Anatomical and physiological observations on lesions in the cerebellar nuclei in Macacus rhesus (preliminary report). By Ernest Sachs MD and Edgar F. Fincher, Jr MD, Fellow in Neurological Surgery, St Louis, Missouri, USA (Brain 1927; 50: 350–356); with Symptomatology of a group of frontal lobe lesions. By Ernest Sachs MD, St Louis, Missouri, USA (Brain 1927; 50: 474–479); with The subpial resection of the cortex in the treatment of Jacksonian epilepsy (Horsley operation) with observations on areas 4 and 6. By Ernest Sachs MD, St Louis, Missouri, USA (Brain 1935; 58: 492–503); with Observations on the pathways transmitting the sensation of taste. By Henry G Schwartz (St Louis) and Graham Weddell (London). From the Neurosurgical Service of Barnes Hospital and the Washington University School of Medicine, St Louis, Missouri, USA (Brain 1938: 61: 99–115)

It turns out that despite writing an article now regarded as a classic in the history of neuroscience (listed as Garrison and Morton #4879.1), Robert Clarke (1850–1926) and (Sir) Victor Horsley (1857–1916)—sportsman and raconteur of ghost stories; and pioneer of neurological surgery, politician and patron of the National Temperance League, respectively, but with a shared interest in gadgets—never finished their work (‘The structure and functions of the cerebellum examined by a new method’, Brain 1908; 31: 45–124; and see Brain 2007; 130: 1449–52). In April 1924, Ernest Sachs (1879–1958) receives a letter to this effect from Dr Clarke asking Sachs to reinvestigate connections of the cerebellar nuclei using the original stereotactic instrument. Starting work in the autumn of 1926, Sachs now has sufficient material to publish some further, but preliminary, details on 12 of 28 monkeys in whom ‘satisfactory lesions’ were established. The controversy is Horsley and Clarke’s claim that all cerebellar fibres only go to thepons and medulla—a conclusion that André Thomas (1867–1963) has contested, arguing that there is also projection to the spinal cord via the restiform body. Dr Edgar F(ranklin) Fincher Jr (1900–69), Fellow in Neurological Surgery, makes the lesions, and the histological material is prepared by Mrs J. L. Ford (nd). Their experiments involve stimulation and lesioning of the dentate and emboliform nuclei, the nucleus fastigius and globosus, and some areas in between these structures. In agreement with Horsley and Clarke, stimulating the dentate produces bilateral movements of all four limbs and head deviation to the opposite side; although consistent with Dr Sachs’s observations in clinical practice, the eyes move conjugately and the pupils constrict with activity of the median cerebellar nuclei fastigius and globosus (Fig. 1). The authors find that the dentate nuclei project through the superior cerebellar peduncle to cross the midline and reach thered nucleus and, perhaps, also the region of the third nerve nu-
cleus. Based on patterns of degeneration, the cerebellar cortex has
the same connections. ‘Our sections, we believe, show beyond all
question that the original contention of Horsley and Clarke, that
cellar fibres only go to the (ponsine and medullary) nuclei,
is not correct’. And that is all that delegates attending the sym-
posium on the cerebellum at the joint meeting of the Section of
Neurology of the Royal Society of Medicine and the American
Neurological Association held in London on 26–28 July, 1927,
learn concerning this particular topic.

Next day, Dr Sachs describes his experience of symptoms attrib-
utable to frontal lobe lesions. He wishes to draw attention to two
aspects that he has found reliable in identifying these quintessen-
tial reticent brain lesions. His experience is based on 25 cases: 15
with left-sided lesions and 10 on the right representing, between
them, ‘ten gliomata, seven endotheliomas, two gummas, two
abcesses, one arachnoid cyst, one chronic inflammatory mass
around a foreign body, one basal cell carcinoma, and one degen-
eration of the frontal lobe’. Six patients have died, of whom one
was not operated on. As for the classical symptoms, emotional
changes are seen in a minority; and impulsive laughter and
crying—‘which, from the literature, one is led to believe is such
a common occurrence’—is noted in only five cases. The ‘eye-
grounds’ (fundal appearances) often show bilateral choked disc
but with no example of the features emphasized by Foster
Kennedy [(1884–1952): ‘unilateral atrophy and contralateral swel-
limg’]; only when searched for carefully by perimetry and when the
tumour encroaches on the temporal lobe, are field defects as
described by Harvey Cushing (1869–1939) apparent. Unilateral
tremor, ipsilateral or contralateral, occurs in five cases. The ab-
dominal reflexes are altered, on one side or the other, in seven
cases, with brisk tendon reflexes in seven and ‘an Oppenheim or
Babinski’ in nine others. That the reflexes are often normal reflects
seeing the cases ‘at a time when not enough intracranial pressure
had developed to involve the pyramidal tract’. Smell is usually
normal, anosmia being a symptom in only two instances.
Different varieties of aphasia occur in the patients with left-sided
lesions, rarely with those on the right. ‘X-ray findings’ including
ventriculography are rarely helpful in localizing the lesion, other
than in one case who had undergone two explorations of the
posterior fossa; and, even then, establishing the site of the
lesion depended more on clinical examination than asymmetric indenting of the lateral ventricle.

As these classical features are present in only half of all cases, Dr Sachs has found it preferable to rely for diagnosis on two less well-recognized symptoms or signs. First, he has observed that 20 of 25 cases have contralateral facial weakness; this is noticeable as drooping or lagging of the lower face during conversation rather than being apparent on voluntary movement. It represents the location and sensitive nature of the ‘specialised…face centre’ in the proximity of frontal lobe lesions. Even more discriminating is the almost universal presence of mental change. The pattern is characteristic: the patients are indifferent to their plight and future management, merely shrugging their shoulders in calm acceptance when the nature of the problem is explained and management outlined. This is associated with a loss of memory for recent events. Both aspects may improve after surgery, the patient then regaining insight into his or her earlier state of indifference; and resuming professional and domestic responsibilities. ‘This mental disturbance, when present and unchanged for some time, and associated with some facial weakness and headache, in the absence of a positive Wasserman, indicates a frontal lobe lesion and entitles the patient to an exploration. Operation has repeatedly revealed a focal lesion, usually a tumour, in the frontal lobe’. In discussion of this and another article from St. Louis on changes in personality seen with tumours of the frontal lobe [given by Sidney I(saac) Schwab (1871–1947)], (Sir) Gordon Holmes (1876–1965) politely points out that he too has observed these patterns in the many soldiers with focal frontal lobe lesions caused by gunshot that he studied during the Great War. He adds that the frontal lobe is not a single structure but consists of many parts, each having different functions; and he urges more precise correlation between localization of the lesions and clinical deficits. Moreover, lest there be any unfair criticism, Holmes points out that (Sir William) Gowers (1845–1915) and Foster Kennedy confined their observations on atrophy and disc swelling to basal lesions of the frontal lobe. And he offers an observation of his own: the patient with a frontal lesion is often slow in moving his eyes in the horizontal plane to command. Dr (James) Collier (1870–1935) agrees with everything that has been said by others. Dr Sachs replies that time did not permit him to specify the location of lesions more precisely. He stands by his conviction that the Foster Kennedy syndrome rarely occurs even with appropriately placed lesions, and he has not observed the sign mentioned by Dr Holmes.

Dr Sachs’s next article in Brain also returns to his training with Horsley and is based on an article given at the Second International Neurological Congress in London between 29 July and 2 August 1935. He reports the adaptation of a procedure...
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procedure. These are from a series of 69 cases amongst 189 with
and considers that 11 others might have benefitted from the pro-
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Medical Journal, 17 July 1909: Figs 1–22, pp. 121–4; Text,
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ment of Jacksonian epilepsy. He has treated 11 cases surgically
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cedure. These are from a series of 69 cases amongst 189 with
focal epilepsy—not due to tumour—in which the operative find-
ings are extensive cortical degeneration (n = 11), focal scars
(n = 19) or nothing abnormal (n = 39). Because resection was ex-
tensive and often macroscopically incomplete in the first two
groups, his interest lies in the third (Fig. 2). Even here, Dr Sachs
has proceeded cautiously in removing subpial brain tissue, but he
considers that the 11 cases of interest allow some conclusions to
be drawn. The histology is examined by Professor James Lee
O’Leary (nd) who confirms that Brodmann area 4 of the motor
cortex, as indicated by the presence of Betz cells, is contained
within the specimen. The surgical procedure involves electrical
stimulation, often inducing a seizure heralded by hyperaemia of
the unstable cortex, and so allowing histological assessment of
peri-ictal alterations in the affected tissue. That said, none are
described in the article. With removal of Brodmann area 4, par-
alysis ensues lasting 7–10 days, but occasionally more prolonged
and presumably depending on the relative distribution of efferent
fibres in this and neighbouring gyri. In contrast to the claims of
(John) Fulton (1899–1960), initially it is not flaccid. Good control
of the seizures is achieved in 10 of 11 cases compared with only
10 of 19 in whom attempts are made to remove a macroscopic
scar. In describing the 11 cases, Dr Sachs draws attention to one
in particular: C.W., a physician, aged 28 years when operated on,
on 30 January 1924, at Barnes Hospital, sustains a comminuted
right parieto-occipital skull fracture in an automobile accident in
1922 complicated by a subdural haematoma, which is removed,
after which he develops focal convulsions preceded by a sensory
aura and followed by loss of consciousness with automatisms on
recovery. The seizures are not controlled by luminal. At operation,
an adherent cortical scar measuring 5 × 5 cm is removed together
with the motor cortex. Re-exploration is needed after 48 h when
Dr C.W. develops a large intracerebral haematoma; and a bleeding
point is clipped. Apart from one brief convulsion confined to the
left hand, he has since been well and takes up appointment as pro-
fessor of physiology in a mid-western Medical School in 1930.
For Dr Sachs, ‘it requires a little courage, I have found, to remove
what appears to be a normal looking cortex; but in the light of the
experience herein cited, if I am convinced that I am dealing with a
true Jacksonian epilepsy, I now have no hesitation in carrying out
a subpial resection of the cortex. This procedure I have designated
the Horsley operation’.

Dr (Henry) Schwartz (1909–98) and Graham Weddell (1908–90: Dr Lee’s reader in anatomy, 1947–73, and later professor of
atomy in the University of Oxford, 1973–75), who worked with
Schwartz as Commonwealth Fund Fellow in Neuroanatomy and
Neurological Surgery between 1935 and 1937, also write from the
Neurological Service of Barnes Hospital in St. Louis. No one much
disagrees with the conventional anatomical description of the gus-
tatory pathway carried in the seventh cranial nerve described by
(Filippo) Lussana (1820–97: Fig. 3); and this has been amply
confirmed by Cushing, and by (Dean) Lewis (1874–1941) and
(Walter) Dandy (1886–1946). Yet there are dissenters who
favour a pathway involving the fifth nerve. Schwartz and
Weddell study 16 cases of whom 11 have undergone trigeminal
sensory nerve root section for the relief of tic douloureux, and five
treated for middle ear disease by radical mastoidectomy. The pro-
cedures are carried out by Dr Sachs or Dr Leonard T(hompson)
Furlow (1902–80), Dr M.F. Arbuckle (nd) and Dr A.C. Stutsman
(nd–1977); and the cases are tested using the Lewis and Dandy
techniques whereby diffusion away from the point of contact,
olfactory cuing and guessing are all carefully avoided. They stimu-
late the protruded tongue held in gauze with crystalline cane
sugar, sodium chloride and quinine sulphate, but not acid or
sour tastes, applying these separately to the anterior two-thirds
and posterior one-third of the tongue. Outcome is judged by
pointing to a card with optional responses required within 60 s
of the application; the tongue is bathed in water between stimuli.
There are no examples of ‘taste-blindness’. In four cases with
differential section of the fifth nerve sparing the petrosal and
facial nerves, achieving anaesthesia of the face, there is no post-
operative loss of taste. Therefore, taste is not transmitted in the
second or third divisions of the trigeminal nerve. The procedure in
four patients, in whom differential trigeminal nerve root section is
complicated, results in facial palsy with loss of taste over the ipsi-

deral anterior two-thirds of the tongue. These patients have not
been examined in any detail before surgery, and all that can be
said is that hemigeusia may have resulted from collateral damage
to the greater superficial petrosal nerve or geniculate ganglion.
More revealing are two cases, complicated by undoubted injury
to the petrosal and facial nerves, which had partially to be

Figure 2 Removal of the convolution after pia arachnoid has
been pushed back. This is an absolutely bloodless procedure
(From Sachs, 1935).
sacrificed per-operatively because of local adhesions. Each develops postoperative loss of taste on the ipsilateral anterior two-thirds of the protruded tongue. The preferred explanation is either damage to the superficial petrosal nerve or, more probably, the geniculate ganglion. One patient undergoing differential fifth nerve root section loses the petrosal nerve per-operatively, but both the geniculate ganglion and facial nerve are spared. Taste is lost on the ipsilateral anterior two-thirds of the tongue. Four patients treated by radical mastoidectomy require section of both the facial nerve and chorda tympani. Each has postoperative ipsilateral loss of taste on the anterior two-thirds of the tongue. Two others undergoing the same surgical procedure experience section of the chorda tympani close to its point of union with the facial nerve which is, itself, preserved. Surprisingly, despite conventional wisdom, taste is preserved in both of these patients undergoing radical mastoidectomy.

(Hugo) Schiff (1834–1915) has proposed that taste is conducted through the chorda tympani to the geniculate ganglion; from there to the sphenopalatine ganglion via the greater superficial petrosal nerve; and thence to the central nuclei of the brainstem in the second division of the fifth cranial nerve. Whilst accepting that the chorda tympani and greater superficial (but not the small superficial nor the deep) petrosal nerve contain sensory fibres of the seventh cranial nerve originating in the geniculate ganglion, they do not agree that the trigeminal nerve is ever part of the peripheral gustatory pathway. The proximal arrangements are less well demarcated; and Shiff has also had his doubts about this aspect of the anatomy, concluding that taste may be conducted in the lingual and small superficial petrosal nerves, as well as the chorda tympani, before reaching the geniculate ganglion.

Reluctantly, the authors accept the possibility that fibres in the lingual nerve sometimes conduct taste to the otic ganglion; but explaining the preservation of taste with section of the chorda tympani is not so easy. Their preferred anatomy is the classical pathway of Lussana: taste being conducted in the chorda tympani to the geniculate ganglion and, from there, to the brainstem via the nervus intermedius. But an alternative involves taste being transmitted through the chorda tympani to the otic ganglion; from there within the internal sphenoidal to the greater superficial petrosal nerve; then to the geniculate ganglion and nervus intermedius and hence to the brainstem (Fig. 4). They suggest that those who invoke an anatomical role for the fifth nerve have been led astray by the results of alcohol injection of the trigeminal ganglion with inaccurate placement or leakage away from the site of injection—including Gowers and Willfrid Harris (1869–1960). It follows that every effort should be made to spare the greater superficial petrosal nerve when operating in the temporal fossa.

These contributions to Brain by Ernest Sachs and Henry Schwartz, godfathers of the Washington University neurosurgical mafia, published between 1908 and 1938 illustrate the authority of their work and the influence of each man on the development and establishment of neurosurgery as a speciality, extending beyond the north-east of the USA, in the first half of the 20th century—as Samuel Greenblatt explains in his review of Neurosurgery at Washington University. A century of excellence by Robert Grubb (page 355).

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