THE SYPHILITIC FACTOR IN THE HEMI- PLEGIAS AND DIPLEGIAS OF INFANCY AND CHILDHOOD.*

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AMONG the children who attend the out-patient departments of hospitals there are none whose condition is more pitiable than those who suffer from the physical and often also the mental disabilities consequent upon hemiplegia or diplegia.

Our knowledge as to the actual causes of these types of paralysis is, unfortunately, much less precise than it is with regard to similar cerebral paralyzes occurring in the adult. Many efforts have been made by able observers in the fields of neurology, of general medicine, and also of surgery, to refer infantile cerebral palsies to their ultimate causes, and it is only by an accurate knowledge of the latter that anything like success in dealing with these conditions can be attained.

CLASSIFICATION.

At present the most useful classification of these early upper neurone paralyzes is that which divides them into three classes according to the supposed time of onset—that is, antenatal, natal, and post-natal. It is obvious that such a subdivision as this, depending as it does on no pathological basis, although at first sight satisfying, is really far from final. The one useful feature about it is that it clearly defines the position of the cerebral palsies due to natal accidents—that is to say, to haemorrhages. In order to clear the ground, I will state briefly the characters of this class, and then, since syphilis has no part in its production, refer to it no more.

Natal Paralysis.

In birth palsies due to haemorrhage the lesions are more often on the surface of the brain than in its substance. As pointed out by Harvey Cushing, the veins which ascend from the middle part of the convexity of the hemisphere to the superior longitudinal sinus are the vessels most liable to rupture. So long as they follow the cerebral sulci these veins are protected, but at the vertex where they cross the subdural space to enter the sinus or its para-sinoidal recesses, they are unsupported. Undue overlap from cranial moulding may tear one or more of the veins away from the sinus walls. As a result of the laceration, free intradural haemorrhage occurs, the bulk of the clot being near the vertex, and on one or other side of the falx cerebri. Consequently the damage to the motor centres for the legs is more extensive than that to the motor centres of the arms.

The clinical characters of the natal paralysis are as follows: Firstborn children more often suffer than the subsequent offspring, and male children, having larger heads, are more likely to be injured than females. The children of elderly primiparae run especial risks.

The occurrence of *post-partum* asphyxia is common. The fontanelles bulge, and may be so tense that they cease to pulsate. Convulsions are frequent, but may not appear until several days after birth. The fits are often unilateral. Vomiting is sometimes a marked feature.

There is great exaggeration of the deep reflexes, and may be, even at this stage, evidence of paralysis of the limbs. Ocular palsies are common, especially a difference in the size of the pupils. In severe cases signs of pressure on the medulla may occur in the form of alterations in the respirations and disturbance of the cardiac rhythm. Epilepsy results in two-thirds of the cases, the convulsions often being Jacksonian in character.

Immediate craniotomy, the whole parietal bone being raised as an osteoplastic flap, for removal of the clot, has given excellent results. Little or no good can be expected from cranial operations performed months or years after the incidence of the lesion.

SYPHILIS AND UPPER NEURONE PARALYSES.

Having eliminated the natal or traumatic paralyzes, we may now proceed to the proper subject of this paper,

which is the consideration of the relation of syphilis to the upper neurone paralyzes of infancy and childhood.

The knowledge that syphilis is such a potent factor in the production of paralyzes in the adult has led to much critical inquiry into the part it may play in producing the paralyzes of the early years of life.

The late Dr. Hadden conducted one of the most interesting investigations of this nature amongst his out-patients at Great Ormond Street. He embodied his results in a paper entitled *Infantile Hemiplegia with Acute Onset*.¹ To his conclusions I shall refer later.

The general consensus of opinion is that syphilis is *not* a common factor in the upper neurone paralysis of childhood, and this opinion appears to be based on very good grounds. Nevertheless, a reconsideration of the part syphilis plays may be instructive and appears to be desirable for two reasons: First, because, in some cases at least, a permanent and crippling disability of both body and mind may be prevented by an intelligent anticipation of events; and, secondly, because, thanks to the methods of diagnosis perfected of late years, it may yet turn out that syphilis is a more important factor in the production of these paralyzes than many have suspected.

Characters of the Lesions Found.

We are not concerned with cases of juvenile general paralysis and cases of juvenile general paralysis with tabes. These are not at all common; but we have it on the authority of Dr. Mott, based on his experience in the London county asylums, that, even in comparison with degenerative syphilitic lesions, cases of infantile syphilitic hemiplegia and diplegia are rare. Many other writers who have had occasion to search for the records of such cases agree as to their fewness. A plausible suggestion is that their scarcity may be explained, not by immunity of the cerebro-spinal structures in infants and children, but by the death of those severely infected with syphilis, either before birth, or at all events shortly afterwards, from such causes as convulsions, meningitis, or hydrocephalus.

Accounts have been published by Sir Thomas Barlow, Judson Bury, Angel Money, and George Carpenter, amongst others, which permit the formation of a definite conception of the morbid anatomy of the disease, and warrant the general conclusion that the changes likely to be found in the brains of paralysed syphilitic children bear a close resemblance to those which may occur in the syphilitic adult. The multiplicity and combination of the lesions are striking characteristics—that is to say, leptomeningitis, pachymeningitis, endarteritis, and even gummata, are associated in varying degrees.

The cases recorded by Sir Thomas Barlow are as follows:

1. A full-term child suffered shortly after birth from snuffles, thrush, and persistent soreness of the nates. The succeeding child was healthy, but there had been one miscarriage at ten weeks. The patient was emaciated, suffered from diarrhoea, and had small shotty glands in the neck. At the age of 15 months there was general weakness of both sides of the face, with frequent laryngeal spasm, and inconstant, rather coarse, nystagmus. No antisyphilitic treatment was attempted, the case being regarded as one of tuberculosis. The child soon died. A gummatus infiltration of the cranial nerve trunks was found; it involved the fourth, fifth, sixth, seventh, and eighth pairs. The basilar artery and all the component vessels of the circle of Willis presented opaque white thickenings which narrowed their lumina. By use of the microscope typical syphilitic endarteritis was revealed.

2. A girl baby, who died at 10 months, but was first seen four weeks after birth. The father had suffered from syphilis, and the child had snuffles. Between the ages of 1 and 4 months she had eleven fits. She was wasted, and serpiginous ulcers appeared on the nates. Treatment with mercurial inunction and cod-liver oil was instituted. At eight months she was worse again with tetany and laryngismus. Early disseminated choroiditis was discovered in each eye. At 10 months a *post-mortem* examination was obtained. The surface of the pia mater was thickened in many places, and a small patch of calcification lay on the convex aspect of the left parietal lobe. The basal arteries were normal, but some of their cortical branches were extensively diseased. There were a few spots of superficial cortical softening. Endarteritis was proved by microscopical examination.

The following, a very striking case, was recorded by Dr. Henry Ashby.

3. A male infant was seen at 3 months of age, suffering from coryza and a well-marked rash. A month later multiple syphilitic epiphysitis occurred. When 7 months old he began

* A paper read at St. Thomas's Hospital before the Lambeth Division of the British Medical Association.

to suffer from convulsions. At first these were mostly confined to the left side, but later they became general. In the course of a few months the left arm and leg, which were more or less paralysed, began to draw up and become rigid. Later the right arm became affected in a similar manner. The child gradually became idiotic and finally died. It was under mercurial treatment from 3 months of age. The arachnoid was of a milky colour and there was an excess of subarachnoid fluid. The superficial layer of the grey matter on the convex surface of both hemispheres, especially the right, was softened and could readily be scraped away. The superficial layers of the caudate nucleus and of the optic thalamus were in the same condition of softening. Microscopically the grey matter showed widespread fatty degeneration; the minute arteries were extensively blocked with old thrombi, their inner coats being thickened and their nuclei increased in numbers. The large arteries were normal as far as could be made out. There seemed to have been extensive syphilitic arteritis of the meningo-encephalic arterioles leading to thrombosis and secondary softening of the grey matter.

4. In an infant of 15 months Chiari records the discovery of chronic meningitis, endarteritis, and small foci of cerebral softening.

5. Multiple grey gummata, the size of cherries, were found by Hensch in a child aged 2 years. These lay under the pia mater and also in different parts of the cerebrum and cerebellum.

Can Syphilis Produce Diffuse Cerebral Sclerosis?

It is necessary to inquire whether there is any evidence that syphilis can produce diffuse cerebral sclerosis, since it is a matter of common knowledge that more or less induration is often found in old cases of hemiplegia or diplegia of childhood.

The available evidence goes to show that the inference that this sclerosis is always due to syphilis is quite unwarranted. Those who have investigated the condition have been unable to agree that syphilis plays more than an unimportant part in its production; in other words, the sclerotic condition must, as a general rule, be attributed to other and at present obscure factors.

On the other hand, if we assert that syphilitic infection never produces diffuse sclerosis, we shall be going too far, for there is evidence that such sclerosis may result from syphilis. The induration must be induced either by a diffuse specific encephalitis or be dependent upon diffuse endarteritis of the minute cortical arterioles. In some instances the sclerosis is associated with diffuse leptomeningitis and pachymeningitis, leading to adhesion of these membranes.

Dr. Angel Money recorded the following case:

6. A child was found at 11 months to be suffering from hydrocephalus and a disseminate ocular choroiditis. The father admitted syphilitic infection and presented an extensive abdominal syphilide. Under antisyphilitic treatment the fontanelles duly closed. Sight began to fail at 3½ years. Within six weeks a left hemiplegia with spasticity developed; this was followed, after an interval, by right hemiplegia and loss of speech. The boy died shortly afterwards. The dura mater was adherent and chronically inflamed. The leptomeninges were greatly thickened. Dilatation of the cerebral ventricles and a granular appearance of the ependyma testified to the old hydrocephalus. The right cerebral hemisphere, which was the site of the first paralyzing lesion, was shrunken and indurated. Its arteries were grossly diseased. The left hemisphere was hyperaemic and some blood was extravasated on its surface.

Another case was recorded by the late Dr. George Carpenter:

7. The child concerned was the second of the family, the first having been born dead. Snuffles occurred at 1 month, and were soon followed by convulsions, which started in the left arm. At 2½ months the patient was supposed to be blind. When she was examined at 5½ months the head was retracted, the arms rigidly extended, and the knees flexed. A macular syphilide was present, and disseminate choroiditis was detected in the fundi of the eyes. The child was then suffering from bronchopneumonia, and death soon ensued. Sclerosis was found in both cerebral hemispheres, involving the central and frontal convolutions as well as part of the right temporo-sphenoidal lobe. The induration implicated the white as well as the grey matter, and the affected areas were in parts cystic. The cysts contained opalescent fluid, but presented no indications of past haemorrhage, either in the presence of blood or of blood crystals. The arteries at the base of the brain were healthy.

The following instructive case was published by Dr. Abercrombie:

8. At the age of 6, a girl suddenly fell down with a right hemiplegia. She came under observation four years later. No conclusive evidence of syphilis could be obtained, but, the child dying, a post-mortem examination was performed. The middle cerebral arteries were diseased. The left cerebral hemisphere

was atrophied and its convolutions sclerosed. The ascending frontal and parietal convolutions were most affected. The meninges were thickened and adherent to the brain, more so on the left than on the right. A small adherent thrombus of old standing was found in the superior longitudinal sinus.

The brain is not the only part of the central nervous system which may be damaged by the infection of congenital syphilis. The spinal cord is not immune from the disease. In addition to the system degeneration of juvenile tabes, focal lesions may occur. Specific endarteritis of the spinal arterioles may lead to localized sclerosis or the spinal cord may be compressed by gummata of the meninges or of the periosteum of the spinal canal.

The following case was recorded by Dr. H. D. Singer:

9. A boy of 12 years, who was the subject of congenital syphilis, rapidly developed a paraplegia, the paralysis reaching its acme in two or three days. He died about seven weeks later. The post-mortem examination revealed the presence of gummatous meningitis, but, in addition to this, a limited transverse lesion was found in the sixth dorsal segment of the spinal cord. The vessels of the damaged area showed typical syphilitic endarteritis with small-celled infiltration of their perivascular sheaths. The larger vessels, however, were not completely occluded, although some of the capillaries appeared to be. In place of softened patches there was an overgrowth of the perivascular connective tissue; this was especially evident in the posterior columns close to the commissure, the posterior median vessels being surrounded by a greatly thickened coat. The vessels in other regions of the cord were practically healthy. In his remarks on the condition Dr. Singer draws attention to the fact that the cord was sclerosed and not softened. He points out that in cerebral thrombosis the result in the adult is as a rule brain softening, but the effect produced in infants and children is very often sclerosis.

In addition to the lesions already mentioned, an arrest of development of the brain and ganglion cells has been ascribed to the action of the virus of syphilis. On this point I am not in a position to offer any evidence.

CLINICAL ASPECTS OF THE DISEASE.

For the purpose of inquiry into the clinical aspects assumed by the syphilitic varieties of upper neurone paralysis it will be convenient to consider first the general characters of the antenatal and the post-natal forms.

The Antenatal Form.

Antenatal paralyses, whatever their causation, possess as a group the following characteristics:

The palsy is bilateral and spastic. It is frequently accompanied by athetosis.

Mental weakness and a tendency to convulsive seizures often accompany the disease. Although present at birth the paralysis may escape detection for a few weeks, sometimes even until the period when the child should normally walk and talk.

In addition to spasticity of the limbs, which as a rule is best marked in the legs, there may be some difficulty in swallowing, and squint or nystagmus may be present. The early presence of symptoms such as these may be of material assistance in tracing the lesion right back to the earliest days of life.

The family history of these cases, too, has important characteristics. Contrary to what occurs in the haemorrhagic form of birth palsy, the youngest children of large families are most prone to suffer. When one child of a family is born diplegic the mortality among the other children is considerable, and miscarriages are common.

The birth of a child suffering from antenatal diplegia is apt to be premature, and the labour may be precipitate. If the labour in such a case is difficult the delay is rarely due to mechanical obstruction, but is caused by uterine inertia, the consequence of some abnormal state of the mother. Even where labour is precipitate there may be an asphyxial condition of the child, and considerable difficulty in establishing respiration.

Antenatal diplegia is usually, and no doubt properly, attributed to some condition of ill-health of the pregnant mother. The actual nature of the maternal disease is obscure. Various acute diseases which may have occurred during the period of fetal development, and among these certain of the acute specific fevers, have been looked upon as possible causes. Intrauterine injury may also be a factor.

The possibility that syphilitic infection may be concerned has not been overlooked. In fact, according to Risien Russell syphilis is by far the most important and mos-

common maternal disease standing as a causal antecedent. Oppenheim expresses a similar opinion, and quotes several other observers agreeing with it.

This view is borne out by the report that, in France, Delbet obtained a positive Wassermann reaction in 13 cases of this disease. On the other hand, H. R. Dean examined, among other cases, at Potsdam, 15 patients with congenital spastic diplegia, and only obtained one positive result. But on inquiry he informed me that the histories were insufficient for the purpose of determining how many of these paralysees were antenatal and how many due to traumatism at birth.

The following two cases of my own are examples of antenatal diplegia in which syphilis appeared to be the cause:

10. K. A., a female child of 3 years, was brought to the hospital by her mother. There had been fourteen pregnancies. Four children were living, the mother had five times miscarried, and five children had died. A perforation, due to the rapid development of a large gumma, had partly destroyed the mother's hard palate. The child was premature, being born at seven months. The labour was easy. For two years she had snuffles, but no rash was noticed. Although three years old she had never learnt to walk, and had only spoken during the last twelve months. Lately she had begun to use her hands for grasping purposes. She was mentally deficient and diplegic. The legs were quite stiff, the knee-jerks exaggerated, and an extensor plantar response present on each side. The pupils were active, and there was no optic neuritis. The sight of the right eye appeared to be defective. Constipation was a great trouble. The child was treated with grey powder and attended for fourteen weeks, after which she was lost sight of. After several years had elapsed the mother came to the hospital with another infant and, in response to inquiries, said that she had been persuaded to take the child to another hospital. A series of convulsions had occurred, and the child died.

11. R. A., a male aged 3½ years, was sent up from the country for advice as to his condition. He was the firstborn and the birth was natural. There was a history of thrush in infancy, with soreness of the nates and eczema in the groins, armpits, and behind the ears. He was a backward child with spastic diplegia, most marked in the legs, but also present in the arms. The deep reflexes were exaggerated and ankle clonus was present. His head was small, the forehead being narrow; he possessed very little power of speech and was dirty in his habits. In each eye there was evidence of old iritis, which the ophthalmic surgeon who examined him considered very suggestive of syphilis. No gross changes were discovered in the fundi.

In 4 other cases of diplegia which were judged to be examples of antenatal paralysis the cerebro-spinal fluid was examined for me by Dr L. S. Dudgeon: the Wassermann reaction was negative, and the fluid free from globulin; no lymphocytes could be detected in it. In two of these patients the blood was also examined for the Wassermann reaction with negative result.

The Post-Natal Form.

The post-natal form of infantile hemiplegia or diplegia is, as a rule, acquired early in life. The first three years are those in which the liability to this type of paralysis is greatest, but the onset has been known to occur later.

Briefly summarized, the clinical characteristics of this group are as follows:

With very few exceptions the onset is sudden and definite, not gradual; this is so constant that it is important to bear in mind that cases of infantile cerebral paralysis in which there is no history of definite onset are likely to be of antenatal origin.

The paralysis is hemiplegic or, much less commonly, diplegic in distribution. It may at first be quite flaccid in type, but soon becomes spastic. Hemianaesthesia, if present, is transient. It is sometimes possible to demonstrate astereognosis in older children. The tendon reflexes are exaggerated on the paralysed side. Often the opposite knee-jerk is unduly brisk at the same time. Ankle clonus is rare, but not unknown; an extensor plantar reflex is the rule. The power of speech may be lost. This occurs, according to Hadden's computation, in about one-fourth of the cases. The loss, as might be expected, is more often associated with right hemiplegia than with left, but this is not invariable. Articulation usually reappears in a few weeks or months. It is remarkable that these patients never show true aphasia in after-life.

There is but little permanent paralysis of the face. The laugh or cry may begin later on the paralysed side, and this side of the mouth open less widely.

The tongue will rarely be found to deviate towards the hemiplegic side in cases of some standing, although it may do so in the earliest stages of the disease.

Convergent squint may occur.

In old cases the arm is found to be affected more than the leg, and the shoulder makes better recovery than the hand. As a sequel to the paralysis, flexor-adductor contracture of the arm and extensor-adductor spasm of the legs are common, but spasticity is not invariable.

Post-hemiplegic perverse movements occur. These mainly affect the upper extremity, and either are deliberate, of the nature of athetosis, or more abrupt, when they are known as post-hemiplegic chorea. The paralysis rarely improves in limbs affected by these movements. The growth of the paralysed side of the body is retarded, and any degree of mental defect may accompany the motor palsy. Nearly all the patients show some mental weakness by the time puberty is reached. Epilepsy supervenes sooner or later in at least one-third of the cases.

The causal factors of this group are diverse. Cerebral haemorrhage and cerebral embolism undoubtedly account for a few cases. The idea is steadily gaining ground that an acute encephalitis (owning as its excitant the same infection as that which causes acute anterior poliomyelitis) is responsible for many more. Cortical venous thrombosis also has its advocates.

But, as with the former group, it is profitable to inquire how far syphilitic brain lesions lie at the bottom of the condition. By most clinical observers the syphilitic variety of the disease is held to be rare. This was the opinion of Hensch, Sir Jonathan Hutchinson, Marie, and Fournier. Professor Osler found but 1 case in 120. Abercrombie described 4, or perhaps 6, in 50 cases. Isolated instances have now and again been reported by other writers. Hadden, in 1891, suggested, on clinical grounds, that the syphilitic variety was more common than was at that time believed. In a series of 40 odd cases, he quotes 2 which were undoubtedly syphilitic, and 12 others in which there was a suspicion of the taint. Of 5 cases of my own, examined by Dr. Dudgeon, only 1 gave a positive reaction. In this case (quoted later) the Wassermann reaction was obtained from the cerebro-spinal fluid, which also contained much globulin and an excess of lymphocytes.

For the recognition of the syphilitic cases which occur in the group of post-natal paralysees attention should in the first place be directed to the family history and previous history of the patient. Next the mode of onset of the paralysis should be investigated carefully, and, in the last place recourse should be had to the various clinical tests of the blood and cerebro-spinal fluid which are now available. It may be possible, as in some of the cases I have already narrated, to recognize in the parents lesions which are unmistakably syphilitic. Too much weight should not be attached to the occurrence of a miscarriage.

In the infant snuffling, soreness of the nates, and unexplained wasting may give rise to suspicion as to the nature of the infection, whilst the detection of syphilitic epiphysitis, iritis, disseminated choroiditis, or interstitial keratitis is of the greatest diagnostic value.

Sometimes a peculiar mode of onset of the paralysis gives the clue to the nature of the case. These suggestive onsets are of several types: One is the development of a hemiplegia which is preceded for some time by premonitory and recurrent convulsions. The fact that convulsions are frequent in syphilitic infants and may be succeeded after a longer or shorter time by hemiplegia was pointed out by Sir Thomas Barlow and Dr. Bury. The primary convulsions are epileptiform in nature and may appear simultaneously with or shortly after the development of rash and snuffles. The succeeding hemiplegic attack may occur after an interval of months or even of years. This mode of onset is well illustrated by the following case which was recorded by Hadden:

12. A female child was seen at the age of 9 years. The soft palate was ulcerated and cicatricial; nasal discharge was present and also caries of the nasal bones. A miscarriage had preceded her birth but there was no history of snuffles or rash. She was treated with grey powder. Shortly after the first examination she had several convulsive fits. These occurred at intervals but left no paralysis. Iritis and keratitis developed in the left eye. After further interval of six months she began to complain of headache and suddenly became hemiplegic on the left side. The hemiplegia was spastic with exaggeration of the reflexes. There was no affection of sensation. Memory and intellect slowly failed.

A second characteristic development of symptoms, and one which, according to Rotch, is extremely characteristic of intracranial syphilis, consists of *separate attacks of paralysis which succeed each other at intervals of days or months*, and are not associated with any such obvious cause as a slowly-growing tumour or recurrent embolisms or intracranial tuberculosis.

13. A child of 18 months was brought to my out-patient department with a flaccid paralysis of the right arm. The paralysis had been discovered accidentally, and the arm was blue and cold. The patient had been treated with mercurials for sore bottom, and her birth was preceded by that of a premature infant. The true nature of the paralysis was overlooked, the condition being attributed to acute anterior poliomyelitis. Steady improvement occurred with simple massage and cod-liver oil. Eleven months later she had a series of six fits in the out-patient room. These fits were stronger on the right side, the eyes deviating also in this direction. The true nature of the case was at once suggested by the second attack, and energetic treatment by mercurial inunction and grey powder was instituted. Notwithstanding this a spastic right hemiplegia rapidly developed. The child became unable to walk, and only acquired a vocabulary of a few words. She is now left-handed. Both legs and the right arm are spastic. The knee-jerks are both exaggerated, and an extensor plantar response can be obtained on each side. Well-marked choroiditis has been found. The blood gives a positive Wassermann reaction, as also does the cerebro-spinal fluid. In addition, the latter contains much globulin and an excess of lymphocytes.

Yet a third method of onset is that in which *prodromal headaches and vomiting* occur, just as they do in many cases of syphilitic cerebral thrombosis of the adult. Even here the actual installation of the paralysis is sudden and definite. (An onset of this character may also occur with tuberculous meningitis.)

I should also be inclined to suspect cases in which the *silent development of acute hemiplegia* is the first sign, although fits may occur later.

Hadden recorded the following case:

14. A boy suffered with thrush and snuffles in infancy, but had no rash. There was no history of miscarriages in the mother. At the age of 1 year and 9 months, with no premonitory or accompanying convulsion, a complete right-sided hemiplegia suddenly declared itself. Convulsions followed the paralysis for one or two days. Improvement was gradual. The boy became left-handed, and speech was delayed until he was 2 years old. The fits did not recur. When seen at the age of 10 he was suffering from interstitial keratitis, and showed extensive choroidal atrophy.

But the family and previous history, and also the mode of onset, will often leave us in doubt as to the cause of the paralysis. The hemiplegia in syphilitic cases may arise out of a first convulsive seizure with or without coma, just as occurs in other forms of the post-natal group. Under such circumstances as this the final, and indeed the only means of arriving at an accurate diagnosis is by examination of the blood and of the cerebro-spinal fluid—the blood for the Wassermann reaction, and the cerebro-spinal fluid not only for that reaction, but also for the possible presence of globulin and an excess of lymphocytes.

REFERENCES.

¹ St. Thomas's Hospital Reports, 1891. ² Brain, 1902.

THE "X" RAYS IN TREATMENT.*

BY

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WITHIN the last few years the question of "dosage" in the administration of x rays for therapeutic purposes has received much consideration. In the appendix to the late Dr. Kassabian's excellent work on electro-therapeutics, published as recently as 1907, the technique of many eminent radiologists is described. In the majority of the descriptions the details give no indications as to the quality of the rays produced or the quantity absorbed by the patient; indeed, the dosage is largely a matter of guesswork based on experience. The following quotation illustrates this point:

¹ Dosage is determined by a light or dark green fluorescence of the tube. The best time to discontinue treatment is at the appearance of any erythema, taking into consideration the number and duration of exposures.

* Read before the Glasgow Medico-Chirurgical Society, March 3rd, 1911.

Now it is true that a certain skill in this guesswork may be gained in time. But I contend that a course of minute doses can be administered in this manner only by one of great experience; even then it is risky; it is unscientific, and happily it is quite unnecessary. To administer the rays in definite measured doses is more troublesome and more expensive; but, when one realizes the extreme potency of the agent and the havoc its injudicious use has already wrought, one must welcome these means of measurement, and use them regardless of either expense or trouble. Already many valuable lives of both medical practitioners and patients have been lost through exposure to the x rays; and Dr. Kassabian himself, one of the latest victims, says in his book (*ibid.*, page 407): "The number of operators who have been disfigured, maimed, and injured would form a deplorable list of tremendous proportions."

I do not think that it can be too much insisted on that the administration of the x rays, except in carefully measured doses, is absolutely inexcusable.

It is not my intention to discuss minutely the question of "dosage." The problem is very complicated and difficult, and I admit that no one of the methods of measurement at present in use is completely satisfactory. I intend rather to give in detail the application of measurement I have found most simple in the daily routine. Regarding the rays themselves, there are two factors to be considered: (1) The quality of those falling on the part treated, and (2) the quantity absorbed by the tissues. Any system of measurement must necessarily take cognizance of both.

"Hard" and "Soft" Rays.

A beam of x rays is composed of radiations of different penetrating powers, just as a beam of ordinary light is composed of rays of different wave length. Rays are described according to their powers of penetration; those of low penetrating power are called "soft," those of high penetrating power are called "hard," while an intermediate degree is spoken of as "medium." The proportion in which "soft," "medium," and "hard" rays are mixed depends on the degree of vacuum of the tube from which they emanate. When the vacuum is low the "soft" rays abound; when the vacuum is high the "hard" are proportionately increased. When it is desired to affect any deep-seated tissue a tube emitting "hard" rays in abundance is selected, while for superficial effects a tube of low vacuum, giving off chiefly soft rays, is used.

Our means of controlling the penetration are: (1) The regulation of the vacuum, and (2) the use of screens.

1. For the first we use a tube having a self-regulating vacuum (such as Müller's heavy anode automatic vacuum regulating tube). It is so set that the vacuum shall not rise above a certain degree, and as the vacuum tends to rise while a tube is in use (provided it is not over-heated) this device keeps it fairly constant.

2. Screens are used to absorb the softer rays—that is, those whose chief effects are on the skin—when the object is to affect deep structures. They may be made of various substances, such as wash-leather and gelatine. I use sheets of aluminium, 1 mm. in thickness, superimposed on one another, the number used depending on the effect desired. Belot, in a recent number of *Archives of the Roentgen Ray*, states that aluminium is the most satisfactory material for screening purposes.

Action on the Tissues.

The administration of a dose of x rays is followed by a period of time during which no change is observed in the tissues irradiated. This is known as the *latent period*. The latent period is shorter after large doses than it is after smaller ones.

1. A *small dose* stimulates all epithelial structures; there is increased secretion from the sweat and sebaceous glands, and the hair grows more vigorously. In the course of a few days or longer, these effects gradually pass off, and the part returns to its normal condition.

2. A *full dose* may be followed by a slight erythema after a latent period of ten days or a fortnight; the hair will become pale in colour, and in about three weeks it will drop out, the bulb being temporarily atrophied. The erythema passes off in a few days, the hair roots gradually recover, and in from six to twelve weeks a vigorous growth