megalocytes were present; there was no excess of leucocytes or of blood plates; and good rouleaux were formed on the microscope slide.

It may be worthy of note that before medicinal treatment had been commenced, and in the earlier periods of treatment when the patient was receiving large doses of iron and arsenic, the blood plates were conspicuously deficient in number; while soon after the administration of bone-marrow had been commenced, a great increase occurred in their number, which was followed by a reduction to a moderate number during the later periods when the blood had been restored to a nearly normal condition.

The frequent failure of therapeutic measures in pernicious anæmia confers an interest upon any remedy which appears capable of controlling this malignant disease, even although the evidence is derived from one case only, and notwithstanding the circumstance that temporary improvement occasionally, though very rarely, appears to occur spontaneously. The facts now stated appear to justify the hope that bone-marrow will be found to have a remedial value in some at least of the cases of pernicious anæmia.

It is with much satisfaction that I express my obligations to Mr. D. A. Welsh, M.B., C.M., resident physician in my wards, for the efficient aid he has given me in conducting the numerous microscopic and other observations that were made in connection with this case, and to which only a general reference has been made in this abstract.

ACUTE DISSEMINATED MYELITIS.

[WITH COLOURED PLATE.]

By JULIUS DRESCHFELD, M.D., F.R.C.P.,

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ACUTE myelitis, according to the situation, distribution, and extent of the affection, is divided into poliomyelitis, when the affection is confined to the grey matter of the anterior horns; and leucomyelitis, when the white matter is chiefly implicated; of the latter we distinguish again between transverse or circumscribed myelitis, diffuse myelitis, and disseminated myelitis. (As in many cases of leucomyelitis the grey matter is also involved, this term, which was introduced by Vulpian, is now rarely used.) The terms diffuse and disseminated myelitis are not always used in the same sense by different writers—thus, while some, like Ross, include under acute diffuse myelitis all acute inflammations of the cord with the exception of the systemic diseases, others use the terms diffuse and disseminated myelitis as synonymous; while others, again (Leyden), group diffuse myelitis with transverse or circumscribed myelitis. Both from an anatomical and from a clinical point of view the classification of Gowers is to be recommended. He divides myelitis into—transverse myelitis, when the whole thickness of the cord is affected in a small vertical extent; diffuse myelitis, when an extensive area of the cord is inflamed in continuity; focal myelitis, when one small area is affected; and disseminated myelitis, when there are many foci contiguous or distant.

Of the different varieties of acute myelitis, the disseminated form is one which presents many points of interest, both on account of its etiology—it being frequently observed after infectious and toxic diseases—its symptomatology and its relations to other spinal diseases.

As but few cases of acute disseminated myelitis with *post-mortem* records have as yet been described, I wish to record the following case:

J. R., aged 23, was seen by me on May 20th, 1893, in consultation with Dr. Harris and Dr. Rigby, of Chorley.

History.—He had enjoyed good health and had been very fond of athletics; he had been very regular in his habits and there was no history and no sign of syphilis. Several members of his family had died of tuberculosis. Whilst out shooting in the middle of April, 1893, he fell into a pond and had to walk two miles to change his garments. About ten days after the accident he suffered from severe pain on the right side of the forehead and rapidly lost the sight of the right eye. He consulted Dr. Little, of Manchester, who found pronounced optic neuritis of the right eye. He improved under treatment and regained vision to some extent. About the middle of May he had much pain and swelling in the right leg, for which he was confined to bed for a few days. The pain and swelling subsided but on attempting to get up he found that he could not use the left leg. Soon after both legs became weak, he complained of numbness and cramps in the legs, and shortly after the left leg became

completely paralysed and the right very weak; he had pain round the body in the region of the ensiform cartilage. Incontinence of urine for one night was followed by retention necessitating the regular use of the catheter. Constipation was also present.

When I saw the patient, I found him to be a muscular, well-developed youth; he was in bed and unable to walk or stand, and with some difficulty could raise himself in bed. He did not complain of any pain then. The vision of the right eye was very much diminished, the pupil dilated and sluggish in its reactions, and the optic disc greyish in colour and showed marked swelling; its outline was indistinct and its vessels were hyperæmic. The fundus of the left eye was normal. All other cranial nerves appeared normal. The upper extremities were normal, both as regards motion and sensation; the radial and triceps reflexes were increased, there was no inco-ordination of the upper extremities, and the muscular sense was intact; the upper part of the trunk appeared normal. About 2 inches below the ensiform cartilage there was a line of anæsthesia on the right, of paræsthesia on the left side; the anæsthesia on the right side affected the whole of the right half of the abdomen, right half of the scrotum and penis, and the whole right lower extremity; the anæsthesia was complete for tactile impressions, for pain, and for temperature; on the left side the loss of sensation was not quite so marked, but some patches both on the abdomen and on the left lower extremity appeared quite insensible. There was complete loss of power of the left leg and thigh and marked weakness of the right lower extremity; the superficial and the plantar reflexes were diminished on both sides, the knee reflexes increased, cremaster and abdominal reflexes were absent, and the epigastric reflexes increased on both sides. The chest and abdominal organs showed nothing abnormal; the pulse was 80, fairly strong, the temperature normal. There was retention of urine, but it was free from pus and mucus, and in other respects normal.

Diagnosis.—The motor and sensory symptoms left no doubt that we had to do here with acute myelitis affecting the lumbar region of the cord, and extending almost across the whole thickness of the cord. The optic neuritis in the absence of other cerebral symptoms except a localised pain was looked upon as either due to meningitis, or as due to an inflammation of the optic nerve. As optic neuritis had been noticed in several cases of disseminated myelitis, I ventured upon the diagnosis of disseminated myelitis, and expressed the fear that the disease would extend. The cause of the affection remained obscure, for there was no history of syphilis, and the patient had not suffered from any acute infectious disease. The symptoms having come on soon after the immersion in the ditch, it was thought that this probably might have acted as the exciting cause.

Treatment.—Iodide of potassium, mercurial inunction, salicylate of sodium, and liquor strychnine.

Progress.—The patient was removed to Manchester and placed under the care of Dr. R. Wild, with whom I saw him several times. The symptoms rapidly got worse and the affection spread, as will be seen from the following short extracts from Dr. Wild's notes:

May 26th. Complete paralysis and anæsthesia of both lower extremities; knee-jerks absent, no ankle clonus; band of hyperæsthesia about 2 inches broad passing all round the body at the level of the ensiform cartilage, and the patient complained of girdle pain in this region; the skin of the abdomen below this line completely anæsthetic; complete retention of urine; the rectum anæsthetic; the bowels were moved by an enema.

June 1st. The urine drawn off by the catheter contains blood, pus, and mucus; the temperature 100°, pulse 90, tongue furred; bed sores began to form on the heels, back, and over the left trochanter.

June 16th. Sensation has partially returned over the whole of the abdomen and part of the right thigh; the left leg and thigh remain anæsthetic. The symptoms of cystitis have disappeared; the bed sores are healed.

June 23rd. Can move the right foot and also the right thigh slightly; complains of painful spasmodic twitchings in both legs.

June 24th. Headache over left side of forehead and in left orbit; the sight of the left eye is rapidly failing, left optic disc very much swollen.

July 4th. Vision of left eye slightly improved, pain over right side of forehead and orbit, total blindness of right eye with increase of papillitis, condition otherwise the same except that there are signs of beginning contractures in the lower extremities, the knees slightly flexed and pressed together.

July 21st. The spastic condition of the legs more marked. The patient now complains for the first time of a hot feeling in the left arm with occasional numbness and tingling. Motor power in both arms still very good.

July 22nd. Marked tremor in the left arm and hand when any movement is attempted, the grip of the left arm is weaker, the right arm shows marked hyperæsthesia, and is painful on the slightest touch.

July 23rd. Tremor and weakness in left arm more marked, and there is slight rigidity to passive movement; the reflexes in left arm are much increased. In other respects the condition the same, patient more emaciated.

July 24th. The right arm is now almost completely paralysed, but still hyperæsthetic; a band of hyperæsthesia passes across the chest at the level of the third costal cartilage; complete anæsthesia and motor paralysis below this level except intercostal muscles which act, but somewhat feebly; the diaphragm is contracting feebly, the upper part of the chest moves well, the lower intercostal muscles, are nearly paralysed; speech and deglutition not affected. The lower extremities are completely paralysed and rigidly contracted. The temperature is slightly raised, pulse quickened. The patient is quite conscious, and there is no affection of any of the cerebral nerves except the optic nerves; he is now completely blind.

July 26th. The respiratory movements are gradually failing, face and lips are cyanosed.

The patient gradually sank and died from asphyxia on July 27th.

Briefly summed up we had here the case of a young man who, in the midst of good health, was suddenly seized with optic neuritis of one eye, followed by a gradually ascending

spinal paralysis, affecting both motion and sensation, beginning first in the legs and followed soon after by optic neuritis of the other eye and then by paralysis of the upper limbs, death being caused by paralysis of the respiratory muscles.

Post-mortem Examination Fourteen Hours after Death.—Body much emaciated, especially the left leg; brain normal but oedematous; optic nerves presented red points on transverse section, there was no distension of the sheath; spinal cord, dura mater distended by fluid, but there was no pus or blood; the pia mater not thickened; the upper part of the cord from the second cervical to the lower end of the cervical enlargement was soft and on section almost diffuent; the dorsal and lumbar region appeared firmer. Fragments of the pulp of the cord, examined microscopically, showed fat cells, masses of compound granular cells, small round cells, and blood corpuscles. The cord was placed at once in hardening fluid for microscopic examination in the clinical laboratory of the Owens College by my assistant, Dr. Williamson. After hardening in Müller's fluid numerous disseminated patches of pale colour were seen on transverse section; these patches were scattered throughout the cord in the most irregular manner. The subjoined diagram shows the distribution of the larger patches of myelitis as seen with the naked eye.

1 Cervical

5 Dorsal

2 Cervical

6 Lumbar

3 Dorsal

7 Lumbar

4 Dorsal

8 Lumbar

DISSEMINATED MYELITIS

In sections stained according to Weigert's method and examined under a low power, numerous pale brownish-yellow patches, irregular in shape and size, were seen in all three regions of the cord. (See coloured drawings.) In these pale brownish-yellow patches the nerve fibres were absent, completely or partially. The small vessels at the pale (myelitic) patches were greatly dilated and filled with red corpuscles. Under a higher power their perivascular sheaths were seen to be greatly distended with cells. The whole of the more recent patches of myelitis were seen to be densely infiltrated with round cells—compound granular cells and leucocytes. In those parts at which these patches extended to the grey matter of the cord the ganglion cells and fine nerve fibres were absent, and their place occupied by leucocytes and compound granular cells.

In the older patches there was an increase of neuroglial connective tissue. The posterior median columns in the cervical region presented the usual changes found in ascending degeneration of these columns—absence or marked degeneration of nerve fibres and increase of neuroglial connective tissue to a marked extent; this new-formed connect-

ive tissue contained numerous nuclei, and many round and oval spaces, from which the nerve fibres had disappeared. In sections stained according to Marchi's method, numerous black dots (degenerated fibres) were seen in the patches of myelitis, and also in the parts affected by ascending degeneration (Goll's columns cervical region). The chief points respecting the changes in the cord were:—

1. The irregular distribution of the myelitic patches.
2. The irregular size and shape of the patches. At some places the myelitis occupied one half of the white matter of the cord; at other parts the patches were so small that they could only be recognised under the microscope.
3. The changes were most marked around blood vessels, and where the myelitis patches were small they were limited to the neighbourhood of small arteries.
4. Another striking feature was the marked distension of the perivascular sheaths with leucocytes and compound granular cells. In every section the vessels at some part were markedly dilated, and the perivascular sheaths distended with cells.
5. There was well-marked ascending degeneration in the posterior columns (in disseminated sclerosis ascending degeneration is generally absent).

OPTIC NERVES.

On transverse section of the optic nerves marked changes were found in both nerves. In sections stained according to Weigert's method, there was almost complete absence of nerve fibres in the centre of the optic nerve. At the periphery of the transverse sections there was a narrow irregular rim of bundles of healthy nerve fibres, stained black, whilst the centre was occupied by brown stained neuroglia connective tissue, from which the nerve fibres had disappeared; this connective tissue was well supplied by nuclei, as seen in the sections stained with logwood. The sections presented many greatly dilated blood vessels, especially in the central part of the nerve. The perivascular sheaths of the vessels were enormously distended with cells—compound granular cells and leucocytes.

Specimens stained according to Marchi's method showed that around the periphery of the sections there were numerous degenerated fibres, stained black, mixed with the healthy fibres, whilst in the centre of the sections, where the connective tissue was in great excess, the degenerated myelin had for the most part disappeared. Both optic nerves presented similar appearances. Sections of the cord of the optic nerve were examined for micro-organisms after the usual methods, but with negative results.

A case similar to the above both in its symptoms and in its combination with optic neuritis I described in the *Lancet*, 1882, p. 14; a similar case is given by Archard and Guinon,¹ and another by Sharkey and Lawford.² Other fatal cases are those by Westphal,³ Kussner and Brosin,⁴ Th. Barlow,⁵ Francotte,⁶ and Gowers.⁷

That so few cases have been recorded is partly due to the fact that primary acute myelitis is not a very common disease, as is shown by the observations of Oppenheim,⁸ who during a period of eight years in the Charité Hospital, at Berlin, could only record the necropsy of two cases of pure myelitis (I am bound to state that the *post-mortem* records of the Manchester Infirmary show a larger number, most of which had been carefully examined and shown to be pure and uncomplicated cases of myelitis), chiefly because many cases of acute myelitis do not run a fatal course and may often undergo a complete cure.

It may not be without interest briefly to refer now to some of the chief features of acute disseminated myelitis.

ETIOLOGY.

1. In a few cases we cannot find any definite cause, except cold or excessive muscular work, and these we may look upon as belonging to the so-called rheumatic form.
2. In by far the greater number of cases the affection comes on during or after an acute infectious or contagious disease such as measles (Barlow), erysipelas, small-pox (Westphal), typhoid, malaria, scarlet fever, gonorrhoea (Gowers). I may here mention a case which I saw with Dr. Massiah, of Didsbury, and Dr. Little, of Manchester, where a young lady whilst suffering from an attack of measles, was seized

with intense headache and optic neuritis, which caused total blindness, followed by paralysis and by numbness of all the four limbs, and where eventually complete recovery took place.

3. In a number of recorded cases syphilis is cited as the etiological factor, and several of these were associated again with optic neuritis," whilst in a few they have been found associated with the puerperal state, with tuberculosis or cancer.

4. The toxic agents which so often give rise to peripheral neuritis may also affect the spinal cord, but the peripheral changes are here much the more prominent ones.

5. In pernicious anæmia Lichtheim¹⁰ and others have often found marked disseminated changes in the cord, affecting, however, chiefly the posterior and lateral columns.

6. Occasionally disease or injury of peripheral nerves may give rise to acute myelitis, probably by extension.¹¹

SYMPTOMATOLOGY OF ACUTE DISSEMINATED MYELITIS.

The affection consists in a more or less complete paralysis, first of one, then of both limbs, and extending gradually to the upper extremities, with the accompanying sensory disturbances and affections of bladder and rectum. From transverse myelitis and compression of the cord the disease is often at first indistinguishable; but the extension of the affection, and especially the early disappearance of the deep reflexes which is noted in nearly all the cases quoted above, will often enable us to distinguish between the two affections. Not that in transverse myelitis or compression myelitis the knee reflexes are always found exaggerated, for we know from the observations of Bastian and others that where there is a complete transverse myelitis affecting all motor and sensory tracts the deep reflexes are absent; but while this occurs usually only in a few cases—unless the transverse myelitis is traumatic as in the cases recorded by Thorburn,¹²—and late in the disease, in disseminated myelitis it occurs early, and, as the *post-mortem* examinations have shown, there is no complete transverse myelitis in these cases. The presence of optic neuritis due to a distinct acute inflammatory process appears to be a not infrequent complication of acute disseminated myelitis, and may thus form another diagnostic feature; other parts of the central nervous system—medulla, pons, and brain—may be affected in acute disseminated myelitis, and the combination of symptoms which thus arise will vary considerably.

Leyden describes under acute ataxia a form of disseminated myelitis, about which I would like to make some observations.

Acute ataxia includes a variety of diseases. We have the acute pseudo-ataxia, which is a form of peripheral neuritis; and acute ataxia, which is an affection of the central nervous system. This latter may either prove quickly fatal, or undergo complete cure, or improve for a time, and eventually show all the signs of ordinary ataxia. Of this type I have seen lately two cases where after a severe cold marked ataxia came on in two days. After a week other typical signs of locomotor ataxia developed; marked improvement took place in both cases, so that the patients could again follow their employment; but both cases relapsed, and are now suffering from advanced locomotor ataxia. Some of the cases of acute ataxia, of the bulbo-spinal type, Leyden regards as cases of acute disseminated myelitis. Up to now there appears to be only one *post-mortem* record¹³ of such a case, for the recorded case of disseminated myelitis quoted above, and, indeed, the other cases quoted also, showed no marked ataxia.

The following case, which I saw with Dr. T. M. Helme, of Manchester, evidently belongs to the class of cases mentioned by Leyden, and may be briefly described.

A boy, of 10, was taken ill with some febrile symptoms of an indefinite nature, lasting a few days. He complained of sore throat, but no membrane was seen on the fauces. The boy quickly recovered from the acute attack, but three weeks after was taken with weakness, first in one and then in the other leg, and had such marked ataxia that he was unable to walk. He became feverish with quick pulse and considerable prostration. When I saw the boy he was in bed. The legs were weak but he could move them, but the movements were very inco-ordinate; he was able to stand, but only with his eyes open and his legs apart, and, when attempting to walk, his legs jerked from excessive inco-ordination; he could not stand or walk with his eyes shut; there was some dyæsthesia of the lower extremities, but, owing to the prostrate condition, the degree and extent of this could not be made out satisfactorily. The knee re-

flexes were absent, the superficial reflexes diminished; the arms showed little weakness and only slight inco-ordination; the breathing was quick and shallow, the diaphragm and intercostal muscles appeared very weak, and it was noticed that on the left side of the chest the respiratory movements were much weaker than on the right side; there was difficulty of swallowing, the voice was weak and husky, the uvula moved slightly, there was marked weakness in the movements of the lips and of the tongue; the upper part of the facial nerve appeared unaffected, and he was able to close both eyes; there was no affection of the eye muscles, and the pupils reacted to light and accommodation. The pulse was 130 and very weak. The condition rapidly got worse, the paralysis of the diaphragm became soon complete, and the boy died with all the symptoms of suffocation. The *post-mortem* examination, which was made by Dr. R. Wild, showed the cord and peripheral nerves to be normal, and only a small patch of softening was found in the left half of the medulla, extending to the periphery implicating the restiform body, and the nuclei of the pneumogastric and hypoglossal nerves. The part affected was almost diffuent, and microscopic sections were obtained only with great difficulty; they showed small hæmorrhages, engorgement of vessels, cell proliferation in the perivascular sheaths, and large collections of round cells in the neighbourhood of the vessels. Of the nerve elements little more than masses of broken up myelin could be observed. A few similar, but much smaller, foci of inflammation were found on the right side of the medulla.

This case, which resembled in its symptoms post-diphtheritic paralysis, was evidently one of acute myelitis of the medulla, and the ataxic symptoms could be readily explained by the situation of the myelitis, which had destroyed the greater part of the restiform body on the right side. Whether the attack from which the boy suffered three weeks before the onset of the nervous symptoms was diphtheria could not be ascertained.

The pathological changes in acute disseminated myelitis appears sufficiently obvious from the cases so far recorded; in all of them we find an inflammatory process most marked round the blood vessels, which are dilated, their sheaths distended with round cells and cell accumulation in the neighbourhood. From this it would appear that the inflammatory process starts from the blood vessels and extends thence to the surrounding tissue, and that the nerve elements are, to a great extent, only secondarily affected. The pathological changes found are very similar to those seen in other forms of acute myelitis and of many cases of acute poliomyelitis, as pointed out by Goldscheider¹⁴ and Reinhold.¹⁵

PATHOLOGY OF ACUTE DISSEMINATED MYELITIS.

Considering the principal causes of disseminated myelitis, which are either of an infectious or a toxic nature, and the distribution of the inflammatory changes near the blood vessels, we naturally connect the two, and look upon the lesions as the effect of some toxic agent circulating in the blood. This view, which is now held by many observers, explains the close connection between peripheral neuritis and the various forms of myelitis, and it is now well established that in peripheral neuritis we do occasionally find inflammatory changes of the cord. It explains also the irregular distribution of the affection, and how it may affect other portions of the central nervous system and cranial nerves, such as the optic nerve, where, indeed, the perivascular inflammatory changes are most marked, and best studied. It is interesting to note that in multiple peripheral neuritis very similar changes are sometimes seen in the optic nerves.¹⁶ As the process affects at first only the interstitial tissue we can also readily understand that absorption of the cells infiltrating the tissues may take place, and if the nerve elements are not irreparably damaged, marked improvement and even a complete and permanent cure may ensue, as no doubt it does in many cases.

We may go further, and compare acute disseminated myelitis with another affection of the cord—namely, disseminated sclerosis—and we notice a close relationship. Here we must often assume an infective agent as the primary cause; we notice, also, the irregular distribution of the lesion in various parts of the central nervous system, and the almost constant implication of the optic nerve; further, we find here, as Déjerine, Hess, Buss, Babinsky, and others have pointed out, the changes proceeding from the blood vessels; and, lastly, we often observe here, also, great amelioration of the symptoms.

About the treatment of acute disseminated myelitis little need be said, as it does not differ from that of other forms of primary myelitis. It is essential as soon as the affection is recognised, and before there is complete paraplegia, to confine the patient to bed, and enjoin him to keep absolute rest.

The application of a spinal ice bag, of counter-irritation to the back, at the outset of the disease may have some tendency to check the exudation. Bearing in mind the toxic nature of the affection, and the good effects seen in multiple peripheral neuritis and retrobulbar optic neuritis (not infrequently seen after epidemics of influenza), large doses of salicylate of sodium and of iodide of potassium should be given, and the internal or subcutaneous administration of strychnine, even if spastic symptoms are present, may well be tried, though it should be given cautiously. The marked improvements which have been noticed after the administration of mercury in some cases where there was a syphilitic history may perhaps have been due to an antitoxic property of this drug, and it might, therefore, be tried in non-syphilitic cases. The same applies to arsenic and quinine. As arsenic, even when administered in medicinal doses, may occasionally produce peripheral neuritis, we should be careful in using it; on the other hand, the beneficial effects of arsenic in pernicious anæmia, where, as pointed out above, a disseminated myelitic affection has repeatedly been noticed, is a strong recommendation for a trial with this drug. Massage and electricity had better be deferred to a later stage of the disease.

EXPLANATION OF COLOURED PLATE.

Sections of the cord and optic nerve stained after Weigert's method (low power). Fig. 1. Cervical region. Fig. 2. Dorsal region. Fig. 3. Lumbar region. Fig. 4. Optic nerve. The areas of degeneration in the white matter are stained brownish yellow.

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A NOTE ON A NEW METHOD OF PREPARING CULTURE MEDIA.

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ONE of the chief sources of the difficulty which is experienced by bacteriologists in regard to the cultivation of pathogenic microbes lies in the fact that the composition of many of the media used for the purpose differs so widely from that of the blood and other fluids found in the animal tissues. By means of the method here described media can be prepared directly from these fluids by a process which reduces the difficulties of manipulation to a minimum.

The one perfectly satisfactory method of sterilising any medium is to raise it to the temperature of boiling water. What is required, therefore, is that from these fluids solid media should be prepared which are perfectly transparent and yet easily sterilised at 100° C. or over it.

Ordinary serum, when coagulated by raising it to the boiling point of water, forms an opaque mass. If on the surface of this the germ of anthrax be planted, it grows with the greatest readiness. As the germ continues to grow, it can be observed that in the layer of the coagulum immediately in contact with the anthrax the opacity begins to disappear. This change proceeds till finally there remains in place of the opaque mass a clear transparent jelly. The most probable explanation of this change is that it is due to a body of an alkaline nature which diffuses into the medium and combines with the proteid. We are already acquainted with a form of proteid, first described by Lieberkühn in 1848, which closely resembles this substance; it is known as "Lieberkühn's jelly," and is formed by adding potash to egg white. By this means he obtained a solid and very insoluble proteid in the form of a clear jelly. It was pointed out by Lieberkühn that if this alkali-albumin be washed till the free alkali is completely removed, it has a neutral reaction

to litmus. According to Mörner, who has more recently investigated the question, the reaction is weakly acid. It is important to bear this in mind, because it shows that in the process of combination with the proteid, the alkali disappears. If, therefore, we add to a solution of proteid such as a tissue fluid, a quantity of free alkali just sufficient to form the combination, we do not seriously alter the weak alkaline reaction which it normally has. In any case, the amount of alkali needed to prevent the occurrence of opacity in coagulation by heat is very small.

A fact illustrating the ease with which the opacity may be prevented in coagulation is that if serum be in a sufficiently thin layer in a glass vessel, the result of raising it to the boiling temperature is a clear jelly. The rule for a method of solidifying solutions of proteid in a clear form must be to add an amount of alkali which is just sufficient to discharge opacity. If we add to ox serum 0.1 per cent. to 0.15 per cent. caustic soda, and heat the solution to 120° C. in the autoclave, we obtain the desired transparent jelly. Too little alkali gives a coagulum with a greyish appearance. Care must be taken to have clear serum, and in particular it must be free from hæmoglobin. The amount of alkali required varies a little with the character of the serum. In regard to egg white the same facts hold. Here, again, the amount of alkali to be added depends on the nature of the egg used. Certain kinds of eggs coagulate in a fairly transparent form without any addition.

In the case of the hen's egg the following method has been found reliable. Break up the white with an egg beater till it loses its consistency. Add to it 40 per cent. of water, and mix well. Pass the mixture through muslin to remove any shreds of insoluble material, or let it stand overnight in a cylinder till these settle to the bottom. Add 0.1 per cent. caustic soda, and solidify in the autoclave. Egg white is naturally more free from pigment than serum, and with a little care in clearing it a jelly can be obtained closely resembling gelatine in transparency. Substances like glucose may be added as desired.

In regard to the question of growth, experiments have been done to show that a large variety of bacteria grow on this medium with great readiness. The effect of the alkali on the pathogenic character of the bacteria or their products has not been to any extent determined. A few preliminary experiments on the diphtheria bacillus seem to indicate that virulence is maintained as efficiently as in the case of agar or beef-broth cultivations. It may also be noted that alkali albumin in the liquid form has been extensively used as a culture medium. The ease with which the solid form can be prepared renders its introduction desirable.

HOSPITALS AND BACTERIOLOGICAL STATIONS IN TURKEY.—The Sultan of Turkey has decreed that each of the ten municipalities of Constantinople is to be provided with a hospital with a sufficient staff in order that day and night a medical man may be in attendance. A great many have what is called a hospital, organised in a private house, therefore very imperfectly. The Constantinople Bacteriological Laboratory is finished and will soon be opened; analyses of water and food will be made in it. The Bacteriological Institute has received a grant of fr. 480, and will receive an annual income (fr. 320). Hydrophobia patients will be treated at Stamboul at a hospital which will be built for the purpose with a bacteriological laboratory attached to it; meanwhile, the Constantinople institute will receive patients from all parts of the Turkish Empire.

MEDICAL EDUCATION FOR WOMEN IN RUSSIA.—The question of the establishment of the proposed school of medicine for women in St. Petersburg will shortly come before the Imperial Council. An endowment sufficient to produce a revenue of 63,000 roubles (£6,300) a year is required. Of this revenue only 42,000 roubles can be counted on. The city of St. Petersburg has promised a subvention of 15,000 roubles annually, and the rest is made up from legacies and funds supplied by private persons. Apparently the State does not see its way to unloose its purse strings. The Municipal Council has given a site for the building, and the city hospitals have promised to throw open their wards to the female students.