With his manifesto for disconnectionist modelling, first published in German (Über Aphasie. Deutsches Archiv für klinische Medizin 1885; 36: 204–268), Ludwig Lichtheim (1845–1928) added a third, classic, 19th century account of aphasiology to those already written by Paul Broca (Perte de la parole, ramollissement chronique et destruction partielle du lobe antérieur gauche. [Sur le siège de la faculté du langage.] Bulletin de la Société d’Anthropologie de Paris. 1861; 2: 235–238) and Carl Wernicke (1848–1905: Der aphasische Symptomencomplex. Breslau, 1874). In 1885, Brain published a version in English of Lichtheim’s paper, translated and somewhat condensed by the Editor, Armand de Watteville: ‘the whole task was by no means an easy one; the careful revision bestowed by Professor Lichtheim to the proofs, and for which I beg to return him my best thanks, has, however, materially relieved me from the responsibility I had assumed’.

Lichtheim aims to settle controversies relating to aphasia by observing and then explaining the ‘experiments of nature’. Specifically, ‘our task is to determine the connections and localisation of the paths of innervation subservient to language and its correlated functions … accounting for its manifestations with the same precision … as for those of a motor or sensory paralysis depending on a lesion of the peripheral nerves’. He predicts that today’s clinical curiosity will become tomorrow’s laws of aphasiology: thus, ‘Broca was led, after many mistakes had been made, to bring into sharp relief aphasia in its narrower sense … Wernicke first … distinguished between … motor and sensorial aphasia … and a third … designate(d) as commissural aphasia (Leitungaphasie)’. As for the history of aphasiology, nothing of substance could be added to the account written by Adolf Kussmaul (1822–1902: translated in Ziemssen’s Cyclopedia, volume xiii, Lepizig 1877). Lichtheim indicates that he intends to elaborate on Wernicke’s three types of aphasia and to add (four) new categories of his own.

His model encapsulates four principles: speech is a learned behaviour dependent on two main centres and a reflex arc—the way in and the way out; a third centre applies intelligence, or the concepts of language, to this circuit for the raw comprehension and delivery of speech; the acquisition of reading and writing adds another layer of anatomical arrangements; and, in many components of spoken and written language can be damaged by discrete lesions to produce seven discrete types of aphasia (see Fig. 1).

Language is acquired by imitation in the child, who etches memories of word-representations in Centre ‘A’ connected by a commissure to the coordinated motor images in Centre ‘M’; later, these are modulated by Centre ‘B’, adding the elaboration of concepts to the slavish activities of ‘A’ and ‘M’ (see Fig. 1). Whilst claiming general consensus amongst contemporary diagram-makers for this arrangement, Lichtheim hints at a controversy in the direction of traffic from ‘B’: Kussmaul has it as ‘B A M’, whereas Lichtheim prefers the direct route ‘B M’. But to complete the circuit for spoken language, there exists a reflex arc having an afferent component ‘a’ that transmits acoustic impressions, and a motor pathway ‘m’ projecting to the organs of articulation. Reading requires a visual Centre ‘O’, connected to ‘A’ in order to link existing auditory impressions to the written symbols of words. Writing requires an executive Centre ‘E’ (see Fig. 2). Lichtheim illustrates the theoretical authority of his diagram by suggesting that reading aloud involves the pathway ‘O A M m’, and intelligent writing must connect ‘B’ with ‘E’, but Lichtheim is initially not too sure whether this goes directly through ‘M’ or via ‘A and M’. Having devised and tried out this comprehensive diagram of speech mechanisms, Lichtheim has waited to publish until such time as he had cases that supported his schema. Now, he turns to the seven types of aphasia.
1. Lesions at ‘M’ are the equivalent of Broca’s ‘aphasia’, Wernicke’s ‘motor aphasia’ and Kussmaul’s ‘ataxic aphasia’. Carelessly, Lichtheim had not thought to determine when previously seeing such a case whether reading aloud is disturbed whereas comprehension of silent reading is preserved. Nor is the literature helpful on this point: only Trousseau has pointed out that these cases appear eager in their reading but do not understand what they see. But Lichtheim has more to say later on this issue.

2. A lesion at ‘A’ corresponds to Wernicke’s ‘sensorial aphasia’. Here, there is one point of contention: Wernicke would have it that the disturbance of speech output—paraphasia—results from an interruption in ‘BMm’ and failed routing of traffic that normally connects ‘B’ to ‘M’ by also looping through ‘A’; Lichtheim considers that paraphasia represents partial damage in the circuit ‘B M A B’ such that language is not altogether arrested. But after thinking it through and examining a case in which ‘inner speech’ was assessed without the intrusions of auditory representations whilst ‘speaking aloud’, he concludes by disagreeing with Kussmaul who considers the pathway for voluntary language as ‘B A M m’ and the seat of Broca’s aphasia as within ‘B A’ (see Fig. 3). This cannot be correct, because such patients are unable to repeat aloud; rather, there must be an interruption somewhere in ‘a A M m’. On balance, Lichtheim concedes that Wernicke’s interpretation of paraphasia as resulting from a lesion of ‘B A M’ seems preferable.

3. The third type—an interruption in ‘A M’—leaves open the route through ‘A B M’ so that only repetition, reading aloud and writing to dictation are impaired; Lichtheim’s analysis of this situation focuses mostly on writing and leads him to conclude that this depends on the integrity of ‘B M A E’ (see Fig. 4) and not a straight connection between ‘B M E’ (see Fig. 2). Here, Lichtheim repeats a principle that he clearly favours both for spoken and written language: intelligent selections are often rehearsed or checked back through the auditory word-representation centre, resulting in many of Lichtheim’s proposed circuits always including a route through ‘A’. Assuming the same requirement for the involvement of ‘A’ in paragraphia as in paraphasia, the contest between the accuracy of Fig. 2 versus Fig. 4 might be resolved by observing whether a lesion of ‘M A’ is associated with paragraphia or agraphia, as expected from Figs 2 and 4, respectively. However, the matter is not resolved by the otherwise promising case of JSB: despite having paraphasia, inability to repeat or read aloud, and preserved comprehension of speech and copying, the clinical observations made in life did not satisfactorily resolve the distinction between his ability to write volitionally and to dictation—not least because, when repeating more than the few words that make up a sentence, there is the added task of
memory, and repetition therefore becomes partly an act of volition. The same issue of memory and retention of material applies to writing spontaneously and to dictation. Taking everything together, apart from the inability of patients with Broca’s aphasia to write to dictation, Lichtheim prefers the schema of Fig. 4 to that of Fig. 2.

4. Damage to ‘BM’ mimics Broca’s aphasia with the added features of preserved repetition, reading aloud and writing to dictation. W(illiam) A(lexander) Hammond had described such a case in his Treatise on Diseases of the Nervous System (1871) and Lichtheim mentions his own patient, Dr CK, who had an accident in his carriage and thereafter could say very little or write, but read well—silent and aloud—and copied and wrote to dictation.

5. Lesions at ‘M m’ prevent speech output, volitionally or in response to repetition and reading aloud, but with intact comprehension of the spoken and written word and of copying, and with preserved writing (volitionally and to dictation). Kussmaul had already described this constellation but Bastian’s concept of aphemia (Bastian HC. On different kinds of aphasia. British Medical Journal 1887; 2: 931–936 and 985–990) and speech apraxia lay ahead. Lichtheim seeks greater precision in the interpretation of these cases—often showing a mixture of features—than Kussmaul, who attributed the defects to a lesion of the motor tracts of the (left hemisphere) speech and writing centres. Rather, Lichtheim works up an analysis of writing as dependent on activity in both hemispheres. His argument turns partly on the issue of mirror writing, which is often seen with lesions of the left hemisphere: left-handed writing is not lost in dense left hemiplegia; and his own skill with the non-dominant left hand all seem to argue for the existence of an independent writing executive in the right hemisphere. Thus, he adds ‘E1’ in the right hemisphere to the Centre ‘E’ already mapped to the left (see Fig. 5). And Lichtheim makes the distinction between agraphia and ability to execute the motor act of writing: again, what is lost is the concept and reference back to auditory representations of words to be written rather than the motoric act. Because ‘A E’ and ‘A E1’ (sadly, our printers of 121 years ago allowed a misprint and the paper refers to A C) run together for some distance before parting for each hemisphere, isolated agraphia (with intact speech) may affect both hands or the right only.

6. Lichtheim was especially pleased to have identified examples of his two remaining categories of aphasia—lesions at ‘A B’ and ‘a A’, respectively. ‘A B’ is expected to be a version of Wernicke’s sensorial aphasia with paragraphia, and preserved repetition and copying, reading aloud and writing to dictation. Mr JU Schwartz, admitted to the Inselspital in Berne on May 19, 1884, had just such a constellation of language disorders, as revealed by Lichtheim’s inventive clinical examination (the translation of German paraphasic and paragraphic details proving particularly challenging for Dr de Watteville). One nuance of interpretation is the addition of repetition of a question—the echolalia of Romberg—as a device for reading aloud, adding the confound of an auditory word-representation of the written word. But this detail has a significance that takes Lichtheim into the 21st century: for he interprets the language-based abilities of Mr Schwartz as indicating that a new set of pathways involving ‘A’ has been opened up in response to the loss of ‘A B’ (see Fig. 6)—anticipating the adaptations and plasticity described in the current issue by Dorothee Saur and colleagues (page 1371).

7. With a lesion of ‘a A’, there is loss of understanding or repeating speech, but preserved volitional speech, reading, copying and writing: ‘BM A B’ being intact, there is no paraphasia. The appropriate designation of this lesion as speech-deafness is revealed by examining Mr L, a teacher and journalist. His case is unique. However, taken together,
the retrospective analysis and interpretation, associated genuine deafness, complex history of several apoplectic fits and discrepancies between observations made by Mrs L and the attending physician (Dr Burckhardt: Ein Fall von Worttaubheit. Correspondenzblatt für Schweizer Aerzte 1882: No. 20) make it likely that Mr L also had a transient lesion at ‘A’, resulting in Wernicke’s sensorial aphasia, and therefore slightly confusing the picture.

Reassured that examples of each are to be found, Lichtheim is nonetheless anxious to reconcile previous descriptions with his designation of seven subtypes of aphasia. That said, so few previous accounts could boast the detail with which he now formulates his classification. Even so, if the involvement of more than one pathway, the impact of extensive lesions that disrupt all the core circuits, and differential rates of recovery in separate locations (word-deafness resolving fast, and all disorders affecting writing more slowly) are allowed, almost every case can be accommodated. Because, unlike Kussmaul, he is espoused to the need for most language activity to make reference to word-representations by connections to ‘A’, in order to account for cases in which there are dissociations between defects of spoken and written language, Lichtheim postulates that disorders of writing and reading occur with intact speech because the pathways ‘AO’, ‘AE’ and ‘OE’ are separable from the ‘BM’ circuit. The crucial role of ‘B’ in the otherwise linear connection between ‘A’ and ‘M’ is highlighted by the need for conceptual reference when naming objects, compared with volitional speech in which the overall significance of the sentence provides the validation, rather than a word-by-word checklist. Wisely, Lichtheim assigns amnestic aphasia (nominal difficulties) rather a low currency in terms of localization (or pathways), this being seen in most generalized degenerative conditions. Central aphasia occurs with lesions of ‘B M m’, and speech-deafness with interruptions of ‘B A a’. Each of these two main types may arise from lesions of the Centres (‘M’ and ‘A’) or their inner (‘B M’ and ‘B A’) or outer-commissural (‘M m’ and ‘a A’) pathways. That affecting ‘M A’ is designated commissural paraphasia. Anatomically speaking, as it were, ‘M’ and ‘A’ are in the inferior frontal and temporal convolutions, respectively; ‘M A’ is in the insula; ‘B’ is anatomically distributed—across the entire hemisphere; and ‘B M’ and ‘B A’ constitute diffuse radiations converging on these two centres (see Fig. 7); the anatomy of ‘m’ is that of the inferior frontal convolution, across the corpus callosum to both descending corticobulbar pathways (see Fig. 8: on which point, Wernicke classifies anarthria as a variant of aphasia); the convergence of bilateral afferent auditory pathways to form ‘a’ occurs in the left temporal lobe close to ‘A’; the anatomy of word-blindness (alexia without agraphia) is at ‘O A’ in the left anterior frontal lobe (we now place it in the territory of the left posterior cerebral artery).

‘Though I have ventured the above remarks concerning the probable localisations of aphasic disturbances, I am well aware of the restricted foundations on which we may safely build, and what a space theoretical reasoning has still to occupy in the discussion. If I have not kept my views to myself, it was on the principle that we must not recoil from the consequences deducible from our hypotheses...even erroneous assumptions may prove of advantage in the search for truth’. Although a ferocious and rigid maker of diagrams, contemporary neuroscience indicates that Ludwig Lichtheim got it more right than wrong.