Mortality rates for respiratory tuberculosis for both sexes over 65 show a rise from 58 in 1938 to a peak of 81 in 1951 for men, but a steady decline from 22 to 13 for women.

All figures show a worsening for men over 65 compared with women in the last 17 years, so that by 1954 male notification and death rates were about five times the female rates.

Even if rising notification rates in old men are due to increased case-finding there is evidence that a large number of these cases exist to be found, and it is suggested that the problem of reactivation in old men merits further epidemiological study.

A series of 35 cases is presented of men fit up to age 65 who subsequently developed active disease, probably usually due to reactivation of old lesions. Cases are classified according to mode of onset, 46% showing no prominent chest symptoms, often in spite of extensive active disease.

Well-known social and medical predisposing factors were found in only about half the cases.

Some suggestions are made to promote earlier recognition of these cases in view of their obvious danger to the community.

I acknowledge with thanks the help received from Dr. S. A. C. Hunter, consultant chest physician, under whose care these cases have been treated; from other chest physicians in the area for the loan of radiographs; and from Dr. D. Thomson, of the Ministry of Health, for providing statistics. I would also thank Sister E. B. Wright and the nursing staff of St. George's Ward, Dorking General Hospital, Miss Mary Wright, the tuberculosis almoner, and Miss D. K. Mould, of the medical office, for their helpful co-operation. I am also grateful to Dr. J. G. Scadding, of the Institute of Diseases of the Chest, Brompton, for detailed criticism and advice.

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Discussing the extreme case of those with a total lack of capacity for guilt or moral sense, in the course of the second Freud centenary lecture on April 17, Dr. D. W. WINNICOTT said it was tempting to postulate a constitutional factor. But psycho-analysis pointed to another explanation—namely, that for these individuals the early environmental setting was not reliable enough for the development of the capacity for guilt sense to take place. It was in fact possible to watch the loss and the regain of the capacity for guilt sense in a child or adult with antisocial tendencies, and to assess the variations in environmental reliability which produced these effects. It was at this point of loss and regain of moral sense that one could study delinquency and recidivism. According to psychoanalytic theory, antisocial behaviour was never the same as the crime that belonged to the prototype of guilt feeling in the individual. In other words, the antisocial act was either done to make sense of inexplicable guilt, the prototype for which was under repression; or else it was done in an attempt to reach to guilt feeling that was repressed and could not be experienced. In the first category came all the relatively unimportant crimes of children and adults, such as bed-wetting, lying, and stealing; in the second the rarer and more ugly criminal acts.

ALLERGY TO MOULD SPORES IN BRITAIN

BY

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Untoward reactions from mould spores were first recorded by Charles Blackley (1873). The suggestion that moulds might be among the causes of asthma was revived half a century later by van Leeuwen (1924), and in the same year Cadham (1924) described three cases of asthma resulting from sensitization to the spores not of a mould but of another fungus—namely, wheat rust (*Puccinia*)—present in the atmosphere of the Canadian wheat fields. Van Leeuwen's views gained ground widely during the next few years (van Leeuwen, 1927; Hansen, 1928; Jiménez-Diaz and Sanchez Cuenca, 1931; Ellis, 1933). Sensitivity to *Alternaria* was described by Hopkins *et al.* (1930), to *Aspergillus fumigatus* by Bernton (1930), and to *Cladosporium* by Cobe (1932). Apart from Cadham's observations, respiratory allergy to fungi was at this period generally thought to be related to indoor habitats such as haylofts, granaries, damp cellars, and the like, and therefore of little general significance.

The first to focus attention on moulds as allergens of importance to the ordinary asthmatic was Feinberg (1935), who found that out of a group of 243 patients with respiratory allergy, 68 (or 28%) gave positive reactions to one or more mould extracts. He next showed, in collaboration with Little (Feinberg and Little, 1936), that mould spores were abundant in the air of Chicago during summer and autumn only, and went on to correlate the seasonal frequency of one kind of mould spore (*Alternaria*) with symptoms experienced by patients who were skin-sensitive to extracts of the same kind of spores (Feinberg, 1937). Later he made similar observations regarding *Cladosporium* (also known as *Hormodendrum*) and certain other kinds of mould spores, and stated that at least 20% of his allergic patients (as determined by both skin testing and history) were allergic to some kind of mould or other (Feinberg, 1944).

To-day in the U.S.A. mould spores are widely accepted as common allergens (Sheldon *et al.*, 1953) and indications of their probable significance in Europe have come from Sweden (Nilsby, 1949), Denmark (Flensborg and Samsøe-Jensen, 1950), France (Vallery-Radot *et al.*, 1950), and Spain (Alemany Vall, 1955). It is the purpose of this article to consider the evidence for the occurrence of mould allergy in Great Britain.

Preliminary Considerations

In order that any kind of plant spore shall be held responsible for giving rise to allergic symptoms, it must first be shown to be antigenic. Secondly, any antigenic kind of spore, in order to affect a significant number of people, must occur in the air in large numbers and over a wide area. Thirdly, before any given patient's illness can be diagnosed as an allergic reaction to a particular kind of spore it must be shown that he experienced his symptoms at a time or

times when the spores concerned were present in large quantities in the air. It follows that spores which have a marked seasonal periodicity are specially suitable for investigation.

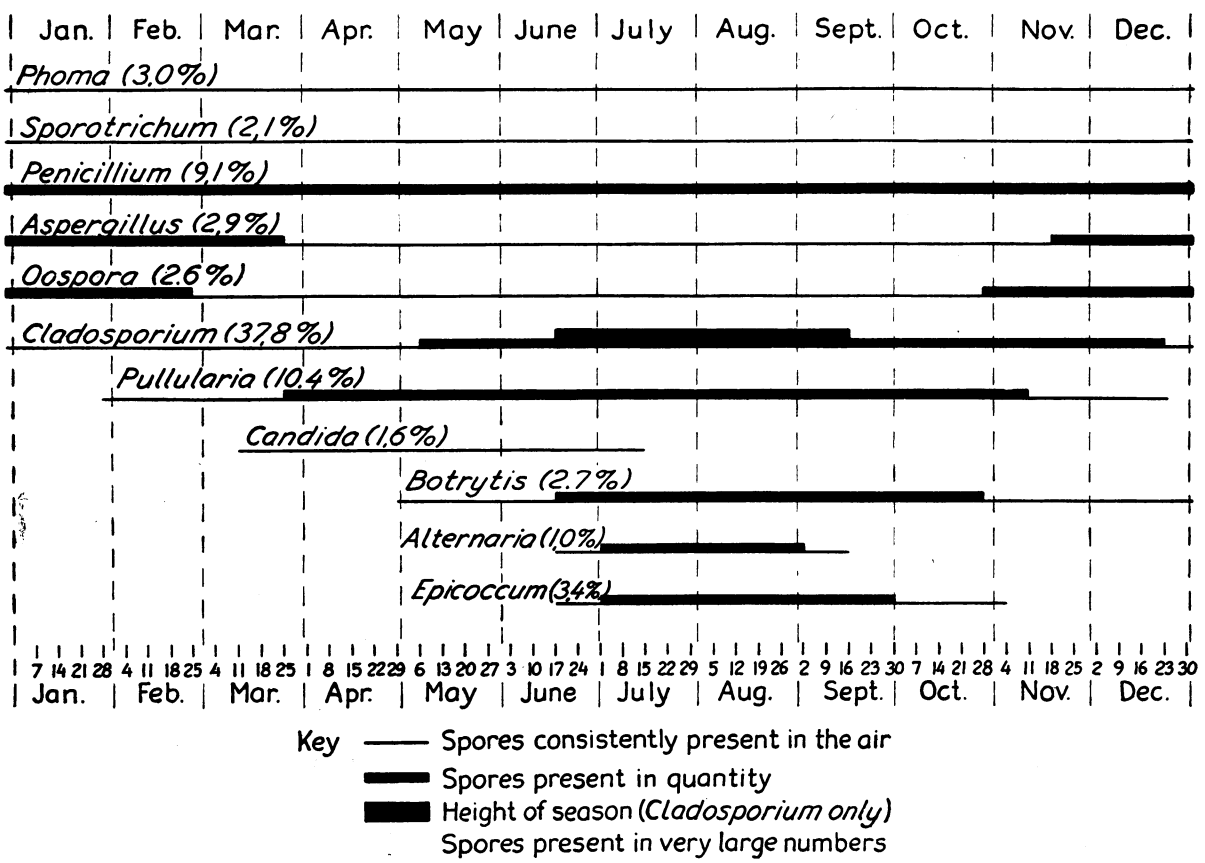
The above requirements were first laid down for pollen allergy: they hold also for mould allergy, but here they are more difficult to comply with, for two reasons: (1) the spores of but few mould genera are determinable without culturing; (2) the sources of mould spores—the mould plants—are microscopic and difficult to trace. The search for likely mould allergens, therefore, demands widespread atmospheric sampling at short intervals all the year round. The methods employed involve spore trapping with or without the use of culture media: no method so far described is completely satisfactory for continuous use, but, as is shown, the results obtained to date justify the presumption that certain kinds of mould spores prevalent in Great Britain act as allergens.

Aerobiological Observations

In view of the importance attributed to *Alternaria* in the U.S.A., Hyde and Williams (1946) made daily counts of the spores of this mould caught on slides which had been exposed primarily for pollen at Llandough, near Cardiff. They found that the total annual catch at Cardiff was less than one-tenth that at Chicago, and the maximum daily catch relatively still lower: the Cardiff counts were comparable with those on the eastern seaboard of the U.S.A. They also found *Alternaria* spores in large numbers in the dust from threshing machines. Later counts, on slides exposed at a number of centres and over several years, have provided additional evidence that, although sensitivity to *Alternaria* might be expected to occur in this country, it is unlikely to be as common here as it is in the American Middle West.

The same authors carried on a mould survey by means of Petri plates at Cardiff for over three years from late 1947 onwards (Hyde and Williams, 1949, 1953). Their counts appeared to indicate a higher incidence of *Cladosporium* than had been recorded anywhere else. Very high concentrations of *Cladosporium* spores during summer have also been observed in this country by Gregory (1952), Ainsworth (1952), and Hirst (1953). Other moulds, notably *Penicillium, Pullularia, Phoma, Botrytis, Aspergillus,* and *Alternaria,* appeared regularly on the Cardiff plates though in much smaller numbers (Hyde and Williams, 1949). This survey was continued by Richards from 1951 onwards (Richards, 1953a) and, with the assistance and collaboration of several colleagues, was extended to seven other stations in Great Britain—namely, Cambridge, Harpenden, London, Southampton, York, Liverpool, and Edinburgh. During the three years 1951 to 1953, over 1,900 plates were exposed outdoors at Cardiff alone, and nearly 16,000 colonies were determined. While nearly a hundred genera were represented, 96% of the total catch (other than sterile colonies) belonged to one or other of 11 genera only—as follows: *Cladosporium* (*Hormodendrum*) 37.8%, *Pullularia* 10.4%, *Penicillium* 9.1%, *Epicoccum* 3.4%, *Phoma* 3.0%, *Aspergillus* 2.9%, *Botrytis* 2.7%, *Oospora* 2.6%, *Sporotrichum* 2.1%, *Alternaria* 1.0% (plus a possible 1.3% represented by dark but non-sporing colonies), *Candida* 1.6% (Richards, 1954a). Seasonal variation was more or less marked in the majority of the genera named; six had a summer predominance—namely, *Cladosporium, Pullularia, Epicoccum, Botrytis, Alternaria,* and *Candida*; two, *Aspergillus* and *Oospora,* were more common in the winter; only the remaining three, *Phoma, Sporotrichum,* and *Penicillium,* showed no seasonal variation. A mould calendar based on the three years' observations is shown in the Diagram (Richards, 1954b).

The general pattern of atmospheric mould incidence at Cardiff was found to hold good for the out-stations also. No mould other than the 11 named above occurred in quantity at any station, and all these 11 exhibited the same



A calendar of the predominant atmospheric moulds. The thin line indicates weekly catches of 2 or more colonies; the thicker line, weekly catches of 12 or more colonies. The very thick line, for *Cladosporium* only, indicates mean weekly catches of between 50 and 250 colonies. (Reproduced from *Acta Allergologica,* 1954, 7, 357.)

seasonal variation everywhere. Two outstanding differences between the various stations were noted, however; first, *Cladosporium* was much more abundant in rural areas, where it formed 70 to 80% of the total catch, as against 40% in the large towns; second, *Penicillium* was more abundant in the large towns than elsewhere, though at all stations its annual incidence was lower than that of *Cladosporium* (Richards, 1953a).

These results permit of two inferences: (1) when summer asthma is suspected of being due to culturable moulds inhaled outdoors, the 11 genera listed above (all of which have been considered, if not proved, to be antigenic) would seem to be the only likely causes; (2) some, perhaps the majority, of the patients concerned might be expected to experience an exacerbation of symptoms on going from the town into the country.

The above conclusions apply to outdoor air. The mould flora of houses has not so far been extensively studied. Richards (1954a) has shown that the atmospheric mould-spore content of a bedroom systematically sampled by him for a year was only a pale reflection of the outdoor spora. Other houses, the walls of which were visibly mould-infested, gave evidence of higher concentrations. The contents of all the various rooms concerned had been left undisturbed for at least one and a half hours before the exposures were made. Disturbances of bedding, and brushing of carpets and walls, as might be expected, and as was shown by Maunsell (1952), cause a marked rise in the number of mould spores in the surrounding air.

Clinical Investigations at Cardiff

Clinical investigations have followed on the lines suggested by the botanical findings. Routine skin-testing has been carried out on the ordinary cases of asthma seen at the Cardiff clinic, and these, together with a comparatively small number from one outside clinic, form the basis of the results presented. Skin tests have been made intradermally with extracts (all made in our own laboratory) from cultures of the principal moulds caught from the air. A minimal quantity (0.02 ml.) of 1/100 extracts was introduced into the skin. No skin test was recorded as positive unless confirmed at least once by further testing.

We have come to recognize a case of mould sensitivity by the occurrence of seasonal asthma accompanied by relatively little or no rhinitis or conjunctivitis: this is so different from the clinical picture of grass-pollen sensitivity, with its typical conjunctivitis and rhinitis, as to be almost diagnostic. Nevertheless, we have regarded a patient as clinically sensitive to one or other kind of mould spore if, and only if, (*a*) asthma was confined to, or exacerbated during, that part of the summer in which the mould spores concerned were known to be abundant in the air; (*b*) the skin test repeatedly gave an undoubtedly positive result; and (*c*) other factors, such as sensitivity to other allergens, psychological influence, or infective conditions, even if present, appeared to be playing no important part in the aetiology of the summer asthma. In order to be sure that this last condition held, the patients, in all but clear-cut and severe cases, were kept under observation but without specific mould treatment for a year, or even longer, before specific mould therapy was begun.

Strict adherence to the above requirements has resulted in the exclusion of many cases with positive skin tests to moulds, and possibly in an underestimation of the part played by mould spores in asthma; but we have thought it prudent first to establish that clear-cut cases of mould sensitivity do occur and to become familiar with the clinical histories of such cases. Details of our findings are given in the following paragraphs.

Sensitivity to Cladosporium

Of 627 patients skin-tested, 52 (8.3%) gave repeated positive reactions. For various reasons, however, 26 of these cases were not regarded as clinically sensitive. Ten were excluded at once because there was no convincing evidence that their asthma was exacerbated during the *Cladosporium* season; a further seven were written off as essentially cases of sensitivity to house dust; four more, though considered sensitive to mould spores, were adjudged not to be sensitive predominantly to *Cladosporium*; two were sensitive to grass pollen, both clinically and by skin-testing; one was predominantly a psychological case; and, finally, one, although apparently worse in the summer, appeared to be basically a case of infective asthma. All these were therefore excluded for the reasons stated.

There remained 26 other patients (4% of the 627 patients tested) who were finally adjudged to be clinically sensitive to *Cladosporium*. In all of these, asthma was confined to, or occurred predominantly in, the summer, and thus coincided with the *Cladosporium* season. Eleven of them gave no other positive skin reactions or any indications of other aetiological factors. The other 15 gave positive reactions to different allergens—namely, house dust (all 15), grass pollen (4), or other mould spores (1). In none of these 15, however, was house dust considered, after the usual "anti-house-dust" advice and observation, to be clinically important; none of the four who gave positive reactions to grass pollen showed any symptoms of hay-fever, and in the one case which also gave a reaction to another mould—namely, *Botrytis*—reference to our charts of mould phenology suggested that *Botrytis* was playing no part. Only one patient in this group was tested by inhalation: ground-up *Cladosporium* spores inhaled through the nose produced a severe attack of asthma in under one minute and his vital capacity fell from 4,500 to 1,900 ml.: a similar test with *Trichothecium* provoked no such attack. Two patients, whilst undergoing hyposensitization, each had a sharp attack of asthma within 20 minutes of an injection of *Cladosporium* extract; both needed adrenaline.

The pattern of *Cladosporium* sensitivity may be illustrated by the three following cases:

Case 1.—The patient was an accountant aged 33. Asthma began with a few sharp attacks at the age of 11 years in September, 1933, and recurred regularly each summer, but did not cause incapacity until he was 25 (1947). Although quite well each winter, he was incapacitated with persistent asthma from June to November in the summers of 1947 and 1948 and from May to November in 1949. When first seen in the summer of 1949 his skin tests resulted as follows: *Cladosporium* ++++, *Alternaria* −, *Botrytis* −, *Penicillium* −, pollen −, feathers −, house dust −. There was no infection in his sinuses or sputum. Pre-seasonal hyposensitization to *Cladosporium* was given in 1950, 1951, and 1952. During these summers, apart from a little wheeziness at night immediately eased by his inhaler, he was well. In 1953, following three satisfactory summers, hyposensitization was omitted, but his asthma returned, and, although not as severe as previously, it interfered with his work and necessitated a short stay in hospital. In 1954 and 1955 hyposensitization was resumed: his condition again became satisfactory and he needed his inhaler only occasionally at night.

Case 2.—A hospital executive aged 44 began to get asthma in June, 1936, at the age of 25. For seven years, 1936 to 1942, he had severe asthma (which was confined to the period June to September inclusive); he was away from work for long periods each summer and had repeatedly to be admitted to hospital. From 1942 to 1948 his periods of illness lengthened, now extending from April to December, and he began to have attacks of asthma when he had a "cold" in the winter months. In 1948 his skin reaction to *Cladosporium* was found repeatedly to be triple positive and his symptoms corresponded closely with variations in our graphs of the incidence of *Cladosporium* spores in the air. Other skin test results were: house dust +, *Botrytis* +, *Alternaria* +, grass pollen −. Each year since, for seven years, this patient has had a pre-seasonal course of hyposensitization to *Cladosporium*. He has steadily improved: his summer asthma has almost ceased, his attacks of infective

asthma, due to "colds" going to his chest, have steadily diminished, and he has not been off work since 1948.

Case 3.—A shop's buyer aged 58 began to get asthma in the summer of 1948, when aged 51. It recurred each summer (June or July to mid-September) for six years. In the winter of 1952–3, for the first time, the attacks failed to clear: when seen in June, 1953, she had had asthma continuously for 18 months, had been away from work for long periods, and had lost 28 lb. (12.7 kg.). There was pus in her sputum, and her asthma appeared to be infective in type. Skin tests were: *Cladosporium* ++, grass pollen −, house dust −, *Alternaria* −, *Botrytis* −, *Penicillium* −, *Aspergillus* −, foods −. Her asthma continued in spite of antibiotics and general therapy until September, 1953, when the attacks ceased. In January, 1954, *Cladosporium* hyposensitization was begun, but severe local reactions occurred and for a period no injections were given. She was prevailed upon to continue on a smaller dosage. In 1954 mild asthma occurred in May and moderate asthma in June and early July, but there was no incapacity. Hyposensitization was repeated pre-seasonally in 1955, and during the summer the patient had only mild attacks which were easily controlled by mild symptomatic therapy.

Sensitivity to *Cladosporium* as described above means sensitivity to the two saprophytic species *C. herbarum* and *C. macrocarpum*, which, as has been shown repeatedly, occur in enormous quantities in the ordinary atmosphere (Richards 1953c). We have observed one case of sensitivity to the spores of a third species, *C. fulvum*, which occurs not as a saprophyte but as a parasite on the tomato plant and is, in fact, the cause of tomato-leaf mould: in this case skin tests were negative to *C. herbarum* and *C. macrocarpum* but double positive to *C. fulvum*; the patient responded satisfactorily to hyposensitization. Out of 18 cases which gave positive skin reactions to *C. herbarum*, 13 gave negative reactions with *C. fulvum*, and only five gave positive reactions. Sensitivity to *C. fulvum* may therefore be quite distinct from sensitivity to the saprophytic species. Our findings in this respect are in accord with those of Rackemann *et al.* (1938) and Samsøe-Jensen (1955)—namely, that cases of specific sensitivity to particular species of *Cladosporium* do occur; we cannot agree with Feinberg (1944) that sensitivity necessarily applies to the genus as a whole.

Sensitivity to Alternaria

Of 638 patients tested to *Alternaria*, 13 (2%) gave positive skin reactions, but seven of these were excluded: three of them were not worse in the late summer, three gave reactions to other moulds and did not experience exacerbations of their symptoms during the *Alternaria* season, and one improved while being kept under observation. (This last case may well have been a case of clinical sensitivity and have outgrown it, but in view of his improvement he also was excluded.) The remaining six (1% of all patients tested) were diagnosed as clinically sensitive. All these had symptoms coinciding with the *Alternaria* season; three had no other positive skin tests, and, of these, one gave a positive skin reaction with a dilution of the extract down to 1/1,000,000 and another down to 1/10,000; three also gave positive reactions to house dust, but all of them had troublesome asthma limited to the late summer and house dust appeared to be playing no part in their illness.

An illustrative example of *Alternaria* sensitivity follows.

Case 4.—The patient was a shorthand-typist aged 21. In August, 1948, at the age of 13 years, she had a series of severe attacks. These recurred each August for five years, while during the rest of the year she had only occasional and mild attacks. The skin tests were: *Alternaria* ++++, *Cladosporium* −, *Pullularia* −, *Botrytis* −, *Phoma* −, house dust −, grass pollen −. In view of the limited period of severe asthma (four to five weeks), it was at first hoped that symptomatic therapy might suffice, but the attacks in August of 1952 and 1953 were so uncontrollable that hyposensitization was begun in the winter of 1953–4. In the summer of 1954 she was quite well until mid-August, when she went on holiday for 14 days to Cornwall (St. Austell), where she had an attack of asthma each night; but the attacks were easily controllable with "franol" and cleared completely after her first night home. In the summer of 1955 she again had only mild asthma, easily controlled symptomatically.

Sensitivity to Botrytis

Of 325 patients skin-tested to *Botrytis*, seven (1.1%) gave positive skin reactions. Of these, six were rejected: two gave positive reactions also to *Alternaria* and *Cladosporium*, and, although the periods of exacerbation coincided in the main with the *Botrytis* sporing period, it was not clear that *Botrytis* sensitivity was a dominant factor in their illness; two were judged to be predominantly sensitive to house dust, the part played by *Botrytis* being uncertain; one completely failed to respond to *Botrytis* hyposensitization; and one was lost sight of.

In only one case, therefore, could a firm diagnosis of clinical sensitivity be made. This was in a woman of 50 who had severe exacerbations of her asthma in August, September, and sometimes October for seven years (1946–53), and who had to be admitted to hospital in status asthmaticus in August, 1952, and August, 1953. A positive skin test to house dust had resulted in her having dust injections in 1952 and 1953, with little benefit. After *Botrytis* hyposensitization in 1954 and 1955 she was practically free from asthma in the late summer of both years, and had no period of incapacity. Her skin reactions to all the other moulds were negative.

Skin Tests with Other Mould Spores

Skin tests were also carried out with extracts of *Penicillium*, *Aspergillus*, *Phoma*, *Pullularia*, and *Candida*. The frequency of positive reactions was as follows: *Penicillium*, 1 (0.8%); *Aspergillus*, 6 (5.4%); *Pullularia*, 10 (7.8%); *Phoma*, 4 (4.3%); and *Candida*, 5 (8.9%). In none of these cases were we convinced that there was definite clinical sensitivity, and many homes were examined for sources of heavy mould concentrations, but without result.

Although no systematic cultures of the sputa of these patients were carried out, a number of those who gave positive skin reactions to *Candida* were found to have that mould growing in the mouth or sputum.

Conclusions

The investigations described have established (1) that certain kinds of mould spores occur in quantity in the atmosphere in England and Wales, especially in the summer months; (2) that many cases of summer asthma are due to sensitivity to the particular kinds of spores referred to, especially to *Cladosporium*; (3) that the knowledge of moulds as allergens so gained has enabled us to treat, with a large measure of success, a number of asthmatics whose complaints were not previously understood; and (4) that allergy to mould spores in all probability accounts for some 5% of all cases of asthma and may well play a subsidiary part in many more.

The investigations by one of us (M. R.) described herein were carried out during the tenure of a full-time grant from the Asthma Research Council.

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PLACENTA PRAEVIA

A REVIEW OF 286 CASES

BY

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The island colony of Singapore now has an estimated population of approximately 1.4 millions. This includes Chinese, Malaysians, Indians, Pakistanis, Europeans, and Eurasians, and is increasing rapidly: in 1931 there were 20,000 births as compared with 54,000 in 1953. The city becomes more crowded every day, and in the outskirts the expansion overruns the supply of urban amenities and leads to slum conditions. Even apart from this the older areas of the city are already badly overcrowded.

Since the end of the second world war the demands of this increasing population have been made more urgent by a growing confidence in Western medical techniques. During 1953, 156,984 patients attended the Kandang Kerbau Hospital and 23,807 cases were admitted. The number of deliveries in the hospital during the two years 1953 and 1954 was 38,259, so that it is inevitable that over this period a considerable number of cases of placenta praevia have occurred.

Incidence

The actual number of cases of placenta praevia met with during these two years was 286, an incidence of 1 in every 133 deliveries, or 0.7%. Other centres report incidences ranging from 0.32% to 2%. A series of 112 cases reported by Schmitz *et al.* (1954) represented an incidence of 0.6%; Paalman and Hunt (1949) record an incidence of 1.12% at the Mayo Clinic; and King and Chun (1945) one of 0.78%. This last figure, obtained in Hong Kong, is very close to that found in Singapore. In the present series the greatest incidence was in the age group 30–39 years (138 cases); in the age group up to 29 years there were 110 cases, and from the age of 40 years onwards 38 cases. Similar figures have been noted by other writers (Berkeley, 1936; Aldridge and Parks, 1938; Reycroft and Platz, 1942; Seeley, 1945).

Parity and Duration of Pregnancy

Only 19, or 6.6%, of the patients were in their first pregnancy, and 267, or 93.4%, were multigravidae. Berkeley (1936) in an exhaustive investigation found that, of 4,406 cases of placenta praevia, 20.1% were in primigravidae.

In the present series 78.3% of the patients had their first haemorrhage between the 34th week of gestation and term. This would appear to indicate a high proportion of viable babies. Eadie and Randall (1954) noted that the first haemorrhage occurred after the 34th week in 42.6% of their cases. The figure 78.3% cited above seems high, but this may be explained in part by the inaccuracy of the history given by the patient or by failure to obtain a full history because of language difficulties. Hendry and Baird (1936–7) reported that, in a series of 258 cases, warning haemorrhages had preceded the patient's admission in 38% of cases "admitted in good condition" and 60% of those "admitted ill."

Clinical Grouping

The type of placenta praevia was noted, the classification used being that commended by Macafee (1945), as follows. Type 1: The greater part of the placenta is attached to the upper uterine segment, but the lower margin extends into the lower segment. Type 2: The lower edge of the placenta reaches down to the internal os. (This is further subdivided into Type 2 anterior and Type 2 posterior, according to whether the placenta is attached mainly to the anterior or posterior uterine wall.) Type 3: The placenta overlaps the internal os when closed, but only partially so when the os is fully dilated. Type 4: The placenta completely covers the os even when fully dilated. Of patients admitted with an antepartum haemorrhage, only those in whom the placenta was palpated in the lower uterine segment at vaginal examination, or seen to be attached to the lower segment at caesarean section, were classified as having placenta praevia.

The incidence of the four types is shown in Table I.

TABLE I.—*Incidence according to Type*

Type	Number of Cases	Incidence (%)
1	67	23·4
2	86	30
3	64	22·3
4	69	24·3

Treatment and Method of Delivery

Vaginal delivery was effected in 142 cases and abdominal delivery in 143 cases. One patient died undelivered. The method of treatment adopted depended on three major factors: (1) the severity of the blood loss; (2) the period of gestation; (3) the type of placenta praevia. The last consideration was important only in so far as it indicated the ultimate type of delivery in many cases. In general, conservative treatment was adopted where the patient was far from term, in order to reduce foetal mortality from prematurity. When the haemorrhage was so severe as to preclude further expectancy the patient was examined in the operating theatre, the diagnosis confirmed by vaginal examination, and the treatment necessary for the type of placenta praevia present carried out. Under conditions prevailing in Singapore many patients came into hospital bleeding severely, a vaginal examination having been made outside. The blood loss, added to the already anaemic state of the patient, in a large number of these cases made conservative treatment difficult. Apart from this, labour had started in 83 cases (29%) at the time of admission. Blood transfusion was required by 183 patients (64%).

The methods of treatment used and the results obtained are shown in Table II.

In general, caesarean section was the treatment favoured in the major degrees of placenta praevia. The majority of cases with a Type 1 or Type 2 placenta praevia were dealt with by artificial rupture of the membranes. Willett's forceps were used on only two occasions—once for a Type 2 and once for a Type 3 placenta praevia. Version and breech pressure were employed on 27 occasions, mostly for a Type 2 condition.