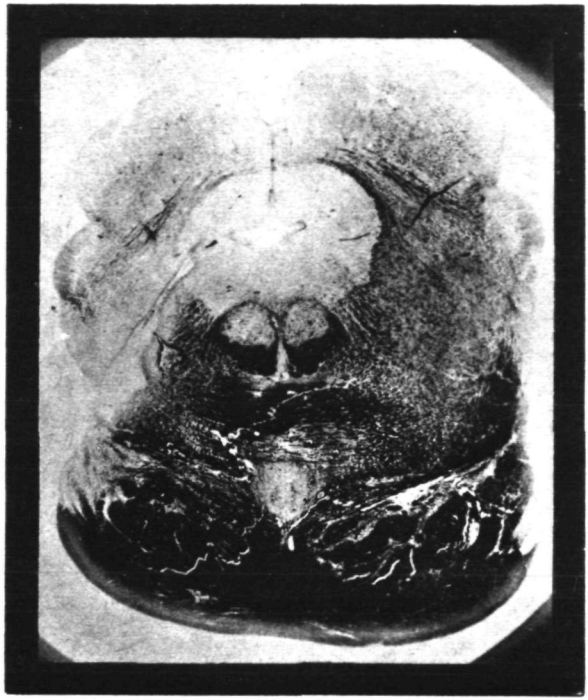
UNILATERAL DESCENDING ATROPHY OF THE FILLET, ARCIFORM FIBRES AND POSTERIOR COLUMN NUCLEI RESULTING FROM AN EXPERIMENTAL LESION IN A MONKEY.

BY F. W. MOTT, M.D., F.R.S.

FOUR years ago when making experiments upon the posterior column nuclei (an account of which was published in this journal), I made an attempt to divide the fillet unilaterally on the left side above the region of the pons. The operation was performed upon a young and vigorous callithrix monkey, with a specially constructed knife which was inserted into the cranial cavity through an opening made in the occipito atlantal ligament. When the effects of the anæsthetic had passed off I noticed nystagmus, but it was inadvisable to test it further that day. Subsequently the following notes were made:—

1st day after operation.—Continuous *oscillations* of the eyeballs towards the *left; right hemianopsy* ascertained by the fact that it does not appear to see a piece of apple until it has passed the middle line to the left; discharge from the left nostril. It can move the right arm and leg in associated movements, but not independently. If the finger be placed against the right hand it will not grasp it. A piece of apple is placed upon the left hand (the animal being prevented from seeing it), it tries to look down and seize it; no such attempt is made when the piece of apple touches the right hand. When a clip is fixed on the left hand or left foot it is immediately taken off, even a weak clip; no notice whatever is taken when it is put on the right foot; the animal does not even stop eating, a clear indication that it does not feel on that side. Conjunctival reflexes are present on both sides.

2nd day.—Dr. Hughlings Jackson saw the animal with me, and he considered that there was considerable weakness on the



I.—Photo-micrograph of a section through the seat of the lesion of the fillet on the left side. A scar can be seen extending right through the lateral fillet from the periphery nearly up to the iter.

II. –Photo-micrograph of a section of the pons below the lesion. The side of the lesion is unfortunately reversed, the left side of the pons being on the right side of the plate. Atrophy of the fillet, especially of the medium portion, will be observed, also of the middle and superior cerebellar peduncles. There appears also to be some atrophy of the pyramidal system.

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left side, and in this I quite agreed. Besides the nystagmus and right hemianopsy, one notices most marked rhythmical tremors of the left hand and arm. There is a little power returning in the right arm and leg.

3rd day.—Can now move both legs when struggling; it can grasp with the left foot, but not the right. There is want of co-ordination with the left hand, and rhythmical tremors like those of disseminated sclerosis on attempting to use it. Sensibility on the left side of the body is apparently perfectly normal; whereas on the right side it takes no notice of a clip when fixed upon the arm, leg, trunk, or ear. When touched with a piece of apple on the right side of the mouth it takes no notice, but as soon as the mid line is crossed it bites at it. Knee jerks are exaggerated on both sides.

9th day.—Movement and sensation are both returning on the right side. There is, however, still nystagmus, right hemianopsy, and a tendency to go to the left in progression. Rhythmical tremors of the left hand. Knee jerks exaggerated both sides.

16th day.—Movement and sensation has apparently returned on the right side; it was observed to scratch that side, and it can now hold a piece of carrot in its hand. Nystagmus tremors and hemianopsy as before.

35th day.—Nystagmus still present; tremors less noticeable; still right hemianopsy; sensation is apparently still blunted on the right side, but movements fairly good. The left hemiparesis still manifest. Knee jerks exaggerated both sides.

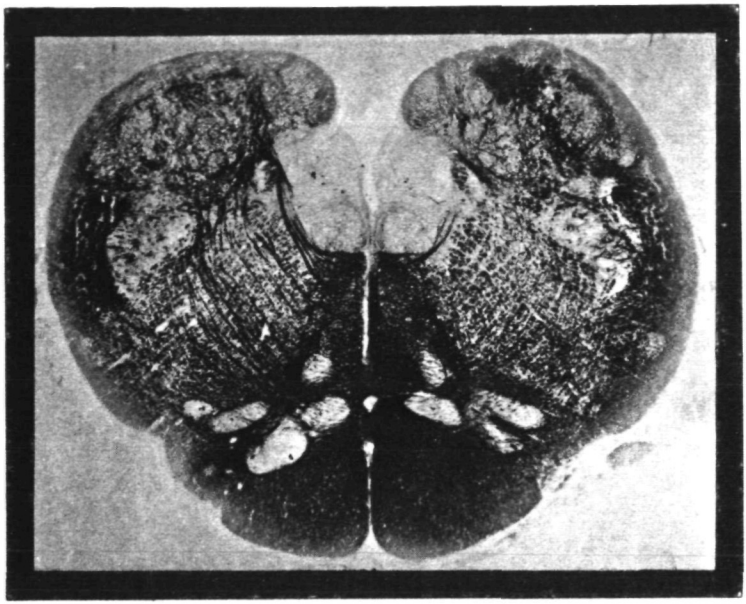
Gradually these symptoms appeared to pass off, leaving no contracture, and six months later it required a very careful examination to detect any loss of function in the animal. The animal became a pet and possessed great strength and cunning, apparently running and climbing like a normal animal; in only one respect did it show any abnormality in its mode of progression, and that was when placed in an open space it was invariably noticed that it made towards the left, the side upon which its field of vision was unimpaired. It died eventually from pneumonia three years after the operation.

The brain and cord were removed and hardened in Müller's fluid, and sections were cut after embedding in celloidin and then stained by the Weigert and carmine methods. Serial sections showed that I had injured the optic thalamus slightly, that I had cut through the inferior quadrigeminal body and the external geniculate body; but

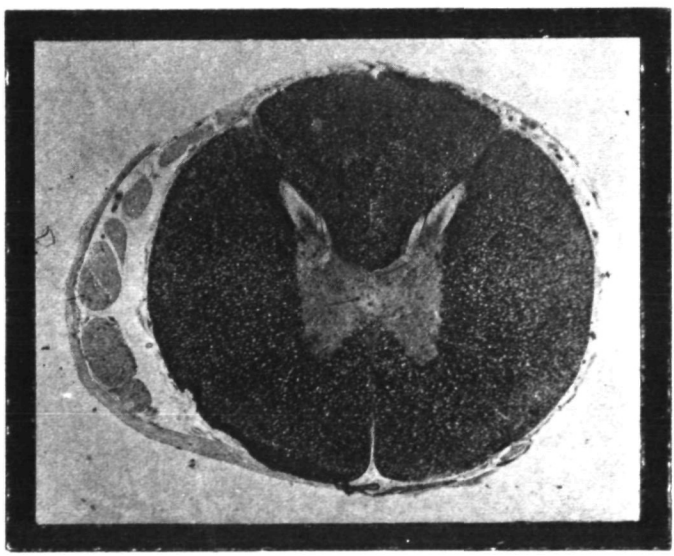
the most extensive destruction was in the lateral fillet just above the pons (*vide* photo-micrograph I.). There was injury of the three cerebellar peduncles and a considerable atrophy of the left lateral lobe of the cerebellum, in fact this was the only naked eye noticeable change on removing the brain.

The lesions were all limited to the left side, and there was but very little scar tissue. I think we may fairly attribute the nystagmus, rhythmical tremors and left hemiparesis to the injury of the cerebellar peduncles; the right hemianopsy to the injury of the external geniculate body; and the hemianæsthesia of the right side to the injury of the fillet and optic thalamus, especially the former. As there is absolutely no sign of degeneration in the crossed pyramidal tracts of the spinal cord, I cannot think that this system was *directly* injured, and we must explain the right hemiplegia by the injury to the fillet and functional disturbance in the motor fibres of the internal capsule, crus and pons in consequence of the lesion.

As the lesion was not a pure one we can only be certain of one or two facts as regards the degenerations that were found. They are—(1) Absence of degenerative sclerosis in the cord, showing that the pyramidal systems were not directly injured. (2) Atrophy of the fillet, especially of the median portion, and atrophy of the corpus trapezoides and inter-olivary layer on the *left side*, with some overgrowth of the glia tissue in these situations (*vide* photo-micrograph II.). (3) Atrophy of the internal arciform fibres and the cells of the posterior column nuclei of the *right side* (*vide* photo-micrograph III.). The fibres and cells are here less numerous and considerably smaller than on the left side. (4) Although Deiter's nucleus was injured I could detect no degeneration in the spinal cord (possibly the injury was insufficient to produce a degeneration extensive enough to be revealed by any other than the Marchi method), (*vide* photo-micrographs IV. and V.). (5) The extensive injuries to the peduncles of the cerebellum of the left side did not produce any evident sclerosis in the spinal cord (*vide* photo-micrograph VI.). The sections were shown at a meeting of the Physiological Society, held at King's College, February



III.—Section of the medulla, showing atrophy of the internal arciform fibres of the right side, and of the inter-olivary layer of the left.



VI. -Section of the spinal cord in the lower dorsal region. There is no apparent sclerosis in the pyramidal system.

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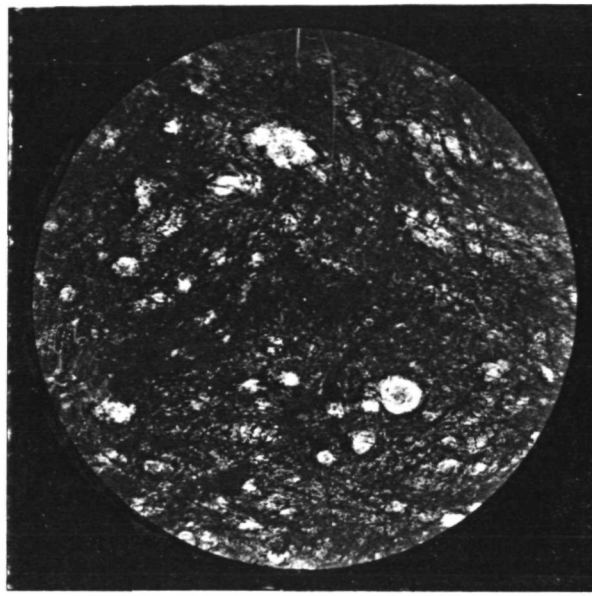
12th, 1898. The accompanying photo-micrographs illustrate the lesion and the descending degeneration and atrophy. Similar appearances of secondary atrophy of the fillet arising from porencephalon have been described in this journal by Alex. Bruce, also by Flechsig and Hösel, Mahaim and Déjérine. Flechsig and Hösel maintain that there is a direct connection between the posterior column nuclei and the central convolutions; that is, that *some* of the axons of the cells of the nuclei of Burdach and Goll pass uninterruptedly to the sensori motor cortex of the Rolandic region, forming what is termed the cortical fillet. Mahaim, Déjérine and Von Monakow maintain that there is not an uninterrupted connection between the posterior column nuclei and the cortex. (6) My observation on monkeys in which I destroyed the posterior column nuclei certainly supported the views of the latter authorities; for in five animals in which I produced most extensive degeneration of the fillet I could find no evidence of degeneration in the internal capsule or cortex. Moreover, the view that the fillet fibres end in the optic thalamus receives strong support from the observations made by Von Monakow, Langley and Grünbaum, Bielchowsky and Jacob, upon material obtained from animals in which extensive *cortical lesions* had been made. They found that the fillet does not degenerate, *but it may atrophy* in consequence of these cortical lesions. I am indebted to Dr. Langley for kindly allowing me to examine his specimens, and I could find no evidence of degeneration of the fillet, although the pyramidal system in the crus on the side of the lesion was entirely destroyed.

Jacob examined the brains of two dogs operated upon by Professor Goltz. In one there was complete interruption of the fillet in the sub-thalamic region, and a complete disappearance of the fillet fibres below the lesion. In the other the sub-thalamic region was not involved; but there was complete destruction of the cortex, capsule and putamen. In this case there was simple atrophy, but not complete degeneration and absence of fibres in the fillet. He concludes, therefore, that *some fibres do degenerate downwards*, and he puts forward the hypothesis that cells in the optic

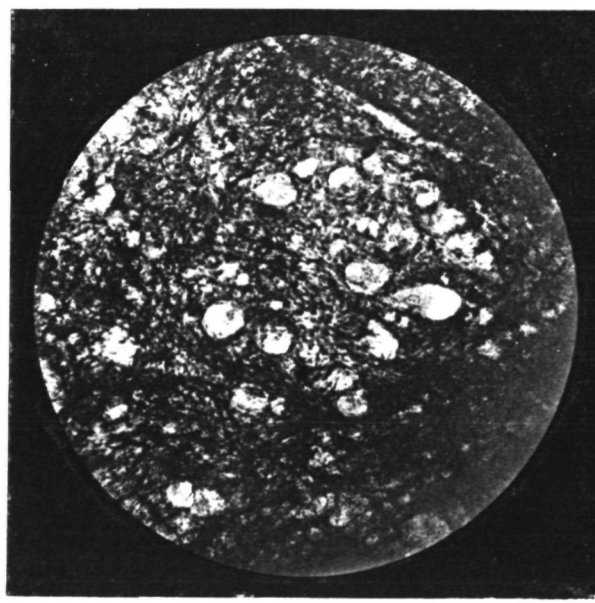
thalamus give off processes which divide, one branch going to the cortex and the other downward in the fillet. A lesion *below* the thalamus would, however, naturally produce a more marked effect upon the fillet than a lesion above, because if we assume that the fillet fibres all enter the thalamus to come into relation with thalamo-cortical neurons, these latter would have to atrophy and disappear first before the whole of the fillet fibres underwent atrophy.

As a result of the secondary degeneration of the fillet, Von Monakow and Spitzka observed that the arciform fibres of the nuclei of Goll and Burdach undergo a proportional atrophy, and Hösel, Henschen, Bruce and Jacob point out that the cells of these nuclei undergo a secondary atrophy proportional to the degeneration of the fillet. Déjérine believes that in cases of thalamic, subthalamic, or peduncular lesions, there is a retrograde cellulipetal atrophy extending from the periphery of the neuron towards its cell of origin. Bielchowsky examined the brains of two dogs in which Goltz had removed the central cortex and the corpus striatum. The fillet was intact; it showed no sign of descending degeneration. He concludes, with Mahaim and Monakow, that if the thalami are spared, ablation of a hemisphere does not lead in the dog to any secondary degeneration of the fillet. After very extensive cortical lesions involving the whole sensori motor area in monkeys, I have never observed any degeneration in the fillet, although the pyramid was stained black throughout by Marchi method. Redlich has, however, recently described scattered degenerated fibres in the fillet after extensive motor cortical lesions in cats.

The sections from the pons of this monkey, however, show an apparent excess of neuroglia tissue in the interolivary layer of the left side, more, I think, than can be accounted for by condensation due to atrophy of the nerve fibres, so that had I examined the brain by Marchi method, within six months of the operation, I might have found evidence of actual degeneration. The main change is undoubtedly one of *secondary disuse atrophy;* but Jacob's observations suggest that there may have been some true



IV.—Section of the posterior column nuclei to show cells in Burdach's nucleus on the side opposite the lesion. A portion of the section was photographed, which showed the largest cells. *Magnification* 200.



V.—Section of the posterior column nuclei, showing normal cells in Burdach's nucleus on the same side as the lesion. The cells are on the whole much larger than those in the atrophied nucleus. *Magnification* 200.

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descending degeneration in the fillet, as well as disuse atrophy.

As I injured the thalamus, the examination of the internal capsule would have afforded no positive evidence whether there is in the monkey a direct cortical fillet or not. Moreover, no method other than that of Marchi would have been suitable for showing a slight degeneration.

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