GENERAL DISCUSSION.

Dr. JOHN BROWN (late M.O.H. Bacup) said that in temperate climates flowers, vegetation, and children thrived under substantially identical conditions of sunlight and air. When vegetation was grimy to the touch it was stunted, and children were physically deteriorated. Some of our industrial towns remained virtually under a sunblind, with serious results to health. He believed that the generation of electricity at large centres would make for economy in industry as well as improvement in the public health. The diminished ratable value of property in the immediate vicinity of gasworks and similar undertakings was a measure of their cost to the public, and this would be saved by placing the power-generating stations of the future at a distance from towns.

Dr. EIDINOW (National Institute for Medical Research), who had been closely associated with much of Professor L. Hill's recent work, described the action of ultra-violet radiation on chronic fibroid phthisis and tuberculous adenitis. He was satisfied that one of its effects was to promote calcification in tuberculous lesions. The reaction of the skin and other tissues differed in adults and children, and he considered that in the latter exposures should be very brief.

The PRESIDENT (Dr. Eustace Hill) said that Professor Hill's paper had revealed what would have seemed not long ago like a scientific fairyland. The great benefits that had already accrued from the application of light, both natural and artificial, were due mainly to the labours of Professor Hill and his colleagues in research. He considered that the Section was to be congratulated in having had the opportunity of listening to an account of his own work from so eminent an authority.

Professor HILL, in replying briefly to the discussion, said he regretted that Dr. Eidinow had not referred to the observations he had recently made on the increase of the bactericidal power of the blood as a result of ultra-violet irradiations. This was very marked in certain experimental animals. In human blood the bactericidal power was already high, but it also was increased by irradiation.

During the week the medical officer of health for Bath, Dr. J. F. BLACKETT, kindly provided opportunities for members of the Section to visit the various child welfare centres in the city, the school medical department, the municipal fever hospitals, waterworks, sewage disposal works, and the meteorological department.

THE EXTENSOR PLANTAR RESPONSE (BABINSKI'S SIGN) IN THE PRESENCE OF COMA.

BY

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THE extensor movement of the great toe in response to plantar stimulation is frequently accepted, and rightly accepted, as a proof of the existence of organic change in the pyramidal tract. There are, however, exceptions to this conclusion, and the recognition of these exceptions has considerable diagnostic importance.

In 1911, in a communication¹ made to the Association of Physicians, I suggested (1) that a bilateral extensor response present in a comatose patient does not necessarily mean an organic lesion, and (2) that coma, however produced, may be sufficient to establish in the nervous system the conditions (whatever these may be) upon which the extensor response potentially depends; and in support of these propositions I quoted certain experiences recorded by various authorities, together with some clinical observations of my own. Since 1911 observations on the plantar reflexes have been multiplied, and among these are cases of coma consequent on various narcotic poisons, not associated with evidences of organic disease but accompanied by an extensor response. For the most part writers have been disposed to suggest that the extensor response in such circumstances is to be explained by a direct and specific action of the toxic agent on the appropriate portion of the central nervous system. In each instance, that is, the coma and the extensor response are placed as separate and independent consequences of the poison—the coma from the action of the poison on the cerebral cortex and the extensor response from some other action of the poison on the part of the nervous apparatus related to the mechanism of the plantar reflex. As, however, the agents or poisons so accused are numerous, and as in some of their properties they differ widely from one another, this hypothesis of direct or specific action may reasonably excite some degree of doubt. In any event, the facts leave room for an alternative suggestion—namely, that the agents concerned produce an extensor response because they produce coma; that is, that coma is the common agency through which they reach the common end. It will be generally accepted that the circumstances in which the extensor response appears include influences that disturb the functional relations normally existing between the cerebral cortex on the one hand and the motor cells of the spinal cord on the other. The

suggestion here made is that coma may be one of these disturbing influences, and that therefore coma *per se* may be sufficient to explain the presence of an extensor response.

It is certain that an extensor response may result from an abnormal functional state existing in the cerebral cortex and unattended by any evidence of organic change either in the cortex itself or in any other part of the nervous system. Such a possibility is illustrated in the occurrence of Babinski's sign as a temporary event after an epileptic or other convulsive seizure. Recently I have seen a young man the victim of severe and periodic attacks of hemiparesis associated with homonymous hemianopsia and an extensor response, the visual fields and toe movements becoming normal when the headache ceased. Such an experience may be quoted as a convincing proof of the ability of a pathological state limited to functional disturbance of the cerebral cortex to explain the appearance of an extensor response. But if the functional disturbance of the cortex announced by convulsions, or, as just described, by homonymous hemianopsia, may cause an extensor toe movement, it is surely not improbable that the cortical condition announced by coma may equally produce this result. And the position is carried a stage further when it is remembered that the extensor response may sometimes be elicited during sleep—for the most part in young children, but in occasional instances quite definitely in an individual who has reached maturity. Of particular significance in this respect are some observations made by Kleitman (quoted in a recent paper² by Professor T. R. Elliott and Dr. F. M. R. Walshe) to the effect that an extensor response was present in healthy young men during the deep sleep that followed deprivation of sleep for a period of 40 to 110 hours. Between the effect exercised on the rest of the nervous system by the cortical condition existing in deep sleep, and that associated with coma, it is difficult to believe there is any essential difference, and if one is an efficient cause of the extensor response why not the other?

When coma and an extensor response follow the administration of a drug or poison it is of course possible to regard both the one and the other as equally independent and specific results of the drug action. Yet the very number and variety of the agents which have to be included in this group (morphine, hyosine, sulphonal, veronal, coal gas, trinitrotoluene, chloroform in the delayed form of poisoning) render not unlikely the suggestion that the extensor response which may follow their administration is due to some influence that they share and exercise in common. Coma is such an influence, and as the extensor response is here, in view of recorded recoveries, certainly not due to organic change, it is, in the circumstances, not unreasonable to claim that each of the drug agents

above named produces an extensor response because it establishes a condition of coma, and that in this fashion the normal relations between cerebral cortex and spinal centres are prejudiced.

The position is somewhat less confident when coma and an extensor response appear in the course of a general toxæmia; indeed, short of a complete and microscopic examination of the nervous apparatus, the exclusion of the possibility of organic change can in such circumstances hardly be an unqualified one. Even here, however, there are experiences which suggest that coma may play an important part in determining the extensor response. In this relation may be briefly stated the case (acute atrophy of the liver) of a woman, aged 37, who, as later information showed, had recently been treated by intravenous injections at a hospital clinic. When first seen she was jaundiced and unconscious, and ankle clonus and an extensor response were obtained in each lower limb. After three days the coma disappeared and each plantar response became flexor. It is at least within the limits of the possible that the coma was here the agency responsible for the extensor response. In the paper² already quoted Professor T. R. Elliott and Dr. F. M. R. Walshe record four cases of hepatic atrophy, each patient showing a double extensor response, and they argue that this response in cases of hepatic disease means the onset of "cholaemia." It is noteworthy that in each instance the extensor response appeared only when the patient's consciousness was more or less seriously compromised. The descriptions run (1) "semi-comatose . . . double extensor response"; (2) "plantars flexor . . . next day mild coma . . . and double extensor response"; (3) "plantars flexor . . . next day lethargic with double extensor response"; (4) "plantars flexor . . . two days before death coma . . . and an extensor response." Similarly Sir William Willcox³ in describing cases of icterus gravis writes: (1) "comatose . . . extensor plantar response"; (2) "comatose in morning . . . extensor plantar reflex 10 p.m."; (3) "unconscious in evening . . . ankle clonus and extensor plantar reflexes present." These experiences obviously are not inconsistent with the suggestion that coma due to hepatic disease, even as coma due to other causes, may determine an extensor response, and this proposal is strengthened by the record above quoted where on disappearance of the coma the extensor response also disappeared and was succeeded by the ordinary flexor movement.

Another toxic state in which unconsciousness may be associated with the extensor response is uraemia. But the association can hardly be pressed into the present argument, for uraemia is often manifested by convulsive seizures, and these, even as in epilepsy, may explain the extensor movement. Moreover, uraemia may be accompanied by organic changes in the central nervous system, including the pyramidal tracts, so that here also a possible explanation of the extensor response other than coma presents itself.

So far as changes in the pyramidal tract are concerned the toxæmia known as diabetic coma may perhaps stand in much the same position as uraemia. Personally I have recorded a case of diabetes,⁴ the patient a child of 3 years, with bilateral extensor response not associated with coma, and the explanation would appear to be either pyramidal degeneration or the re-establishment under some unknown influence of the ordinary infantile extensor movement. In the adult I am able to mention two examples of diabetic coma each with a double extensor response. One, a woman aged 56, brought into hospital in a state of unconsciousness and found to yield a bilateral extensor response; later, glycosuria was discovered, and a history of treatment for diabetes during some five years. At the outset the case was regarded as one of cerebral hæmorrhage, but *post-mortem* examination showed the brain and other organs to be free from obvious disease. Whether there were or were not microscopic changes in the spinal cord must remain an open question, but the record plainly announces that diabetic coma must have a place among the clinical possibilities marked by coma and a double extensor response; and it admits the suggestion that such a response may owe its existence to the presence of coma. For the notes on a second case of this order I am indebted to Dr. P. P. Dalton.

The patient, a woman aged 36, had been under treatment for diabetes for some months, when, somewhat suddenly, she became very excited and, later, comatose; there were evidences of acidosis and a bilateral extensor response.

In opposition to all that has here been written it may be said, and said truly, that in many cases of coma the plantar response is not of the extensor type. But this negative admission by no means destroys the positive evidence. The exact mechanism of the extensor response may be outside a confident definition, but it is not difficult to believe that to set it in readiness some delicate degree of adjustment or of maladjustment is required. In sleep, for example, the issue is plainly a matter of degree or measure; not sleep, but a certain depth of sleep, is essential to the extensor response. Such a consideration must apply equally to coma, and indeed with greater force, for coma varies not only in its degree but also in its associates, and some of these include agencies that in the highest degree of probability affect widely and in various fashions the several parts of the nervous apparatus, including that (whatever it may be) on which the extensor response directly depends. Hence it is possible to picture a degree or phase of coma which allows a flexor response, or one which decides an extensor movement, or a third, in which the plantar response is altogether abolished. The unquestioned experience that in many cases of coma, in the large majority indeed, the extensor response is not obtained, does not destroy the claim that, given the appropriate conditions, coma may determine this response. Granted a suspension of the cortical functions in the form of a particular degree or depth of sleep and the demonstration is simple, but amidst the conflicts and complications so frequent in clinical cases an experience far from uniform must be judged inevitable. Further, while the extensor response is not always present in coma, neither is it always present in pyramidal disease. Yet no one on this account will deny pyramidal disease to be potentially a cause of the extensor response, and if negative experiences do not exclude the one claim neither can they be quoted as conclusive against the other. The positive evidence broadly presented shows that coma arising in many and varied circumstances and apart from organic disease is, as a matter of confirmed clinical observation, not infrequently associated with an extensor response, and it is on this generalization that the thesis presented in this paper mainly rests.

A few further notes may be permitted. First, while recognizing that an extensor response may occur apart from pyramidal disease, it is well to remember that this response may be the sole objective evidence of such disease, perhaps in an early stage, perhaps for the time being latent. In a series⁴ of cases collected to illustrate this proposition I suggested that an investigation of the plantar responses was a necessary part of any satisfactory scheme of clinical examination. Professor Elliott and Dr. Walshe² think that the "general physician" is in this respect less thorough than the "neurologist," but I hope that few of us come under this reproach. I agree, however, that in so far as we do so we run the risk of serious error.

A second practical point is that when, in testing the plantar reflex, there is so much movement of the foot and toes that it is difficult to be sure whether there is or is not an extensor response, help may be obtained by applying the stimulus along the groove below the external malleolus instead of along the sole. By this method the toe movement may sometimes be obtained free from the confusion which is apt to follow stimulation of the sole in a highly sensitive and explosive patient. It has further been claimed⁵ that an extensor response following a stimulus applied below the external malleolus may detect a pyramidal lesion which might escape the test applied in the more usual fashion. Whether this is so or not the method is certainly a useful one, and particularly in the presence of the difficulties and ambiguities just suggested.

As a third memorandum it may be added that an extensor response may be produced voluntarily and for purposes of deception. Some few years ago I was asked to see a man who, after an alleged accident in a railway station, was said to be hemiplegic, incontinent, and unconscious. No signs of injury to the head could be detected,

but the man appeared to be only partly conscious and to be paralysed in his right limbs; and certainly his right pupil was dilated and fixed and his right plantar response was extensor. After some weeks he began to get about in a halting fashion, and in due course he advanced a claim on the railway company. For various reasons this claim was questioned, and an examination in the presence of several practitioners was arranged. At this examination the patient appeared to make two blunders. First, on request, he promptly protruded his tongue, and this with a pronounced deviation; but the deviation was not toward but away from the paralysed limbs. Secondly, he produced unfailingly a right extensor response, but he did this on the slightest provocation and almost without regard to the site of the stimulus—calf, shin, sole, dorsum, each alike received the characteristic reply. In the first examination the possibility of the man being a malingerer never occurred to me, and the majority of my colleagues at the later consultation concluded that an ingenious patient by the aid of atropine and of some carefully garnered medical knowledge had proved too much for me and for some others equally guileless. Assuming them to be right, I registered a mental note that among the possible causes of an extensor response a place must be found for the deliberate malingerer. Certainly the trick, if once known, is easily repeated, and the experience is worth adding to a list of the circumstances in which an extensor response does not mean organic disease.

REFERENCES.

¹ *Practitioner*, vol. xciii, p. 330. ² *Lancet*, January 10th, 1925; see also *Diseases of the Liver* (Sir Humphry Rolleston), second edition, p. 587. ³ *Medical Society's Transactions*, vol. xlii. ⁴ *Medical Press and Circular*, August 11th, 1915. ⁵ *Lancet*, December 16th, 1911.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

WEBBED HAND WITH ABNORMALITIES OF BONES.

A boy, aged 4, presented webbing of the left hand as to digits 2, 3, 4, and 5. The webs between 3 and 4 and 4 and 5 were complete up to the tip of the nails. That between 2 and 3 was less complete. The palm of the hand appeared long in comparison with the fingers, and only one row of joints distal to the metacarpo-phalangeal joints could be made out. X rays showed that one row of phalanges, presumably the middle, was absent. The ossification centres for the metacarpal of the thumb are abnormal. Usually the shaft and head of this bone are developed from a primary centre, while the base has a secondary centre, appearing about the third year. In this respect therefore, ordinarily, metacarpal 1 differs from metacarpals 2, 3, 4, and 5, and resembles the ossification of a phalanx, a circumstance which has given rise to discussion among anatomists as to whether the thumb consists of three phalanges and no metacarpal or one metacarpal and two phalanges. In the present case metacarpal 1 has a primary centre for the shaft and base, and a secondary for the head. Ossification centres are present for os magnum, unciform, cuneiform, semilunar, trapezium, and lower end of radius. As there was no bony fusion between the individual fingers, separation of the soft parts was performed by Didot's method, in two stages. Digits 4 and 5 were first separated, and afterwards 2, 3, and 4. The anatomical and functional results have been good.

Ruabon.

A. H. TURNER, M.B., B.S.Lond.

ACUTE INTESTINAL OBSTRUCTION DUE TO A GALL STONE: VOLVULUS OF SMALL INTESTINE ABOVE THE OBSTRUCTION.

Impaction of a gall stone is one of the rare causes of acute intestinal obstruction. As the grouping of the symptoms is frequently unusual, the exact diagnosis is often very difficult. It seems right, therefore, to report the following case, which occurred in the practice of the West London Hospital. I am indebted to Mr. Donald Armour for permission to do so.

A woman, aged 54, was admitted on May 6th, 1925, complaining of abdominal pain. The pain had come on suddenly three days previously, was colicky, and situated round the umbilicus and in the hypogastrium. Vomiting had commenced shortly after the onset of the pain and had been repeated several times daily. The vomit was at first bilious, but had become faeculent. Absolute constipation of faeces and flatus had existed from the onset. She had had occasional attacks, lasting one to two days, of slight epigastric pain, unaccompanied by vomiting, for the past seven years, but no symptoms between the attacks. The abdomen was only moderately distended. Coils of gut were visible through the thin abdominal wall, and there was shifting dullness in both flanks. There was no visible peristalsis, tenderness, or rigidity. No mass was felt on palpation of the abdomen or on rectal examination. Shock was absent, except for slight lowering of the temperature (97° F.) and raising of the pulse rate (100).

I opened the abdomen by a right paramedian incision, its centre opposite the umbilicus. Free serous peritoneal fluid immediately escaped. Distended and congested small intestine was traced to a point 9 inches from the ileo-caecal junction, where a gall stone was found loosely impacted in the gut. The bowel below was pale and collapsed. The loop of ileum immediately above the site of obstruction had undergone axial rotation in a clockwise direction, forming a volvulus with one complete twist, presumably from the irregular muscular movements set up in the intestine by the presence of the gall stone. Owing to the position of the incision examination of the gall-bladder region could not be satisfactorily performed. The wall of the gut at the site of impaction of the stone appeared undamaged and the stone was easily displaced upwards. After unwinding the volvulus this was done, and the stone removed by opening the bowel. The stone was ovoid, 1½ inches long, with a diameter of 1 inch, and tuberculated, except at its narrow pole, which was smooth and presumably had projected into the fistulous communication between the gall bladder and the small intestine, which doubtless existed before the stone escaped to bring about the obstruction. The abdominal wound was then closed with a drainage tube to the site of enterotomy.

The patient made an excellent recovery.

London, W.1.

N. ROSS SMITH, F.R.C.S.Eng.

ACUTE APPENDICITIS IN THE AGED.

THE following case seems worth placing on record because of the unusual features which were present.

At 10 a.m. on April 6th I was summoned to a frail old lady, aged 75, who, about twelve hours previously, had been seized with vomiting and abdominal pain which had gradually become more severe in the right iliac region.

When I saw her the physical signs were rigidity of the abdomen and pain and tenderness (this last very marked even on gentle palpation) over the appendix area. The temperature was 99.2°, and the pulse rate 58. Her normal pulse is 60, and her normal temperature 98°. There were no signs of any external hernial strangulation, and the very definite local signs made the diagnosis of acute appendicitis fairly certain.

Dr. Morland Smith saw the patient with me at midday, and agreed with the diagnosis and necessity for immediate laparotomy. This he performed in the early afternoon as soon as the patient had been removed to the Southbourne nursing home.

The appendix was found to be very distended and discoloured and tortuous, with commencing ulceration near its base. The usual operation was performed, the appendix removed, and, as investigation showed nothing abnormal elsewhere in the abdomen or in the pelvis, the wound was closed. Convalescence was uninterrupted and she left the nursing home quite well four weeks later.

The unusual features which make this case interesting were: (1) The pulse rate did not increase, and the rise of temperature was negligible. A marked increase in the pulse frequency is often insisted upon as an indication for operation. Possibly the slow pulse and low degree of fever may be explained by the way aged persons react to a toxæmia. (2) The advanced age of the patient; acute appendicitis is rare in patients over 70. When the local signs are definite, to wait for a marked rise of temperature, or an increase in the pulse rate, or to deem the age of the patient as beyond the probability of an acute appendicitis may lead to grave consequences; in this case delay would certainly have been serious. The happy result obtained was due to the speed of the operation (fortunately the absence of any complication assisted in this, because the patient's frail condition demanded quickness), and the careful nursing the patient received.

Bournemouth.

FREDERICK C. FORSTER.