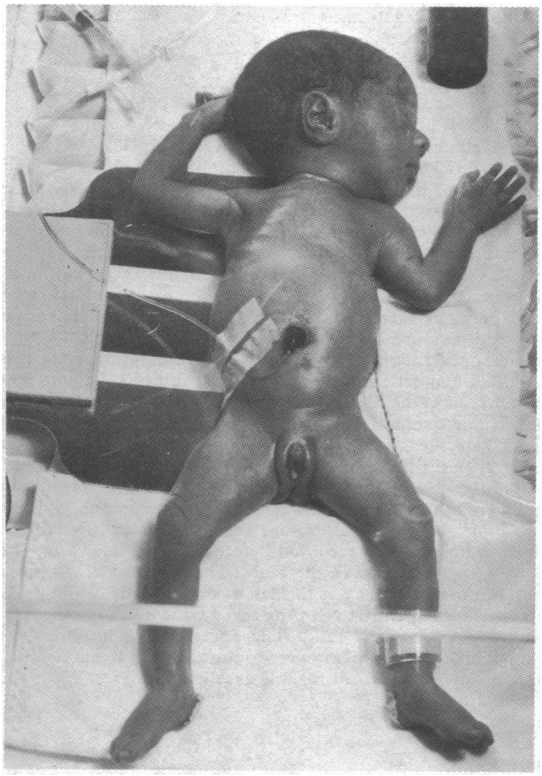


An infant of 1200 g with the leading edge of a shortened electrode mat inserted under her right side and lying on an apnoea mattress.

give rise to a false alarm unless the electrode is reduced in size and inserted so that it makes direct contact only with one side of the infant, leaving his other side lying directly on the apnoea mattress (as in the figure).

When monitoring during transport, the electrode mat was connected to a battery powered ECG monitor† which performed well and without interference during ambulance travel. Recent reviews from two centres indicate the great value of continuous heart rate monitoring during transfer.[4 5] Adequate observation during transit is difficult for many reasons: background noise renders heart and breath sounds inaudible and vibration obscures respiratory movement and accurate palpation of pulses. Bradycardia is an extremely important sign in any sick newborn infant, and particularly during transport, when a high proportion of babies (49%) have endotracheal tubes in situ.[5] Here, early recognition of bradycardia provides immediate warning of accidental extubation or similar impending problems.

Because the mat warmed quickly in the incubator infants were placed naked on the electrode and then wrapped round with warm blankets. Continuous monitoring ensures better protection from heat loss as no part of the infant need be left exposed for observation of respiratory movement or skin colour. In both babies transported a small rise in rectal temperature occurred during the journey.

Conclusion

This electrode mat is not intended to replace conventional electrodes. Nevertheless, it has great advantages of speed and ease of insertion, lack of trauma and minimal disturbance, together with the benefits of simplified monitoring during transport. No important problems were encountered provided the mat was reduced in size for small infants and excessive movement did not occur. We suggest that wider use of this electrode mat could simplify ECG and heart rate monitoring.

We are grateful to Dr G Katz and Dr K Norton for allowing us to study their patients and for the interest and care given to these infants by the medical and nursing staff of the units concerned.

*Intek Cardiomat ECG Electrode, Intek (UK) Limited, London.
†Visicard 8 portable cardioscope, Linton Instrumentation, Harlow.

[1] Julian, D G, *Medicine*, 1973, **17**, 1089.
[2] Klaus, M H, and Faranoff, A A, *Care of the High Risk Neonate*. Philadelphia, Saunders, 1973.
[3] Blake, A M, *et al*, *Lancet*, 1970, **2**, 183.
[4] Storrs, C N, and Taylor, M R H, *British Medical Journal*, 1970, **3**, 328.
[5] Blake, A M, *et al*, *British Medical Journal*, 1975, **4**, 13.

Intensive Therapy Unit, Barnet General Hospital and the Special Care Baby Units of Barnet and Edgware Hospitals

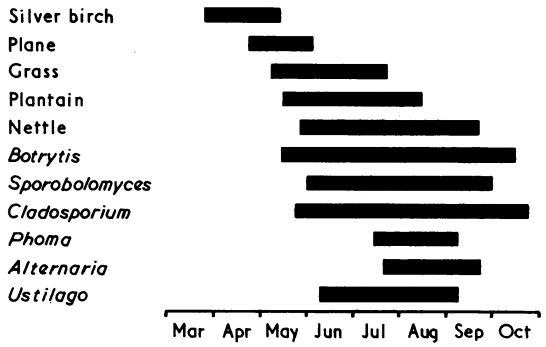
ANDREW J MACNAB, MB, BS, acting senior paediatric registrar (present address: Department of Paediatrics, University of British Columbia, Vancouver, BC)
ROSEMARY POPE, SRN, nursing officer, intensive therapy unit

Seasonal allergic symptoms due to fungal spores

The commonest cause of seasonal hay fever is a type 1 IgE-mediated allergic reaction to grass pollen, which is sometimes accompanied by asthma. In England several grasses release their pollen at about the same time; pollens from different grasses are antigenically similar and all can cause seasonal conjunctivitis, rhinitis, and asthma in susceptible patients. The diagnosis is suggested by the seasonal nature of the symptoms, which begin in the south of England towards the end of May, reach a peak towards the end of June, and diminish gradually during July. A simple prick test with a commercially available extract of grass pollen will produce an immediate weal and flare response, which correlates well with the results of direct intranasal challenge.[1] Some patients, however, complain of seasonal respiratory symptoms that do not coincide with the release of grass pollen. At any given time the atmosphere contains several other pollens[2] and a large variety of fungal spores,[3] some of which may cause seasonal respiratory symptoms. With the use of a Hirst spore trap[4] the air may be sampled continuously throughout the 24-hour period, both qualitatively and quantitatively, and the "count" of any particular pollen or spore expressed as the number of particles per cubic metre of air per 24 hours. The typical seasonal pattern of the common pollens and spores in central London is shown in the figure.

Patients, methods, and results

Patients who were referred to the allergy clinic with a diagnosis of seasonal rhinitis or asthma were asked to define closely the weeks of the year when their



Seasonal pattern of common pollens and spores in central London.

symptoms were present and at which period they were at their worst. They were then given prick tests with all the common airborne pollens and fungal spores. Allergy to a given pollen or spore was considered established if the symptoms occurred when the allergen was at maximal aerial concentration and if the patient showed a weal and flare response to the allergen greater than 5 mm diameter when prick tested and the results of prick tests with other allergens were negative.

Out of 2916 patients seen during 1973 (1425) and 1974 (1491), 2637 (90%) were allergic to grass pollen and 279 to other airborne allergens. Of these 279 patients, 86 (30·8%) were allergic to pollen of either the silver birch (*Betula*) or plane tree (*Platanus*) and had their symptoms between the end of March and the middle of May, and 156 (55·9%) were allergic to spores of *Alternaria tenuis* and had experienced their symptoms between the second half of July and the beginning of September. The remaining 37 patients were allergic to

spores of *Phoma betae* (19 patients; 6·8%), *Botrytis cinerea* (8 patients; 3·2%), *Cladosporium herbarum* (8 patients; 2·9%), *Sporobolomyces roseum* (1 patient; 0·4%). No patient was found to be allergic either clinically or on prick testing to *Penicillium notatum* (a perennial spore) or to *Ustilago tritici*. None were allergic to the pollen of nettle (*Urtica*) or plantain (*Plantago*), both pollens being abundant during the appropriate seasons.

Comment

The commonest fungal spore to cause seasonal allergy (*Alternaria*) did not exceed an aerial concentration of 900 spores/m³ air/24 hours in either year, and the most abundant spore (*Cladosporium*) was present in concentrations as high as 24 000 spores/m³ air/24 hours but rarely caused seasonal allergic symptoms. The absence of ocular symptoms in patients allergic to fungal spores may be due to the larger size and weight of spores compared with pollen grains, making them less likely to be blown into the eyes. This can be a helpful diagnostic pointer. When patients complain of hay fever or seasonal asthma in the early spring, allergy to tree pollens should be considered. Those with such symptoms in late summer and early autumn may be hypersensitive to the spores of *Alternaria*. In either case the diagnosis is strengthened if a prick test result with the suspected allergen is positive.

I thank Drs A W Frankland and M A Ganderton for allowing me to study patients under their care, Dr R R Davies for advice and criticism, and Brenda Gann for secretarial help.

[1] Morrow-Brown, H, in *Proceedings of 5th European Congress of Allergy*, p 301. Basle, Schwabe, 1962.
[2] Hyde, H A, *Acta Allergologica*, 1959, **13**, 186.
[3] Davies, R R, *Methods in Microbiology*, p 795. London, Academic Press, 1971.
[4] Hirst, J M, *Annals of Applied Biology*, 1954, **39**, 25.

Allergy Clinic, St Mary's Hospital, London W2 1NY
PAUL D BUISSERET, MB, MRCP, registrar

Prazosin: severe side effects are dose-dependent

Prazosin (Hypovase or Sinetens in Great Britain, Minipress in South Africa) is a new antihypertensive agent, whose efficacy has been established in many clinical trials.[1] Recently there have been several reports[2 3] of severe hypotension and collapse 30-90 minutes after the first 2-mg dose of prazosin. In a leaflet warning of this side effect of prazosin, the Committee on Safety of Medicines estimates that at least 1% of patients treated have experienced this reaction. In the course of two carefully controlled clinical trials of prazosin, the incidence of these first-dose reactions in our clinic was 16%. Most of these reactors were later re-challenged with 0·5 mg of prazosin. This paper is, to the best of my knowledge, the first published report of a re-challenge study of first-dose reactors to prazosin.

Methods and results

Seventy-four volunteers (who gave informed consent) attending the hypertension clinic at the Johannesburg General Hospital with mild or moderate[4] essential hypertension were admitted to either a double-blind crossover study[4] of prazosin *v* methyldopa, or to a double-blind crossover study of prazosin plus a beta-blocker *v* prazosin plus placebo. Twelve patients (16%) experienced (in descending order of frequency) dizziness, weakness, sweating, faintness, blurred vision, syncope, nausea, chest pain, or palpitations 5-180 (mean 43) minutes after the first dose of prazosin, 2 mg by mouth (see table). There was no correlation between the incidence of side effects and previous therapy, sex, age, weight, or pretreatment supine or standing blood pressure. Also there was no predictive value of the electrocardiogram, appearance of the chest *x*-ray film, intravenous pyelogram, urinary vanillyl mandelic acid, urine analysis, full blood count, blood urea and electrolytes, alkaline phosphatase, bilirubin, transaminases, Coombs test, antinuclear factor, or plasma renin activity—all of which were done routinely.

At a later date seven of the 12 patients volunteered for the re-challenge study. After at least three days off all antihypertensive treatment and at least eight weeks after the last dose of prazosin, the patients were given 0·5 mg prazosin by mouth. Blood pressure, supine and standing, was measured before and at half-hour intervals for one and a half hours after the test dose. The electrocardiogram was recorded before and one hour after the tablet. The results are summarised in the table. The standing blood pressure fell in all cases, but in no patient was this fall to anything that could be described as a hypotensive level. Only two of the seven patients had dizziness; in both cases this was transient and mild. There were no changes in the electrocardiogram. Three of the patients were continued on prazosin for 1-12 weeks; in all cases blood pressure control was excellent on 1·5 to 2 mg per day in divided doses.

Discussion

A 16% (12 out of 74) incidence of severe reactions to the first dose (2 mg) is unacceptable with any antihypertensive agent. The smaller dose (0·5 mg) produced mild dizziness in two of seven patients re-challenged. The predicted incidence of first-dose reactions in the original population of 74 patients can then be calculated as $2 \times 12 \div 7 \times 100 \div 74$ —that is, 4·6% of mild dizziness, which compares favourably with many other antihypertensives.

It is concluded that the hypotension and syncope reported after the first dose of prazosin is dose-dependent, and that the incidence of mild dizziness is brought down to tolerable levels by using an initial dose of not over 0·5 mg, and building up the daily dose slowly to the required level.

[1] Cotton, D W K (editor), *Prazosin—Evaluation of a New Antihypertensive Agent*. Excerpta Medica, Amsterdam, 1974.
[2] Gabriel, R, *et al*, *Lancet*, 1975, **1**, 1095.
[3] Bendall, M J, *et al*, *British Medical Journal*, 1975, **2**, 727.
[4] Bloom, D S, *et al*, *Current Therapeutic Research*, 1975, **18**, 144.

Departments of Physiology and Medicine, University of the Witwatersrand Medical School, Johannesburg, and Johannesburg General Hospital
CLIVE ROSENDORFF, PHD, MRCP, professor of physiology, senior physician, and director of hypertension clinic

Results in 12 patients who experienced symptoms after first dose of prazosin

Patient data					Symptoms after first dose (2 mg prazosin)										Re-challenge 0·5 mg prazosin			
Case No	Sex	Age	Weight (kg)	Pretreatment standing blood pressure (mm Hg)	Onset (min)	Sweaty	Faint	Weak	Dizzy	Blurred vision	Nausea	Syncope	Chest pain	Palpitations	Standing blood pressure (mm Hg) fall at ½ h	1 h	1½ h	Symptoms
1	F	58	68·5	205/109	5				+	+		+						
2	M	50	54·0	164/102	180		+		+		+				14/4	+4/2	+10/6	None
3	F	52	77·1	206/104	15		+							+				
4	F	58	78·5	165/114	60	+		+							5/6	1/10	7/20	None
5	F	71	62·6	180/110	60			+										
6	M	70	82·6	175/104	10	+		+	+	+					29/9	5/4	45/14	Slightly dizzy at 2 h
7	F	58	106·6	164/100	10				+							+28/10	2/0	None
8	F	54	64·4	170/108	60	+		+	+				+					
9	M	61	85·7	160/108	10				+			+			26/22	32/28	8/18	Slightly dizzy at 1 h
10	F	76	61·2	188/115	30			+	+	+					34/18	46/20	16/6	None
11	F	65	62·6	238/108	60			+							62/20	50/8	44/0	None
12	F	62	59·2	242/108	15		+		+									
Total		61	71·9	188/108	43	3	3	6	8	3	1	2	1	1	28/13	15/8	16/7	5 none, 2 dizzy