But for the most part these have been "voices crying in the wilderness." Anything like routine appears to be abhorrent to some surgeons, yet the reaction to injury must at least be always in the same direction, and if bruised brain always reacts by swelling, treatment surely should always be directed to providing for the swelling to take place.

Conclusion

To sum up, then, we may inquire whether the Monroe-Kellie doctrine does hold in regard to the reaction of the bruised brain to injury. Is the encapsulation of the brain by cranium, meninges, and cerebrospinal fluid of a comparatively rigid and inelastic character like the tunica albuginea of the testis, as has already been suggested, or does it by some physiological mechanism readily accommodate to cerebral swelling as does the thin elastic renal capsule to swelling of the bruised kidney? If, as it seems to me, the encapsulation behaves in a comparatively rigid and relatively inelastic manner, there can be no questioning the desirability of directing our treatment of the bruised brain towards an attempt to render the cerebral encapsulation more accommodating and thus to maintain the blood supply to the contused area by permitting it to swell in response to the law of its reaction to injury. It is further contended that the best method of doing this is by continued gentle dehydration to facilitate brain-swelling and so prevent the reduction in the blood supply to the damaged areas of the contused brain.

There is some reason to think that our forefathers may have had some inkling of the benefits of dehydration. Ambroise Paré over 300 years ago advocated restriction of fluids, purgation, and sweating for cases of head injury (Turnbull, 1939); and the prescribing of calomel and salts and otherwise purging of patients with fracture of the base of the skull, which was common practice early in this century, was probably introduced quite empirically, but was nevertheless essentially sound.

Perhaps in addition to the striking clinical improvement which is so often observed as soon as this treatment is instituted, the absence of sequelae in a long series of cases is the best criterion on which to base its claims. It is simple—so simple that it tends to be neglected or perhaps rather scornfully regarded, as Naaman was inclined to treat the advice of Elisha (ii Kings v). The Syrian captain was angered and disappointed that the prophet had not come down and performed some miracle. In these cases of severe closed head injury with profound unconsciousness it would be so much more dramatic to perform wide decompressive craniotomy, but the simpler treatment is so effective that, like bathing in the Jordan, it suffices. To those who are still not convinced by the arguments here put forward, the figures presented, or the cases instanced, one may quote what was once graphically said of some numerical evidence: "An emaciated child suffering from hunger is a more impressive sight than any number of mortality statistics." A few cases treated by this method will prove more convincing than any theoretical considerations.

Summary

Prolonged unconsciousness following a severe head injury, particularly when blood is intimately mixed with the cerebrospinal fluid, would indicate that the brain has been bruised or torn. Evidence is presented that the brain reacts to such injury by attempting to increase its volume.

No two head injuries are exactly comparable, and it is well known that remarkable and complete recoveries are often made from extensive injuries to the skull and brain. It is difficult, therefore, to assess the value of any particular type of treatment. The absence of sequelae following the consistent application of any particular form of treatment, however, may be urged in its favour.

Dehydration is a method of permitting brain-swelling to take place after injury, and the evidence in favour of its application is here examined. It should be effected by rectal magnesium sulphate and not by intravenous methods, which probably produce undesirable reactions.

Smoothness of convalescence and an absence of sequelae—e.g., the so-called post-concussional state—have been noticed after treatment of a large series of cases by continued gentle dehydration.

It is suggested that the post-concussional state may, in many cases at least, be the result of failure to introduce effective treatment in the early stages and represent a condition of unresolved contusion, it being maintained that resolution takes place readily and completely if the requisite condition for reparation is provided. It is suggested that this is the provision of space so that brain-swelling can occur and is not restricted by the cerebrospinal envelopes, with consequent impairment of the cerebral blood supply. Provision for such swelling is made by sustained dehydration, which reduces the incompressible water content of the skull.

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THE NATURE OF THE RHEUMATIC CHILD

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(Concluded from p. 125)

Aetiological Factors in the Rheumatic State

It should be generally conceded that statistical inquiry has a more limited value in such a disorder as the rheumatism of childhood than it has in many other diseases. Diagnosis is more than usually difficult, so that the outlook, the experience, and the bias of the individual clinician have to be assessed. The conclusions, too, are valid only for the time and place in which they are made. This may be true in some degree of all statistical investigations, but it is most true of diseases that are disorders of personality and environment. So it should surprise no one that careful investigations by experienced observers at different times and in different places arrive at contrary conclusions with regard to the aetiological factors of rheumatism. The influences of climate, of heredity, of poverty and its accompaniments, are still keenly debated. It may be said of climate that its influence is equivocal; the disease is certainly not limited to northern latitudes and temperate climates as was once taught (Swift, 1940; Fernando, 1939), and it seems probable that the higher incidence of rheumatism in cold than in hot climates depends on the increased liability to respiratory infections in these areas. In England the geographic distribution of the disease cannot be shown to depend on climatic factors (Morris and Titmuss, 1942).

It is generally admitted that the disease shows a well-marked familial incidence, but in Great Britain most observers agree that this depends more on exposure to similar environmental stresses than on an inherited quality. In America, observers are inclined to attach more importance to the influence of heredity, but there are many facts (some of which are dealt with below) which suggest that the heritable factor cannot be of dominant importance. There are, however, three factors—age, social grouping, and the haemolytic streptococcus—about which opinion may be more dogmatically stated. Of these factors it may be said that unless an individual is between the ages of 3 and 15 it is uncommon for him to develop the disorder for the first time; that if he belongs to the wealthier classes he will with great probability escape it altogether, and that, if he does develop it, then at some phase of the illness, but not always at the start, the presence of the haemolytic streptococcus can be demonstrated. These factors deserve more attention here.

Age

There is no other infection which, while conferring no immunity, limits itself so accurately to an age group. This fact alone should be sufficient to characterize it not as a disease caused by an invader but rather as a disease conditioned by the host. This age group is paramount. Why?

The peculiarity of this age group is growth, and yet children grow more quickly in the cradle and continue to grow after puberty, so that physical growth does not suggest a solution. The fact that chorea is limited to this same age group confirms those who believe that rheumatism is an infection in their belief that chorea too is caused by the rheumatic invader. On the other hand, those who believe that chorea is a profound nervous instability, proved to follow psychic stress, will be encouraged by this age-grouping to look for a comparable nervous instability in non-choreic rheumatism. Physical growth is a silent progress to predestined stature (always granted a guaranteed nutrition). The growth of personality is a pilgrimage attended with difficulty and beset by dangers. The rheumatic age embraces the years when the child steps down from its cot into the family, and then out into the world—the years of integration and adjustment. I take it to be no coincidence that these are the years of growing pains, of chorea, and of rheumatic carditis.

Social Grouping

There is general agreement on both sides of the Atlantic that child rheumatism is much more frequent in the poorer classes. There are no comparable groups of statistics for the children of the poor and the rich; but Still (1927) found it to be nearly 20 times more common among his hospital out-patients than among the children of the same age group in his consulting practice. Glover (1930) suggests that it is 30 times more common among the poor. This is not to suggest that it is an unknown disease among the well-to-do. American writers often emphasize this (Kaiser, 1934), and epidemics among relatively closed communities of wealthier children in this country have been described (Bradley, 1932).

Large-scale investigations in Great Britain made by the Medical Research Council (1927) have established the fact that the incidence of rheumatism is not in direct relation to the degree of poverty.

"Indeed," writes Still, "if the hospital patients are divided into three classes according to the degree of poverty, the poorest of these three, the very poor and destitute, show a lower incidence of rheumatism than the other two. It would seem, therefore, that if poverty is a factor in the incidence of rheumatism it must be on account of some condition or conditions associated therewith which are not equally present in all degrees of poverty."

Although this conclusion has been contested by some, there should be no doubt that it was valid for the time and place at which the "investigations" quoted were made. Morris and Titmuss (1942) have questioned it on the grounds that the mortality rates show a steep rise in deaths from heart disease coincident with decline in social status right through to the lowest social group. This criticism carries an obvious fallacy, since it might be expected that once the extremely poor child had become rheumatic its chance of survival was less than that of its wealthier brethren; mortality rates from heart disease in social groups cannot be accepted as an adequate reflex of the incidence of rheumatic disease in those groups.

Findlay defines this social status more closely: "It would seem, however, that it is not the very poorest but rather, if one may so express it, the better-class poor families that have been better off and have fallen on evil days." The Medical Research Council report thus described these families, which it labels "Group B," Group A being the less poor and Group C the very poor: "They are normal households, for the most part with adequate furniture, but have no margin for anything beyond the bare necessities of life, and are liable to periods of deprivations should loss of work occur. They have few outside interests, and are mainly occupied with the struggle to make ends meet."

No observer has suggested that the mental and nervous stresses of poverty on children may be as important as the physical. It needs little imagination to see that the nervous stresses of poverty will be greater in Group B than in Group A or C, greater than in the less poor and in the more poor—that is, in the family which has known failure and is pre-

occupied with the fear of falling again, with its struggle to keep up appearances, with its avoidance of social life, with its emphasis on hard work as a prelude to success, and with the distortion of its emotional life by fear and insecurity: this is the domestic background to Group B families. Is it fantastic to see in this environment one of the breeding-grounds of child rheumatism? In which class should we expect to find that education imposes a greater nervous strain? Although this factor of nervous instability may explain the higher incidence of rheumatism at this particular economic level of poverty, the general association of rheumatism with poverty at all levels probably depends, as a host of observers have assumed, on the physical accompaniments of poverty, poor clothing, bad and damp housing, malnutrition, and overcrowding. While most of these factors operate by reducing the resistance to infection, others such as overcrowding presumably act by increasing the number and virulence of nasopharyngeal infections.

Strong evidence against the importance of a heritable factor was provided by the M.R.C. report. Evidence was collected from the Poor Law residential schools, and it was found that children living in these institutions much less frequently developed the rheumatic state than children of the same class living at home. At two of these schools, in 22 and 18 years respectively, there had been only five cases of chorea and five cases of acute rheumatism out of more than 6,000 children who had passed through them. It may be estimated that 200 of these children would during that time have developed rheumatism or chorea had they remained in their own homes. Rheumatism in childhood is a disorder based on environment, although a predisposition may be inherited.

The Haemolytic Streptococcus

There is in any rheumatic child a phase in the development of the rheumatic state which is intimately associated with infection, and this infection can usually be shown to be associated with the haemolytic streptococcus. Sometimes, when the rheumatic state develops three weeks after an acute tonsillitis, the "invader" seems to have sufficient power by its own strength and numbers to overwhelm the body defences; sometimes, as in the subacute rheumatic state and in chorea, there has been preliminary "fifth column activity" in the nervous system which has weakened the capacity for resistance to the invader. There is no means, except by clinical approach, of assessing the relative importance of these various factors in any individual or of estimating their aetiological importance in various peoples. It is certain that these factors will vary in importance with race, with country, with social grouping, and with sex.

The view that emotional disorder may lower the resistance to infections may be difficult for some minds to accept, but it is in fact a commonplace of paediatrics. Cameron (1929) writes of the nervous child:

"Lowering of resistance to infection of all sorts is clearly incurred. The frequent illnesses of the delicate only child have a real existence. They are not to be explained as imaginary ills due to the too great anxiety of the mother. The timid excitable child, with his faulty physique and wrong posture, not only catches every infection possible but responds to each with a violence peculiarly his own."

This review of the aetiological factors in the rheumatic state suggests that the nearly absolute limitation of that condition to an age group and its common association with poverty do not refute the theory that nervous instability in the child is part of the rheumatic diathesis, nor does the recognized association with infection in some of its phases controvert such a theory.

What Manner of Child is This?

Less than 10% of the children entering the rheumatic state do so by way of subacute rheumatism, and in my own cases these children have more often shown evidence of continued nervous instability than of infection. More than 25% of the children entering the rheumatic state do so by way of chorea, and it is suggested that this condition also is evidence not of infection but of profound nervous instability. If these aetiological assumptions are accepted then a third of the children who develop rheumatic carditis show evidence of preceding nervous instability. If this nervous instability is a

component of the rheumatic state can it be shown to be present in other children with active rheumatism who have not suffered either from subacute rheumatism or from chorea?

I attempted to find evidence of this nervous instability in 21 consecutive cases of active rheumatism, none of these patients having suffered from chorea or from anything resembling subacute rheumatism. I did not regard the history as displaying nervous instability unless the child showed some disorder of physical process such as insomnia, somnambulism, or night terror; anorexia, diarrhoea, or acquired enuresis; fatigue, fidgetiness, or habit-spasm; pains, whether of head, abdomen, or limbs—and in addition some evidence in the mother's story of emotional disorder or environmental difficulty.

The results of this inquiry were disappointing—only three of the 21 children showed evidence of nervous instability when judged by this exacting standard. The parents of two other children, however, told a story of environmental stress that seemed so closely related to the onset of the rheumatic infection as to provide an aetiological factor. Neither of these two children showed any previous psychological abnormality. One other point of importance emerged. The parents of 16 of these children described them as unusually excitable—such excitability, for the most part, going back to the "toddler" age. It was notable, too, that eight of these mothers used the expression "inwardly excitable." Of these 21 children 12 were described by their mothers as unusually timid or shy. It was as a rule easy to detect evidence of excitability or timidity in these children. This inquiry disappointed me until a year ago, when I read an article by Neustatter (1937) which began with a remark made by Prof. H. A. Harris of Cambridge, "The rheumatic child is highly strung but he isn't neurotic." I realized then that I had been "barking up the wrong tree" in looking for evidence of neurosis in these children. The important fact is their excitable temperament, demonstrated again by this small series of mine, but emphasized over many years by a number of clinicians. Cheadle wrote in 1889:

"I would insist on the fact that there is nothing antagonistic between the agency of the nervous state or mental excitement and rheumatism. They are concerned together as factors in many cases of chorea." And again: "I should like to call attention to the relation of nervous excitability to rheumatism. The rheumatic children who develop chorea are intellectual, highly strung, excitable, nervous, often emotional. The question arises, Is the nervous instability due to the influence of the recent active rheumatic state and induced by it; or is the nervous instability part of the original constitution of the child, and chorea produced when this is acted upon by a second fact—viz., rheumatism? Or do the two tendencies run together in the original diathesis—the child with a tendency to rheumatism inheriting thereby a mobile nervous system?"

Still (1912) writes:

"To my mind one of the most striking features in such children is the nervous temperament, to which Goodhart has specially drawn attention. The rheumatic child is *par excellence* the nervous child. In many cases this is seen in an undue excitability: such children will become almost uncontrollable in their excitement over games and amusements or, on the other hand, there may be excessive timidity; in some of the older children this is replaced by a sensitivity or shyness which is almost morbid."

This description of the rheumatic child's temperament has been elaborated by many authors, but the quotation from Still described it well enough—the excessive excitability disguised often by shyness but wildly expressed in moments of acute emotion. It has often been suggested that this nervous excitability is the result of rheumatic infection, but Neustatter's investigations do not support such a theory. He concludes that the "rheumatism may aggravate the nervousness but does not cause it." This has also been my experience. The mothers of these children related that the excitability was present for years before the development of the rheumatic state. Therefore studies of the child's personality during and after the development of the rheumatic state will have equal validity with studies made before; and they should be more informative, since the child's nature has been exposed to the most searching of all tests—a recognition that the heart, the organ whose beating signifies life and whose stillness means death, is diseased. Poynton (1936) said of this emotional disturbance, in the Harben Lectures:

"We must understand the child with acute heart disease. It may be instinctively terrified to a high degree, just as an adult may be when he or she feels that the very pivot of life is in danger."

Winnicott (1938), who has studied these children for many years as a paediatrician and later by psycho-analytical technique, writes:

"When we examine the child's heart we do not always realize that in the child's unconscious fantasy we are testing his ability to keep his ideals alive and unhurt. The child does not necessarily understand the life and death of his body as we understand it, but he understands something deeper which is the life or death of what he believes in and values most in himself. What we say, therefore, about the things inside to which we listen with a stethoscope and discuss with our colleagues over a bedside comes to the child as a matter of life and death in an acutely real sense. It is a matter of reality and of the right to live. In adult terms, it is to be or not to be, to continue to live or to seek death. These are the anxieties that belong to psychiatric depression."

It makes little difference whether we choose to talk of instinctive terror with Poynton or in terms of unconscious fantasy with Winnicott, the important fact for practising physicians to realize is that we are dealing with children who are suffering from fear—unexpressed, unrecognized, and undischarged.

Neustatter suggested that the symptoms—night terrors, restless sleep, worrying disposition, timidity, swings of mood, and emotional instability—which were commoner in his rheumatic groups have an aetiological distinction from the neuroses: "the former are possibly of primarily physiological origin and the latter due to psychological causes." This idea is an illuminating extension of Prof. Harris's remark quoted above, but its expression and the juxtaposition of "physiological" and "psychological" are not happy. It is preferable to regard the temperament of these children as showing a quantitative difference from normal—a speeding-up of both motor and emotional reactions—while the neurotic displays a qualitative deviation from normal. It is possible that the electroencephalogram may one day diagnose and measure for us this quantitative deviation.

No one can deny the emotional and kinetic lability of the child with chorea, but it is as legitimate to deny as it is to assert that this excitability exists also in many rheumatic children. Nor do I see how, in the present state of our knowledge, these matters can be put to scientific proof. There is no laboratory test for acute rheumatism, nor is there any objective measurement of excitability; investigation can only be by question and answer and by subjective observation. Any one physician caring for any one rheumatic child has to attempt the assessment of these factors for himself.

It is concluded that where the rheumatic child displays neurotic symptoms, as he or she infrequently does, these symptoms are superimposed on a nervous temperament and stand in no aetiological relation to the rheumatism. It is suggested that the personality of the rheumatic child shows a quantitative increase in emotion and kinesis—which has long been recognized by many clinical observers—and it is suggested that this nervous instability is an important factor in the development of the rheumatic state.

Treatment of the Nervous Rheumatic Child

There are two principles in the treatment of these children which are implicit in all that has been written here. The first is that the child, whether suffering from nervous disorder, growing pains, arthritis, or chorea, should not be regarded as suffering from the rheumatic state until the diagnosis of carditis is definitely established. The second is that evidence of nervous excitability or instability should be sought and, if found, treated in all such children before, during, and after the development of this rheumatic state. The first principle will prevent the formation of cardiac neurosis, and the second principle, I believe, gives the best hope of limiting the extent of the rheumatic disorder in childhood.

Nothing recondite is required in the way of psychiatric diagnosis and treatment for these children. They are nervous and timid, excitable and fearful, and the appropriate treatment is by way of reassurance, recognition, and solution, if possible, of environmental difficulty, and by sedatives.

Reassurance.—The need for reassurance should be remembered at every stage of treatment. Words spoken at the bedside should be definitely encouraging; no hint of diagnostic difficulty, of therapeutic failure, or of doubtful prognosis should be allowed to reach the child. Attempts should be

made by parents, doctors, and teachers to overcome the child's timidity and fear. He should be encouraged by every means to believe that his background is stable and his environment secure. The best means to this end is to surround him with a confident family circle. The already anxious mother will be quick to catch the physician's dominating fear, which is the fear of relapse, and the family atmosphere seldom brings reassurance to these children. Fortunately, most of these children are much less fearful of the effect of physical activity than are their mothers and doctors.

Winnicott (1938) has written: "It may be said that once we have diagnosed rheumatism implying that the child is liable to heart disease our job is to produce in the child an *induced hypochondria*. This is our first task, and the future depends on our success rather than on drugs." No doubt this holds a certain element of truth, but it also holds a greater element of danger. Hypochondria will not help to return the child to normal living, and this last must surely be the object of all therapy. It is important to recall constantly, when treating rheumatic heart disease in children, that a cardiac neurosis is frequently imposed on these children. Paul White (1937) reported that 4.7% of patients with organic heart disease had "neuro-circulatory asthenia"; and Paul Wood (1941) found that out of 100 recruits with early organic heart disease 13 showed the symptoms of Da Costa's syndrome. I believe that once the active rheumatic process has subsided (and of this we can to-day furnish good evidence with the sleeping pulse rate and the sedimentation rate) the child should be returned to normal living as quickly as possible, and its activity should then be limited, not by some imposed standard but only by the heart's capacity. I believe that nervous relapse (chorea and subacute rheumatism) is best prevented by the methods outlined here. The prevention of further pharyngeal infection presents a difficult problem. The removal of large numbers of these children to Florida has been shown not to prevent it. Schlesinger (1938) has shown that continuous aspirin therapy reduces the liability to streptococcal relapses; it is probable that continuous sulphonamide therapy will prove itself more effective (Thomas, 1942). I believe that no measures should be taken to prevent infection which interfere with the child's normal living.

Environmental Difficulties.—These may be related to the family or to school. In neither case is removal from the family or prolonged absence from school likely to offer a permanent solution. It is amazing how light-heartedly children are kept away from school. The child that cannot adapt itself to its family has failed at its first hurdle, the child that cannot adapt itself to the normal educational stresses has failed at its second, and such children are unlikely to succeed at the later stages of the race. An attempt should be made to iron out the grosser environmental stresses, but the aim should always be to alter the child's abnormal reactions to its environment.

The Value of Sedatives.—I believe that continuous sedation by phenobarbitone is first-class therapeutics for these children with growing pains and the subacute rheumatic state, with chorea, and with active rheumatism. It will no doubt be objected that there are dangers in this continuous treatment by sedatives. I can only say in reply that I have watched these children for several years and am convinced that their physical and mental performance is improved by such therapy.

Growing Pains.—The child is depressed, weeps easily, sleeps badly, and is worried by pains. In time its anxieties appear to be related entirely to its physical symptoms. "There, I'm flushing again: My heart's beating away: I can't eat my dinner: I've got that pain in my legs: Why can't I stop twitching?: I shall never get to sleep." These are the cries of the child, and phenobarbitone causes the symptoms to disappear.

Chorea.—I believe that every child who has had chorea should be treated with phenobarbitone daily until after puberty. I have had 12 unselected children who have suffered from chorea under continuous observation and such treatment for periods varying from three to six years. Only one of them has had a relapse of chorea and only two have had another rheumatic episode in the period of observation. This is much less than the expected relapse rate for such children. Other observers have found that it is possible to reduce the expected

relapse rate in chorea by the attempt to remove environmental stress but without continuous sedation. Gerstley and his collaborators recorded a relapse in only 6 children out of 45 with chorea over a period of seven years, and "three of those had not been faithful attendants at the clinic." Their prophylactic treatment was "by keeping the youngsters under constant observation, obtaining co-operation from the school teacher, and avoiding the radio and thrilling movie."

The Rheumatic State.—All children with acute rheumatism should be given sedatives, since all are liable to chorea. Such therapy is more important for girls than for boys, and more important for children who have had previous attacks of chorea. As to the need for sedation in the other phases of the rheumatic state, the physician must assess the instability of the individual child. I have formulated for my own use the following rules for the administration of phenobarbitone to these children. It should be given to all rheumatic children who (1) show undue excitability, timidity, or motor instability; (2) show disorders of sleep, digestion, or appetite; (3) complain of fatigue or other "circulatory" symptoms unrelated to physical activity (i.e., the common complaint of tiredness in the morning). As an example of the results of this treatment the following case is related:

A girl aged 12 was admitted to the hospital for the first time in April, 1941. She had been known to have a "weak heart" since an examination by the school medical officer at the age of 5. She had not been allowed to play games, go to the gymnasium, swim, or cycle. She has always slept badly and had a poor appetite. Her weight was 4 st. 12 lb. before her present illness. Her mother said she was very excitable and, although usually happy, was liable to violent outbursts of temper. These outbursts were generally related to imposed restriction of her physical activities. She had an attack of rheumatism after measles at the age of 8, but had had no other rheumatic episode since. Examination of her heart showed pericarditis and fully developed mitral stenosis. Her sedimentation rate was normal in six weeks, and she was allowed out of bed in ten weeks. She returned home and has had 1½ gr. of phenobarbitone night and morning since. She sleeps well, has a good appetite, and has gained 20 lb. in the last year. She is said to be happy, even-tempered, and much less excitable. She rides a bicycle, has learnt to swim, has spent a week under canvas at a girls' camp, and has had no time away from school.

It is suggested that the treatment of these children by simple psychotherapy and by sedation removes their growing pains, reduces relapse in chorea, and improves the mental and physical performance of the rheumatic child.

Summary of the Argument

The rheumatic state in childhood has two main components—nervous excitability and infection. These two factors are present in varying degree in any one rheumatic child, so that it may be impossible in one child to distinguish any nervous instability or in another to detect any certain evidence of infection. In one, the profound motor and emotional instability which is called chorea may possess the child and infection be either absent or a late invader; while in another, infection dominates the scene at the abrupt onset and throughout the subsequent course.

It is concluded that growing pains are usually the result of some difficulty attending the growth of personality, not of stature, and that it is more easy to find evidence of nervous instability than of infection in the subacute rheumatic state.

The nervous instability of the rheumatic child has no relation to neurosis; it is suggested that it is a quantitative increase in emotion and kinesis. The relationship between the nervous excitability and the infection is difficult to determine. It has been generally assumed that such nervous instability as is allowed to the rheumatic child is the direct result of the "rheumatic infection or toxæmia," and this view is supported by the fact that chorea may develop for the first time during an attack of acute rheumatism. There is more evidence to support the contrary view that this nervous excitability in the child commonly precedes the onset of infection and may even prepare the ground for the rheumatic invader.

The various aetiological factors in the rheumatic state are potent in so far as they predispose to these two components—nervous excitability and infection. Age, sex (as in chorea), and the nervous stresses of poverty are important since they contribute to the appropriate nervous excitability; climate, cold and damp, overcrowding, and the other physical accompaniments of poverty are important because they increase the liability to droplet infection. The heritable factor in the

rheumatism of childhood cannot be of great importance, since there are many facts that declare it primarily an environmental disorder.

Glover (1930) has called rheumatism an obsolescent disease, and this no doubt depends on the slow removal of the appropriate environmental stresses amongst the poor. Such "gradualness may be inevitable," but it is suggested that the health of the rheumatic child is improved and stabilized by treatment of its nervous instability. This is done by the removal of the grosser environmental stresses, if possible, by the adjustment of the child's reaction to its environment, and, most important of all, by continuous sedation with phenobarbitone.

I am grateful to Lieut.-Col. Paul Wood, R.A.M.C., for much helpful criticism in the revision of this paper.

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MACROCYTIC ANAEMIA OF PREGNANCY AND THE PUERPERIUM

BY

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It is generally believed that the macrocytic anaemia, or so-called "pernicious anaemia," of pregnancy is due to a lack of the anti-pernicious-anaemia principle brought about by one or more of the following causes: (a) a temporary depression of secretion of the specific intrinsic factor of Castle (Strauss and Castle, 1933; *Brit. Ency. med. Pract.*, 1936); (b) a deficient intake of the extrinsic factor (Strauss and Castle, 1933; *Brit. Ency. med. Pract.*, 1936); and (c) a diversion to the foetus of the anti-anaemic principle itself (Vaughan, 1936). The usual conception of treatment is that it is similar to that of Addisonian pernicious anaemia except that in the macrocytic anaemia of pregnancy it may be discontinued after delivery, when the demands of the foetus no longer exist and a return of the secretion of intrinsic factor has been presumed to occur (Strauss and Castle, 1933; Vaughan, 1936). The three cases to be described are of interest because there was little or no improvement in the blood level after liver extracts given parenterally, but rapid regeneration occurred when this treatment was supplemented by the ingestion of whole liver.

Case I

Mrs. A., aged 39, was admitted to hospital in Nov., 1941, during the 36th week of pregnancy. For two weeks before admission she felt tired and breathless and vomited frequently. The pregnancy had until then been uneventful, and she had

taken a diet which was notable in only one respect—namely, that she had never cared for and seldom ate any red meat. She had five children, all alive and well. The last child was born in Nov., 1937, and at that time she was noted to be severely anaemic and was given a blood transfusion after labour. Next day the haemoglobin was 30% and a film was described as showing the features of pernicious anaemia. In the next 13 days 40 c.cm. of campolon was injected. Reticulocyte counts apparently were not done, but it is of interest that 15 days after the first injection the haemoglobin had fallen slightly to 28%. Large doses of campolon were continued, and recovery was then rapid. She was discharged with a haemoglobin of 70%; no further treatment was given. There seems little doubt, therefore, that she had had "pernicious anaemia" of pregnancy in 1937.

When seen in Nov., 1941, she was obviously severely anaemic. Physical examination revealed no notable abnormality. The tongue and nails were normal. Free hydrochloric acid was secreted after the injection of histamine—maximum, 30 units at one hour. The haemoglobin was 20%, and the other findings were compatible with Addisonian pernicious anaemia except that anisocytosis was less than would be expected at this level. A Price-Jones curve gave a mean cell diameter of 8.5 μ .

In the first three days 10 c.cm. of campolon and 8 c.cm. of anahaemin were injected (see Fig. 1). Three days after the

FIG. 1.—Chart of Case I.

first injection there was a reticulocytosis of 9.7%. On this day delivery occurred and a blood transfusion of two pints was given. The blood loss after delivery was estimated at about one pint. The slight improvement produced by the transfusion was transient and the blood count had fallen almost to the initial level on the tenth day. From the eighth day onwards daily injections of anahaemin or neo-hepatex and small doses of campoferron orally were given. On the sixteenth day there was a reticulocyte crisis of 40%, but very little rise in the red cell count occurred. On the fourteenth day treatment with whole liver was begun. The reticulocytes were 29% on the thirtieth day, and the blood count then rose rapidly.

Case II

Mrs. B., aged 39, was admitted to hospital in June, 1941, five weeks after giving birth to her sixth child. She insisted that she had no untoward symptoms until three weeks after delivery, when she began to be breathless and tired; but she was inclined to belittle her symptoms, and it is quite possible that their onset was earlier than this. At the same time there developed a burning pain in the tongue, and her appetite, which previously had been excellent, began to fail, so that shortly before admission she had literally to force herself to eat. Her diet appeared to have been satisfactory, and was