Neurology. — *Some Clinical and Anatomical Observations on the "Paraplegie en Flexion" and Related Motor Disturbances.* By Dr. W. J. C. VERHAART. (From the Laboratory in the Mental Hospital "Oud Rosenburg", The Hague.) (Communicated by Prof. B. BROUWER.)

(Communicated at the meeting of September 28, 1929).

The researches made into this paraplegia, covering the last 30 years did not result in showing a clear anatomical localisation, since the post mortem examinations of the greater part of the cases showed degenerations in nearly all parts of the central nervous system.

A short historical outline of the subject under discussion will prove this assertion.

In 1911 BABINSKI ascertained that in this paraplegia the patellar and Achilles jerks were not increased, while the reflexes "dits de défense", the reflexes of flexion of the leg obtained by stimulation of the foot were always present. BABINSKI noticed this dissociation of reflexes in cases of compression of the spinal cord, of the Pons, and in some cases of Sclerosis Multiplex, and admitted that it was possible that this phenomenon was caused by cerebral lesions.

Cases of paraplegia in flexion of central origin were described by MARIE and FOIX in 1920, by ALAJOUANINE in 1923, by WILLIAM HERMAN in 1926, by R. LEY and LUDO V. BOGAERT in the same year. These researchers noticed multiple degenerations and foci in the cortex, in the marrow of the hemispheres, and in the basal ganglia, as well as in the cord in some cases. Especially the region of the paracentral gyrus of both sides was affected, more or less complete degeneration of the pyramidal tracts was always to be found. The symptoms of this paralysis were only found in patients suffering from organic dementia.

A circumscript anatomical substratum could not be given. ALAJOUANINE, however, was struck by the frequency of bilateral lesion of the paracentral region, where the cortical centrum of the motility of the lower limbs must be supposed to be located. He is of opinion that this bilateral lesion is the cause of paralysis in flexion of the lower limbs. The same view is held by R. LEY and LUDO V. BOGAERT.

In 1928 I published a case of paraplegia in flexion, in which many lacunae were found in the cortex and basal ganglia, as well as a considerable loss of ganglion-cells in the frontal and the central gyri. There was a marked loss of fibres in both pyramidal and fronto-pontine tracts, the mid-brain and the lower parts of the nervous system showed no primary

degenerations. I, therefore, thought that the combination of frontal and central lesions were the cause of the paralysis in flexion of the lower limbs.

Also in German publications similar cases are to be found, although they are not described there as instances of paraplegia in flexion. JACOB, STIEF, KASHIDA, O. FÖRSTER and KIRSCHBAUM describe cases of Arterio-sclerosis cerebri, "spastische Pseudosclerose", and other diseases, which led to paralysis in flexion of the lower limbs. Foci were found in most parts of the nervous system, degeneration of the pyramidal tracts nearly always occurred to a greater or smaller extent.

In the mental hospitals, where patients with disseminated cerebral lesions will frequently have to come, symptoms of paralysis in flexion of the legs are rather common, which enabled me to observe a small number of such patients in a comparatively short time, in each case checking the conclusions made by means of a post mortem examination. When examining more closely the symptoms of motor disturbances, it soon became obvious that there existed great differences between the symptoms of the various patients, and that a classical "Paraplegie en flexion" was only present in some of them.

Clinically the patients must, therefore, be divided into 3 groups, the first of the three being the pure "paraplegie en flexion", with both legs paralysed in flexion. The second group consists of patients suffering from a *hemiplegia* with flexed lower limb, without motor disturbances of the contralateral arm and leg. The third group was formed by patients with both legs in flexion, who, when examined clinically, were found to have absolutely no signs of paralysis, being able to stretch their legs actively, and to make all other movements without help. Notwithstanding this, they were always found with their legs sharply flexed. The reflexes "dits de défense" were increased.

The first patient to be described suffered from a paraplegia in flexion during the last year of his life. He passed the last 6 years of his life at Oud Rosenburg and other hospitals because of his general paralysis. Specific treatment, Recurrens and Malaria could not arrest a steady mental and physical decay. During the last months of his life he could hardly speak any more, both his legs were constantly flexed, while he was unable to change this attitude. No mention is made of his reflexes.

A histological examination made after death confirmed the diagnosis. Macroscopically the brain showed a marked atrophy of the windings, the frontal and temporal pole were extremely atrophied the other windings of these lobes were also atrophied, though to a smaller degree, except the back of the middle frontal gyrus. The frontal operculum of both sides was atrophied, as well as the supramarginal and the angular windings, both Fossae Sylvii were surrounded by atrophied windings. The upper part of the Gyr. centr. ant. of the right hemisphere was atrophied, the rest of the brain seemed unaffected. Sections showed that the left lateral ventricle

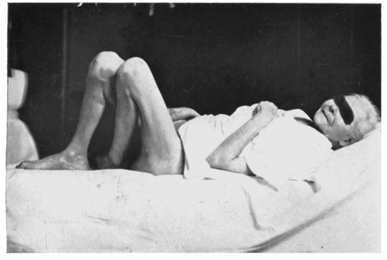

Fig. 1.

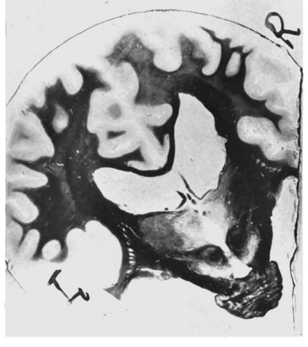

Fig. 2.

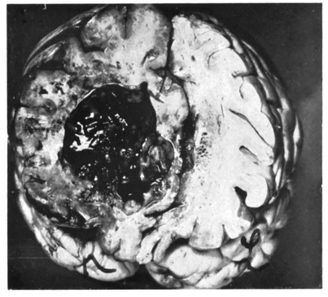

Fig. 3.

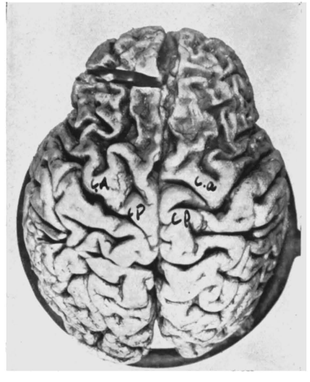

Fig. 4.

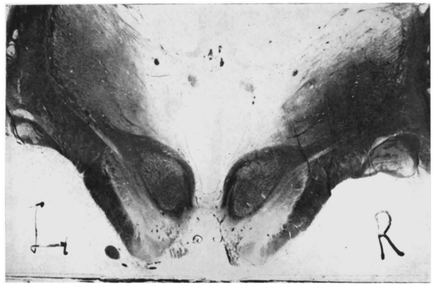

Fig. 5.

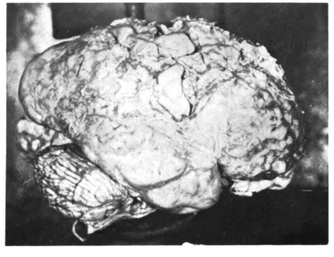

Fig. 6.

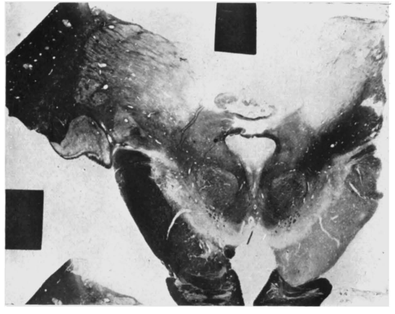

Fig. 7.

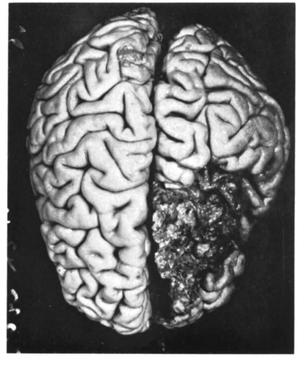

Fig. 8.

Fig. 9.

was much enlarged, especially in the neighbourhood of the paracentral gyrus, the wall of the ventricle was uneven, under the ependym many lacunae were found. The right lateral ventricle was somewhat enlarged, the wall was smooth. Both nucl. caudat. were much atrophied, macroscopic haemorrhages or softenings were not present.

WEIGERT—PAL preparations showed a great loss of fibres in the atrophied winding, the marrow-fibres of the left praecentral winding were much reduced by the subependymal necrotic process. In the midbrain the inner part of the Pedunculus Cerebri was somewhat paler than the rest, a complete degeneration of the fronto-pontine tract could not be detected. Both pyramidal tracts were too pale in the Medulla Obl., as well as in the cord, while in the lower dorsal and in the lumbar segments these tracts were wholly degenerated. In this case both frontal lobes and both pyramidal tracts for the lower limbs were degenerated. In this case the result was a paraplegia in flexion.

Another patient, who also showed symptoms of paralysis in flexion, however in a somewhat different constellation, suffered from an organic dementia in the latter half of her 7th decennium. During life the nature of her disease was not clear, after death it proved to have been a case of general Palsy. She died at the age of 70 ; shortly before her death the following symptoms were noticed :

The patient is confused in mind, her speech is disjointed, she does not answer questions, but seems still to understand simple sentences. In her bed she lies on her back with both legs sharply flexed. The left arm is flexed into a right angle in the elbow, the right limbs can be moved actively, the left limbs are paralysed. The left lower limb is totally flexed in the knee, the right is bent into an acute angle Ph. I. The left leg can be moved passively in the knee, triflexion reflex immediately appearing when the foot is stimulated. Patellar and Achilles reflexes are unobtainable, BABINSKI's reflex is positive. The left arm is bent in the elbow, and lies motionless on the breast, the fingers at the ulnar side are bent in all joints and rigidly contracted. Passive movement is possible only to a limited extent in the elbow, the biceps and triceps jerks are obtainable, spontaneous movements do not occur in the arm. At the right extremities, no motor disturbances are to be observed, except the constant flexed condition of the leg with limited capacity of stretching ; the tendon reflexes are normal, the sole-reflexes are plantar, active movements are possible. The abdominal reflexes are obtainable on the right side, but not on the left.

After death it appeared that the cerebrum showed considerable atrophies: At the right hemisphere the entire frontal lobe was much atrophied as far as the Gyr. centr. ant., except the agranular zone of the upper winding, likewise the frontal Operculum and the base of the foremost central winding. All of the three temporal windings were much atrophied, the Gyr. central. post. was softened as far as the lower half, likewise the parietal and the occipital windings. The left hemisphere showed an atrophied frontal

lobe, just as the right, the anterior central winding, the frontal Operculum and the temporal windings were, however, in much better condition. The parietal and occipital windings showed no degenerations.

On the transverse sections an enormous Hydrocephalus was found, which extended upwards to a considerable degree into the central region, in this way undermining the fibres from the paracentral windings for the greater part. (Ph. 2). The Nucl. Lentiformes were somewhat atrophied on both sides, the Nucl. caudat. were very narrow.

The sections, which were stained according to the method of WEIGERT—PAL showed a marked difference between the left and right hemisphere. From the central and paracentral windings on the left side strong bundles of fibres extended to the Capsula Int. The bundles of the right side were very poor in fibres owing to a lack of fibres of the windings themselves, and the enormous enlargement of the lateral ventricle (Ph. 2). Similar sections stained with BÖHMER's haematoxyline showed excessive paralytical degenerations and a total destruction of the cyto-architectonics in the greatly atrophied windings ; the slightly atrophied windings showed an inconsiderable degree of peri-vascular infiltration and vascular growth, the cyto-architectonics, however, were still clearly recognisable.

In the basal ganglia only extremely slight deviations were to be found, ganglion-cells were present in great numbers everywhere. The fibre-systems in the midbrain were not degenerated, the systems H1 and H2 of Forel, the Ansa Lenticularis, and the fibres about the Nucl. Ruber were entirely intact. In the Pons the right pyramidal tract was too pale, the transverse pons-fibres and also the pons-ganglia showed a normal aspect. The pyramidal tract from the right hemisphere was degenerated in the Med. Oblongata and the cord, and could be followed as such into the lumbal segments ; in the lumbal segments it was found that the other pyramidal tract was not wholly intact ; however, it contained a great many more fibres than the first-mentioned.

Therefore in the case of this woman the greater part of the right hemisphere had disappeared, in consequence of a paralytic process, while of the upper part of the anterior central winding, which was still preserved, the greater part of the descending fibres close under the cortex was interrupted by a subepedendymal necrosis. Of the other hemisphere only the base of the anterior central winding and the frontal lobe was affected, the lower parts of the brain showed no lesion that was worth mentioning.

In consequence of this the left extremities were both paralysed in flexion, while the right leg showed a pronounced tendency of flexion without actual paralysis. In this case there is already an indication of the analysis of the paraplegia in flexion in the tendency of flexion and the paralysis ; in both legs the tendency of flexion has become manifest, only the leg, the pyramidal tract of which is degenerated has got paralysed in flexion.

With the patients now to be discussed, we shall be able to study more

closely the tendency of flexion, either combined with paralysis caused by attendant pyramidal disturbances or not, and localise it anatomically. This tendency of flexing the legs is invariably attended with incapacity to walk erect, that is to say, the movements required for walking can be executed, so that the patient, if supported, can walk a few paces, but very soon his knees begin to give way, or his legs slip away under him.

Symptoms of lesion of the pyramidal system occur only by way of exception, or are merely indicated, the "reflexes, dits de défense" are clear. In a very demonstrative way these symptoms are met with in the case of Mrs D.

In 1924 this patient began to complain of headaches, dizziness, and impaired eye-sight. On examination she was found to have a right-sided homonymous hemianopsia and optic neurotics. She was therefore trepanned occipitally on the left side, but no tumour tissue was found, though the cerebral pressure proved to be increased. In the course of years the condition grew worse and worse, the right arm became paralysed, while at the same time the deep sensibility in that arm practically disappeared. It was observed that the patient, who had, in the meantime become bedridden, was always lying in bed with her legs flexed in knees and hips, it appeared that she had lost the power to walk erect, in the same way as described above, although her legs were not paralysed. In connection with the hemianopsia the paralysis, and the sensibility disturbances in the right arm, it was now supposed that the tumour was to be found at the foot of the central windings, and tending downwards would destroy the Tract. Opt.

Shortly before death the general symptoms of increased intracranial pressure suddenly grew much more pronounced, the flexed condition of the legs at the same time gave way to a strongly marked stretched condition. More than 4 years after the beginning of the illness, death occurred under increasing coma.

On opening the cranium it was found that the windings of the cerebrum were flattened everywhere, the sulci were closed up, the right frontal lobe was about twice as thick as the left. Transverse sections brought to light a recent haemorrhage in a tumour in the front part of right frontal lobe, also in the left lobe tumour tissue was present. (Ph. 3). The right frontal lobe was considerably dilated owing to the haemorrhage and the tumour, so that the left lobe was compressed. On sections made farther back the tumour extended through both frontal lobes, terminating in the region of the Foramen Monroï. The base of the left central winding was compressed by the tumour, in the same way as the left For. Monroï, so that fibres radiating from that winding had greatly decreased in volume, and the left temporal and occipital ventricles were considerably dilated owing to the impeded passage of the ventricle-humour.

In the midbrain, the cerebellum and the Med. Obl. there were no deviations.

In the WEIGERT—PAL preparations the compressed central windings

of the left hemisphere proved to be poor in marrow-fibres, in the Pedunculus Cerebri the fibres on the left side going to the arm were degenerated.

In the case of this patient the symptoms pointing to lesion had been caused by pressure of the tumour on adjacent parts of the brain, the actual havoc made by the tumour was not diagnosed during life. The destruction of the frontal lobes caused a tendency of flexion of the legs, which did not culminate in a paraplegia in flexion, because there was neither paralysis nor lesion of the pyramidal tracts. The tendency of flexion disappeared towards the end, when through the considerable haemorrhage in the tumour of the right frontal lobe, the intercranial pressure increased so greatly, that the cerebrum ceased to function, causing the tendency of flexion to give way to decerebrate rigidity.

Another woman, who showed a constant flexed condition of the legs without paralysis, was the patient J.

Already at the age of 46 she began to suffer from a rapidly progressive organic dementia, so that in a short time she unlearned to perform her daily work, began to make mistakes in speaking and writing, and did not know her way about in the house any more. Epileptic or apoplectic attacks did not occur, neither did paralysis, only the left leg became a little shaky. An inquiry into the antecedents of the patient's parents brought to light, that the patients mother had shown similar symptoms, which, however, did not appear before she was 74, death ensuing in a few months.

Two years after the beginning of the disease the patient was admitted into the hospital, she was much demented then, was quite incapable of speech, walked badly, and wanted assistance with everything. The very simplest actions, as e.g. conveying a piece of bread to her mouth were performed imperfectly, other actions serving a practical purpose were not noticed, she did not at all react on verbal orders given to her.

An examination, shortly before death gave the following results:

The patient lies in bed practically motionless, legs sharply flexed in hips and knees, and arms folded across breast. She can actively stretch her legs to the full extent, though the movements are slow. The patellar and Achilles jerks are not increased, neither knee nor foot-clonus are obtainable, the sole-reflexes are mostly plantar, sometimes uncertain on the right side, the OPPENHEIM reflex is dorsal on the right side. On stimulation of the foot or the lower part of the leg the triflexion reflex is immediately excited. In the arms the mechanical irratibility of the muscles is very great. The reflexes are unchanged, the fingers of the left hand are contracted in a flexed position. The suction reflex is not clearly obtainable, although she seeks and follows with her lips an object held out to her. The "RÜSSEL reflex" is lively, the threat reflex is positive on both sides.

This patient died at the age of 53, in consequence of broncho-pneumonia. The disease was diagnosed as PICK's Atrophy, as the symptoms corresponded exactly to those mentioned in GANS's publication on this affection

and its differentiation from ALZHEIMER's disease. The total apraxis, the absence of hallucinations and delusions, the apathic nature of the disease, the absence of logoclonus, so typical for ALZHEIMER's disease, vacant facial expression, all these characteristic symptoms were uncommonly completely displayed by this woman.

On the cranium being opened this diagnosis was confirmed at first sight, as shown in Photo 4. An extreme atrophy of the frontal lobes, with a sharp demarcation on the line between BRODMAN's Regiones 6 and 4, could be ascertained immediately. Moreover region 32 on the medial side of the frontal lobe and the lowest temporal winding of the right hemisphere, the temporal pole, and the G. Submarg. were atrophied corresponding to regiones 20, 21, 38, and 40, likewise sharply demarcated from the surroundings. The atrophied windings were discoloured to a yellowish brown, and were somewhat firmer to the touch than the other windings.

When transverse sections were made, it was found that there was a considerable Hydrocephalus, especially in the frontal parts of the front cornua, the upper part of the Nucl. Caud. was greatly atrophied. Neither haemorrhages nor softenings were found, the vasa were not calcified, the cerebrum weighed 690 grams.

The histological examination showed a nearly total absence of ganglion-cells in the atrophied regions, senile plaques or ALZHEIMER's fibrillary degeneration were not present, further the typical degenerations as described by GANS were found.

WEIGERT—PAL preparations proved that the marrow fibres in the frontal windings had almost entirely disappeared, just as the frontopontine tracts in the Capsula Int. and in both Pedunculi Cerebri (Ph. 5). In the right Pedunculus Cerebri some medial bundles from the pyramidal tract were lacking. The fibre systems of the mid-brain, H1 and H2, the Ansa Lentic., the fibres about the Nucl. Ruber, the transverse Pons fibres were intact, only the hilus of the Nucl. Ruber was less clear than in normal cases. The Nucl. Ant. and Med. of the Thalamus seemed to be a little atrophied, the cells of the Subst. Nigra were pale and imperfectly formed.

This patient showed, even more than the preceding, a syndrome that had much in common with the classical cases of "Paraplegie en Flexion", dull facial expression, extreme organic dementia, uncleanliness, spasmodic fits of weeping and laughter, lack of spontaneous movements, flexed condition of the legs, unchanged tendon reflexes, increased flexion reflexes in the legs. The great difference is the absence of paralytic symptoms of the legs, although the power of walking erect had been lost. An anatomical examination revealed no pyramidal tract lesions, except some fascicles situated medially in the right Pedunculus Cerebri, which caused the paralysis of the left hand. The destruction of the frontal lobes had, therefore, given rise to predomination of the tendency of flexion of the lower limbs and the loss of the power to walk erect. A more complete,

perfect destruction of the frontal brain than took place in this woman suffering from PICK's Atrophy, can hardly be imagined.

The patients that have been discussed so far showed especially degeneration of the frontal brain, either combined with pyramidal tract lesion or not. In consequence of this a flexed condition of the legs had resulted, attend with paralysis, if the pyramidal tract system was degenerated. The lower portions of the brain were in all cases wholly or, for the greater part, intact. Another combination of lesions, unilateral degeneration of the frontal and central brain appears also to be able to cause symptoms of paraplegia in flexion as will be illustrated by the two cases now to be discussed.

The patient K. had to be admitted into the Ramaer Hospital, at the age of 63, because she had become paralysed on the left side in consequence of an attack of apoplexy.

In the hospital it was ascertained that the patient was quite paralysed atonically on the left side, arm and leg lacked all active motivity, tendon reflexes were unobtainable, with the exception of the triceps tendon reflex of the arm. The leg was kept flexed constantly in knee and hip, the BABINSKI and OPPENHEIM reflexes were negative. The left facialis was paralysed centrally, the ocular movements were possible in all directions, the pupils reacted on light.

After death, about 6 months after paralysis set in, it was found, when the cranium was opened, that the entire right hemisphere had softened, in the thickened pia mater hung a pulpy greyish yellow mass, in which it was still possible to recognise remains of the windings. (Ph. 6). The left hemisphere did not show degenerations externally, the wall of the vascular corona was thickened here and there. On making a section it was found that the whole right hemisphere, where it had not softened owing to softening of the marrow, was separated from the brainstem. Moreover the Nucl. Lenticularis, the lateral Nucl. Thalami, part of the Pedunculus Cerebri, and the greater part of the Pulvinar had disappeared.

No serious deviations were seen in the left hemisphere. Serial sections through the brainstem and the remains of the softened hemisphere stained in the usual ways, to study nerve fibres and cells, revealed the following results :

The Thalamus did not contain any recognisable ganglion-cells in a single nucleus, everywhere there was a considerable infiltration, at least, if larger and smaller foci had not dissolved the tissue. The Capsula Int. was not present, the Fasc. Lent. of FOREL did not contain fibres, the Fasc. Thalamo-Mammillaris was on the right side half as voluminous as on the left, but was well tinctured, the Corp. Mamm. was on the right side half as big as on the left, in the most lateral part nervecells were recognisable. The Fasc. H2 was poor in fibres on the right side, the Fasc. Retrofl. was the same on the right and left sides, the Nucl. Ruber was on the right

side a little flatter than on the left, the hilus was not perfectly formed, the Corp. Geniculata were lacking on the right side, the Pedunculus Cerebri did not contain fibres. (Ph. 7). The Subst. Nigra was not poor in cells on the right side, the cells, however, were all paler, and smaller and more irregular in form than on the left side, between the cells there was much pigment. In the Pons all fibres extending longitudinally were lacking on the right side, the Pons-ganglia and the transverse Pons fibres were the same on both sides. The region of the medial Pedunculus Lemnisci was fibreless on the right side, and could therefore be followed far into the Pons, in the interolivary region of the lemniscus fibreless regions were likewise to be seen. The pyramidal tract in the oblongata was entirely fibreless, just as the uncrossed tract. ant. and the crossed tract. lat. in the cord, the first-mentioned was to be seen as far as the lowest dorsal segments. In the basal ganglia of the sound hemisphere a number of lacunae occurred.

This patient differs indeed greatly from the ones that have just been described, although here also, just as with the others, the frontal and the central lobes, are quite destroyed, but the decay did not stop here, also the ganglion-cells were rendered quite incapable of function, as far as the Nucl. Ruber everything is decayed. This lesion consequently amounts to the decerebration, which ZELJONI, ROTHMANN, GOLZ and others performed on their animals, without observing a change in the tonus of the muscles of the extremities. With human beings such a decerebration apparently results in serious functional derangement. In the case of ordinary paralysis, owing to decay of the pyramidal tract, generally in the centre of the capsula int., the arm is indeed rendered powerless ; the leg, however, in its excessively stretched condition can still be used for walking in the erect posture characteristic of man. In cases of paralysis in a flexed condition this difference between arm and leg is not extant, we might even say that in such cases it is just the other way round.

The leg in a flexed condition is eminently unfit to perform the movements required for walking, even without actual paralysis. Therefore it was impossible for the two first patients to walk. Consequently tendency of flexion of the legs must be looked upon as the expression of a regression of motility, the stretched leg being typical for the human species, just the same as the erect posture. As O. FÖRSTER remarks, theorising about the phylogenetic moment in the case of spastical paralysis, this tendency of flexing is still extant in the infant ; paralysis occurring in the first months after birth results in a flexed condition, and not in a stretched condition, as is generally the case with adults. He is of opinion that subcortial influence is pre-eminently favourable for the flexed condition, and that the development of the pyramidal tract causes cortical influence to take the lead, and the stretched condition to pre-dominate. From the above clinical experiences I think I am justified in concluding, that this cortical influence should be ascribed to the frontal lobes ; also with the infant the tendency of stretching gets the upper hand, not when the pyramidal tract is getting

fully developed, but much later, at the time of the myelinisation of the frontal lobes. Also phylogenetically this is more probable; not the pyramidal-tract system is the latest acquisition of the human species, this system being pretty fully developed with the higher mammals with their extremities flexed in walking; the strongly developed frontal lobes are much more specifically human, just as the erect posture with the tendency of extension of the legs.

The last patient shows us, where the tendency of flexion of the legs is located. With her, everything above the Nucl. Ruber had disappeared, causing a permanent flexed condition to become manifest. We know, however, from RADEMAKER's, R. MAGNUS's, and SHERRINGTON's experiments with animals, from clinical observations made by KINNIER WILSON and STENVERS, that decerebrate rigidity with extremities stretched to the utmost extent occurs in decerebration close under the Nucl. Ruber. Consequently in this nucleus or its immediate vicinity, the tendency of flexion is located, which during life is kept in check by the influence of the frontal lobes, to become manifest as soon as these lobes disappear. If there is at the same time a considerable degeneration of the pyramidal tract of the relative extremity, a paraplegia in flexion makes its appearance, otherwise the only consequence is a habitual flexed condition, and loss of the power of walking erect.

As a symptom of regression this syndrome is apt to occur with old persons, even if the whole frontal lobe has not disappeared, if, owing to many disseminated foci all possibilities of compensation are lost, this syndrome occurs, and is characterised by the lingering nature of the disease. Many patients mentioned in the literature on this subject show similar symptoms, although the entire frontal lobe proved not to be degenerated.

In contradistinction with this the brains of young persons can stand a considerable lesion of the frontal and central windings, which does not necessarily result in a paraplegia in flexion. SOUCQUES, for instance, described in the "Revue Neurologique" a 30 year old lueticus, with whom an entire hemisphere was practically decayed, in consequence of which an ordinary hemi plegia occurred, so that the man could use the stretched leg in walking. With old people paralysis in flexion can arise in a clearly localised way, as was the case with a patient, I could observe.

As early as 1918, at the age of 60, the patient E. was admitted into the hospital, because she had attempted suicide. During the 10 years she stayed there, she continually displayed the varying symptoms of a maniacal depressive psychosis, organic dementia was not diagnosed, neurological deviations were not found.

Her physical condition never gave rise to anxiety, till in November 1927, it was noticed that the patient walked with a certain amount of difficulty, caused by a little stiffness in the right leg.

A physical examination revealed the following facts:

Heart enlarged on the left side, at the apex systolical souffle, pulse celer

and hard, abdominal reflexes all positive on the left side, on the right side positive in the upper region, for the rest negative. Patellar and Achilles jerks all increased, on the right side patellar clonus. Solereflexes dorsal on the right, on the left plantar. Standing and walking uncertain, spastic — paretical with the right leg.

In the early part of January of the next year the patient became very lethargic, she could not stand anymore, she proved to be suffering from a rightsided hemiplegia, with arm and leg in a sharply flexed position, also the right facialis was centrally paralysed.

Examination on the 3rd April '28.

The patient is always lying in bed turned on her left side, she is quite confused in mind, and continually of uncleanly habits, she can speak only a few monosyllables (yes and no). The right leg is sharply flexed, the right arm lies on the breast, flexed in the elbow, active movements are not possible in those extremities. The patellar and Achilles reflexes are lively on the right side, clonus is not obtainable, the triflexion reflex is very lively, the sole-reflex is dorsal.

On the right arm the ulnar fingers are contracted in a loosely flexed position, the arm can be a little stretched in the elbow, the right facialis is paralysed round the mouth.

The left leg and the left arm are not paralysed, the reflexes are normal, the left leg is often in a flexed position.

The facial expression is dull, occasionally interrupted by spasmodical fits of weeping and laughing, the eyes can be moved in all directions, the field of vision does not seem to be limited, speech is monotonous and restricted to a few words, simple questions are sometimes understood.

In May '28 the patients condition grew much worse, swallowing became difficult, broncho-pneumonia caused death in June.

The disease was diagnosed as a softening of the frontal and central windings of the left hemisphere. As this was a case of acute hemiplegia in flexion the expectation of finding a circumscript defect of the parts mentioned was justified.

On the cranium being opened, it was found that this diagnosis was fully confirmed, as will be seen in photo 8, the frontal and central windings of the left hemisphere have indeed disappeared, only the orbital windings of the left hemisphere were still preserved, all the other frontal, and likewise the anterior and posterior central windings were quite softened. On dissecting the cerebrum also the marrow-cone of those windings proved to be softened, just as the marrow of the paracentral winding and the Insula. On the right hemisphere a slight softening proved to be present. The basal ganglia and the Caps. Int. were intact. The wall of the vascular corona showed slight protuberances in many places.

A histological examination of a winding that had remained intact, showed degenerations of the vascular walls, senile plaques were not found.

An examination of the brainstem by means of serial sections showed the following facts :

The medial and the anterior nuclei of the Thalamus have, for the greater part been destroyed by softenings on the left side. The Nucl. Lenticularis shows some lacunae, the Ansa Lentic., and FOREL's H2 are, however, well provided with fibres. The Caps. Int. does not contain any transverse fibres, only EDINGER's comb-system and a few fibre fascicles between the Nucl. Caud. and the putamen show marrow-sheaths. The temporal lobe is in a good condition.

In the Mesencephalon the left Pedunculus Cerebri contains marrow fibres only in the third lateral part, the Nucl. Ruber is well provided with fibres on both sides, still the hilus is better formed on the right side than on the left. (photo 9). The subst. nigra is the same on both sides. VICQ D' AZYR's Fasc. and FOREL's H1 are sufficiently tinctured. In the Pons the fibres of the fronto-pontine and of the pyramidal tract are lacking, the fibres of the temporal-pontine tract terminated in the distal pontine nuclei. The Ganglia of the Pons and the transverse pontine fibres are not degenerated.

The Pyramidal tract from the left hemisphere was entirely degenerated in the Med. Oblongata and in the cord. The Pedunculus Lemnisci Med. was fibreless on the left side, in the interolivary part of the Lemnisci fibreless fascicles were also to be found.

This woman, therefore, suffered from paralysis in an acute form with the leg in a flexed position, owing to a circumscript softening, while, although the cerebrum showed a few slight degenerations in the windings that had been spared, only one focus of any significance was to be found.

This case, of course, is a great deal different from the "Paraplegie en Flexion", which, indeed, seems to be the most usual form, in which paralysis of flexion of the legs occurs. The comparative frequency of this form can be accounted for by assuming, that the centra for maintaining an erect posture in walking are located in the frontal lobes of the cerebrum. These centra will, from the nature of things, have little or no influence on the arms, whereas for walking it is necessary that both legs co-operate to this end in the same degree, so that bi-lateral influence of the centra is essential. Consequently, if in a senile cerebrum there is something wrong with these centra, with slight degenerations in the pyramidal systems, this will be fatal, especially for the legs, and cause symptoms of paralysis with a tendency of flexion in the legs. Especially with the diffuse cerebral diseases of the senium, in which those portions of the brains which attained their full development latest, are often affected first and most seriously, and in which the tendency of regression is greatest, these symptoms will appear. The "Paraplegie en Flexion" should still be considered as the "Paraplegie des Vieillards", the name that the older French physicians gave to it.