Winkler, C., On localised atrophy in the lateral geniculate body causing quadrantic hemianopsia of both the right lower fields of vision, in: KNAW, Proceedings, 15 II, 1912-1913, Amsterdam, 1913, pp. 840-850
3. Messrs. SMITS and DE LEEUW write: "Why in reference to these
experiments COHEN and GOLDSCHMIDT give 195° for the point of transition
in the "Chemisch Weekblad", and 170° in the "Zeitschrift für
physikal. Chemie" is quite unaccountable." The difficulty disappears
immediately when one refers to the said paper 2); it then appears
that the following sentence has escaped Messrs. SMITS and DE LEEUW's
notice. "Wir setzen hier vorläufig 170°, doch beabsichtigen wir auf
die genaue Bestimmung dieser Temperatur noch später zurückzu-
kommen. In der Figur steht irrtümlich 195°." 3)

I will refer again to the transition: tetragonal tin -> rhombic tin
as soon as the investigations announced in my above paper shall
be concluded.

Utrecht, November 1912.

van 't Hoff-Laboratory.

Physiology. — "On localised atrophy in the lateral geniculate body
causing quadratic hemianopsia of both the right lower fields
of vision". By Prof. C. WINKLER.

(Communicated in the meeting of November 30, 1912).

In 1904 BEWOR and COLLIER 1) observed blindness in the upper
quadrants of both the left fields of vision by an invalid, who after
death proved to be the bearer of a focus in the right hemisphere,
through which the surroundings of the calcarine fissure, from
the occipital pole to the confluence with the parieto-occipital fissure were
destroyed.

This observation is one of the few, in which quadratic-hemianop-
sia responded to a focus, which chiefly destroyed the cortex, although
the optic radiation, as shown in the drawings of BEVOR and COLLIER,
here too was not spared in the least, on the contrary it was de-
stroyed to an important extent (especially the medio-ventral part).

BEVOR and COLLIER pointed out, that already at that time in the
literature there was sufficient ground to suggest, that foci in the
dorsolateral division of the strata sagitthae of the occipital lobe can
cause blindness in the lower quadrants of the crossed optic fields.
On the other hand foci in the ventro-medial division of these strata

1) These Proc. XV, p. 677.
2) Chem. Weekblad 1, 487 (1904), special p. 449.
4) C. E. BEWOR AND JAMES COLLIER. A contribution to the study of the cortical
1904. XXVI p. 158.
Fig. 1. Lateral surface of the left hemisphere in quadrant-hemianopsia of both the right lower fields of vision.

Fig. 2. Medial surface of the same hemisphere. (The lines indicate the place of the sections, drawn with the same number on Plate II and III).

Fig. 11. Medial surface of the right hemisphere, with a defect in the proximal part of the G. cuneus, lingualis, fusiformis and occipito-temporalis. (The lines indicate the place of the sections, drawn with the same number on Plate II and III).

Fig. 17. Basal surface of the left hemisphere with a defect in the ventral occipital convolutions. (Cf. Psych. Neur. Elsdan. 1910 p. 1–10. Plate IV. fig. 1–6. Plate V. fig. 12).
C. WINKLER. "On localised atrophy in the lateral geniculate body."

NORMAL. Lateral geniculate body covered with its capsule by fibres, which enter the geniculo-cortical radiation through the area of Wernicke, penetrating the flimsy-capsular bundle and the retro-tectal cuneus capsulae, and pass into the striate sagittal cortex.

Fig. 4. Normal preparation.

Fig. 5. Quadrantanopia.

Quadrantanopia of both the right lower fields of vision. The laminated areas of the occipital cortex are marked in black. The entire area of the geniculate body is totally intact.

Fig. 6. Quadrantanopia.

Quadrantanopia of both the right lower fields of vision. The long area of the occipital cortex is marked in black. The entire area of the geniculate body is totally intact.

Fig. 7. Quadrantanopia.

Quadrantanopia of both the right lower fields of vision. The long area of the occipital cortex is marked in black. The entire area of the geniculate body is totally intact.
sagittalia can cause quadratic hemianopsia in the crossed upper fields of vision (HENSCHEN, FÖRSTER, WILBRAND etc.) 1)

VON MONAKOW 2) proceeds still more in the here taken direction. If the dorsal division of the occipital lobe (Upper Cuneus, $O_1-O_4$) incl. the dorsal part of the optic radiation is destroyed, then exclusively the dorsal layer of the lateral medullary capsule of the lateral geniculate body degenerates, and of this body the fronto-medial part.

On the contrary after destruction of the ventral convolution of the occipital lobe (ventral lip of the calcarine fissure, the Gyrus lingualis, the Gyrus occipito-temporalis) it gives rise to a secondary degeneration of the ventral division of the geniculo-cortical radiation and, degeneration of the ventro-lateral part (cauda) of the lateral geniculate body.

The projection of the retina on the cortex could no longer be interpreted as simple as HENSCHEN had taught us. It was not limited only to the surroundings, of the calcarine fissure and had to be regarded from a different point of view.

It had to be borne in mind that in each lateral geniculate body there was already a first field of projection for the two homonymous retinal halves. Another projection, secondary to this, took place through the geniculo-cortical radiation, which united this body with the cortex. But in a particular way.

As long as the dorsal division of the radiation and the caput of this body did not show secondary change, the vision in the lower crossed quadrants of the fields of vision was intact. (BEVOR and COLLIN).

As long as the ventral division of the radiation and the cauda of the body lacked these changes, the vision in the upper crossed quadrants of the fields of vision could remain intact.

The radiation from this body spreads itself however to a greater area of the cortex than to the surroundings of the calcarine fissure only. Without doubt also the upper Cuneus, $O_1-O_4$, i. e. the whole

La projection de la rétine sur la partie corticale calcarine. Sem. med. 1908.

WILSHAND'T' Hemianopsische Gesichtsfeldformen. Wiesbaden. 1890.

WILSHAND'T' and SÄNGDR. Neurologie des Auges. 3 Bde 1900-1904.


2) VON MONAKOW. Gehirnpathologie. 1905. S. 757.
of the occipital pole has to be taken in account as von Monakow desires, but most probably even more.

The retinal projection on the cortex, secondary to that of the lateral geniculate body is therefore without doubt much more complicated than Haukscher had figured to himself.

In 1909 I myself 1) could prove that the geniculo-cortical radiation and the geniculate body reacted differently, if by dorsally situated foci the dorso-lateral division of the strata sagittalia was cut through, than they did, if ventrally situated foci destroyed the ventro-medial division of these strata in the occipital pole. In the first case, with incomplete quadraantic hemianopsia of the lower fields of vision, the dorsal division of the radiation and the medial part of the geniculate body was greatly, but not altogether degenerated.

In the second case the degeneration took place in the ventral division of the radiation and the cauda of the body. Both degenerations were incomplete. At present I can communicate two new cases, this time of complete partial atrophy of the lateral geniculate body (cauda or caput), of which one with exquisite quadraantic hemianopsia, and through which I am obliged to extend even more than Monakow did, the areae of the cortex for the lateral geniculate body.

1)

Nephritis. Attack of unconsciousness on Dec. 9th 1910, followed by transitory sensory aphasia, alexia and permanent quadraantic hemianopsia in the lower right fields of vision, which in July 1911 is tested through the ophthalmologist. In January 1912 second insult, which causes death. Autopsy: Old haemorrhagic cyst in the Gyrus temporalis II and the Gyrus angularis, sectioning completely the dorsal optic radiations. Fresh bleeding immediately next to this in the dorsal strata sagittalia.

Miss C. P. S., 37 years, is the eldest of 9 children, of which 5 are still living. The mother of this family died 60 years old of apoplexy, the father 75 years old of nephritis. Mental or nervous diseases did not exist in the family.

Noabusus alcoholicus, no syphils. Before this present illness she had nothing to complain of.

On the 9th of December 1910, she all at once fell unconscious, remained unconscious for 10 days. After coming to, she spoke with much difficulty, she could not find the words, asked for "syrup" (zuur) when she meant "butter milk" (karmelk), etc.

She soon regained a certain quantity of words, although she did not understand everything alright, but even now (July 1911) she names with difficulty the objects, which she recognises well. Especially proper names and nouns she often uses in the wrong way. Moreover after the attack she could not read, partially, as she says, because she soon grew tired, partially because she did not understand much of what she read.

Lastly after the attack she had been paralysed on the right side, but the faintness had passed off completely after three weeks.

Afterwards she often had been giddy, in March, on the 4th of June and on the 15th of June; but this always happened at the beginning of the menses, which were very irregular after the attack. She noticed that after the attack she did not see very well to the right; it seemed as if white spots were there. The electric light on the market-place seemed to hang lower than formerly to her, and now and then it was, as if brown spiders hung in front of the right eye. Since the 9th of December she sees worse through the right eye. She also often complains of headache, vomiting at the same time. Moreover the urine contains 4½% albumen and many cylinders covered with epithelium of the kidneys.

On account of these complaints she was brought into my ward of the University Hospital (Binnen-Gasthuis).

The patient looks very ill. is a woman of middle height. Anaemic. Much arteriosclerosis. Somewhat enlarged heart. The second tone over the valvula aortae is loud. Pulse 90 - 120.

Her attitude is active, she takes interest in her surroundings, is well orientated in time and in space. She can walk and makes every movement.

Nowhere on the trune or extremities any trouble of motility or sensibility is to be found. Except a lowered abdominal reflex at the right side, all the reflexes of the extremities are within normal limits. No sign of Babinski. There are impediments in speech. She understands simple commands without an exception and follows them out. Her abundance of words is unlimited but she often mispronounces them. Most of the objects are well named; they are always well recognised. Now and then she has to think long over them and after all uses the wrong word for them.

She recognises every letter of the alphabet and pronounces them correctly. Also short words. She can read loud, but she reads paraphastically and the longer words are regularly badly reproduced. She does not comprehend the reading or only insufficiently. To comprehend the reading she repeats it several times loudly and then as a rule she does not understand it, she forgets many things. Yet she can do light work. She manages her little affair in pottery.

The smell is not affected.

The pupils are equally wide, the right one does not react on light as correctly as the left. She cannot converge and the reaction of the pupils by convergence is not to be seen.

The vision of the right eye is 1/6; of the left eye 1/2.

There is quadrantic hemianopsia in both the lower quadrants of the right fields of vision (s. figure).

Dr. Sarr, the ophthalmologist writes about the fundus oculi: "There is no trace of papillitis. On the right the borders of the papilla are clearly limited, but there have been bleedings and there is still some oedema of the retina (retinitis albuminurica). On the left the papilla is also clearly limited, but here too are rests of haemorrhages.

There is exquisite hemianopsia in the lower quadrants of the right fields of vision. That the macula vision is lost in the right anoptic sector is probably due to the bad vision of that eye.

The eye-movements, especially by their turning to the right and more so of the left eye, are limited. The left eye deviates to the temporal side. It is impossible to direct both eyes to one point.

- 8 -
The hearing has not been strongly disturbed, certainly not on one side only. A ticking watch can be heard on both sides at a distance of 1 Meter.

Field of vision on July 6th 1911.

The diagnosis was made of nephritis with retinitis albuminurica and a focus in the left Gyrus angularis, cutting through the dorsal strata sagittalia. July the 11th she left the hospital. On the 10th of January 1912 she was brought in unconscious and died three days later.

The account of the section shows: Hypertrophia cordis with nephritis interstitialis chronica and a focus in the left hemisphere, in the Gyrus temporalis II and the Gyrus angularis. The brown coloured focus spreads itself out in a straight direction along the distal third of the fissura t., and follows this along its ascending branch. The dorsal bounder of the Gyrus temporals II and the ventral Gyrus angularis are sunken in (s. fig. 1 and 2). On the section the focus proves to be a cyst with orange coloured walls, sectioning the strata sagittalia, in the neighborhood of the retro-lenticular internal capsule and sectioning them completely in more distal slides (fig 6 and 7). More distally, it soon retracts from the strata.

There is however a second fresh focus in the strata sagittalia, an haemorrhagy of bright colour, consisting of scarcely altered blood corpuscles (See fig. 7 in y).

In resuming the clinical data, it is not to be doubted that the second fresh focus caused the lethal ending insult on the 10th of January 1912 and that the first apoplectic cyst responds to the insult of the 9th of December 1910, which brought forth the quadratic hemianopsia as well as the secondary degenerations.

The importance of this observation lies in the first place in the fact, that a quadratic hemianopsia of both the right lower fields of vision, noted with all possible precaution, is caused by a focus cutting "completely" through the dorso-lateral division of the strata sagittalia. Therefore too the secondary degenerations are of great importance. They lasted for 13 months and
made alterations proximally in the lateral geniculate body and distally in the occipital lobe.

As the reproduction of the WEIGERT—PAL preparation 1) (fig. 6 and 7) and photo 1 and 2 show, the two foci are thus situated that the older cuts the dorsal division of the strata sagittalia over the whole width.

This focus — the important one of the two — reaches close up to the lateral geniculate body (fig. 6, pointed out by the first line through fig. 1 and 2) and stretches cutting through the strata sagittalia, along the dorsal boundary of the cornu inferius and posterius (fig. 7, pointed out by the first following line through fig. 1 and 2), where the fresh focus too is found. It ends about 2 c.m. proximally from the distal end of the cornu posterius. Nowhere the ventro-medial division of the strata is affected directly by the focus. In fig. 6 and in fig. 7, this is intact.

According to the destruction by the focus, totally different fibre-systems are affected and a massive degeneration towards the occipital pole takes place.

The degenerated mass of fibres has been drawn on a more distally situated section (s. fig. 8, line 8 through fig. 1 and 2) 1 c.m. distally from the focus 1). In this is visible, that the tapetum-fibres are very soon restored after their destruction, showing nearly a normal tapetum and forceps posterior round the very wide ventricle. In a less degree this is also the case with the stratum sagittale internum. It has fewer fibres than normal, and between them are spread degenerated fields in different spots. But the loss of fibres in the stratum sagittale externum is enormous. No normal fibres are to be found in it. This mighty black layer in WEIGERT—PAL preparations is here replaced by a white band, as well in the dorsolateral as in the ventro-medial division.

Smaller white stripes, coming from the degenerated band round the ventricle penetrate to far into the medullary cones of the convolutions, surrounding the calcarine fissure, also to the precentral and to the gyrus angularis. The gyri occipito-temporales and fusiformis have suffered least.

The massive degenerated ring round the ventricle is always found distally from the ventricular-end till the occipital pole. About 1/2 c.m. behind this end (s. fig. 9, line 9 from fig. 1 and 2) the distal point of the restored stratum sagittale internum is still touched and lies as a black island within the white degenerated mass of the stratum sagittale externum, while nearly all the medullary cones of the convolutions are degenerated and only fibres arcuatae seem to be left.

The precentral has suffered least. In the section, which falls about 1 c.m. from the occipital pole (s. fig. 10, last line through fig. 1 and 2) it is likewise. From the massive centre degenerated stripes penetrate in every convolution.

All this proves that perception in the upper fields of vision is still possible, notwithstanding the stratum sagittale externum in the occipital pole is missing. If therefore the fibres, used for visual perception are to be looked for in that layer, as seems probably to me,

1) All these figures have been drawn with the greatest care; they are enlarged 2 1/2 times and reduced to 7/18 of their size at the reproduction. Photos would have shown the same things, but drawings are more instructive as combinations of several sections are possible.
those which are spared here, do not at all belong to the occipital pole, but they must issue from far more proximal parts of the Gyrus occipito-temporalis.

This conclusion is the more valuable, if we look at the influence which the focus has had on the geniculo-cortical radiation and on the lateral geniculate body.

To make this clear I have drawn in fig. 4 a normal section of the surroundings of this body and in fig. 3 a cell-preparation 1 of the same, to make comparison possible.

In these figures one sees the lateral geniculate body, which shows on frontal sections the form of a shoe (s. fig. 3) and in which can be distinguished a dorso-medial part: the caput, and a latero ventral one: the cauda.

Within its own fibre-capsule covering the whole of it, (s. fig. 4) layers of fibres — laminae medullares — are alternately followed by layers of cells. The cells in the ventral layers are large, those in the dorsal ones much smaller, although, especially in the capital part large cells penetrate in these dorsal layers. The size of the dorsal cells differs a great deal between themselves. Many of them are very small.

In the normal fibre preparation the cauda contrasts but little against the caput, because the radiation of the optic tract has already begun in this proximal section. On the dorso-lateral side the lateral geniculate body is covered by the triangular area of Wernicke through which the geniculo-cortical radiation penetrates. In the dorsal part of this area (s. fig. 4) the fibre-direction is totally different from the transverse sectioned fibres of its ventral part.

A rather thick layer of very thin subependymal fibres surrounds the area of Wernicke against the ependym of the ventricle. As soon as the geniculo-cortical radiation has freed itself from this area, it opens its way in elegant curvings through the fronto-occipital bundle and the retro-lenticular division of the internal capsule to the stratum sagittale externum. So it seems at least, although nobody will dare to make a decided conclusion about the origin of these fibres, crossing here in all directions.

If we compare the above described area of the normal brain with an identical of our quadrant, hemianopsia, it then follows, (not to mention the degenerations in the fronto-occipital bundle, in the more proximally situated parts of the corona radiata, etc) that the dorsal layers of the geniculo-cortical radiation, and more in particular of the area of Wernicke, are totally degenerated. The ventral division of this fibre-area on the other hand, is not much injured, neither is the neighbouring dorsal and ventral part of the proper medullary capsule of the lateral geniculate body (s. fig. 6). In the cauda of the body we find intact laminae medullares. In the caput (in its dorso-medial part) the proper medullary capsule is dorsally and ventrally gone as well as the striae medullares. All the cells of this caput are (s. fig. 5) vanished, the dorsal as well as the large ventral ones. The layers in which they were situated are to be seen as thick layers of glia. The whole body

1) The cell-preparations of this body have been drawn with the camera of ZEISS; they are enlarged 20 times and reduced to 7/12 of their size by the reproduction. Idem with the retro-lenticular area.
is reduced to almost half its normal size\(^1\), but in its cauda the small dorsal
and the large ventral cells (s. fig. 6) are completely intact; there too the striae
medullares as well as the proper capsule are on the whole untouched.

The conclusion is readily made: the possibility of sight in the
upper quadrants is due to the conservation of the cells and fibres
in the cauda of the lateral geniculate body, their projection on the
cortex being preserved by the ventral layer of the area of Wernicke
and of the geniculo-cortical radiation.

But where do these cells find their projection on the cortex? Not
in the occipital pole which in my opinion was totally separated
by the focus from the lateral geniculate body, as is shown by the
complete degeneration of the stratum sagittale externum and all the
medullary cones of the occipital convolutions (only fibrae arcuatae
remained). Perhaps from the gyrus occipito-temporalis, its medulla
being but partly cut through by the focus (s. fig. 7). Distally from
it (s. fig. 9) the medullary cones of the temporal circulations
were normal, those of the occipital lobe (s. fig. 9) were degenerated.
Proximally from it this convolution with normal medullary cone
contributed to the forming of the intact ventral division of the strata
sagittalia.

The answer to the question where the field of projection of
the lateral cells of this body was situated, was brought to me by a very
remarkable right hemisphere, given to me by Professor Bolk. He
had found it by accident in the corpse of a woman of whose ante­
cedents nothing was known.

II.

This right hemisphere carries the rest of a very old pathological process, which
has reduced on the transition of the basal temporal and occipital lobe all the
corvolution with their medullary cones to a thin membrane. When the pia mater
was removed it was torn near the cuneus. (s. fig. 11). The occipital pole is intact
On the middle of the cuneus the defect begins with a sharp edge. The proximal
end of the cuneus, of the gyrus lingualis and of the gyrus fusiformis, as well
as the medial part of the gyrus occipito temporalis (as far as near to the f. rhinæa)
are replaced by a thin membrane (s. fig. 11, 13, 14, 15 and 16).

The series of sections show the following\(^2\). The first remarkable alteration
is drawn in fig. 16 (pointed out by the line 16 on fig. 11 and comparable with
fig. 9 of the first observation). Twice the distal end of the defect has been cut.
Firstly in A in the depth of the fiss. calcarina. There the cortex is gone and the

\(^1\) The enlargement is similar to that of the normal figure. (s. fig. 3).

\(^2\) In order to give an easy survey the sections are reversed and drawn as if
they came from a left hemisphere.
The medulla of the cuneus lies uncovered. The line of Gennari ends on both sides sharply against the defect, is not atrophied, even mightier than usual and formed by thicker fibres; secondly in B, where ventrally from the f. parieto-occipitalis the medulla of the cuneus lies uncovered and in C, where the defect begins in the gyrus fusiformis.

In the white matter opposite the fissura calcarina a triangular degenerated field is to be seen. It is situated for the greater part ventrally, but also a bit laterally round the sectioned distal end of the strata sagittali.

In figure 15 (pointed out by line 15 of fig. 11 and comparable to fig. 8 of the first observation, the defect is found distally from the confluence of the fiss. calcarina and f. parieto occipitalis. All the basal convolutions are missing.

Cuneus, lingualis, fusiformis, as well as the medial border of the ventricle are entirely gone. The medial medullary cone of the g. occipito temporalis lies uncovered. The degenerated field is larger, lies partly in the ventral, partly already in the latero-dorsal division of the stratum sagittale internum, but also in the stratum sagittale externum. Especially there where the ventral division of it passes into the lateral. For the rest the stratum sagittale externum is seen quite distinctly here (in fig. 8 totally gone), a proof that this area consists of more fibres than the geniculo-cortical radiation only (all gone in fig. 8).

In fig. 14 (pointed out by line 14 of fig. 11 and comparable to fig. 7) the splenium corporis callosi is sectioned.

Except a rest of the Cornu Ammonis no convolutions are to be found ventrally from the cornu inferius. The greater part of the gyrus occipito-temporalis is gone. The intact ventral strata sagittali, as were found in fig. 7 are missing. The degenerated field (due to the defect) lies laterally and dorsally from the ventricle in both the strata sagittali.

A great part of the dorsal stratum sagittale externum is intact. In fig. 7 exactly this large layer was totally destroyed and therefore also the geniculo-cortical radiation to the occipital lobe.

In fig. 13 (pointed out by the lines 13 of fig. 11, comparable to fig. 6) the retro-lenticular area is sectioned.

As if this section were the negative of that reproduced in fig. 6, one hardly finds here normal fibres in fields, which were there the best preserved. In the ventral part of the geniculo-cortical radiation and of the area of Wernicke all the fibres are gone. The ventral and lateral part of the proper capsule of the lateral geniculate body scarcely consist of normal fibres, the striae medullares in the cauda are gone, and the body is reduced to half its normal size.

On the other hand the dorsal part of the geniculo cortical radiation and the area of Wernicke, the dorso-medial proper capsule and the striae medullares in the caput of the geniculate body are only relatively changed.

The same reverse is shown in the cell-preparations of the body itself. Latero-ventral, in the cauda of the body not one cell is to be found.

Thick layers of neuroglia, where once the cells were alternate with less thick layers of neuroglia (now representing the striae), but all cells, the dorsal as well as the ventral, have disappeared. On the other hand, the dorso-medial part, the caput of this ganglion contains well ranged cell layers, small dorsal ones as

1) Here, as well as before, purposely I do not point out several other degenerations. To make things still less complicated I do not even mention the influence upon the pulvinar of both these foci.
well as a number of ventral large cells. This geniculate body is in every respect the negative of fig. 5.

The result of this observation is clear enough: The important defect in the occipital lobe above mentioned, was not sufficient to produce an atrophy of the dorso-medial division of the lateral geniculate body. The cauda on the other hand lost all the cells and fibres. From our first observation we learned that the cauda remained uninjured, when the focus (s. fig. 6 and fig. 7) totally destroyed the dorsal layer of the strata sagittalia. There (according to the spot of degeneration in our second observation in fig. 14) the geniculo-cortical radiation from the ventral occipital convolutions is already situated dorsally from the cornu inferius.

Moreover on the same sections in our first observation the ventral strata sagittalia are intact, and exactly these are completely missing in the second (s. fig. 13). New was to me the exquisite total loss of all the cells and fibres, either in the lateral, either in the medial half of the geniculate body, as is found in both these observations, although I possess many other partial atrophies of it after occipital-lesions.

Generally spoken, lesions of the medio-ventral occipital convolutions cause atrophy of the latero-ventral part of the geniculate body, but in my cases it has never been a total one.

As long as the gyrus occipito-temporalis proximally from the calcarine fissure is uninjured, not all the laterally situated fibres disappear, but cells often remain in the ventral, occasionally also in the dorsal layers. 1) Only after the knowledge of such extremes as above described, I have learned to appreciate the incomplete atrophies. Wedges turning their base to the dorsal part of the geniculate body, fall out. Their localisation differs by the place of the focus, although they never touch the dorso-medial part of it, as long as the focus only destroys the ventro-medial occipital convolutions.

In this way e. g. must be considered the ventral occipital foci with atrophy in the cauda of the lateral geniculate body, described by myself in 1910. At present I complete this observation referring to the same figures in order to describe that geniculate body exactly.

III.

A basal defect in the left hemisphere (s. fig. 17, also Psych. and Neurol. Bladen 1910. p. 16 more precisely the photos on plate IV and fig. 12 on plate V) elimi-

1) Nearly the same can be said of dorsally situated foci (mutatis mutandis) which section the optic radiation either close to the geniculate body or further off. I shall refer to this later on.
nates the O, the gyrus lingualis and fusiformis to the confluence of the calcarine fissure with the parieto-occipital fissure (s. Psych Bladen Pl. IV, fig. 6). Also a part of the gyrus occipito-temporalis, lying more proximally, is injured.

Through this lesion the ventral division of the geniculo-cortical radiation as well as that of the area of VRECKEC is degenerated, but in less degree its most ventral layer (c.f. Ps. Bladen, Pl. V, fig. 12).

The geniculate body belonging to this is drawn in fig. 18. It is smaller than normal, but not as far reduced as in both the former observations. The proper capsule is not changed dorso-medially and the same can be said of its cells, dorsal as well as the ventral ones, belonging to the caput of the ganglion.

The cauda is for the greater part atrophied but not the most laterally situated division of it. There, ventral and dorsal cells are to be seen within an almost normal capsule. Between caput and cauda, not or only little changed, one finds in the middle a part, where all is destroyed; the dorsal and ventral cells, the spheric medullaries, the proper fibres and the proper capsule.

In this case an example is shown of an incomplete atrophy of the cauda of the lateral geniculate body, incomplete because the focus did destroy the ventral occipital convolutions, but had not touched the gyrus occipito-temporalis far enough proximally. Therefore the most ventral layers of the geniculo-cortical radiation and the most lateral parts of the cauda remained free from degenerative atrophy.

Recapitulating I come to the following conclusions:

1. Vision in the upper quadrants of the field of vision is possible, notwithstanding the total loss of all the cells and fibres in the medial (caput) division of the crossed lateral geniculate body, as long as the cells and fibres of the cauda (origin of the ventral geniculo-cortical radiation) are intact.

2. It is not sufficient that the ventral occipital convolutions are destroyed to make all the cells disappear out of the lateral (cauda) division of the geniculate body. This only occurs when more proximally situated parts of the gyrus occipito-temporalis are destroyed.

3. The cortical areas belonging to the lateral geniculate body are not only limited to the cortex of the occipital lobe.

Chemistry. — “On the occurrence of metals in the liver”. By Prof. L. van Itallie and Dr. J. J. van Eck. (Communicated by Prof. Einthoven). (Communicated in the meeting of November 30, 1912).

In the analysis of organs as to the presence of metallic poisons, we found in the liquid obtained after destruction of 170 grams of liver, kidney and heart, in addition to traces of arsenic and copper, as much zinc as corresponds with 80 mgs. of zinc oxide per kilogram of organs. As there was no reason to suppose that a poisoning