Adolf Meyer’s insights into the neuroanatomy of the optic radiation play an important role in understanding the development of visual field deficits after temporal lobe resection. He studied medicine in Zurich, where his interest in neuroanatomy was influenced by his teachers, Constantin von Monakow and Auguste-Henri Forel. After graduation in 1890, he studied in France, Scotland and England for 2 years. Here he came into contact with well-known neurologists, including Jules Dejerine and John Hughlings Jackson, and the neurosurgeon Victor Horsley. He enriched his anatomical knowledge with a doctoral thesis on the structure of the forebrain of reptiles. In 1892, he emigrated to the USA and started teaching neuroanatomy and pathology. Particularly in the 19th century, these disciplines played a significant role in psychiatry that was still closely connected to neurology. The pinnacle of his career was attained in 1910 with his appointment as director of the psychiatric clinic at Johns Hopkins Hospital. This historical essay aims to depict the neuroanatomist behind the well-known psychiatrist Meyer, and especially the less known historical context in which he described, in 1907, that part of the optic radiation, which, coming from the lateral geniculate body, bulges ventrally to the temporal pole, and then bends posteriorly, along and beneath the temporal horn to the calcarine cortex, known today as ‘Meyer’s loop’.

One of the most frequently reported complications of temporal lobe surgery is a visual field defect due to optic radiation injury, in particular the part that became eponymously associated with the name of Meyer. Although Adolf Meyer’s role in American psychiatry is well known and described by several authors, here we discuss his neuroanatomical work, in particular his description of the temporal loop of the optic radiation. Following a short biographical sketch, in which his education by Swiss neuroanatomists and European neurologists is emphasized, using the German language report of his postgraduate peregrination, we discuss the circumstances in which he published his major article on the visual pathways, in a period when he was director of the New York State Pathological Institute. This is followed by a description of his interaction with Harvey Cushing after Meyer’s appointment as first Chief of Psychiatry at Johns Hopkins Hospital, where he set up the first academic department of Psychiatry in North America and subsequent publications on visual field defects in temporal lobe surgery.
Adolf Meyer (Fig. 1), principle architect of American psychiatry, was born on 13 September 1866 in Niederweningen, a suburb of Zurich, Switzerland. He grew up in a harmonious family, free of religious, philosophical and scientific dogmas. After completion of the Gymnasium, he doubted between the studies of medicine (he had helped his mother’s brother in a rural medicine practice) and theology (his father was a Zwinglian minister). Finally, he decided to study medicine in Zurich and graduated in 1890. From 1890 to 1892 he made a neurological peregrination to Paris, Edinburgh and London, and wrote an extensive report of his experiences. He came into contact with well-known neurologists, including Jean-Martin Charcot, Joseph Jules Dejerine and John Hughlings Jackson.

A most interesting part of the report of his peregrination is on meeting Horsley.

‘The most news was given to me by Victor Horsley. In the past, when I read the communications on his experimental work or on his operations on the human brain, I had imagined him as an elderly surgeon and found a most friendly man in his early thirties, who deserves his fame in all measures. … He was so kind to invite me for several of his operations.’

Meyer mentioned a tumour in the motor centre, a case of focal epilepsy by depression of bone splinters and another case of focal epilepsy without a distinct sign with respect to the nature of the lesion. He described the latter case more extensively. It was a 41-year-old female with ~50 daily left-sided seizures, starting on the left side of the mouth, radiating to the arm and the leg, ‘so the only thing one knew was that it originated from the right facial region’. A complete surgery report follows, mentioning the use of an electric motor to drive the circular saw and electric (otolaryngologist) lamp on the forehead. After opening the dura mater, the pia mater was only slightly injected in the facial region. Following tying of some blood vessels he made an extensive incision. ‘Except for a somewhat suspicious place the size of a pea nothing special was observed.’ To give the patient a chance of cure, he removed another part of the facial gyrus after binding the vessels. The wound was then closed. At that moment the patient had a seizure, starting in the arm, during which the face remained unaffected. The first day she had a few more seizures and then none thereafter. He later received a letter from Horsley stating that the patient had no more seizures (Meyer, 1891a, b, c).

During that period, Meyer had a special interest in anatomy and neurology, which resulted in a doctoral thesis, supervised by Auguste Henri Forel, about the structure of the forebrain of reptiles. Attracted by what he learned from American visitors and others, he decided to go to Chicago in 1892, at the age of 26. He obtained a teaching commitment in neurology and worked as pathologist at the Illinois Eastern State Hospital in Kankakee. In 1895, he moved to Worcester State Hospital (Massachusetts), where he was appointed pathologist and, later, in addition, clinical director. He continued to publish in the area of neurology, but also in psychiatry, as can be learned from the titles in his bibliography. He began to concentrate on and intensify his attention to psychiatry and classification of his own clinical cases. In 1902 he was appointed director of the Pathological Institute of the New York State Hospitals for the Insane. Besides his clinical and administrative tasks, he continued brain anatomy studies and was particularly interested in white matter brain pathways in patients with aphasia. In the same year, he married Mary P. Brooks, who tried to help many psychiatric patients in their home environment and so became the prototype of the psychiatric social worker. In 1904, he was appointed Professor of Psychiatry at Cornell University Medical College (New York). In 1905, William Osler, in his farewell address to Johns Hopkins Hospital, described the desolate state of psychiatry in the USA, compared to the situation in Europe. Henry Phipps, a rich steel magnate, donated $500 000 and the first American university clinic for psychiatry was built. The position of chairman was offered to Adolf Meyer, after which he moved from Cornell University to Baltimore to be appointed director of the psychiatric clinic of Johns Hopkins Hospital in 1910, his final academic working environment up to 1937, when, at 70 years old, he retired. In the meantime he built up his clinical and scientific department and as an ‘accredited representative of American psychiatry’ was recognized for his knowledge and advice by governmental and educational agencies as well as many universities (Lamb, 2012). In those days, Adolf Meyer was the most influential and prominent psychiatrist in America. He continued his work...
in the anatomy laboratory and organized anatomical courses for medical students. His anatomical papers on neurodegeneration, apraxia and aphasia and the anatomical trajectory of the optic radiation, central theme of this manuscript, are of special value. With respect to the latter subject, it is of interest to note that in 1893, he wrote a series of short articles on ‘Neurological Work at Zurich’ in the *Journal of Comparative Neurology* (Meyer, 1893), the third of which was on Constantin von Monakow and his work on the neural connections between the eye, the lateral geniculate body and the occipital cortex, applying Wilhelm von Gudden’s degeneration method.

Both Monakow, founder of the diachisis theory, and Forel, co-founder of the neuron theory, and alumni from the University of Zurich, Switzerland, had inspired him and most probably aroused his early interest in neuroanatomy.

### Origin of the eponym

**‘Meyer’s loop’**

In 1907 Meyer published his first work on ‘the peculiar detour of the ventral portion of the geniculocalcarine path’, nowadays also known as the ‘Meyer’s loop’ of the optic radiation in the temporal lobe. In that manuscript, he pointed out that the calcarine cortex ‘is a very specific structure’ recognized and described by the German neuroanatomist Korbinian Brodmann, and that the ventral part of the geniculocalcarine tract ‘plunges first forward from the external geniculate body into the temporal lobe’, becomes part of the inferior longitudinal fasciculus and then curves backward to finally end ‘in the anterior part of the lower lip of the calcarine cortex’. He clearly stated that there exists a somatotopic organization in the optic radiation fibre tracts and the calcarine cortex: from Meyer’s own observations of case histories (Meyer, 1907), combined with the theories of his teacher von Monakow (and his Swedish colleague Salomon Henschens with whom Monakow had a dispute) about the organization of different connections in the brain, he concluded that the dorsal tracts have an upper retinal distribution and correspond with field defects in the lower quadrants (blindness of the upper retina quadrant) and that the ventral tracts (Meyer’s loop) have a lower retinal distribution and therefore correspond with field defects in the contralateral upper quadrants (blindness of the lower retina quadrant). This signifies that the disposition of the fibres in the optic radiation is practically the same as in the optic nerve. Central (macular) vision will, in most cases with a lesion lateral and posterior to the lateral geniculate body, be spared as the macular fibres are represented in the most mesial part of the optic radiation bundles in the parietal and high-dorsal temporal lobes and most of the lateral temporal lobe lesions will thus not affect them. Another key finding, which was published by Cushing 4 years later (Cushing and Heuer, 1911), was that ‘the field defect in the eye on the same side as the lesion is apt to be in advance of the other’, which could not be confirmed in the recent study by van Lanen et al. (2018). In that study, our findings suggested the contrary, namely a more anterior location of the contralateral eye fibres in Meyer’s loop compared to the ipsilateral fibres, making the contralateral eye more susceptible to injury by anterior temporal lobectomy (ATL), resulting in more extensive visual field deficits.

In 1910, when he started in Baltimore, Meyer was consulted at the neurosurgical ward of Cushing (Fig. 2) and saw a patient with a gunshot wound through the left eye and inferior temporal lobe. After the house officer concluded that the perimetry was normal, Meyer advised to perform a perimetry with <30° intervals after which a contralateral quadrant anopia was diagnosed in the right eye (Cushing and Heuer, 1911) (Fig. 3). This Meyer-Cushing encounter was a pivotal moment for the discovery of and further elaboration on the anatomical course of the optic radiation, the interpretation of visual field defects and the consequent anatomical localization of the lesion, which was crucial in an epoch without sophisticated neuroimaging. Subsequently Cushing concluded that Meyer’s 1907 publication received a clear lack of recognition:

‘Meyers’s own observations were not supported or confirmed by perimetric tests, which were difficult or impossible in the clinical material of the type he had at his command. Nevertheless, he shrewdly surmised in connection with the few reported cases of quadrant hemianopia in which small defects had been found post mortem that the dorsal bundles have an upper retinal distribution and hence correspond with field defects in the lower quadrants, whereas per contra involvement of the ventral bundles is responsible for defects of the upper field quadrants’.

In Cushing’s paper, from which this citation was taken, he studied visual field deficits produced by temporal lobe lesions (Cushing, 1922). He described 10 clinical cases with temporal lobe tumours and summarized that the primary most important localizing symptom of these tumours are the so-called ‘uncinate fits’, nowadays classified as focal epileptic seizures with sensory (olfactory) symptoms. In the second place he distinguished both negative (visual field defects) and positive (visual hallucinations) ophthalmological symptoms. He concluded that ‘perimetry is of paramount diagnostic value’, i.e. for the anatomical lateralization and localization of the pathological lesion, ‘particularly in the early recognition of temporal lobe tumours’, and that ‘partial or complete homonymous field defects’, i.e. quadrant anopia/hemianopia and hemiachromatopsia, were ‘especially characteristic of involvement of the temporal lobe optic radiation’. The paper is accompanied by ‘superb diagrams’ made by the well-known medical illustrator Max Brödel, after ‘a glass-model reconstruction of these fibres … made by Dr Meyer. … They render superfluous any further written description’ (Cushing, 1922).

The next influential paper on the architecture of the optic radiation was by Van Buren and Baldwin (1958). They
studied 41 patients, who underwent temporal lobectomy for chronic epilepsy, which is definitively another category of patients compared to the tumour patients described by Cushing. One of the most evident differences is the probability that in the epilepsy group the pre-resection distortion of the optic radiation is near zero, whereas the distortion in the tumour group will be more impressive, because of mass effect by the tumour itself. The authors made five clear statements, notably (i) ‘that the radiation does not cap the temporal horn’; and (ii) ‘that the most anterior loop of the radiation in the temporal lobe represents the retina on either side of the vertical meridian’. Besides this, they stated that (iii) ‘the representation of the retina on either side of the vertical meridian lies farthest forward in the radiation’; (iv) ‘a lesion from the lateral aspect of the radiation would injure the ipsilateral retinal fibres more than the contralateral’; and finally (v) ‘a lesion from the anterior aspect of the radiation would injure ipsi- and contralateral retinal fibres to an equal degree resulting in congruous defects’. This would signify that after an anterior temporal lobectomy for epilepsy, in which case the optic radiation will be injured from the lateral side, the quadrantanopsia will begin in the vertical upper part of the quadrant and can be incongruently, usually larger on the side of the surgery. The start of the defects in the vertical upper part was confirmed by the recent study of van Lanen et al. (2018), but again there is controversy about the side where the defect will be the largest. In the same year, Murray Falconer (1958) published on visual field changes after temporal lobectomy in 50 consecutive epilepsy patients. The discussion about (in)congruency of the
visual defects continued in that they always found congruent contralateral, mostly upper quadrant defects immediately adjacent to the vertical meridian and they rejected the older theories of incongruent defects caused by pressure on the optic tract or pressure on the anterior choroidal artery. Furthermore, they concluded that all temporal lobe resections with a dorsal resection border of 8 cm measured from the most anterior part of the temporal pole along the superior temporal gyrus and parallel to the sylvian fissure can have non-significant differences in the range of extent of quadrantanopia, but the chances of hemianopia greater than a quadrant increase with resections of 8 cm and more.

Relevance of Meyer’s loop in temporal lobe surgery

Most frequent indications to perform elective temporal lobe surgery are chronic drug-resistant epilepsy or tumours sometimes leading to visual field defects due to optic radiation injury. In many patients with brain tumours or trauma there is diffuse tissue compression and injury, with consequent white matter shifts. At present, new preoperative MRI techniques are available to compose a realistic fibre tract image between key anatomical structures, such as the lateral geniculate body and the calcarine cortex, and to counsel the patient preoperatively about possible complications.

In most patients selected for resective temporal lobe epilepsy surgery, the ‘normal’ grey and white matter anatomy is preserved without distortions of white matter tracts by tumour and oedema. Therefore, to investigate visual field deficits in relation to temporal lobe surgery, patients indicated for epilepsy surgery are the most suitable group. Epilepsy surgery for temporal lobe epilepsy has been found particularly effective with reported seizure freedom rates of 70–80%. Temporal lobe surgery encompasses ATL with or without amygdalohippocampectomy or tailored variants. Complications related to ATL include, among others, visual field deficits, cognitive complaints, other neurological deficits, and infections. The most common reported visual field deficit is a contralateral homonymous partial superior quadrantanopia, clinically often referred to as a ‘pie in the sky’. The reported incidence of postoperative visual field deficits ranges widely from 15% up to 90% (Barton et al., 2005). This deficit is caused by damage to the anterior part of the optic radiation extending from the lateral geniculate body of the thalamus in the anterior part of the temporal lobe, bending around the tip of the temporal horn and then passing backwards, first lateral, and then beneath the ventricle on its way to the calcarine cortex. This anterior bending of the optic radiation in the temporal lobe is also known as ‘Meyer’s loop’ (Meyer, 1891a, b, c; Yogarajah et al., 2009). The distance between the most anterior part of Meyer’s loop and the temporal pole varies widely between individuals (Barton et al., 2005; Yogarajah et al., 2009). Recent reports have estimated a temporal pole to Meyer’s loop distance variation of 22–44 mm (Barton et al., 2005; Yogarajah et al., 2009). As temporal lobe resections for epilepsy surgery may extend up to 90 mm, Meyer’s loop can easily be injured. Nevertheless, there is controversy on whether the size of resection correlates with the size of visual field deficit. Recently, we published a method to quantify the postoperative visual field deficit after temporal lobe surgery and proposed a new quantitative scoring method independent of the perimetry procedure. Using this method, we assessed the relation between the length of temporal lobe resection and postsurgical visual field deficit and compared differences between right- and left-sided surgery (van Lanen et al., 2018). In concordance with most studies, we found a significant correlation between resection length (anterior-posterior resection minus anterior temporal-occipital pole distance; AP-ATOP distance) and visual field deficit, although only for right-sided surgery. The suggestion of a more anterior located Meyer’s loop in the left hemisphere might explain why we did not find this relation for left-sided ATL. As a consequence, a smaller resection length in the left hemisphere causes relatively more damage to Meyer’s loop, resulting in a larger visual field deficit, while on the right side there is a more gradual increase in visual field deficit with resection size.

In conclusion, Meyer’s contribution to the anatomy has been only sketchily discussed, and the lasting value of this anatomical work is sometimes questioned. The aim of this paper was not to detail or summarize Meyer’s achievements in psychiatry, but to emphasize the importance of his description of the peculiar anatomical course of the optic radiation in the temporal lobe as recognized in particular by Cushing. Although his anatomical work has received limited attention, there is no doubt that it attained a profound significance in current neurosurgical practice.

Competing interests

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