occupation of the colony this long-stored rice was severely rationed, so that individuals received approximately only one-third of the amount previously consumed. Consequently undernourishment was added to B₁ avitaminosis, with the result that the secretion of lactating women either considerably diminished or completely ceased. Thereafter one could see many undernourished infants with the symptoms of chronic milk intoxication, while acute milk intoxication had apparently disappeared. It may seem incongruous, but the fact remains that a high incidence of acute human-milk intoxication in infants indicates a certain degree of prosperity in the case of the native population. The observations of Bray (1929) on the island of Nauru support this statement. There the natives were given royalties for their phosphate deposits. As a result there was a distinct drop in their production of native foods, which were replaced by imported white flour and tinned meat. As palm toddy was prohibited they consumed large quantities of sugar-water instead. This change of diet resulted in the appearance of acute human-milk intoxication, and the infantile mortality rate rose to 40%.

Human-milk intoxication occurs whenever the staple foods of women are vitamin-B₁-deficient carbohydrates. Admittedly, in Great Britain such staple foods as the national loaf and potatoes have enough vitamin B₁ for their own metabolism; however, if the bulk of the diet consists of carbohydrates, some of them refined, the balance may be upset and result in deficiencies. In fact, deficiencies of other components of the vitamin B complex—such, for example, as riboflavin and nicotinic acid—have been reported recently in England (Slater, 1942; Duckworth, 1942; Clarke and Prescott, 1943; and others).

An apparently healthy mother with latent B₁ avitaminosis may produce milk containing intermediary metabolites just above the infant's tolerance, which would lead to predisposition to infantile complaints such as respiratory infections or persistent intestinal catarrhs. Even before the war Geiger and Rosenberg (1933), in Palestine, reported that Jewish sucklings suffering from persistent diarrhoea with toxic symptoms and excreting methylglyoxal in the urine, were cured after the administration of vitamin B₁. Popoviciu and Munteanu (1934) observed similar gastro-intestinal disturbances in Rumanian infants, which they also attributed to deficiency of vitamin B₁ in human milk. Occasionally in the paediatric wards in continental Europe and in the United States of America one encounters cases of sudden dyspnoea and cyanosis or learns of cases of sudden death in breast-fed infants. Where no pathological lesions are found to account for these occurrences, death is usually presumed to be due to status lymphaticus, laryngeal spasm, or oedema, to asphyxiation caused by blankets, pillows, or overlaying, or to strangulation with the strings of a jacket. May it not be possible that some, at least, of these puzzling and inadequately explained cases are due to human-milk intoxication?

Causes other than B₁ Avitaminosis

Furthermore, it is quite possible that accumulations of intermediary metabolites may also occur in other conditions in which there is a disordered carbohydrate metabolism, as in cases of glandular dysfunction—e.g., diabetes; or in other vitamin-deficiency diseases such as nicotinic acid and riboflavin deficiencies (the last two conditions most probably associated with B₁ avitaminosis).

It is conceivable that human milk intoxication occurs whenever B.B.S. are increased in the human body; and their increase has been found in various conditions other than B₁ avitaminosis—viz., acidosis, ketosis, anoxaemia, toxæmia, and uræmia (Wilkins, Taylor, and Weiss, 1937), as well as in diabetes, anaemia, epidemic dropsy, and splenomegaly (Wilson and Ghosh, 1937). However, the correlation between the nature and amount of intermediary metabolites in human milk and the symptoms in an infant fed on such a milk have yet to be definitely established. To the best of my knowledge such work has not yet been undertaken, my own investigations in that field having been curtailed after a short time.

It would seem that the statement that breast milk is the best food for an infant should be qualified by the proviso that it is secreted by a healthy woman.

Summary

It is suggested that the term "breast-milk intoxication" or "human-milk intoxication" be substituted for the term "infantile beriberi."

It is also suggested that in addition to methylglyoxal, hitherto considered to be the only toxic factor in the milk of B₁-avitaminotic women, other intermediary metabolites, in concentration encountered in the milk, may play an important part in human-milk intoxication.

The action of these intermediary metabolites appears to be synergic and cumulative.

The symptoms of human-milk intoxication are discussed.

It is pointed out that accumulation of intermediary metabolites in the milk of lactating women may be encountered in conditions other than B₁ avitaminosis and thus cause an intoxication.

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ACUTE BRACHIAL RADICULITIS

BY

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Spillane (1943) has drawn attention to the comparative frequency of cases of neuritis of the nerves around the shoulder girdle in his experience with the M.E.F. Cases also occur in which the incidence is predominantly on the roots of the brachial plexus rather than on the peripheral nerves, and combinations of these conditions are seen in other patients. This paper is based on 36 cases—24 personally observed in various stages of the disease and 12 seen by Major Philip Buckley, R.A.M.C.

The essential clinical picture is a simple one: severe pain starts across the back of the shoulder and radiates down the outer side of the arm and sometimes on to the upper part of the chest; the pain remains severe for a few days and then weakness of the shoulder develops, sometimes accompanied by numbness. As the paralysis appears the severe pain usually passes off and is replaced by a dull ache, the paralysis persisting.

Illustrative Case History

An R.A.F. corporal aged 33 was admitted to hospital on Sept. 15, 1942, with the history that six weeks previously he had developed very severe pain across the back of both shoulder-blades, which radiated down the outer side of the right arm and forearm; the severe pain persisted for about five days and was then replaced by a dull ache. On the third day of the severe pain he noted considerable weakness of both arms, but especially the right, and this weakness had persisted unchanged. There were no general symptoms of malaise or fever at the onset of the illness, and his past history was uneventful except for attacks of migraine.

On examination he had winging of the left scapula due to paralysis of the serratus magnus. In the right arm there was considerable wasting of the spinati, deltoid, biceps, supinator longus, and the clavicular head of the pectoralis major. There was minimal voluntary power in the supinator longus, while the other wasted muscles were weak; the biceps and supinator jerks were absent, and the triceps jerk just present. On sensory testing there was impairment of cutaneous sensibility down the outer side of the right arm

and forearm in the distribution of C.5 and 6 segments. There were no other abnormal neurological signs. The W.R. was negative. The C.S.F. was under pressure of 150 mm. and there was no block on jugular compression; it contained 1 lymphocyte per c.mm. and the protein was 30 mg. per 100 c.cm.

He was treated with massage and exercises. In the course of two months there had been considerable improvement in the right arm; the biceps and deltoid were of normal power, the spinati and supinator longus moderately weak. The biceps jerk was feeble and the supinator jerk still absent: the sensory impairment was still present, but over a smaller area than previously. The left serratus magnus remained paralysed.

Anatomical Distribution of the Lesions in the Series

(a) *Cases Confined to a Single Peripheral Nerve.*—The peripheral nerve most commonly involved was the long thoracic nerve, with resultant winging of the scapula and difficulty in raising the affected arm above a right-angle. Isolated palsies of this nerve have been recognized for many years, and Richardson, in 1942, drew attention to their comparative frequency. The reason for including the isolated non-traumatic cases in this series is because in 4 instances paralysis of the serratus magnus has occurred on one side with involvement of other muscles in the other arm, while in 6 cases other muscles on the same side have been involved, together with the serratus magnus. There have also been 11 examples of isolated serratus magnus palsy. It has been found that if the paralysis of this muscle first occurs while the patient is in bed with some other complaint he may not notice the muscle weakness himself; it is usually first noted when he tries to stretch up to take something off a high shelf. The suprascapular nerve has been involved alone in two cases with paralysis of supra- and infra-spinatus and weakness of external rotation of the arm. It was noticeable in one case, seen some months after the onset of the disability, that the two muscles were involved in very different degrees, the infraspinatus being considerably weaker and more wasted than the supraspinatus. There were two examples of isolated circumflex palsies with paralysis of the deltoid and sensory impairment over the nerve distribution. In one case the musculo-cutaneous was the only nerve involved, with loss of power of the biceps and sensory impairment in the distribution of the lateral cutaneous nerve of the forearm. The disability was relatively slight, as good flexion of the elbow was carried out by the supinator longus, the main weakness being in supination.

(b) *Cases of Root Involvement.*—There were 5 examples of involvement of C.5 and 6 roots, of which the case described above is one. In these cases the muscles affected were the spinati, deltoid, biceps, supinator longus, and sometimes the clavicular head of the pectoralis major; and there was impairment or absence of the biceps and supinator jerks, with sensory impairment on the outer side of the arm and forearm. In one case the C.7 root was affected in addition to C.5 and 6, as shown by weakness of the triceps and extensors of the wrist and fingers as well as of the muscles just mentioned.

(c) *Muscle Involvement Not Explicable in Above Groups.*—In a number the muscle weakness was not explicable on the basis of involvement of a single peripheral nerve or in terms of root distribution, as in the following cases: spinati and serratus magnus on one side (2 cases); deltoid and spinati (7 cases); deltoid and biceps (1 case); spinati and serratus magnus on both sides (2 cases); sterno-mastoid, trapezius, deltoid, spinati, and serratus magnus (1 case); while in another case there was widespread bilateral involvement affecting the spinati, deltoid, and triceps on one side, with the spinati, biceps, triceps, and extensors of the wrist and fingers on the other; in this patient, sensory impairment was found over the lateral side of both forearms. In these cases there is often a different degree of involvement of the various muscles affected. It is possible that the distribution is explicable on the basis of involvement of more than one peripheral nerve—for instance, the long thoracic and suprascapular in the serratus and spinati cases—or by partial involvement of one or more spinal roots.

(d) *Bilateral Cases.*—In 9 of the 36 cases there has been bilateral involvement, usually asymmetrical. In 7 cases the two sides were affected at the same time or with an interval of one or two days, but in the remaining 2 cases there was a longer interval: in one, three weeks elapsed before the second

arm was involved; and, in the other, six months between paralysis of the serratus magnus on one side and the spinati on the other.

Other Features of the Disease

Precipitating Causes.—The age incidence has been 16 to 54, and all the cases have been in males; this is probably on account of the selected Service personnel seen. The geographical incidence of the disorder appears to be wide; in this series the onset has occurred in the United Kingdom, Iceland, the Middle East, Tunisia, Italy, Uganda, Nigeria, India, and in one case at sea off the West African coast. In a number of the cases a possible precipitating factor has been found. In no fewer than 9 cases the onset has followed closely on a surgical operation: in 8 of them herniotomy, and in the other excision of a pilonidal cyst. The anaesthetics were varied—spinal in 4 cases, general in 3, spinal in combination with a general anaesthetic in 1, and intravenous in the last. The time interval between the operation and the presenting pain of the radiculitis has been from 2 to 4 days in 6 cases, 6 to 7 days in 2 cases, and 10 days in 1 case. This time interval rules out as the causative factor trauma during the operation from pressure against the side of the table or other accident of that type. Eight cases were related to infections, the onset of the radiculitis usually being during the period of convalescence: malaria in 2, influenza in 2, and pneumonia, glandular fever, dysentery, and an axillary abscess in 1 case each. In this last case the disorder was bilateral and so could not have been the direct result of the abscess. Two cases followed a week after a fall on the shoulder; although pain and muscular weakness in each case developed on the same side as the fall, the trauma was slight and there was a complete absence of symptoms in the week immediately after the fall. One man developed the presenting symptoms of pain during a period of severe exposure to cold and rain while escaping from a P.O.W. camp. Two cases followed diagnostic procedures—one 10 days after a lumbar encephalogram for a suspected cerebral tumour, and one on the day after a lumbar puncture. (This was done on account of a prolapsed lumbo-sacral intervertebral disk.)

The question of prophylactic inoculations in the possible aetiology of these cases has been raised, but it is difficult to draw any conclusions from this series. One patient had an anti-typhus inoculation three days previously and 1 two weeks previously, while 3 patients had had T.A.B., the interval between inoculation and onset of symptoms being 18 days, 4 weeks, and 5 weeks. Another man had had A.T.T. three weeks previously, while in the remaining cases no recent inoculations had been given.

Onset of the Disorder

The absence of general symptoms of fever and malaise is remarkable; in only two cases was there slight malaise when the pain started. The presenting symptom is always pain; it is across the back of the shoulder and down the outer side of the arm; in bilateral cases it spreads to both shoulders and arms, and in cases in which the C.6 or C.7 root is affected it spreads to the outer side of the forearm. The pain is continuous and is usually of considerable severity; in only 3 of the cases was it described as slight.

The duration of the severe pain before muscle weakness first appeared has varied from a few hours to two weeks or possibly slightly longer, the usual length of time being three to four days. The severe pain tends to pass off as the weakness develops, and may be replaced by a dull ache; but in some cases severe pain has lasted for two to three weeks, although the paralysis has occurred within a few days of the onset of the pain. The onset of weakness in the majority of cases is sudden, with a maximum at the onset, but 4 patients said that the weakness had progressed over the course of two to three days.

Sensory Changes

No sensory impairment was found when a motor nerve, such as the long thoracic or suprascapular, was alone involved; in the cases of involvement of the circumflex and musculo-cutaneous there was cutaneous impairment to all forms of sensation in the area expected, and in 5 cases in which C.5

and 6 roots were affected there was impairment down the lateral side of the arm and forearm. Of the mixed cases only two (in which the deltoid was involved) showed some sensory impairment over the skin on the outer side of the arm, and the case previously mentioned in which there was impairment over the lateral side of both forearms. In the other patients no sensory impairment could be found.

Special Investigations

As many of these cases have been seen some time after the onset, detailed investigations in the acute stage have been infrequent, but when they have been possible the C.S.F., blood count, and E.S.R. have been normal, and Spillane also found this in his cases. The only exception was the finding of 10 lymphocytes per c.mm. in the C.S.F. in the case of glandular fever with radicular involvement.

Prognosis is difficult to determine in view of the varying times after the onset at which these cases have been examined. The prognosis in the serratus magnus palsies is on the whole poor: of the 21 cases in which this muscle has been involved 3 were partial from the beginning, and of these one had shown definite improvement after three months, while the other two were apparently stationary in the same period. Of the complete cases, 1 recovered completely in four months, while 5 had shown partial recovery in periods ranging from three to seven months; the remaining cases still had complete palsies when last examined at periods ranging from three to eighteen months after the onset.

Once marked atrophy has occurred in a muscle the prospect of recovery is poor; when localized atrophy is only slight, considerable and even complete recovery may occur, though the onset of recovery may be delayed for as long as six months and possibly more. In a few cases recovery has started within three to four weeks and has progressed rapidly.

Pathogenesis

There has as yet been no pathological material available from cases of this disorder; it appears, on clinical grounds, that the condition can affect single peripheral nerves, nerve roots, or combinations of these. Riddoch (1944) has suggested that the anterior horn cells may be involved in some cases, basing this view on the rapid muscular wasting which may be present and on the scattered nature of the muscular involvement which is occasionally seen. No exactly comparable condition seems to occur in the lower limbs.

The best name for the disorder is uncertain in the absence of a known aetiology or pathology. Acute brachial neuritis is already used for a different clinical syndrome, and the name applied by Spillane of "localized neuritis around the shoulder girdle" is hardly comprehensive enough. For these reasons the term "acute brachial radiculitis" has been applied to it for the time being till more is known about its aetiology.

The differential points from anterior poliomyelitis, to which it bears some clinical resemblance, have been admirably summarized by Spillane, the main ones being: the absence of signs of general disturbance in the acute stage; the normal C.S.F. when this has been examined early in the illness; the sensory impairment in a number of the cases; and the occurrence of these cases in the absence of typical cases of poliomyelitis in the same district at the same time. To these may be added the fact that it may occur at the age of 50—a most unusual happening with poliomyelitis.

The aetiology of the condition is still speculative, and the somewhat mixed group of apparently precipitating causes has not helped on this point. It has been suggested that a virus infection is the cause: the main point against this is the absence of fever or other signs of general infection and the normal C.S.F. findings. In most diseases due to neurotropic viruses—even in zoster, in which the main incidence of the infection is on the posterior root ganglia—an increase of cells is found in the C.S.F.

There is one condition which has a very close clinical resemblance to it, and that is serum neuritis (Kennedy, 1929; Young, 1932). This may occur after the injection of any form of serum, usually about the seventh to ninth day; there are frequently the usual symptoms of serum sickness, followed

within a day or two by severe pain in the region of the shoulder and upper arm; muscle weakness shows within the next few days, and when this happens the severe pain passes off. The paralysis which may be accompanied by sensory changes is most often in the distribution of the fifth and sixth roots or the circumflex nerve, but at times paralysis of the serratus magnus may arise. The exact cause of these cases is uncertain, but it is thought that oedema of the affected nerves occurs as a reaction to the foreign protein in the serum. I think that the possibility of some, as yet uncertain, aetiological relationship between serum neuritis and acute brachial radiculitis must be considered before assuming that a neurotropic virus is the causative agent.

With the lack of knowledge of the aetiology, treatment can be only palliative—analgesics for the pain in the acute stage, support for paralysed muscles to prevent stretching, and active and passive exercises and electrical stimulation of the affected muscles when the painful stage has passed.

Summary

The syndrome of pain around the shoulder, followed in the course of a few days by muscular weakness and wasting and at times sensory changes, is described.

The anatomical distribution of the weakness can be a single peripheral nerve, two or more spinal roots, or combinations of these.

A number of apparent precipitating factors are mentioned, the most outstanding being operation for hernia in 8 of the 36 cases.

The possible aetiology is discussed and an analogy drawn between this condition of acute brachial radiculitis and serum neuritis.

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THE TRANSNASAL INTRAGASTRIC RYLE'S TUBE IN TROPICAL PRACTICE A LIFE-SAVING MEASURE

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Ryle's tubes have been used by us in a series of 355 acute medical cases during the past year. So far as can be ascertained, this represents the first extensive trial, in tropical practice, in which the method has been applied to the problems of hydration and alimentation presented by such acute cases. Its use was introduced, in the first place, to meet the special problem of the dehydrated patient who would not or could not drink that amount of fluid considered necessary for him. Dehydration, among coolies especially, was seen on a scale rarely encountered in civil practice, and in many cases was so gross as to resemble that of cholera. Marriott's (1943) teaching of the fatuity of what he calls "the homoeopathic dosage of a pint or two" is wasted unless we realize that in gross dehydration the fluid loss is to be measured in gallons. Furthermore, some of these patients refused to drink, and spat out the water or saline offered to them. In a series of 30 of our cases this symptom was found to be associated with a low serum chloride figure, and possibly represents a vicarious form of water intoxication. In the more robust patients this can be corrected by the intravenous injection of one or more pints of hypertonic (2 N) saline solution, after which the previously non-drinking dehydrated patient will drink avidly. Other patients, however, are often so enfeebled by disease on admission to hospital that for them to drink is too much effort. It is in such cases that the tube becomes a life-saving instrument, since by its use the body fluid may be restored in a few hours.