Editorial

Fitting two languages into one brain

As Europe moves into the next century, the language barrier appears more formidable than ever. Eleven languages are recognized as official languages of the European community, but the actual number of languages needed to operate with other countries must be closer to 40. Multilingualism is a complex problem for the European administration, which has had to appoint the largest translation service in the world, Brussels’ Joint Interpreting and Conference Service. But multilingualism also poses special challenges to the human brain. How can cerebral circuits that normally handle a single phonology, lexicon and syntax adapt to the storage of multiple language systems? Consider the case of German and English. Verbs are placed at the end of sentences in German, but not in English. How then do English–German bilinguals avoid mixing up the two sets of rules?

The coexistence of multiple languages in the same brain suggests that sophisticated mechanisms of segregation and coordination must exist to prevent cross-talk. In this issue, Cathy Price, David Green and Roswitha von Studnitz use positron emission tomography to throw some light on bilinguals’ cerebral organization (Price et al., 1999). Their results clarify how bilingual brains escape the curse of Babel, and also help make sense of some puzzling reports of bilingual aphasia.

Price and her collaborators studied six subjects whose native language (L1) was German and who became fluent in their second language (L2), English, after they started learning it at about the age of nine. Subjects were scanned while they read or translated written words, one at a time. In distinct blocks, the words were presented only in German, only in English, or alternately in the two languages. This experimental design allowed the authors to image, in the same study, the areas involved in translation, language switching, and first and second language perception and production.

Surprisingly, the main regions that were found most active during translation fell outside of the classical language areas. Translating, relative to reading, activated mainly the anterior cingulate and bilateral subcortical structures (the putamen and the head of the caudate nucleus). Price et al. attribute this to the need for greater coordination of mental operations during translation, during which the direct cerebral pathways for naming words must be inhibited in favour of other, less automatized translation circuits. The supervision of articulation processes may be particularly important, because circuits known to be involved in the control of articulators (supplementary motor cortex, cerebellum and left anterior insula) also showed greater activation during translation.

During language switching, increased activation was also observed in Broca’s area and in the bilateral supramarginal gyri. Since those regions are thought to be involved in mapping orthography to phonology, it suggests that phonological processing is also a source of increased difficulty when having to switch languages.

The bilingual brain, then, seems to address the problem of translating single words as it would tackle many other non-automatized tasks. The attention system of the anterior cingulate kicks in during translation, just as it does during the Stroop test or various word generation task, presumably to control the switching on and off of multiple distributed language circuits that collectively support translation. Anyone who has ever tried this well-known party trick of reciting the number sequence while switching languages (for instance ‘un, two, trois, four . . .’) will recognize that central coordination is a very plausible source of difficulty in translation and language switching.

The activation of control circuits in the anterior cingulate and basal ganglia may also help account for otherwise puzzling reports of bilingual aphasia. In rare cases, brain lesions can leave a bilingual patient impaired in only one language while sparing the other. Yet some reports are even more surprising. On one day, a given patient can be aphasic in L1, but not in L2, while the next day he may show the converse pattern! Stuningly diverse patterns of bilingual aphasia and recovery have been reported, impeding neuropsychologists’ efforts to propose general rules of organization of the bilingual brain (Paradis, 1995). Price et al. convincingly argue that many of these deficits may in fact reflect impairments not of the language circuits themselves, but of their control structures. It will be interesting to see how far the hypothesis of a central attentional or switching difficulty can go in explaining the patterns of bilingual aphasia.

Price’s work emphasizes the importance of control mechanisms in bilingual processing. What remains to be clarified, however, is where the circuits that are being controlled are. Are the brain circuits that support L1 and L2 anatomically segregated, or are they intermingled in the same cortical regions? Price et al. observed that comprehension of words in L1 yielded greater activation of the left temporal lobe, including the temporal pole, than did words in L2. This replicates several earlier studies which all showed that the ‘language organ’ in the left temporal lobe is more activated when listening to the mother tongue than to any other lesser known language (Mazoyer et al., 1993; Perani et al., 1996, 1996; Perrin et al., 1994; Price et al., 1994; Perani et al., 1996).
Other studies capitalizing on the higher spatial resolution afforded by functional magnetic resonance imaging have suggested that, even within a single brain region, there may be smaller-scale circuits specialized for L1 or L2 (Dehaene et al., 1997; Kim et al., 1997). For instance, in bilinguals who learned their second language late in life, sentence production tasks in L1 and in L2 have been found to activate two non-overlapping subregions of Broca’s area (Kim et al., 1997). In that study, only early bilinguals, who received equal practice with their two languages from birth, showed an activation overlap for L1 and L2.

My colleagues and I have obtained similar results in the domain of sentence comprehension, though the critical variable appeared to be the eventual fluency of the subjects rather than the age of acquisition. Highly fluent bilinguals activate strikingly similar left temporal areas for L1 and L2 (Perani et al., 1996), including, in some subjects, small left-temporal and right-hemispheric activation foci that are specific to L2 (Dehaene et al., 1997). In two recent studies, Chee et al. also observed activation overlap for L1 and L2 in fluent Mandarin–English bilinguals, whether the task was sentence comprehension (Chee et al., 1999b) or single-word production (Chee et al., 1999a). The latter study incorporated a group of late learners of L2, but unfortunately it failed to replicate Kim’s finding of segregated activity in Broca’s area. Nevertheless, a weak consensus seems to be emerging to suggest that the level of fluency is a critical determinant of brain activation patterns in language tasks. In fluent individuals, processing differences between L1 and L2 may be supported by differences in cerebral microcircuitry that are hardly visible with the present resolution of brain-imaging methods.

It should be clear that all language-related brain activation patterns are, in the final analysis, images of brain plasticity. Left perisylvian activity is conspicuously absent when subjects are exposed to otherwise normal sentences, but delivered in a language with which they have had no prior experience (Mazoyer et al., 1993; Bavelier et al., 1998). Language acquisition must radically alter the brain through some kind of symmetry breaking, so that it ultimately becomes exquisitely tuned to one language, but unresponsive to another. By improving our comprehension of this tuning process, research on multilingualism may eventually teach us much about brain plasticity and critical learning periods.

References


