some impairment of the functions of the spinal cord as far as conduction in its long axis was concerned.

On stimulating one arm, however, both upper limbs contracted, showing conduction transversely in the upper part of the cord still persisted. After 45 minutes the transverse conduction had also disappeared, for stimulation of one upper extremity only caused contraction of the irritated limb, and not of the opposite one, although when the electrodes were placed over the upper cord itself both limbs responded. On applying the electrodes to the eye directly at this stage, the protected limb contracted well, and the three poisoned ones feebly only, while 7½ minutes later this powerful stimulus produced a reflex action of a very feeble nature in the protected leg only. The heart was still beating, but respiration had ceased for some minutes, the animal being quite flaccid, and apparently dead; the nerve trunks of the limbs were now exposed, and stimulated directly, to ascertain how far the end plates were paralysed, with the following results: The muscles of all four limbs still responded to direct stimulation. The sciatic nerve of the left (poisoned) limb gave no response at all with the secondary coil at 0. That of the right (protected) limb responded with the coil at 15 mm. On testing the arm nerves, contractions were produced with the coil at 7½ mm, but not at 10 mm, showing only partial paralysis of the end plates of the arm muscles at a time when those of the poisoned lower limb were completely paralysed; an important point which must be taken into account in considering how far the changes in the reflexes detailed above can be taken as evidence of loss of function of the spinal cord, as apart from the affection of the motor end plates. The loss of the transverse reflex in the upper cord when the motor end plates of the muscles of the upper extremities were not paralysed, points to a diminution of the reflex functions of the spinal cord. On the other hand, the marked reflex contraction of all four limbs on applying a strong current to the eye just after respiration had ceased, shows that the reflex functions of the cord were not abolished at this period, although they rapidly declined within a few minutes of complete respiratory paralysis, as would be expected. The rapid loss of the upper extremity, as compared with those of the lower limbs, accounts for the ascending paralysis apart from any interference with the functions of the spinal cord itself.

We must conclude, then, that the respiratory paralysis is complete before the reflex functions of the spinal cord are abolished, although they may be diminished at an earlier stage, so that the action of Enhydrin poison on the spinal cord itself is of quite secondary importance as compared with the paralysis of the respiration, and of the motor end plates of the muscles.

Conclusions.

1. In lethal doses, Enhydrin poison has no direct depressing action on the heart. The marked rise in blood pressure observed is secondary to failure of respiration, producing venostasis of the blood.

2. The primary action of the poison is the production of a respiratory paralysis by a direct action on the respiratory centre, this being very quickly followed by paralysis of the end plates of the phrenic nerves.

The latter may occur at a time when the sciatic nerves show no end plate paralysis.

3. The poison has a very marked action in paralysing the end plates of motor nerves, but does not perceptibly affect the conducting powers of the nerve trunks themselves. In this respect it resembles cobra venom and curara.

4. Its action on the reflex functions of the spinal cord is slight, and altogether secondary in importance to its influence on respiration.

Review.

An Atlas of Illustrations of Clinical Medicine, Surgery and Pathology.—Compiled for the New Sydenham Society (a continuation of the "Atlas of Pathology"), Fasciculus XIV (Double Number), Fasciculi I and II of New Series, Framboesial Syphilis (Yaws and Parangi). Plates A to H and LXXV—XCI. London, The New Sydenham Society. Agent: H. K. Lewis, 136, Gower St., W.C. Price to non-members one guinea.

These fasciculi deal with yaws or as the author prefers to call it framboesial syphilis. In Part I the disease is described under the headings of "primary sore, secondary and tertiary stages." This description is good and is supplemented by a number of excellent black and white illustrations; the question as to whether yaws, parangi, coko, &c., are one and the same disease is also discussed.

Part II consists of a large number of coloured engravings of the disease, with case histories, as it occurs in Ceylon, prepared from drawings of Sir William Kynsey; these are of a high standard of excellence. The author acknowledges that these fasciculi are of a controversial nature, and at the end of Part I states his own opinion that "yaws," "parangi" and framboesial syphilis are caused by syphilis modified in the case of yaws, &c., by climate and race. He quotes a large number of authorities in favour of this view, but we cannot help thinking that sufficient prominence is not given to Powell's work which, to our mind, proves that syphilis and yaws are two distinct diseases; no doubt showing similarities, but very less well-marked than in many other diseases which nobody doubts are dissimilar. This is scarcely the place to enter into a long controversy on the subject, but we must take exception to one statement of the author, i.e., that in tropical countries syphilis spreads very commonly as a non-venereal disease; the primary sore being on the limbs or trunk, flies being the carriers of the virus, this is not our experience in India.

To mention one apparently very strong point in favour of the view that the diseases are dissimilar, is that a large number of cases of yaws occurs between the ages of two and fourteen, i.e.,

* Powell, J. M. G., Vol. XXXII, page 365.
before active sexual life begins and later than the usual time of appearance of symptoms of inherited syphilis. Again Powell* has seen cases in which the patient has contracted a hard chancre with the usual sequelae whilst still suffering from yaws.

**Elementary Bacteriology.**—By M. L. Dhingra, M.D., C.M. Published by Longmans, Green & Co. Price, 3s.

The author's aim in this little book is to deal in a concise manner with the fundamental principles of bacteriology, and to so select and arrange the material at his disposal as to meet the requirements of Indian students and practitioners.

The book is arranged in two parts. Part I contains chapters on the Theory of Spontaneous Generation, Fermentation, the Morphology of Bacteria, Putrefaction, Antiseptics and the Preservation of Food Stuffs.

Part II deals with the different Bacteria in Disease.

There are two appendices, one on the principles of Bacteriological Technique, and one on Snake Venom and Antivenous Serum.

When it is seen that the book is comprised of about 140 pages of fairly large print, in which number are included twenty-six illustrations, it must necessarily follow that the twenty-six chapters and the two appendices into which it is divided are extremely brief. In fact each chapter consists of little more than mere scraps of information about the subject with which it deals.

So far as it goes the material it contains is fairly accurate, but we do not consider it at all sufficient to meet the ordinary requirements of Indian students and practitioners.

We have nothing but praise for the way in which the publishers have done their work, the type and paper being excellent.

The plates and illustrations, most of which are copies of illustrations from other works on Bacteriology, are very well selected, and add considerably to the value of the book.

---

**EXTRACTS FROM MEDICAL JOURNALS.**

**MEDICINE.**

The Clinical estimation of the blood pressure and its great value in cases of Cerebral Compression.—In the Journal of the American Medical Association of May 2nd, 1903, there is an article by Dr. Henry Wireman Cook, in which is a description of a manometer for the clinical estimation of the maximum blood pressure, that is the pressure required to obliterate the radial pulse by compression of the brachial artery. It consists of a mercurial manometer connected with a Y-shaped tube, one arm of the Y being attached to an extensible rubber bag which completely encircles the arm about its middle, so that "the pressure is thus transmitted equally through the medium of the tissues to the artery, perpendicular to its wall at every point." To the other arm is attached an air-pumping bulb as is used for a Faquelin's cautery. By means of this the pressure is raised till the radial pulse disappears. The height of the mercurial column is noted. On the cessation of pumping the pressure in the apparatus begins to fall owing to escape of air, and the pulse will again appear at the wrist. The height of the mercury column at this point is the mean of the two readings taken as the maximum blood-pressure. The apparatus having been fixed on, the two readings are completed in the 30 seconds required for the pulse count. The instrument can be obtained from Messrs. Eimer & Amend, 205-211, Third Avenue, New York City, in two forms—for hospital or private use.

An interesting illustration of the sphere of usefulness of this instrument is furnished in The American Journal of the Medical Sciences for June, by Dr. Harvey Cushing, in an article entitled "The blood pressure reaction of acute cerebral compression, illustrated by cases of intra-cranial hemorrhage." The paper brings into line much experimental work on animals with what has been recognised clinically. He shows that when the cerebral compression is severe enough to cause symptoms it is accompanied by an increase in blood pressure, which may range to nearly three times the normal (in one case it rose from 130 mm, the normal, to 360 mm.), and that later this rise gives place to a fall which continues till it terminates in death. His point of view is that the rise of blood pressure is due to the compressing force causing extravascular pressure on the capillaries of the vaso-motor centre and the bulb sufficient to narrow their lumen and produce partial cerebral anemia. This anemia of the vaso-motor centre produces, as bulbar anemia always does, general arterial constriction. The resultant rise in general arterial pressure will force the blood through the capillaries of the bulb against the increased extravascular pressure. It will, at the same time, be likely to force out of the torn vessel any clot which is plugging it so that bleeding may begin again. This will produce further rise of intra-cranial pressure, further anemia of the vaso-motor centre, further vaso-constriction, and further rise in blood pressure, and the consequent restoration of the circulation through the capillaries of the vaso-motor centre. The rise in blood pressure is terminated, either by the cessation of bleeding, in which case with the slow absorption of the clot there will be a gradual fall in blood pressure, or by a continuance of the bleeding till the tired out vaso-motor centre is unable to respond to the stimulus of anemia and fails, the pressure now being of a dilatation being accompanied by a rapid fall of blood pressure. By this time the vaso-motor centre is so exhausted that it will not respond even if the compression is relieved. In accordance with Kocher he divides the stages of cerebral compression as follows:

- **First Stage.**—The compression is slight, and is compensated for by the escape of cerebro-spinal fluid and some narrowing of the venous channels. The symptoms are insignificant.

- **Second Stage.**—The compression causes some cerebral anemia, shown by headache, vertigo, restlessness, excitement or drowsiness. The compression of the larger venous channels inside the skull is shown by venous congestion of the veins outside the skull opening into them, recognized by dilatation of the veins in the fundus of the eye, the "eye-grounds" as he calls them. Bulbar anemia is shown by slowing of the pulse, but this may not be enough to cause a rise in blood pressure.

- **Third Stage.**—The stage in which the symptoms are characteristic, the blood pressure is rising, and the rise may or may not be able to keep the bulb from becoming anemic. The periodicity of symptoms such as Cheyne Stokes respiration, and the varying size of pupil is

---

* Powell, J. M. G., vol. xxxii, page 365.