failure would be more likely to be due to environmental than to inherited causes. In recent years bottle-feeding has become almost as safe as breast-feeding, and a generation of bottle-fed babies has grown up to perpetuate this trait. Hence genetic factors might well be responsible, in part at least, for the rising incidence of bottle-feeding, and they probably now contribute much more towards lactational failure than was the case when potentially poor lactators were eliminated by natural selection in infancy.

Theoretically selective breeding might counteract to some extent the above effects, for, as any film-goer and magazine-reader must know, modern society applauds the well-developed feminine bust. This, however, is not the only characteristic that makes a man choose a mate, and, even if it were, it must be realized that the large breast does not necessarily contain the greatest amount of functional glandular tissue.

Changing fashions in infant feeding have undoubtedly contributed to the decline in breast-feeding. Insistence on exclusive breast-feeding for six months has resulted in inevitable failure and abandonment in many cases, but the "new" vogue for early introduction of solids may serve to eke out an inadequate lactation for longer. No other mammals have demanded so much from the breast as we have.

In the final analysis it is impossible to separate heredity and environment. Genetic factors would be expected to play a part in the lactational ability of all women. It is therefore the result of the interplay between heredity and environment which determines the success of a lactation. The safety and facility of artificial feeding in modern society mean that environmental factors, such as social conditions, appear to control breast-feeding when in fact they may be merely marginal ones which tip the scales against the breast in a constitutionally poor lactator. According to Hytten, one-third of all women are doomed to fail to produce enough nourishment to support the average infant above a weight of about 11 lb. (5 kg.). Even if glandular inadequacy is largely genetically determined, this does not preclude the discovery of a remedy which could overcome the deficiency. Until one has been found, it may be of some consolation to a mother, genuinely and bitterly disappointed by her failure to feed her baby, to learn that it may well have been due to inborn deficiencies outside her control; she deserves to be spared the unkind suggestion that is sometimes made that she could have fed her baby for longer had she tried.

Summary

An investigation is described in which 943 women (43% primigravidae) were asked at the antenatal clinic about the breast-feeding performance of their mothers. The method of feeding adopted by these women was recorded when they subsequently attended the post-natal clinic.

The principal finding was that of 397 women whose mothers were "good" breast-feeders, as defined for the purposes of the comparison, 60% succeeded in maintaining uncomplemented breast-feeding for eight weeks, compared with 43% for the 63 women whose mothers were not "good" breast-feeders.

This association appears to be independent of environmental factors and of the mental attitude of the mothers and their daughters.

In a small series it was shown that the milk yield at the end of the first week is positively correlated with the duration of lactation.

Absolute proof of the influence of heredity is lacking, but the mother-daughter association and the evidence of a twin study (briefly referred to) point to the probability that genetic factors significantly contribute towards lactational failures, 50% of which are otherwise unexplained.

The possible role of heredity and the mechanism of its application in the declining incidence of breast-feeding is discussed.

A large number of people have been involved in this investigation and we are grateful to all of them for their help. In particular, the main part could not have been attempted without the active co-operation of Dr. D. Regan, area medical officer of Edmonton, who kindly put the resources of his organization at our disposal. Of his assistants, Drs. A. Goggin and M. Brown supervised the completion of the forms at the antenatal and post-natal clinics; Miss H. Bridge, non-medical supervisor of midwives, Miss S. Briggs, superintendent health visitor, and Miss R. Saunders, deputy chief clerk, all worked hard to ensure that the requisite details concerning the confinement and housing were collected and inserted and that the forms, after completion, were dispatched to the staff of the Statistics Department, St. Bartholomew's Hospital, to whom we are also grateful for keeping the records and transferring the data on to punched cards. Our thanks are also due to Mr. K. A. K. Hudson and Mr. A. W. Purdie, who permitted us to investigate the 24-hour yield in some of the patients admitted under their care, and to Miss A. L. G. Hammond and Miss H. Stephenson, superintendent midwives, for their part in obtaining this information.

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PNEUMONIA AND BRONCHIAL VESICULATION ASSOCIATED WITH HERPES ZOSTER

WITH REFERENCE TO OTHER VISCERAL ASSOCIATIONS OF ZOSTER

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Of the rarer manifestations of herpes zoster a motor paralysis of the same spinal or cranial nerve distribution as the skin lesion is the one most often recorded and, on anatomical grounds, the easiest to explain. Certain visceral associations have been described with very suggestive clinical features—for example, abdominal pain and gastro-intestinal haemorrhage with a tenth thoracic zoster (Porteous, 1947); dysuria and increased frequency of micturition with a second and third sacral zoster (Chesterman, 1932)—but there has not as a rule been any conclusive evidence of the specific nature of the visceral lesion. Thus one reads differing opinions regarding the existence of visceral zoster. Kinnier Wilson (1954), while quoting various reports of the condition, considers it to be a doubtful entity, and notes that Head (1910) found no sign of pleural, peritoneal, or other visceral damage in 394 cases of zoster. On the other hand, Gais and Abrahamson (1939) record that out of 137 cases of herpes zoster 42 had visceral symptoms, and Dubois (1926) reported a case which seems

indisputable—vesiculation of the mucosa of the bladder being observed, by repeated cystoscopy, to run an identical course with the skin eruption of a second and third sacral zoster.

The illness, being self-limiting and relatively benign, is unlikely to provide opportunity for pathological examination of the affected organ, and endoscopy may give the only support for a diagnosis of visceral zoster. In the following case consolidation of the lung was followed by a typical intercostal zoster and the appearance of vesicles on the bronchial mucosa.

Case Report

A male clerk aged 63 had been in good general health, but for two years had experienced a retrosternal gripping pain on exertion, relieved by resting and eructation. He had had a slight cough for many years, and attributed this to smoking. In mid-May, 1955, he developed a fever with right pleuritic pain, and his doctor noted signs of consolidation in the right lung. The fever and physical signs disappeared slowly during the next four weeks, showing no obvious response to sulphonamide, penicillin, or chloramphenicol, and he was referred to the chest clinic on June 15 in view of an abnormal chest x-ray picture.

On examination he looked well and the only abnormal findings were impaired percussion note and air entry over the right lower lobe. There were no added sounds and his blood pressure was 170/90. On screening the right side of the diaphragm appeared high and sluggish and the barium swallow normal.

Films taken on May 20 had shown dense opacity in the right mid-zone and right lower zone which appeared to be due to consolidation of the lower and middle lobes. There were also calcified lesions at both lung apices and the right hilum, with calcification in the ninth and tenth thoracic intervertebral spaces. Further films on June 23 and July 14 showed progressive clearing of the opacity, and when he was last x-rayed, on November 10, the right lower zone was quite clear, although the right costophrenic angle remained obliterated. The apical lesions were unchanged.

He was unable to produce any sputum, and one's initial assessment was that he had old apical tuberculosis and a recent lobar pneumonia which was resolving so slowly as to make bronchoscopy advisable. Within a fortnight of his first examination at the clinic, however, and within six weeks of the onset of his illness, he developed the typical erythematous stage of a left fourth thoracic zoster and the investigation was postponed until the eruption (Figs. 1 and 2) had progressed through vesiculation to scab formation.

Bronchoscopy (July 16).—The palate, pharynx, larynx, trachea, carina, and left bronchial tree appeared normal. The right middle and lower lobe bronchi also looked normal, some watery secretion being aspirated and found negative for tubercle bacilli. The orifice of the right upper lobe bronchus was reddened and three small glistening vesicles were seen on the anterior wall. The mucosa around these vesicles was particularly inflamed and they could not be removed by suction.

The patient declined a further bronchoscopy a fortnight later and returned to work feeling perfectly well. It was ultimately learnt that he had died suddenly on January 3, 1956, and that a post-mortem examination had shown a myocardial infarction which was regarded as the cause of death. Apart from the apical tuberculous lesions no abnormality was noted in the lungs.

Other Reports of Pulmonary Changes with Herpes Zoster

Of the 42 cases of Gais and Abrahamson, 7 had symptoms referable to the chest. In each of these the zoster was intercostal, but only one showed a radiological abnormality; five, however, had abnormal signs in the form of a pleural rub or rales. Herbeuval *et al.* (1950) collected 16 consecutive cases of intercostal zoster, and three of these had radiological evidence of pulmonary consolidation (which they aptly called "pneumopathie zostérienne"). Brain (1932) and Thursby-Pelham and Floyer (1944) have noted a pleural rub directly underlying an intercostal zoster; and Lingemann (1953) reports that zoster may be followed by an effusion into an artificial pneumothorax or be associated with an exacerbation of pulmonary tuberculosis.

Discussion

It is generally agreed that the basic lesion of herpes zoster is in the posterior root ganglion or corresponding cranial ganglion and that the virus is neurotropic in behaviour. Thus, to accept any visceral complication as primarily due to the zoster virus one should require the affected organ to be innervated via the same neural segment as the area of eruption and that the sequence of events should be closely related in time.

One difficulty in suggesting an association between intercostal zoster and a lung lesion is that the lungs originate from a pharyngeal outgrowth and cannot be related to any thoracic segment. On this ground one might expect pulmonary manifestations to occur rather with zoster of the vagus nerve, but although various authors—for example, Ergström and Wohlfart (1949) and Negus and Crabtree (1943)—have described this condition, with vesiculation of the pharynx, larynx, and sometimes of the external auditory meatus (via the auricular branch of the vagus), they do not appear to have searched for changes beyond the larynx.

However, the pulmonary autonomic plexuses do receive fibres from the second, third, and fourth thoracic sympathetic ganglia and it is thought that the cells of Dogiel in the posterior root ganglia may bring the somatic afferent neurones into relation with those of the sympathetic system travelling via the posterior nerve roots and the white rami communicantes. There are also fibres connecting the pulmonary plexuses of the two sides (Cunningham's *Text-book of Anatomy*, 1951), and one can thus postulate a neural connexion between the lung and the skin supplied by the second, third, and fourth thoracic nerves on the same or opposite side. Here it is interesting to note that in Head's series the

FIGS. 1 and 2.—Skin eruption at time of bronchoscopy which showed bronchial vesiculation.

zoster virus appeared to show a marked predilection for segments having a sympathetic-nerve connexion.

The peripheral lesion of zoster is an inflammation and vesiculation of skin or mucosa associated with degenerative changes in underlying nerve endings, and one might expect any lung complication to be due primarily to such changes, plus oedema, in the bronchial mucosa resulting in pulmonary atelectasis. Personal communication with several thoracic surgeons suggests that bronchial vesiculation is an unusual finding, and it seems reasonable to associate it with the zoster in this case—coinciding as it did with the typical skin lesion.

It is less reasonable, however, to consider that the pulmonary consolidation was a specific manifestation of zoster because it affected the middle and lower lobes whereas the vesiculation was in the upper lobe bronchus; moreover, the onset of the pulmonary episode did not coincide with that of the eruption but preceded it by over a month. The incubation period of herpes zoster is said to vary from 7 to 24 days.

Possibly the pneumonia was a predisposing factor in the manner suggested by Spillane and White (1939), who noted a tendency for patients with angina of effort to develop zoster in the distribution of their cardiac pain—the supposition being that irritation of posterior root ganglion cells by stimuli from a diseased organ might result in trophic changes resembling zoster or, alternatively, in these cells becoming devitalized and susceptible to attack by the zoster virus. This might be compared with anterior poliomyelitis affecting a previously injured limb. Parsonnet and Bernstein (1939) also discussed the association of heart disease and zoster. Wilson considers that all zoster, even those apparently secondary to another disease, are due to an infection with the specific virus—that is, not in some cases to a purely trophic change.

Conclusion

Admitting that both pneumonia and zoster are common and could easily coincide, earlier reports do suggest—and this case would support the suggestion—that visceral disease can be directly connected with zoster; and the practical implication, as stressed by Loiseau (1953), is that one should always question the state of any organ in the same neural segment as the eruption.

In most cases it might be practicable to make only a brief physical examination and inquiry regarding symptoms, but with an upper thoracic or vagus nerve zoster it would be a simple precaution to have the chest x-rayed to exclude an unsuspected carcinoma or tuberculous lesion of the lung.

Summary

A case is described in which pneumonia was followed by intercostal herpes zoster and bronchial vesiculation, and the possible connexion between these lesions is discussed.

Visceral disease in association with herpes zoster has been reported on various occasions, being sometimes probably itself a specific manifestation of the virus infection but at other times perhaps a predisposing factor.

One should consider the health of organs having the same nerve root supply as the superficial eruption.

I thank Dr. W. G. Sutcliffe, who referred the case to me; Mr. C. E. S. Oxley, who performed the bronchoscopy; and Dr. G. B. Forbes, who supplied the post-mortem findings.

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PROLONGED LABOUR

A CLINICAL TRIAL OF CONTINUOUS CAUDAL ANALGESIA

BY

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A first stage of labour exceeding 48 hours in duration due to discoordinate action of the uterus and cervix has a sinister reputation for both mother and foetus. The stillbirth rate attributed to this condition is more than four times that encountered in uncomplicated vaginal delivery. The major causes are foetal injury from ill-advised attempts to expedite vaginal delivery, intrauterine infection (McCredie-Smith *et al.*, 1956) by organisms within the upper vagina, and foetal anoxia from placental failure (Johnson and Clayton, 1955). The potentially anoxic foetus, in such labours, is sometimes further endangered by post-maturity (Stewart and Bernard, 1954) and pre-eclampsia, both of which depress placental efficiency (Walker and Turnbull, 1953).

There is no general agreement about the management of prolonged labours of this type, and treatment largely depends upon the judicious use of sedatives to ease the pains of labour and assist dilatation of the cervix, intravenous fluids to control maternal dehydration and ketosis, and, more recently, antibiotics (McCredie-Smith *et al.*, 1956) to prevent intrauterine infection. Some authorities have advocated uterine stimulants (Theobald *et al.*, 1956), and others claim that certain drugs have a relaxing effect upon the cervix (Gill and Farrar, 1951). In some instances, however, these latter techniques may exaggerate the abnormal uterine behaviour (Jeffcoate, *et al.*, 1952; Embrey and Garrett, 1955).

The serious wastage of foetal life attributed to prolonged labour has resulted in the more frequent use of caesarean section (Baird, 1955) as a speedy and less traumatic means of salvaging these infants, but unfortunately the maternal risk of this procedure increases steadily with the preceding length of labour, especially when this has lasted for more than 48 hours (Marshall, 1949).

More recently, claims have been made for the therapeutic application of continuous caudal analgesia to this problem (Arthur and Johnson, 1952; Johnson, 1954; Ball and Chambers, 1956; Youngman and Lloyd, 1956). This technique interrupts the autonomic visceromotor nerves of the cervix, and allows the dilating powers of the body of the uterus to have unopposed action upon a paralysed cervix and pelvic floor musculature. At the same time it has been pointed out that this method