Sir, It is with great interest that we read the paper from Seeley and colleagues (Seeley et al., 2008) on the consequences of primary progressive aphasia (PPA) on a patient’s pictorial production. However, we are somewhat concerned that the authors have attributed the cognitive disorders developed by Ravel (1875–1937) in his last 4 years to PPA. This diagnosis is mainly based on data published by Alajouanine, the eminent neurologist who examined Ravel over 2 years and suggested the existence of ‘Wernicke aphasia’ of ‘moderate’ intensity (Alajouanine, 1948; Amaducci et al., 2002). However, a careful reading of this article—an MD thesis on Ravel’s disease which has unfortunately never been published (Achache, 1990)—and many accounts from Ravel’s friends, biographers and musicologists, as well as more recent articles (Baeck, 2005) suggests other interpretations.

It can be advanced that the first symptoms were not aphasic in nature but rather apraxic. The preliminary symptoms were indisputable only in 1933. It was not until later, in 1935–36, that clear aphasic signs appeared, such as problems finding words, but, even at the latest stages of the disease, Ravel was able to speak fluently, though slowly and understand what people told him (Alajouanine, 1948; Sergent, 1993). Therefore, it is more accurate to evoke a diagnosis of late anomic aphasia. In contrast, there are overwhelming arguments for apraxia as a presenting symptom. Alajouanine himself described a predominant ‘praxic difficulty’. There was such a striking alteration in Ravel’s handwriting that a musicologist, René Chalupt, erroneously believed that his last production, *Don Quichotte à Dulcinée*, completed in 1933, was written by ‘a friend’s hand’. In June 1933, although he had been a skilful swimmer, he was unable to recall the movements of swimming. At the end of 1933, he was no longer able to sign his name (the reason why he refused to give autographs) or read (text and then music scores). An analysis of letters from this period shows that there are spelling errors but mainly an apraxic agraphia with many erasures and ink blots (Achache, 1990). He said that he had musical projects, such as the opera *Jeanne d’Arc*, but was unable to translate them to a sheet of paper or a keyboard; nor was he able to sing them, as if they were locked within his brain. Brain imaging data show that areas specifically involved in generating and playing scales are centred on posterior cingulate (Sergent, 1993; Parsons et al., 2005).

In contrast to this motor output failure, his auditive perceptual abilities were preserved: he was able to recognize his works when they were played and detect the most subtle errors. In 1937, he was still able to give to a pianist useful advice for playing his *Concerto pour la main gauche* (Alajouanine, 1948; Sergent, 1993). At no time were there arguments for a dysexecutive syndrome. Even at the end of his life, Ravel’s memory, insight, personality and behaviour were preserved (Baeck, 2005).

Hence the usual criteria of PPA, a diagnosis sometimes suggested in the literature, are not met, and attributing the thematic repetitiveness of *Boléro* (Amaducci et al., 2002), written in 1929, to the earliest signs of the PPA or ‘Pick’s complex’ is overly speculative and questionable. Neurosurgical exploration, mistakenly performed on the right side, only excluded a tumour and intracranial hypertension (Sergent, 1993). Although a definitive diagnosis is not available, we suggest that Ravel had a left focal degenerative disease, mainly involving the parietal lobe. It explains the picture of progressive apraxia, with severe alexia and agraphia. According to our current classifications, a corticobasal syndrome is ruled out by the absence of motor signs. Posterior cortical atrophy, with alexia as an early visuospatial disturbance, may be discussed. A focal parietal atrophy is probably the most accurate diagnosis.

**References**