agranulocytosis it occurs in the first few days of treatment. There is a sudden fall in the number of red cells, and a varying amount of jaundice together with haematuria. No fatal cases are recorded, and as a further test course of the drug given to these patients after recovery fails to reproduce the condition it is possible that it depends to some extent on the presence of the infection.

The most troublesome symptoms with M & B 693 are nausea and vomiting; this is more obvious than in the case of sulphanilamide. M & B 693 is more toxic than sulphanilamide in animals when the blood concentrations of the two drugs are compared (Marshall, Bratton, and Litchfield, 1938), but owing to the slower and less complete absorption of the free base, toxic symptoms, excluding vomiting and mental depression, seem to be no more common than with sulphanilamide. The soluble sodium salt may well give rise to symptoms of toxicity; so far it has not been used on a very large scale.

The administration of large doses of M & B 693 to animals causes the deposition of concretions of the acetyl derivative in the renal tract, more especially in the collecting tubules (Antopol and Robinson, 1939). Several cases of haematuria (Southworth and Cooke, 1939; Marshall, personal communication), presumably due to this cause, have occurred in America. Up to the present there have been no reports of the condition in this country.

Summary

The drugs in general use for chemotherapy of bacterial infections are sulphanilamide and certain of its derivatives—notably M & B 693.

These derivatives can be divided into those in which substituents are introduced into the amino group and those in which they are introduced into the amide group.

The action of these drugs is discussed from the aspects of absorption and excretion and of toxic symptoms.

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ACHLORHYDRIC HYPOCHROMIC ANAEMIA ASSOCIATED WITH PERIPHERAL NEURITIS

WITH REPORT OF TWO CASES

BY

C. WORSTER-DROUGHT, M.A., M.D., F.R.C.P.

*Physician to the Metropolitan Hospital and to the West End
Hospital for Diseases of the Nervous System*

AND

J. SHAFAR, M.D., D.P.H.

*Medical Registrar, West End Hospital for Diseases of the
Nervous System and Willesden General Hospital*

The association of neurological lesions with certain disorders of the blood is an established fact. The frequency with which organic changes in the spinal cord and peripheral nerves occur in Addisonian (pernicious) anaemia, however, is in pronounced contrast with the apparent rarity of like changes in idiopathic hypochromic anaemia. This is somewhat surprising as there is considerable evidence to show a relation between these two varieties of anaemia (Heath, 1933). Families in which some members were the subjects of Addisonian anaemia and others of hypochromic anaemia are also described (Witts, 1930; Heath, 1933). There is, indeed, some conflict of opinion as to whether affection of the nervous system occurs in association with hypochromic anaemia, as shown by the literature.

Review of the Literature

Whitby and Britton (1937) state that although numbness and tingling of the extremities are not uncommon in hypochromic anaemia, definite lesions in the nervous system are rare. They quote Daniels and Roclofs, who described migraine, disturbances of voluntary movement, and papilloedema as complications of this malady. On the other hand, Wilkinson (1936) asserts that cases of hypochromic anaemia practically never show involvement of the central nervous system or peripheral nerves, while Vaughan (1936) was unable to find any evidence to support the occurrence of subacute combined degeneration of the cord or other organic lesion of the central nervous system in this form of anaemia. Wintrobe and Beebe (1933)

record that paraesthesiae were present in 33 per cent. of their cases, but that the severity of the symptoms rarely attained the prominence encountered in subacute combined degeneration of the cord, and that they more resembled the acroparaesthesia of the menopause. These authors were unable to trace in the literature any undoubted case in which the anaemia was complicated by objective neurological signs. The observations of Bethell, Goldhamer, Isaacs, and Sturgiss (1934) are also in accordance with this latter observation.

Witts (1930) reports the case of a patient with hypochromic anaemia, sore tongue, and achlorhydria who showed weakness of the lower limbs, without ataxia or paraesthesia, and in whom the right ankle jerk and abdominal reflexes were absent. Cases of postero-lateral sclerosis of the spinal cord associated with achylia and hypochromic anaemia are also described by Witts (1930), but he regarded the blood changes as being in a "pre-pernicious stage" and likely to revert to the true picture of an Addisonian anaemia if treatment had been withheld. Caughey (1936) records an example of a change from hypochromic anaemia to Addisonian anaemia in a patient manifesting signs of involvement of the motor and sensory tracts of the spinal cord. Dameshek (1931) reports a case which exhibited diminution of vibration sense over the right leg. Abramson (1935) found objective neurological changes in several instances: "reduced external sensibility" was found in one; absence of radio-periosteal and triceps reflexes with preservation of the biceps reflex in another; and a third showed signs of involvement of the pyramidal tracts. Following treatment with iron these physical signs resolved. In a series of pathological examinations of the spinal cord in anaemia Weil and Davison (1929) concluded that only in pernicious anaemia are the microscopical appearances of subacute combined degeneration of the cord found, and that anaemia in itself—that is, lack of haemoglobin and oxygen—does not act destructively on the spinal cord. These statements are criticized by Hadden (1936), who points out that the series of cases was too small to warrant such conclusions, and he describes cases in which post-haemorrhagic anaemia gave rise to syndromes in which the spinal cord was affected.

It is of interest to note that mental changes have been met with in association with hypochromic anaemia, the apparent presumption being that the anaemia was the underlying aetiological factor of the psychosis, since the latter disappeared with improvement in the blood picture (Scheid, 1937; Romano and Evans, 1938).

Two personal cases showing objective neurological signs in association with hypochromic anaemia are described below.

Case I

Achlorhydric hypochromic anaemia with peripheral neuritis. A man aged 74 was admitted to hospital on November 30, 1938, suffering from general weakness, and especially weakness of the legs. About two years before he began to complain of substernal pain on exertion as well as after meals, together with fatigue. The pain had improved to some extent, but the weakness had progressed, and his legs became rapidly tired on exertion. His previous illnesses were dysuria, for which prostatectomy was performed in April, 1935, and haematuria in June, 1936, renal calculi being removed from the right kidney by operation. He remained in hospital for seven months, eventually making a good recovery, and had no further symptoms of haematuria, dysuria, or frequency.

Physical Examination.—Moderate development and nutrition, but facies very pale and showing a faintly yellowish tinge;

mucosae and conjunctiva also pale. Tongue smooth, with poorly marked papillae. Edentulous. Heart: Apex beat just palpable in fifth interspace in mid-clavicular line, with systolic murmur at apex conducted towards axilla. Pulse regular, full, and average rate of 90. Blood pressure 145/70. Lungs normal beyond slight crepitations at both bases. Abdomen normal apart from operation scars. Nervous system: Pupils very small but equal and regular, with sluggish reaction to light and normal reaction to accommodation. Other cranial nerves normal. Sensation: Vibration diminished in both legs, especially the right, with patchy diminution to pain and loss to cotton-wool over the distal portions of each leg; similar patchy anaesthesia over hands. The arms showed full movement with slight loss of power. Supinator, biceps, and triceps jerks all absent. Legs showed full range of movement, but with deficient power at all joints and slight loss of tone; knee jerks and ankle jerks absent and plantar reflexes flexor. The abdominal reflexes were normal. Co-ordination and sphincters were normal. A blood count showed: haemoglobin, 58 per cent.; red cells, 3,850,000; colour index 0.75 per cent.; no abnormal red cells apart from deficiency in haemoglobin. White cells, 5,500—polymorphs 59 per cent., lymphocytes 32 per cent., large mononuclears 7 per cent., eosinophils 2 per cent. The cerebrospinal fluid was normal in all respects, with two cells per c.mm.; total protein 0.025 per cent.; negative globulin, Lange and Wassermann reactions. Fractional gastric analysis showed absence of free hydrochloric acid and a total acidity of only 0.036 per cent. throughout the two and a half hours of investigation.

There was no lesion to account for a secondary anaemia, and although at times the motions contained a slight amount of blood, this proved to originate from internal haemorrhoids. An x-ray examination of the stomach and intestinal tract showed no evidence of abnormality; similarly a barium enema passed rapidly through the colon with signs of early diverticulosis. Sigmoidoscopy gave a negative result.

Subsequent Progress.—He was put on ferri et. ammon. cit. 30 grains thrice daily, and by December 31 his haemoglobin had reached 60 per cent. An examination of the blood on February 10, 1939, gave the following result: haemoglobin, 72 per cent.; red cells, 5,150,000 per c.mm.; colour index, 0.7; white cells, 7,000 per c.mm. No abnormal red or white cells were observed in the blood films. The patient was seen again on May 24, 1939, and had improved considerably. He looked well, and stated that he had no complaints beyond an occasional pain across the small of the back after sitting. Muscular power in the arms and legs was good; in the upper limbs the supinator, biceps, and triceps jerks were all present, only the periosteal radial jerks being absent. Knee jerks were definitely present even without reinforcement, but the ankle jerks were still unobtainable. Sensation was now everywhere normal apart from some diminution of vibration sense over both tibiae.

Case II

Hypochromic anaemia with achlorhydria and peripheral neuritis (mainly sensory neuronitis). The patient was a married woman aged 51 with four children, all alive and well; she had had one miscarriage.

History of Illness.—In 1932 the patient first developed dyspeptic symptoms, and an x-ray examination revealed the presence of a gastric ulcer. She was placed on routine treatment and remained well until 1937, when she began to lose weight and to complain of a dull epigastric pain. A further radiological examination was undertaken, and showed an ulcer of the perforating type on the lesser curvature of the stomach. She was admitted to hospital, when the haemoglobin content of her blood was found to be 56 per cent. A partial gastrectomy was performed in September, 1937, only two inches of the lesser curvature being left. The pathological report of a section of the resected piece of stomach showed that a columnar-celled carcinoma was present in the ulcer bed. Her recovery from the operation was uneventful, and she continued well until July, 1938, when she began to experience paraesthesia of the upper and lower limbs and some loss of power in the legs. These symptoms

gradually became worse until the date of her attendance at hospital on January 4, 1939.

Physical Examination.—The patient showed marked pallor of the skin and mucous membranes; glossitis of mild degree and koilonychia were present. No abnormality was detected in the thorax; the spleen was just palpable, and the blood pressure was 130/70. Nervous system: The right pupil was smaller than the left, and the reaction to light was diminished as compared with the other pupil. Other cranial nerves were normal. In the upper limb all the reflexes were present, but were more pronounced on the left side. The knee and ankle jerks were present and equal, and the plantar responses were flexor. There was no loss of power in the muscles of the upper limbs, but in the legs there was a generalized diminution of muscular strength. Co-ordination and sphincters were normal. The examination of sensation revealed some absence of appreciation of cotton-wool in all four limbs, involving the distal halves of the forearms and legs; there was also "blunting" to pin-prick over the same areas. Vibration sense was much diminished in the right leg and to a lesser degree in the left, but was normal in both arms. Pronounced hyperaesthesia to deep pressure in all four limbs was found.

Subsequent Investigations.—Wassermann and Kahn reactions negative. Blood counts: (a) January 12, 1939—red cells, 3,470,000 per c.mm.; haemoglobin, 58 per cent.; colour index, 0.85; white cells, 8,400 per c.mm. (b) February 15—red cells, 4,540,000; haemoglobin, 62 per cent.; colour index, 0.69; white cells, 10,400. (c) March 5, 1939—red cells, 4,560,000; haemoglobin, 84 per cent.; colour index, 0.9; white cells, 7,500. A gastric analysis after a test meal (March 16) showed no free hydrochloric acid in any of the specimens of gastric fluid. The result of x-ray examination (March 14) showed that, although the stomach emptied rapidly at first, there was still a small residue four hours after ingestion of the barium.

Subsequent Progress.—The patient was placed on one tablet of ferrous sulphate thrice daily, and after a month's treatment the paraesthesia had disappeared. Periodic examination showed a gradual improvement of sensation, and in April, 1939, the only sensory abnormalities were a loss of sensation to cotton-wool over the terminal phalanges of the fingers. The reflexes of both upper limbs were now equal and the spleen was no longer palpable. By May, 1939, all sensation was normal.

Discussion

The first case presents several unusual features. It has been observed by many authorities that the age incidence of idiopathic hypochromic anaemia is strikingly constant, the great majority of cases occurring between 40 and 50 years of age. After the age of 50 the condition is uncommon, and it is distinctly rare in the sixth decade. Its occurrence in individuals over the age of 70, however, has been recorded by Haden (1932). The incidence of the disease in females far exceeds that in males, although the latter are by no means exempt (Witts, 1930; Haden, 1932). The question arises as to whether the anaemia in Case I can be designated "idiopathic," since two possible sources of chronic haemorrhage are present which might account for the anaemia. One of these sources can be eliminated, since no haematuria was observed subsequent to the operation of 1936. The possibility of loss of blood from leaking haemorrhoids, however, cannot definitely be excluded, and a chronic haemorrhage from this site has to be considered as the underlying cause of the hypochromic anaemia.

Anaemia as a sequel of gastrectomy is not an uncommon event. In all of Morley and Roberts's (1928) cases the anaemia was of the hypochromic type. Our second case appears to be a typical example of this group. The marked improvement in the anaemia was accompanied by parallel improvement in the neurological signs, and while

the latter mainly consisted of sensory changes and were consequently dependent on subjective sensations, there appeared to be no reason to doubt the evidence of the patient's observations, and she was not in the least a hysterical subject.

Two factors are common to both cases: (a) a hypochromic anaemia; (b) achlorhydria. Douthwaite (1936) suggested the term "gastrogenous polyneuritis" for the clinical syndrome of multiple neuritis associated with gastric dysfunction. While it has been recognized for many years that faulty functioning of the stomach is often associated with morbid changes in the peripheral nerves, it is not clear whether all such cases have a common aetiology. Douthwaite considered that the neuritis might be "dependent on an adequate supply of neuropoietin or some allied substance from the gastric or duodenal mucosa." Recently, much attention has been directed to the part played by possible vitamin B deficiency in polyneuritis, and Ungley (1938) and Laurent and Sinclair (1938) record examples of polyneuritis with gastric abnormality in which the vitamin B concentration of the blood was low and the response to vitamin therapy was pronounced. Hartfall (1934), in an extensive study of anaemia occurring subsequent to gastric operations, was impressed by the inadequate dietary in many cases, and particularly by the low content of vitamin B; he also emphasized the importance of rapid gastric evacuation and small intestinal hurry, both of which would tend to diminish absorption. Sinclair (1938) found low values of vitamin B in the blood of some cases of idiopathic hypochromic anaemia, and he suggests that this result is probably due to an impaired absorption consequent on the achlorhydria present, or to actual destruction of the vitamin in the stomach. He also mentions the relation between vitamin B and normal blood formation.

From a consideration of the above facts it would appear that the two cases we report could be classified as examples of gastrogenous polyneuritis. The criteria of this latter disorder, however, are not sufficiently restricted to warrant such affections being included under the one heading, and while the term "gastrogenous polyneuritis" may be of some value for clinical description, it is reasonable to assume that the underlying aetiology is not necessarily the same for all cases, and that future research will subdivide this group according to the underlying cause. The changes in the peripheral nerves described in association with pernicious anaemia and the concomitant achlorhydria could equally well be included under this category as an example of a gastric dysfunction coexisting with polyneuritis.

The importance of the hypochromic anaemia in our cases is difficult to assess. The anaemia may be coincidental with the changes in the peripheral nerves or may be a basic factor in the causation of the condition. In both cases iron medication was the only therapeutic measure adopted, and the response of the neurological signs was so pronounced as to suggest at least an association between the iron deficiency and the neuritis. It might be argued that the improvement in the patients' general condition was accompanied by an increase in appetite and that the augmented intake of food provided some factor the deficiency of which was responsible for the nervous lesions. An inquiry into the dietaries, however, revealed little discrepancy in the quality and quantity of the food before and during treatment. Since the vitamin B content of the blood was normal, there does not appear to be any reason why a deficiency of this principle should be responsible.

Summary

Two cases showing objective neurological changes are described.

Hypochromic anaemia and a disturbance of gastric function were common to both cases.

The possible aetiology is discussed.

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RHEUMATISM AND CLIMATE

BY

SIR LEONARD HILL, LL.D., M.B., F.R.S.

Rheumatic diseases are prevalent in North Europe, particularly in countries bordering on the North Sea, and to a less degree in Japan, North China, South-East Australia, Tasmania, and New Zealand, where climatic conditions approach those of Britain. The incidence of such disease diminishes progressively with the increase of tropical conditions from latitude 50° to that of 20°, being severe in latitude 50°–40°, not common in 40°–30°, rare in 30°–20°.

Rheumatic fever is reported to be unknown in hot tropical regions where there is no seasonal change of climate—for example, in Perak, Singapore, Central America, West Indies, Nigeria, and Gambia. Cases, however, are known to occur in the Punjab. In hospitals in New Orleans the incidence of mitral stenosis (rheumatic in origin) is stated as one-twentieth of that in Boston hospitals, while in Miami 1.3 per cent. of heart disease is reported as rheumatic in origin, in England about 32 per cent. In Switzerland, in the lower Alps, rheumatism is common; not so in the high Alps, where the dry cold and sunny and relatively calm atmosphere have curative effects. In Finland the greatest amount of rheumatic troubles is in the south-east; there are few cases in the open and colder regions of the north, although lakes and rivers are there abundant.

In a temperate climate such as Massachusetts one in ten of the inhabitants over 40 years of age are reported as suffering from rheumatism, and one in four of those of 70 to 80 years. In the U.S.A. there are twice as many cases of chronic rheumatism as of heart disease, ten times

as many as of tuberculosis, seven times as many as of cancer.

The financial loss due to rheumatic troubles is enormous. Twenty times more prevalent in the wage-earners than in the propertied class, they affect women nearly two and a half times more than men. Dental infections, tonsillitis, and respiratory infections are frequently associated with rheumatic affections: the last seem to be particularly provocative of rheumatic fever. The influence of herd infection is shown by the experience of the Presbyterian Hospital Training School, U.S.A. A group of healthy young women enter every six months as nurse students. They are carefully examined before and after admission. As the result of three months' work in wards full of patients suffering from severe respiratory diseases these students frequently are affected with sore throats and other respiratory infections, and any old rheumatic troubles are then likely to recrudesce.

Influence of Housing and Working Conditions

G. Edström, investigating the influence of housing and working conditions in Sweden upon the cause of rheumatic diseases, concludes that bad housing and working conditions have a large productive and also a deteriorating effect, for the rate of fully recovered patients is much greater among those who have lived under good or fairly good conditions. "It is nearly as important for the causation and prognosis of the chronic rheumatic infections to make more healthy the environment as to stop the local infection." Poynton likewise had drawn attention to the ill effect of the micro-climate of the house. The quickest reaction from rheumatic conditions, says Neergaard, is brought about by staying in a warm dry region, as in the desert sanatoria in Arizona, or in the high mountain sanatoria in Switzerland, where it is sunny, calm, and dry. In both conditions most of the time can be spent in the open air, where the deteriorating conditions of shut-up rooms do not act.

The influence on mortality of (a) social class and (b) crowding is shown by the following figures cited by M. Greenwood.

(a) In 1930–2 the mortality of the highest social class (males) was 13 per cent. better than the average mortality, while that of the lowest social class was 12 per cent. worse. For wives the respective figures were 19 per cent. better and 13 per cent. worse.

(b) For the two worst social classes the worst area had a mortality 50 per cent. greater for males than that of the best, and 47 per cent. for wives. In Durham the cottages in the rural areas are often mere hovels; nevertheless the mortality is 36 per cent. better than that in the county boroughs, and yet educationally and economically the rural folk are just as poorly off as the borough people.

It is noteworthy how sickly-looking people brought into a garden city, such as Letchworth, from some crowded part of London blossom into virile healthy citizens. Many authors regard cold, and above all cold and damp conditions, as causative of rheumatism. As changes of weather are commonly credited with making affected joints ache it is suggested that cold reduces the defensive mechanisms against infection. Against this supposition is the fact that people like the Eskimos and Lapps, greatly exposed to cold and mist, do not suffer from rheumatic troubles. Moreover, civilized people in temperate climates, who suffer from these most, live almost all their lives indoors in badly ventilated, artificially heated, and more or less polluted houses in towns, where the population is dense.