OBSERVATIONS IN ELECTRICALLY-PRODUCED EPILEPTIC CONVULSIONS. PART III: THE POST-CONVULSIVE DECEREBRATE STATE.

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FOR a few seconds after the convulsion has ceased, whilst the patient is cyanosed and the breathing forced, there is complete flaccidity of all the body muscles. The first sign of muscular activity is a contraction of the masseter muscle, which holds the mouth tightly closed. This is followed by rigidity of a definite pattern; in its complete form the neck is retracted, the mouth closed, the shoulder flexed and adducted, the elbow flexed and the forearm pronated, the hip, knee and ankle joint are extended. Wrists, fingers and toes are invariably flaccid, offering no resistance to passive movements. In the extreme forms of rigidity the extension in the lower limbs is so intense that it is impossible to overcome it by force.

In cases in which the rigidity is less complete, the muscles most markedly contracted are the masseters, the extensors of the ankle and flexors of the shoulders; the increased tension of the muscles may be slight and easily escape observation. We considered the presence of ankle clonus as evidence of spasticity, when it was otherwise difficult to assess. There is, however, a small minority of cases in which the muscles of the limbs are entirely without tone. The state of rigidity lasts from one to several minutes. In a small number of cases following it, the muscles of the limbs become flaccid for a period varying from 1–3 minutes, after which time they regain their normal resting tone. In the majority of cases the patient then becomes restless and it is impossible to ascertain the muscular tone. The restlessness consists of mass movements of the extremities or of the body, of rapid, unco-ordinated and aimless movements in the arms and legs, or of turning or rolling movements of the body. In other cases these movements, whilst of the same character, are less generalized, and consist of thrusting or jerking movements in particular joints; they are purposeless, uncoordinated and undirected, of a pseudo-spontaneous character and they may be exaggerated by external stimuli (pin-prick). The next stage of motor reintegration carries the movements to a more co-ordinated and purposeful level; these are evoked by external stimuli and may consist of turning away of the stimulated part of the body or of brushing movements of the hand first directed into space, or at a part of the body some distance from the stimulation until finally a brushing movement is carried out at the point of stimulation.

The rigid limb shows the greatest resistance to the initial passive movement, but persists to some extent throughout the movement. When the rigidity is

of any considerable intensity the limb maintains the position, at any angle in which it is passively placed ; this is especially well demonstrable in the lower extremities. The rigidity has therefore definite plastic character.

The muscular tone can be influenced by neck and labyrinthine reflexes. Passive retroflexion of the head increases the flexor spasm in the arms, and extensor spasms in the legs. Anteflexions of the head causes relaxation of the muscular tone of the limbs. Turning of the head to one side relaxes the muscular tone of the opposite side and increases the tone of the limbs of the same side. This one-sided reaction is still more marked when the whole body is passively turned to one side while the head is kept fixed. When the body is turned from the supine into the prone position all the rigid muscles relax. All these reflexes have a tonic character, the alteration in muscular tone is maintained as long as the passive position of the head or body is kept up. The alteration in tone can only be demonstrated on agonistic rigid muscles ; no change can be found in the antagonistic flaccid muscles nor is there any reflex movement of the limbs when these tests are carried out. As has been mentioned in a previous paper (Klein and Early, 1948) vestibular stimulation by syringing with cold water fails to elicit nystagmus with slow and fast component, instead the eyes go into homolateral deviation. The same homolateral effect upon the eyes can be elicited by turning of the head to one side or by turning of the body to one side while the head is fixed.

During the whole period of rigidity the pupils are usually smaller than before the application of the shock treatment, and do not react to light. The corneal reflexes are absent. There is no reaction to nociceptive stimuli from any part of the body. The superficial reflexes are absent apart from some plantar response which consists of a flexor movement or fanning of one or two toes, corresponding to the area under pressure ; there is no combined flexor or other reaction of the toes. The tendon reflexes do not show any conformity : As has been pointed out by Kino (1944) some are increased, others diminished or absent altogether. Ankle clonus is, however, quite regularly present, even when the signs of rigidity are very slight. It is only absent in those cases where complete muscular flaccidity is maintained throughout the first stage of the post-convulsive phase. When this occurs, and when this phase lasts for any length of time without interference by involuntary movements, all the deep reflexes may gradually diminish or disappear altogether. But when rigidity is found, the briskness of reflexes proved to be dependent upon the tone of the effector muscles. Accordingly, since in the upper limbs a flexor spasm prevails, the biceps reflex is as a rule present or brisk, and the triceps and supinator reflexes are usually sluggish or not obtainable at all ; in the lower limbs where extensor spasm occurs the knee jerk and ankle reflexes are very brisk and ankle clonus is the rule.

Though at the cessation of the convulsions the breathing is difficult and irregular, at the time of the muscular rigidity its normal rhythm becomes re-established. Heart action is accelerated but regular, and the acceleration gradually diminishes during the rigid state. Vestibular stimuli are conducted and lateral eye movements can be reflexly elicited. There is finally a response of the motor branch of the trigeminus expressed by contraction of the masseter

muscles. All this suggests activity or excitability of centres of the brain stem up to the level of the mid-pontine region. Above this level no activity can be ascertained; there is no corneal reflex and the pupillary light reflex is absent.

We obtain a somewhat different picture when successive shocks are applied. A series of twelve patients were studied; we carried out this treatment with the additional purpose of gaining experience of the effectiveness of repeated shocks as a substitute for anaesthesia in transorbital leucotomy. In one session three shocks were applied, each successive shock after reappearance of the corneal reflexes in the post-convulsive phase of the preceding shock. Each patient was observed in four such sessions.

It was found that after the second shock the state of deep unconsciousness lasted much longer than after the first, and again that of the third much longer than that of the second. If after the first shock it took about 2–3 minutes before the patient showed signs of reintegration, it took about 4–5 minutes after the second, and 7–8 minutes after the third shock. In the post-convulsive state, the following observations were made.

The duration and intensity of the rigidity decreased progressively with the second and third shock. At the same time the state of complete muscular relaxation lasted longer after the second, and still longer after the third shock. In patients where, after the first shock an intense and extreme rigidity appeared, the rigidity was only moderate after the second shock and still less after the third. This applied to the muscles of the limbs; the masseter contraction remained the same throughout the three shocks. Immediately the convulsion had ceased the tendon reflexes were the same in the second and third shock as in the first, but the longer the relaxation of the muscles lasted the more sluggish they became, and in some cases they disappeared altogether. Even when the resistance of the muscles at the beginning of the post-convulsive phase showed only a slight increase, ankle clonus was still usual, though this clonus was less marked and of less duration than after the first shock. Throughout the rigid and flaccid stages tonic neck and body turning, eye reflexes were present, and phasic ipsilateral deviation upon syringing with cold water was obtainable. Tonic neck and labyrinthine reflexes were more difficult to assess in successive shocks, as the rigidity in the limbs was less marked. There was no corneal reflex and no pupillary light reflex. The end of this phase was marked by recovery of the tendon reflexes to normal, by the reappearance of the pupillary light reflex and the corneal reflex and of a dorsal response of the big toe. At this stage the following phenomena could be observed:

When the ear drum was stimulated either by tactile stimulus or by syringing, a sudden extension of the limbs occurred. In a complete reaction of this kind the legs became extended in all joints, the feet rotated inwards, the arms were slightly elevated, extended and pronated in the elbow, the wrists and fingers extended; the contraction of the muscles involved the flexors as well as the extensors with the same intensity. The same reflex could be elicited by stimulation of the cornea. In most of the cases this stretch reflex lasted as long as the trigeminus was stimulated, in one case it was extreme, and present for a few seconds after the cessation of the stimulus. In the majority of the observations the reaction was less complete, occurring

in some only at the initial stimulation or intermittently, and in others involving predominantly the muscles of the arms, whereas the legs showed only an abortive contraction. In others the homolateral limbs contracted; but in almost all cases the tendency to this reaction could be observed. From no other area of the body could this reflex be elicited, nor by any other manipulation, but occasionally it appeared automatically. There was no response to nociceptive stimuli from any part of the skin. When the stage of the reflex had appeared, the next phase of uncoordinated pseudo-spontaneous movements or general restlessness usually followed very quickly, and the reactive movements of the patients soon reached a more purposeful level.

Discussion.

Experimental physiologists, Thiele (1905), Weed (1914), Beritoff (1915), Cobb and co-workers (1917), Bazzett and Penfield (1922), Magnus (1922), Rademaker (1926), Pollock and Davis (1930), Magoun and co-workers (1946), have been in general agreement that section of the brain stem at the pontine level in acute, as well as in chronic preparations, is followed by the type of rigidity originally described by Sherrington (1896). This finding is not entirely undisputed. More recently Keller (1939–46) has insisted that, in his experiments on dogs, section from mid-brain to lower pons was followed only for a few minutes by rigidity, which yielded to complete flaccidity with loss of the deep reflexes, and this flaccidity was maintained for several weeks. Magoun and co-workers assert that on stimulation of lateral parts of the reticular formation, limbs previously rigid suddenly became flaccid and collapsed and the reflexes disappeared; they assume that there are facilitatory and suppressor centres subserving the muscular tone in the reticular substance of the brain stem. Decerebrate rigidity has also caused considerable clinical interest, and there are a number of observations published in which a muscular rigidity appeared in the course of a localized lesion of the brain stem, Turner (1916), Wilson (1920), Magnus and de Kleyn (1921), Walshe (1923), Weisenberg and Alpers (1927), Zand (1928), Walschonok (1929), Manson and Ferguson (1930), Epstein and Yakovlev (1930), Carmichael (1932), Thorpe (1935), Davis (1935), Nielsen (1941), Penfield and Erickson (1941), Davidenkov (1946), O'Neill (1946).

The clinical problem was first extensively discussed by Wilson. He took as the criterion of decerebrate rigidity the rigid posture and the muscular spasticity, and describes a variety of clinical pictures.

This rather loose conception has since been applied in most of the publications in spite of Walshe's efforts to bring clinical decerebrate rigidity more in line with the classical experimental picture.

Walshe, in explanation of clinical decerebrate rigidity, postulates the presence of plasticity of muscular tone, of lengthening and shortening reflexes and of the Magnus and de Kleyn reflexes. He would exclude cases in which the rigidity is, in his opinion, an irritative phenomenon and due to discharge, as in anoxaemic conditions, or in unconscious patients with absent light reflexes, raised temperature and pulse irregularities. He also excludes cerebellar fits. Walshe (1923) reported a case of a tumour in the interpeduncular space

in which he could demonstrate all the characteristics of a decerebrate rigidity, whilst the above complicating factors were absent. A similar case has been reported by Davis (1925). There are, however, cases of brain stem lesions described (Turner, 1916, Walschonok, 1929), where the picture was complicated by the symptoms mentioned by Walshe, and yet a rigidity, constant over a period of weeks, could be observed; this long duration is not in favour of the irritation theory. Similarly, it is difficult to accept Keller's (1945) opinion when he regards rigidity following experimental decerebration as an altogether irritative phenomenon. There seems to be no necessity to connect the otherwise typical rigidity in our observations with irritation, although it is likely that the post-convulsive decerebrate state is associated with anoxaemia. Positive evidence of its reflex character will be given later when pathological material is being discussed. Though typical, the rigidity in our observations has some particular features. It was found that in the mild forms of rigidity only one or two joints of the limbs show marked spasm, whilst all other joints appear less involved; there is no similar finding reported either in experimental or clinical decerebration. In animal experiments the method of testing may account for this; postural tests are mainly used for assessing rigidity; spasms of this kind can thus easily be overlooked, so that preparations not entirely free from rigidity may pass as flaccid. Of some interest in our cases is the early transition from rigidity to flaccidity with lowering of the reflexes. This occurrence is emphasized by Keller, although not the rule in animal experiments. The tonic neck reflexes were different from the usual description only in that no tone increase occurred in the antagonists when the agonists relaxed; therefore no reactive movement of the extremities took place. The presence of tonic labyrinthine reflexes could be demonstrated by changing the patients posture from the supine to the prone. The tone of the rigid muscles consequently relaxed, and the relaxation was maintained as long as the patient was kept in the prone position, but there was no reflex movement of the limbs. The phasic and tonic eye reflexes found at this stage were analysed in a previous paper (Klein and Early 1948). The influence on the muscular tone of the limbs was strongest when the patients were moved into the lateral position with head fixed; the response consisted of a contralateral relaxation and a homolateral increase of the rigidity, identical with the reaction seen when the head is turned to one side. Neck reflexes were, however, excluded by fixation of the head. It seems unlikely that stimuli from the body surface could have had any influence, since there is at this stage no response to superficial stimuli. This suggests a labyrinthine origin. It has been found under experimental conditions (Magnus 1921), that tonic labyrinthine reflexes can be elicited by alteration of body posture in the vertical plane, but not in the horizontal. Since this experimental finding was an important point in the explanation of the mechanism of the tonic labyrinthine reflexes, our conflicting observations are of some interest.

The rigidity is only a part of the decerebrate state. Though it has been proved that a local process in the brain stem can produce a rigidity which is closely related to experimental rigidity, there is no actual decerebration under these conditions. As Wilson (1921) pointed out, unless the interruption is

absolute the cortex (we may add subcortical centres) may, and often does, interfere in part with the activity of the lower centres. It is therefore of no great value to make comparisons between our observations and such lesions. We have sufficient clinical evidence to assume that at this stage the central nervous system is acting at a pontine level functionally isolated from higher centres. Experiments in which functional conditions similar to the post-convulsive state have been produced were carried out by Ward and co-workers (1947). These authors found after intravenous injection of sodium cyanide a rigidity indistinguishable from decerebrate rigidity. Electro-encephalographic records demonstrated that the electric activity of all structures above the level of the midbrain was abolished by appropriate doses. If, as we assume, the conditions produced by these experiments are comparable to those of the post-convulsive state, then they confirm our clinical conclusions. However, compared with that in animals, the functionally isolated brain stem in our observations shows considerable differences in function, the pattern is primitive, the reflex response is reduced to a minimum and is of a primitive nature. The tone regulating centres can draw only on two afferent sources from which stimuli are conducted: the muscle proprioceptive and the vestibular system. We do not suggest that this difference is due to a difference in function of the isolated brain stem between human and animal. Under the conditions of our observations a sharp borderline between active and inactive nerve tissue cannot be expected. There may be variations in degree of activity in different cell groups, some areas may be depressed, others may be more active. Such factors could explain why we may fail under these conditions to obtain a complete picture of brain-stem functions. It may be that such factors interfere less in experimental decerebration.

In slowing down the re-evolution by the application of successive shocks, the first sign of re-integration is the recovery of the deep reflexes and the reappearance of reflex movements upon superficial stimulation. It is at this stage that the trigeminal stretch reflex can be elicited. As a muscular reaction, it differs strikingly from the initial rigidity; there is invariably an extensor posture of all the limbs in which all the joints participate, and in which the muscles, agonistic as well as antagonistic, are in intense contraction. The fully developed picture is, so far as the limbs are concerned, indistinguishable from the tonic phase of the epileptiform seizure. The transient character of the stretch reflex did not permit the study of the behaviour of the muscles under passive movements, but in the tonic phase of the epileptiform fit we found the same plasticity as in the initial decerebrate rigidity. The trigeminal stimulation evokes a reaction to which some tendency already exists; at this stage a spontaneous stretch posture, particularly in the upper extremities can be seen intermittently in a number of patients. The response upon trigeminal stimulation is thus comparable to the provocation or intensification of rigidity in decerebrate animals by the application of nociceptive stimuli (Weed 1914), or by direct trigeminal stimulation (Leiri, 1926), and similar to an observation by Carmichael (1930). At a period when the stretch tendency in the decerebrate state occurs, a redistribution of tone can be noted. Instead of the two extremes of a selective muscular contraction or complete muscular flaccidity

found in the initial stage, the tone in agonist and antagonist now becomes equal with no preponderance of either group. Passive movements in the presence of normal deep reflexes indicated that the tone is not different from its normal qualities. The general muscular contraction is thus in line with the basically even distribution of tone between agonist and antagonist. Muscular tone is dependent upon afferent impulses. At the initial rigid state these impulses are limited to proprioceptive and vestibular stimuli. In this later stage extraceptive impulses have started to exert their influcnce, and the increase of afferent impulses may have some relationship to the distribution of tone. The reappearance of the pupillary light reflexes at this period indicates that the activity of the brain has shifted to mid-brain level. The general clinical picture, however, has not changed, and there is no evidence that the change in the muscular reaction is due to influences from higher centres. The picture is still a decerebrate state which probably works at brain-stem level. The obvious structure at this level which might be responsible for the re-distribution of tone is the red nucleus. Though the observations of experimental physiologists as to the significance of this nucleus are conflicting, it is established that a typical decerebrate rigidity appears in sections below the level of the red nucleus. According to Rademaker (1926) who draws his conclusions from the literature and from his own experiments, muscular tone is normal in animal preparations when the red nucleus is left intact, which would explain the redistribution of tone at this stage of reintegration in our observations. But again the functional performances are of much lower order than in animal preparations which might correspond to this level. The number of reflex reactions is still very restricted at this stage ; the only reflex of " local signature " on superficial stimuli is the dorsal response of the big toe, appearing for the first time. There is no body righting reflex upon turning the head, nor can any co-ordinated muscular action be elicited, only mass movements. This low performance level may be due to underlying conditions, but the fact remains that under decerebrate conditions a characteristic redistribution of tone occurs consequent to a shift of activity from lower to higher brain stem levels without interference of higher centres.

In the next stage, involuntary movements of varying type are the most outstanding features. Their character suggests that the re-integration has reached subcortical level. The picture becomes complex, the patient may enter a semi-conscious condition or such condition may soon follow ; they have thus passed the decerebrate state which is the subject of this paper.

Reaction of Cases with Lesions of the C.N.S.

In the following cases with lesions of the nervous system the effect of loss of function upon the post-convulsive decerebrate rigidity was studied.

1. Three patients with unilateral absence of the ankle reflex were investigated. The motor power in these cases was intact and there was no disturbance, either in superficial sensation, position or vibration sense. The post-convulsive spasticity in the extensors of the legs was slight, ankle clonus on the normal side was marked, whilst absent on the side of the missing ankle reflex.

Viets (1920) who studied the physiology of patellar clonus in decerebrate preparations, found that there is a most favourable point of muscular tension for the maintenance of clonus. The presence of the ankle clonus in the post-convulsive decerebrate rigidity is almost the rule, whilst patellar clonus is rare. This may be explained by the fact that rigidity in the calf muscles is more constant and stronger than elsewhere, with tension favourable to clonus. But the ease and regularity with which a sustained ankle clonus can be produced in our observations without appreciable clinical spasticity of the calf muscles is still surprising. This suggests that the tone of the antagonist muscles, their resistance and the relation between tone in agonist and antagonist is significant in the maintenance of clonus. By the nature of this reflex, these factors would be more important for ankle clonus than for patellar clonus. In our three cases the failure to obtain ankle clonus on the side of the missing ankle reflex is primarily a failure of the calf muscles to contract during the rigidity and to set the basic condition for clonus. Sherrington, by cutting the dorsal roots of a particular muscle group, showed that this muscle group failed to contract during the decerebrate rigidity, thus demonstrating the dependence of the rigidity upon afferent impulses and its probable reflex character. Our observations correspond clinically to Sherrington's experimental condition and we regard these observations as strong evidence for the reflex character of the post-convulsive rigidity.

2. Observations on a female patient, 39 years old, who 12 years previously had an acute spinal disease, probably a myelitis. She came into hospital with a depression. On neurological examination she showed a hyperaesthesia for touch, prick and temperature from D.3 downwards. In the left leg there was a slight extensor spasm in knee on initial passive movements, which became flaccid after the first resistance had been overcome. No extensor resistance in hip, slight contraction of ankle in middle position, the active stretching in hip and knee showed reduced muscular power. Active flexion of hip and knee was impossible. There were also reduced active movements in ankle and toes. In the right leg the motor power of all the big joints was good, but there were no active movements in the toes. The muscles in this leg were slightly hypotonic. The position sense of the right big toe was inaccurate. Vibration sense of right much less than on the left. Abdominal reflexes were brisk and equal on both sides. Both knee reflexes were brisk. No ankle reflex was obtainable on the right, but it was very brisk on the left. Definite dorsal response of the big toe on the left; on the right equivocal response. Cranial nerves and upper limbs were intact. The patient had E.C.T. In the tonic phase of the convulsions no difference could be seen in the contraction of both legs, the twitching in the following clonic state was less in the left leg than in the right. In the post-convulsive phase both arms were in slight flexor spasm of the usual type, on the left there was extreme extension in hip and knee, whilst the right leg was completely flaccid in all the joints throughout. Definite neck and labyrinthine reflexes upon the right leg and the arms were present. By bending the head passively forward the rigidity in the left leg diminished considerably; the left leg became completely flaccid when the body was turned head fixed, into right lateral or supine position.

In this case the most striking observation in the post-convulsive stage was the complete flaccidity of the right leg in contrast to the rigidity of all the other limbs.

In investigations of more than 200 patients in the post-convulsive stage of E.C.T. the rigidity of the two halves of the body was always found equal in

distribution and intensity when the nervous system was intact. The incongruity of complete flaccidity of the right leg in this case, therefore, must be related to the neurological lesion. In the right leg there was a deep sensory loss and an absent ankle reflex, but the knee jerk was present and brisk. The absence of rigidity in the calf muscles was in accordance with the findings in the three preceding cases, but could not be predicted in the remaining extensors. An absent tendon reflex seems to indicate that afferent impulses, necessary for the reflex rigidity of the muscles concerned, are not conducted, but its presence does not assure contraction. It follows that the routes on which the afferent proprioceptive impulses travel are not the same as for the spinal reflex arc and for the brain stem centre which is responsible for the rigidity.

In the left leg where pyramidal signs were found together with a weakness in the flexor group, the extensor rigidity was very intense. Extreme reactions are not exceptional, but usually they manifest themselves with equal intensity everywhere. The right leg did not take part in the rigidity. The arms, however, were intact and the rigidity was light, not in harmony with the strong reaction of the left leg. Apparently the reaction of the left leg was intensified by the defect of this extremity, due to damage to its pyramidal pathways. The part played by the pyramidal tract in the decerebrate rigidity has been studied in animal experiments. Weed (1921) in a decerebrate preparation hemi-sectioned the bulb above the pyramidal decussation, and found no change in the rigidity on the contralateral side. Thiele (1905) caused the pyramidal tracts to degenerate prior to the brain stem trans-section and found no change in the nature and extent of the rigidity. He concluded that the pyramidal tracts have no relation to the causation of decerebrate rigidity. As the brain stem trans-section involves a section of the pyramidal tract it is difficult to see why an additional severance of this tract should influence the result of the trans-section. And yet in the functionally isolated brain stem of our observation a long standing degeneration of the pyramidal tract seems to have intensified the rigidity. Two explanations are possible : (*a*) That the functional isolation of the brain stem in the post-convulsive state is not as complete as the clinical picture suggests ; some cortical (pyramidal) impulses may still have been transmitted, and unless there is an additional pyramidal lesion the maximum rigidity does not develop. This explanation is not supported by experience of brain stem trans-sections, nor of the post-convulsive state where, under constant conditions, a great variation in the intensity of the rigidity is observed. (*b*) That the pyramidal lesion, by lowering the threshold of the motor centre of the brain stem and spinal cord to proprioceptive stimuli, has intensified the release reaction. This is the more likely explanation of our case.

3. The next case was seen after a succession of spontaneous epileptic seizures. He was a man of 32 years of age, a chronic hospitalized schizophrenic. Six years ago he developed a left-sided hemiplegia. His cooperation was poor on examination. The left arm was flexed and adducted at the shoulders, flexed and pronated at elbow, flexed at wrist and fingers. The left leg was extended at all the joints. He did not move the left arm actively, and in the left leg only slight flexion of the hip and knee was carried out. On the left there was a severe disturbance of superficial sensory qualities, the position sense was diminished in fingers and big toe. There was strong passive resistance to the stretching of the left shoulder and of the

left elbow, and very strong resistance to the passive flexion of the left hip and of the left knee.

In the last two years the patient has developed epileptiform seizures. He was seen after he had had three successive fits at short intervals. He was unconscious, and it was found that the muscles of the limbs of both sides were completely flaccid. He was again examined after he had two further fits without regaining consciousness. It was then found that the left arm was flexed and adducted at the shoulder and pronated and extended at elbow with contraction of flexors and extensors. The hand and fingers were in the same position as before the fit. There was strong passive resistance to lifting and adducting of the shoulder, as well as to flexion and supination of the elbow. The left leg was extended in all joints and rigid in this position. The rigidity in both these extremities was of " clasp-knife " character, which broke down after the initial resistance had been overcome. In contrast the right extremities showed no tone increase. Passive bending forward of the head immediately relaxed the tone on the left side, and the same occurred by turning the body to the right. The tone of the left side increased when the body was turned to the left. On the left there was a Babinski response with patellar and ankle clonus as before the series of fits. On the right the reflexes were approximately the same as before the fits. After a few minutes pseudospontaneous movements began, and at the same time the left arm went into the same flexed position as before the fits. Tonic neck reflexes could not be elicited any more.

In this case a lesion of the right hemisphere was present by which considerable parts of the right cortical and perhaps also subcortical regions were eliminated or cut off from lower centres. There was evidence of a pyramidal lesion with the characteristic posture and alterations in tone and reflexes. The rigidity found in the post-convulsive phase after a series of fits was not just a reappearance of the hemiplegia. The arm, otherwise flexed in elbow with spasm in the flexor group, became extended with contraction of agonist and antagonist. Tonic neck reflexes could be elicited which were not previously present. This altered picture was thus not solely determined by the motor lesion which existed before, but must be related to the post-convulsive functional decerebration. The right intact extremity did not take part in the rigidity, therefore the post-convulsive muscular rigidity on the left was facilitated by the pyramidal lesion as in the previous case. The picture is apparently similar to that elicited by trigeminal stimulation and to the spontaneous stretch reaction occasionally seen in a later stage of the post-convulsive phase.

4. The next patient, male, 42 years old, had a Parkinsonism for about five years ; he developed depressive-obsessional features and was treated with electric shock. On examination he showed a rigid facial expression, dysarthric speech and a marked right-sided rigidity with cog-wheel character in the wrist. The muscular power was satisfactory. The left extremities showed nothing abnormal. The deep reflexes were brisk, equal on both sides, and there were no pyramidal signs, nor was there any sensory loss.

In this case there was a strong post-convulsive rigidity of the usual type, the upper limbs in flexion, the lower limbs in extension and no difference in intensity nor in distribution between the two sides. The extrapyramidal lesion of the right extremities had therefore no influence upon the post-convulsive rigidity.

5. A 26-year-old schizophrenic who ten years previously had poliomyelitis. On examination he showed a complete flaccid paralysis of the right quadriceps with wasting of this muscle and loss of knee jerk, slight decrease in the right flexor

group of the knee, restriction of the dorsal flexors of the foot with preservation of the extensor digitorum only. The calf muscles were intact. There was no sensory loss on the right. There was no disturbance in the arms nor in the left leg. During the tonic phase of E.C.T. the left intact leg went into extension with contraction of all the muscles, whilst the right leg became flexed in hip and knee. There were no definite movements in ankle and toes. Almost immediately after the cessation of the convulsion the post-convulsive rigidity set in, the arms were flexed and adducted at both shoulders, flexed and pronated at elbow, and there was considerable resistance to passive extension of these joints. The left leg went into strong extension in the usual way; hip, knee and foot were extended. The right leg, however, showed flexor rigidity in hip and knee of much the same intensity as the extensor rigidity of the left leg. There was an ankle clonus though less marked and sustained than on the right leg.

The paralysis of the knee extensors in this case was evidently the cause of an alteration in the rigidity pattern. Instead of the usual extensor spasm in knee and hip a flexor spasm occurred in both joints though the hip extensors were functionally intact. Sherrington's interpretation of the extensor rigidity as a static reflex counteracting gravity is still accepted, but this does not explain why the flexors of the knee should contract when the extensors are paralysed, and still less does it explain the contraction of the hip flexors instead of the intact extensors. In our case the action of the tone regulating centre was apparently not to produce extension but to distribute the tone into the extensor group; as this was not possible for peripheral reasons the impulses of this centre were switched into the flexor group instead. Thus the distribution of tone to synergic muscle groups in a simple extensor-flexor pattern was the over-ruling factor on which the isolated brain stem centre worked. The fact that the flexor group contracts readily when one of the antagonists is out of action suggests also that this contraction has been facilitated by the absence of inhibition, which exists in the flexors under the usual conditions of extensor rigidity. This reaction has some relation to experimental findings (Brown and Sherrington, 1912) in which by repeated stimuli of a particular point of the motor cortex the extensor response was reversed into a flexor reaction or with the reversal of the response by the reversal of the initial position of a responding limb (Ward, 1938). According to Lorento de No' (1939), the transmission of an impulse through any neurone is not a fixed, predetermined event, but to a certain extent optional.

6. A woman, 45 years of age, was treated for an acute excitement; her left leg was amputated at the age of 14, just below the hip joint. The stump was feeely movable and the muscular power for flexion and for extension was very good, not much less than that of the intact right leg. She showed in the post-convulsive state a marked flexor rigidity in both arms and an extensor rigidity in the right leg. The hip joint of the left amputated leg was completely flaccid.

Two explanations can be offered for the non-participation of the stump in the rigidity. (1) Because of the absence of most of the muscles the proprioceptive stimuli may have not been sufficient to activate the brain stem centre, or (2) assuming a tone distribution to synergic muscles as the basic necessity in the activity of the brain stem motor centre, the conditions for this activity were unfulfilled in this case.

Summary.

The post-convulsive state gives an opportunity for analysis of decerebration on a large scale such as is offered by no other clinical condition. Clinical evidence suggests that the functional level at the initial stage of the post-convulsive decerebration is the pontine level. As the decerebration is due to a transient functional isolation and not to an anatomical severance there is no dividing line between functioning and non-functioning nervous tissue. It is therefore possible that within an area or at a level where certain centres are found functionally active, others may be inactive, or various centres may have a varying degree of activity. The picture obtained cannot, therefore, claim to demonstrate optimum functional conditions in decerebration or to be representative of the functional pattern of the isolated human brain stem. Under these special conditions the pattern is well defined. Compared with animal preparations, where the level of severance can be regarded as approximately the same as the level of functional isolation in our observations, the ascertainable functions are inferior. In isolating the various components of the resulting primitive reflex mechanism we found that on the receptive side the muscle proprioceptive and vestibular stimuli were the only afferent impulses. The corresponding picture on the reactive motor side was a rigidity, with phasic and tonic reflexes of the rigid muscles of the limbs and of the eye muscles upon proprioceptive and vestibular stimuli. In distribution and character the rigidity appeared to be closely related to the experimental rigidity; the rigid muscles showed a plastic tone and tonic neck and labyrinthine reflexes were present. We established that muscle tone and deep reflexes were correlated, e.g. the reflexes of the rigid muscles were present or exaggerated, those of the flaccid muscles were diminished or absent. There were, however, some special features. Wrist and fingers were always free from rigidity and ankle clonus was the rule. Among the postural reflexes the influence on rigidity of lateral body movement with fixed head was discussed.

On observation of cases with lesions of the C.N.S. evidence of the reflex character of the rigidity was given. There was some indication from these observations that degeneration of the pyramidal tract enhanced the rigidity in contrast to findings in animal experiments. No difference between the two sides in the post-convulsive rigidity was found in a case of hemi-Parkinsonism. In a case with unilateral paralysis of the quadriceps femoris the usual extensor spasm in the lower limbs was converted into a flexor spasm in the affected leg.

When the decerebration was more prolonged the rigidity could not be maintained and was replaced by a general flaccidity and diminution of reflexes. Following this in the course of reintegration a redistribution of tone took place. The muscular tone became equally distributed between agonists and antagonists, and the tendon reflexes attained their normal briskness. At this period a dorsal response of the big toe appeared. It was at this stage that a general contraction of the limb muscles occurred upon trigeminal stimulation, a contraction which occasionally set in automatically. Clinical evidence indicated a shift of activity to midbrain level.

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