glucose present. Thus in urines containing 0.61–0.8 g. all three tests gave results varying from a trace to 3+. Glucose concentrations over 2 g. per 100 ml. usually, but not always, caused complete reduction in each test. Such discrepancies are likely to be due to the presence in individual urines of substances interfering with the sensitivity of the test as well as to the difficulty of visual assessment of partial reduction. Difficulty was sometimes experienced with the colour charts used with the glucotest for grading the results. The colours did not always accurately represent those observed in actual tests. In this respect the clintest colour chart seemed superior.

It is likely that Benedict's test, when properly performed, is the most satisfactory of the three tests because of its slightly greater sensitivity. However, the simplification and convenience of the tablet technique make it the method of choice in almost all circumstances, certainly in the control of treatment of known diabetics, either in the clinic or by the patient in his home, as well as in the routine testing of urine, whether before operation or in illness generally. In the control of treatment of diabetes, failure to detect an occasional trace or to determine the exact percentage of glucose is of little significance. The routine Benedict test in illness or before operation is seldom well performed because of the difficulties of accurate measurement of reagent and urine and because of the practical difficulties of uniform heating; the much simpler and more readily standardized tablet test is to be preferred, although it is slightly less sensitive. The value of the tablet test in life assurance examination is a problem requiring separate investigation. Most of the subjects are symptomless and may not excrete more than traces of glucose, which might be missed by the tablet test and detected by Benedict's test. Perhaps a screening test using undiluted urine would be advantageous in this respect.

Two serious disadvantages of the tablet test require mention. First, the tablets are highly caustic and should be tipped into the tube, if possible without handling them. Secondly, although the tubes of tablets as manufactured are stable for many months, once the tubes have been opened it would appear that the inside of the cap is disturbed so that the bottle is not completely air-tight. A number of clinicians have reported deterioration in the tablets (turning blue) after only a few weeks, even though the stopper has been replaced tightly. The manufacturers might well effect some improvement in this respect. This would also be an advantage to the patient performing his own urine tests at home: the tablets are so often kept in the humid atmosphere of the bathroom. The manufacturers should describe a suitable method of cleaning the tubes after use. Water alone is not always satisfactory, and a preliminary rinse with acetic acid (vinegar by the patient), followed by water and drying, might be recommended.

The time saved by use of the tablet technique is of special importance. Benedict's test can seldom be done in less than 10 minutes by persons not trained in the laboratory. This allows for measuring two solutions, boiling, standing, and washing out the tube. In the tablet method there is one measurement and the result is read after 15 seconds, thus saving at least five minutes of time for each test. In a hospital of 500 beds with out-patients, an average of 100 routine urine tests a day is not an excessive estimation, and replacement of Benedict's test by the tablet test will save about nine hours of working time each day.

It must be emphasized that a positive result with a tablet test requires just the same consideration in regard to non-glucose-reducing substances as Benedict's test—that is, when the positive test is not accompanied by clinical signs of diabetes the reduction needs to be proved by the customary methods to be due to glucose and not to other reducing compounds.

From the diabetic angle, all that is usually needed is to know whether the urine contains no sugar, a little, or a lot. The three tests investigated show this equally well.

We wish to thank Dr. R. D. Lawrence for his interest and help.

INTRACRANIAL HYPOTENSION ASSOCIATED WITH SUBDURAL HAEMATOMA*

BY

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The first clear account of intracranial hypotension was given by Leriche (1920) in patients with severe cranio-cerebral injuries. Further examples of the syndrome were described (Leriche and Wertheimer, 1921; Leriche and Emery, 1922), and later Leriche (1931) said that the syndrome was seldom recognized and that although there was nothing absolutely characteristic in the symptomatology it formed a striking clinical entity.

Since these early descriptions of the syndrome from Strasbourg, most of the literature of the condition has come from the Continent, mainly from French authors. It was the subject of a joint discussion at a combined meeting of the Société de Neurologie de Paris and the neurological section of the Royal Society of Medicine in 1948 (*Proc. roy. Soc. Med.*, 1948), when it was clear that most of the British members present were unfamiliar with the condition, at any rate in its severe forms.

Most of the reports of the more severe forms of the syndrome have come from neurosurgical clinics, and apart from the minor manifestations such as lumbar puncture headache, it must be regarded as a rare occurrence in the province of a physician. This paper records the only two examples of the condition which I have recognized and treated, and both followed the drainage of fairly large subdural haematomata. Subdural haematoma seems to be associated with intracranial hypotension much more often than any other space-occupying lesion, so far as can be judged from the published reports. For example, Krebs, Puech, and Brunhes (1937) reported six cases of hypotension with ventricular collapse, of which two followed the drainage of subdural haematomata, one of an extradural haematoma and one of an arachnoid cyst following head injury. Lalonde and Gardner (1948) reported nine cases, of which seven followed the drainage of a subdural haematoma, one followed the removal of a pituitary adenoma, and one of a parasagittal meningioma. Chorobski (1950) also records six cases, one with a post-haemorrhagic cyst and one with both extradural and subdural haematomata. Gloor (1951) mentions its association with three cases of subdural haematoma in a series of 23 cases of widely different neurological conditions.

The association of a low cerebrospinal fluid pressure with subdural haematoma and chronic intracerebral haematoma is well known and is indeed an important diagnostic feature in these conditions. It is not so well recognized, however, that the persistence of the "low-pressure state" following the surgical drainage of the haematoma, and its association with severe cerebral symptoms, possibly of greater degree than those preceding operation, is responsive to simple therapeutic measures such as the intrathecal injection of normal saline to restore the pressure to a normal level, the intravenous injection of hypotonic saline or distilled water, or the inflation of the ventricles with saline or air. In

*Based on a paper read to the West Midlands Association of Physicians on May 26, 1951.

the two cases reported here the intravenous injection of distilled water, first suggested by Leriche, was used with striking effect.

Case 1

A married woman aged 73 was admitted to hospital on November 29, 1949. Twelve days before admission she stumbled and struck her head on a rockery stone in her garden. She was slightly dazed, but walked about afterwards for a week, although headache of increasing severity appeared. She had been in bed for five days before admission, and during the past three days had become very drowsy and incontinent of urine and faeces.

On examination she could be roused, but answered questions inaccurately and with difficulty, being very confused in time and space. There was no papilloedema and the cranial nerves were normal. Movements of the left arm and leg were weak and clumsy, and the tendon reflexes were increased on the left side. The left plantar response was doubtfully extensor and the abdominal reflexes were absent. The cardiovascular system was normal: B.P. 150/100, pulse rate 64, temperature 98° F. (36.7° C.). A lumbar puncture showed: C.S.F. pressure 150 mm.; fluid faintly yellow; red cells, 425 per c.mm.; leucocytes, 22 per c.mm. (56% polymorphs, 44% lymphocytes); protein, 150 mg. per 100 ml.; globulin present, glucose, 61 mg. per 100 ml.; chlorides, 720 mg. per 100 ml.; Wassermann reaction negative.

A diagnosis of right-sided subdural haematoma was made and on November 30 bilateral parietal burr-holes were drilled under local analgesia. On the right side a recent subdural haematoma consisting of dark fluid blood containing a few clots was found and evacuated, about 60-70 ml. in all, and the cavity was washed out with warm saline. The surface of the brain was depressed to about 0.5 cm. below the dura.

On December 1 the patient's level of consciousness was higher, but she was still very confused and disorientated. Next day she was stuporous, only just rousable, and incontinent; no focal neurological signs were present. On the third the stupor was deeper. The C.S.F. pressure on lumbar puncture in the lateral position was zero and fluid could be obtained only in small amount by suction with a syringe. Respirations were 18 and shallow, pulse was 80, Temperature 97° F. (36.1° C.), B.P. 120/70. Her extremities were cold and cyanosed. The deep stupor persisted for the next three days, but no focal neurological signs appeared. On December 6 she was in deep coma and unrousable. Respirations were 16 and very shallow, extremities cold and cyanosed; temperature was 97° F. (36.1° C.), pulse 60, and B.P. 80/?. Still no focal neurological signs were present. The C.S.F. pressure was still zero.

In considering the possible causes of the coma and other symptoms it was thought that intracranial hypotension might be the explanation, and it was decided that the very simple procedure of injecting distilled water intravenously or saline intrathecally should be tried before reopening the skull to look for further haemorrhage. At 12 noon 30 ml. of distilled water was injected intravenously and within ten minutes there was a noticeable improvement in the grave condition of the patient. The first change was an improvement in the peripheral circulation; the coldness and cyanosis of the hands and feet disappeared and the blood pressure rose to 100/70. The coma rapidly became less profound and there was a feeble response to painful stimuli. At 3 p.m. the patient could be roused with difficulty and a further 40 ml. of distilled water was given intravenously. At 4 p.m. she was fully conscious and drank a cup of tea without assistance. At 4.30 p.m. the C.S.F. pressure was 60 mm.

On December 7 she was fully conscious, but complained of slight headache; B.P. 145/95; extremities warm; no neurological signs; C.S.F. pressure 80 mm. No further symptoms developed and the patient was discharged from hospital after 10 days. She has remained well since.

Case 2

A married woman aged 49 was admitted to hospital on March 13, 1950, complaining of severe frontal headache and noises in the head. The noises were described as buzzing in the ears on both sides, and had begun about one month before. Two weeks after this she developed pain in the frontal region and behind the eyes, which was constant and gradually increased in severity. She also complained of pain in the right ear on hearing loud sounds. She had been in bed for two weeks before admission with no relief of her symptoms. During the week before admission she noticed a weakness of her right hand and leg, but did not complain of any disability on the left side. She had been adequately treated for Addisonian anaemia for the previous eight years.

On examination she was slightly confused, with considerable delay in answering questions and in performing requests. There was a marked defect of memory for both recent and remote events. Her pulse was 84, B.P. 135/85. There were no abnormal physical signs in the heart or chest, and no signs of Addisonian anaemia. The cranial nerves were normal. There was no papilloedema. Clumsiness and weakness of movement of both arms were present. She had slight loss of motor power, more evident on the right side, but there was astereognosis and gross loss of position sense on the left side of which the patient appeared to be unaware. Fine finger movements were grossly impaired in the left hand and there was also a tendency to a grasp reflex on this side. The left leg also showed gross loss of position sense. The tendon reflexes were slightly increased on the left side as compared with the right, but the left plantar response was definitely flexor and the right extensor. The abdominal reflexes were absent.

A lumbar puncture showed: C.S.F. pressure 60 mm.; fluid faintly yellow, with a slight coagulum; 83 cells per c.mm. (polymorphs 44%, lymphocytes 56%); protein 340 mg. per 100 ml.; globulin present; Wassermann reaction negative. A blood count showed: red cells, 3,393,000; Hb, 78%; C.I., 1; white cells, 5,600; E.S.R., 60-103 mm. (Westergren).

A diagnosis of subdural haematoma was made and bilateral burr-holes were drilled in the posterior parietal region on March 14. A large right subdural haematoma was found, consisting of dark-brown fluid and many clots, about 120 ml. in all. It was washed out with warm saline. The brain surface was compressed to about 1 cm. below the dura. The protein content of the haematoma fluid was 8.12%. On March 15 the patient was rather drowsy, but much less confused. Minimal neurological signs were present in the limbs, but the right plantar response was still extensor. Severe headache was still present. The C.S.F. pressure was 10 mm. Next day she was much more drowsy, but rousable. Confusion was again marked. Her pulse was 64, B.P. 140/90, respirations 20, temperature 99° F. (37.2° C.). There were no focal neurological signs. The C.S.F. pressure was 30 mm. The lumbar puncture needle was left in position and 30 ml. of distilled water given intravenously. Within ten minutes the C.S.F. pressure had risen to 50 mm. and there was a definite lessening of the drowsiness, confusion, and, later, the headache. Later in the day she was quite alert and uncomplaining. On the 17th the headache had entirely gone, and she was alert and feeling well. On March 18 there were no symptoms or neurological signs. The right extensor response had disappeared. C.S.F. pressure was 110 mm.; the fluid was straw-coloured, and contained 120 cells per c.mm. (polymorphs 51%, lymphocytes 49%); protein, 430 mg. per 100 ml.

The patient returned home on March 25 and has remained well since.

Discussion

The clinical picture of intracranial hypotension is seldom clearly defined, and it is usually impossible to make the diagnosis unless a lumbar, cisternal, or ventricular puncture

is done and a low pressure is found. Whenever possible it is preferable to measure the ventricular pressure, as a falsely low manometric reading may be obtained on lumbar puncture in cases in which a tentorial or medullary pressure cone is present, and the withdrawal of fluid by lumbar puncture may be extremely dangerous (Krebs, Puech, and Brunhes, 1937). It is improbable that the diminished volume and pressure of the cerebrospinal fluid is in itself the cause of the many and varied symptoms associated with hypotension, but it is the only objective sign which will confirm an otherwise presumptive diagnosis.

Morice (1944) has classified the symptoms of hypotension under the following headings: (1) Disturbances of consciousness of a progressive nature, slowly changing from stupor to a more or less deep coma. (2) Affective disorders accompanying the impairment of consciousness, most often of a depressive type, but rarely maniacal. (3) Diffuse headache of increasing severity, accompanied by nausea and vertigo. (4) Pyrexia. (5) Dehydration, characterized by dryness of the skin, shrunken facies, ocular hypotension, and polydipsia. (6) Normal respiration and deglutition and absence of bulbar involvement.

This list is not complete and other symptoms may be found. The temperature may be subnormal, as in my first case. Stiffness of the neck and Kernig's sign may occur. Hypothalamic symptoms are common, such as alterations in the pulse rate and blood pressure, diabetes insipidus, alterations in the depth and rate of respiration, and even Cheyne-Stokes respiration has been described. Paralysis and convulsions sometimes occur. Some French authors say that papilloedema has been seen (*Proc. roy. Soc. Med.*, 1948), but it is difficult to imagine why this should occur. Chorobski (1950) says that the alteration in consciousness is distinct from the coma or semi-coma of intracranial hypertension and likens it to the state of "arrested consciousness" sometimes found in tumours of the hypothalamus. We must agree with Leriche that such varied symptoms, however they may be grouped together, cannot be regarded as pathognomonic of hypotension. They could equally well be associated with increased intracranial pressure or with cerebral softening or haemorrhage. In spite of this an awareness of the conditions in which hypotension is likely to occur should enable the clinician to suspect its presence when a clinical picture comprising some of the above-mentioned symptoms is encountered.

The associated pathological conditions are almost as numerous as the symptoms, and were originally classified under the following headings (Leriche and Wertheimer, 1921): (1) Lumbar puncture, especially for spinal anaesthesia, with loss of cerebrospinal fluid into the soft tissues. (2) Closed or compound craniocerebral injuries, with or without loss of cerebrospinal fluid. (3) Cranial operations. (4) Various medical or neurological diseases.

It must be seldom, if ever, that severe symptoms associated with hypotension follow lumbar puncture, but in this connexion Pickering (1948), in a study of lumbar puncture headache in 11 cases, found a low pressure on repeating the lumbar puncture. He ascribes the symptoms to a caudal displacement of the base and posterior parts of the brain, with tension on the anchoring structures, particularly the large arteries at the base. This mechanism may well play an important part in producing the symptoms associated with hypotension and is mentioned later, when the pathogenesis is discussed.

The association of hypotension with closed or open craniocerebral injuries is difficult to determine. The symptoms may be indistinguishable from those of the brain injury which may be present or of cerebral oedema. It is important to make the distinction, as it is dangerous to give hypertonic intravenous infusions when hypotension is present, as was first shown by Clovis Vincent (Krebs, Puech, and Brunhes, 1937). These workers in Vincent's clinic favour the diagnosis being made by ventricular puncture through burr-holes, owing to the previously men-

tioned danger of lumbar or cisternal puncture should a pressure cone exist. These aspects of head injury have not been widely recognized in this country and are worthy of further study. It is still a common practice to give hypertonic solutions intravenously without consideration of the possible harm it may do.

Intracranial hypotension following operations on the brain for the removal of space-occupying lesions is more widely recognized, and most of the recent literature on the condition stresses this association. As stated before, hypotension seems to be much more commonly associated with subdural haematoma than with other space-occupying lesions, and Gerlach (1949) puts forward the theory that the hypotension may actually precede the formation of the haematoma and facilitate bleeding into a potentially empty subdural space. The hypotension is then further increased by the osmotic attraction of fluid into the effused blood.

It is stated by some French authors that hypotension occurs in a large number of apparently unrelated neurological conditions. At the Anglo-French conference in 1948 it was stated that its incidence was 7.2% of all neurosurgical cases, but this is difficult to accept. It has been found in lymphocytic meningitis by Fontan, Verger, and Dabadie (1946) and in meningococcal meningitis by Debré, Thieffry, and Doumic (1946). Puech *et al.* (1942) describe a case in which the hypotension was apparently due to sclerosis of the capillaries of the choroid plexus and absence of C.S.F. production. Gloor (1951) is of the opinion that hypotension may be "primary" and apparently not associated with any other pathological process. Gloor also states that hypotension may be an accidental finding in almost any nervous disease; but as he regards a C.S.F. pressure of below 100 mm. on lumbar puncture in the lateral position as his criterion of hypotension the statement is unconvincing. Pressures of below 50 mm. would probably be a more acceptable criterion to most British neurologists.

Pathogenesis

The decreased volume and pressure of the C.S.F. observed in intracranial hypotension is probably only a secondary manifestation, and other causative mechanisms must be sought to explain the graver forms of the syndrome. "Hypotensive state" would perhaps be a better descriptive term, in that there is less implication that the hypotension is primarily concerned in producing the symptoms.

The simple loss of cerebrospinal fluid may cause hypotension (Jacobaeus and Frumerie, 1923; Alpers, 1925; Kunkle *et al.*, 1943; Pickering, 1948), but the symptoms rarely consist of more than severe headache and perhaps neck rigidity. It is also rare to find severe symptoms following the loss of large amounts of C.S.F. from repeated drainage by lumbar puncture or from leakage through cranial fractures. It seems clear that the simple loss of C.S.F. is not a factor in producing serious symptoms such as occurred in my first case.

The rapid reversibility of the symptoms by the methods originally suggested by Leriche suggests that vascular mechanisms are very largely involved. Leriche suggested that spasm of the cerebral vessels following injury and a consequent ischaemia of the choroid plexuses was responsible for the diminished production of C.S.F. The distortion of cerebral vessels and pressure upon them by large tumours and subdural haematomata could easily produce ischaemia, and the tension on vessels produced by displacement of a brain unsupported by its normal bath of cerebrospinal fluid could easily perpetuate the deficiency of cerebral blood flow, particularly in the basal regions. Pickering (1948) invoked this explanation of lumbar puncture headache.

Chorobski (1950) suggests that the secretion of C.S.F. is controlled by nervous influences from the hypothalamus and that disturbance of blood supply to the hypothalamus may be responsible for the lack of C.S.F. production. It is certain that hypothalamic disturbances accompany the

hypotensive state, but it is not clear whether they are primarily responsible for the diminished secretion of C.S.F. or merely due to a general deficiency in cerebral circulation. Overt signs of hypothalamic dysfunction are found only in the more severe forms of the hypotensive syndrome.

In the present state of our knowledge of the syndrome it seems reasonable to speculate that impairment of cerebral circulation is the most important factor involved in its production, but the mechanisms are still obscure, apart from the possibility of spasm produced by trauma and vascular compression from space-occupying lesions. "Primary" diminution of cerebrospinal fluid production must be a rare occurrence, and it seems likely that in most cases it is a secondary effect of disturbed intracranial haemodynamics, particularly in the choroid plexuses. A brain reduced in size from collapse of the ventricles and lying in a relatively "dry" subarachnoid space could produce further circulatory impairment either by compression of vessels by its unsupported weight or by tension on them from its shifting in the cranial cavity. There is support for this idea in the fact that the symptoms of hypotension are often much relieved by lowering the patient's head by raising the foot of the bed, as, for example, in lumbar puncture headache.

Summary

Two cases of intracranial hypotension following the drainage of large subdural haematomata are described. The treatment, symptomatology, and pathogenesis of the hypotensive state are briefly discussed.

The syndrome is so far little recognized in Great Britain, and most of the literature is continental.

It is suggested that although intracranial hypotension is a definite clinical entity it is not a disease *sui generis*, and is probably a secondary effect of a disturbance of cerebral circulation, particularly in the basal vessels.

ADDENDUM.—Since this paper was written, a full account of intracranial hypotension has appeared in a paper by Page (*Lancet*, 1953, 1, 1). Four illustrative cases are described, including one following the drainage of a subdural haematoma and similar in most respects to my two cases. Page also describes a case with severe symptoms, including deep coma, following a lumbar puncture in which only 7 ml. of C.S.F. was removed. This contradicts what I have said above, but such cases must be very rare. There is an interesting reference to a statement made in 1825 by Magendie, who attributed symptoms following the removal of C.S.F. to pressure of the unsupported brain on basal vessels.

REFERENCES

- Alpers, B. J. (1925). *Arch. Neurol. Psychiat.*, Chicago, 14, 806.
 Chorobski, J. (1950). *J. Neurol. Psychiat.*, 13, 280.
 Debré, R., Thieffry, S., and Doumic, A. (1946). *Arch. franç. Pédiat.*, 3, 586.
 Fontan, A., Verger, P., and Dabadie, J. (1946). *Ibid.*, 3, 552.
 Gerlach, J. (1949). *Dtsch. Z. Nervenheilk.*, 160, 387.
 Gloor, P. (1951). *Schweiz. med. Wschr.*, 81, 326.
 Jacobæus, H. C., and Frumier, K. (1923). *Acta med. scand.*, 58, 102.
 Krebs, E., Puech, P., and Brunhes, J. (1937). *Rev. neurol.*, Paris, 68, 831.
 Kunkle, E. C., Ray, B. S., and Wolff, H. G. (1943). *Arch. Neurol. Psychiat.*, Chicago, 49, 323.
 Lalonde, A. A., and Gardner, W. J. (1948). *New Engl. J. med.*, 239, 493.
 Leriche, R. (1920). *Lyon chir.*, 16, 638.
 — (1931). *Presse méd.*, 39, 945.
 — and Emery (1922). *Lyon chir.*, 19, 201.
 — and Wertheimer, P. (1921). *Ibid.*, 18, 492.
 Morice, J. (1944). *Thèse de Paris*.
 Pickering, G. W. (1948). *Brain*, 71, 274.
Proc. roy. Soc. Med., 1948, 41, 771.
 Puech, P., Lhermitte, J., Buvat, J. F., Leriche-Koechlin, M., and Perrin, J. (1942). *Rev. neurol.*, Paris, 74, 316.

Half a million dollars will be spent on the first phase of a cholera control campaign in East Pakistan, under an agreement signed recently by the World Health Organization and the Government of Pakistan. The first part of the campaign will include demonstrations of cholera control methods, and will be followed by the extension over the entire endemic area of those methods shown to be most successful.

THE LONG-TERM PROGNOSIS IN INFANTILE ECZEMA

BY

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It has long been known that some cases of infantile eczema are liable to develop asthma or chronic skin lesions or both. The particular skin lesions concerned are grouped under various names such as Besnier's prurigo (described by Besnier as "prurigo diathésique" in 1892), asthma-eczema, disseminated neurodermatitis, and atopic dermatitis of Coca and Sulzberger. Taylor (1899) first noted that cases of infantile eczema are liable to become asthmatic, but it was not until fairly recently that attention was drawn to the association of Besnier's prurigo with asthma (Rasch, 1914). In a review of cases of infantile eczema Abt (1909) said: "As regards the aetiological relationship existing between [infantile] eczema and asthma, several striking instances in confirmation of such a view have been noted" (my italics). Modern knowledge would regard such a statement as a fair illustration of meiosis.

Besnier's prurigo is often simply termed chronic or recurrent eczema by others than dermatologists, and may briefly be described as a chronic, pruritic, lichenified cutaneous disease with a predilection for the anterior aspects of the elbows and posterior aspects of the knees, and also usually, but not always, the fronts of the wrists, the forehead, the sides and back of the neck, and the upper part of the chest and back. During remissions there is merely lichenification to view, but during exacerbations itching is intolerable, the lesions may become widespread, papules with surmounting post-traumatic blood crusts are evident, and there may be a superimposed acute eczema from infection.

The literature contains no reference to any investigation aimed at discovering what proportion of babies with eczema of the restricted type implied but inadequately defined by the adjective "infantile" are destined to suffer from the above recurrent or chronic disorders. Consequently, authors of textbooks and articles on this subject are obliged to use vague terms such as "a minority" or "a few" when discussing the prognosis. Hill and Sulzberger (1935), who have contributed much to the knowledge of atopic dermatitis, can only say, "A great many infants with atopic dermatitis recover entirely towards the end of the second year . . . and are bothered no more with atopic symptoms of any sort the rest of their lives."

Bamber (1931) interrogated cases of Besnier's prurigo regarding previous history of infantile eczema and found it present in 51 of the 83 cases examined. The results provide no figures useful for prognostic purposes.

Thus it was thought that an investigation with a primary object of demonstrating what proportion of a group of former victims of infantile eczema has progressed to manifestations of asthma and Besnier's prurigo might prove of some general prognostic value *per se* or, at worst, might be used for such a purpose in later years in conjunction with similar surveys conducted elsewhere.