Compensations for brain deficits

"Every cloud . . ."†

IAN H. ROBERTSON

Those involved in research or clinical work related to brain function will be used to the dinner party question "we only use 50% of the brain, don't we?" The scientist's dismissive sneer is usually well enough concealed, depending on how much he or she has had to drink. Where on earth did this lay myth arise, we chuckle over coffee in the common room on Monday morning? But scientists and clinicians are not immune to myths also. For many decades, neuroscientists preached the doctrine that the adult brain is 'hard-wired'. Perhaps in very early childhood, we conceded, plastic changes in the brain were possible, but after the age of three or four years connections were indelibly made.

DEATH OF A SCIENTIFIC MYTH

This doctrine had huge ramifications, not only in medicine, but also in education and social policy. If brain capacity is largely hard-wired through genetic and early experience factors, then the expectations we have for changing brain function through structured experience are surely limited. Within one branch of medicine for instance – physical rehabilitation – a direct consequence of this in Britain and some other countries was a paucity in the provision of systematic rehabilitation for adults who suffered brain damage: the 'hard-wired' myth led to huge pessimism that the brain's physical recovery could be influenced by that systematic provision of experience we call rehabilitation. Yet recently, for instance, we read in the *Lancet* that the extent of recovery of hemiparetic limb function is proportional to the number of hours of targeted physical rehabilitation that the patient receives (Kwakkel *et al*, 1999).

Also, in psychiatry we find evidence that brain function can be altered by systematic cognitive rehabilitation – for instance in relation to cognitive deficits associated with schizophrenia (Wykes *et al*, 1999). Language rehabilitation following left hemisphere stroke also produces distinct changes in brain function (Musso *et al*, 1999), and there are many other types of effective means of altering the supposedly 'hard-wired' damaged adult brain (Robertson & Murre, 1999). The conceptual justification for much of this activity lies in recent revolutionary findings about the brain. In particular, there has been dramatic disconfirmation of a long-held, central assumption about the brain – that new cell bodies cannot emerge in adulthood. In both humans and animals, recent data show that in the hippocampus, new cells can indeed be produced (Eriksson *et al*, 1998). What is more, this process is partly experience-dependent – animals kept in enriched compared with impoverished environments show more cell genesis in the hippocampus (Gould *et al*, 1999). This finding follows close on the heels of another revolutionary discovery of the past decade – the demonstration that the adult brain can show large experience-dependent change in neural circuits, including dendritic and axonal sprouting (Recanzone *et al*, 1993). I have reviewed the theoretical basis for the design of planned, experience-dependent changes in the brain elsewhere (Robertson & Murre, 1999).

RELEASE FROM INHIBITION

Not all experience fosters positive changes in the brain, however. The complex network of inhibitory and facilitatory connections between networks means that some types of experience or stimulation may actually exaggerate deficits in a vulnerable circuit because it activates a competitor network that further inhibits the malfunctioning module (Kapur, 1996). In the case of unilateral left neglect following right hemisphere stroke, for instance, single left-hand movements can produce improvements in neglect of the left side that are abolished when both hands are moved together (Robertson & North, 1994). The single left-hand movement activates fragile attentional circuits in the damaged right hemisphere that are immediately again inhibited if the competitor left hemisphere is activated by a right hand movement.

The study by Miller *et al* (2000, this issue) in California gives an intriguing psychiatric example of the consequences of a naturally occurring change in the inhibitory dynamics of the human brain – in the context of frontotemporal dementia. Twelve people stricken by this disease process showed either preservation of, or indeed improvement in, musical or visual ability as the disease progressed. One 68-year-old man, for instance, began to compose classical music, with some of his pieces actually being performed. He continued to compose in the year after his language skills deteriorated, linked with a progressive reduction of function in the left temporal lobe. Other individuals developed completely new skills as visual artists, and again this seemed to be linked to a progressive reduction in function of the left temporal lobe. Miller *et al* suggest that such paradoxical enhancement of skills in dementia may be due to the lifting of inhibition by the left temporal lobe over other areas of the brain. In particular, right hemisphere temporal and parietal areas may have benefited from this reduction in inhibition, allowing certain visuo-spatial and musical abilities to flourish.

All this research shows that the neuroscientist's complete rejection of the 'myth' of untapped potential is misplaced (Robertson, 1999). True, there are not great 'silent' tracts of brain waiting to be 'filled-up' with stimulation, but on the other hand there is a considerable potential for the strengthening of connected networks via, among other processes, dendritic sprouting (Donoghue, 1995). And as mentioned above, there is also the potential for hippocampal cell regeneration which is also experience-dependent. In support of such a view, there is evidence for a correlation between density of dendritic trees in the left temporal cortex and number of years of education (Jacobs *et al*, 1993).

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USE IT OR LOSE IT

The converse also almost certainly holds. Lack of use and lack of stimulation should lead to atrophy of neural circuits, with consequent loss of function. Rats kept in impoverished, unstimulating environments show relatively atrophied dendritic trees compared with rats kept in more stimulating environments (Kolb, 1996). It is certainly possible to speculate that conditions such as schizophrenia and head injury that tend to lead to low levels of activity and participation in the world may result in secondary, additional cognitive impairment because of inadequate use and stimulation of the brain. In the case of normal aging, there is clear evidence that continued participation and activity is associated with relatively preserved cognitive functions (Shimamura *et al*, 1995).

In spite of this relatively preserved cognitive function as a result of higher levels of activity, decline of certain aspects of cognition with aging is still a fact. But as is the case with Miller *et al*'s patients, decline in one respect need not mean decline in all cognitive attributes. On the contrary, there is some evidence that certain types of complex problems are better solved by older than younger people, particularly where people can draw on accumulated experience or 'wisdom' to solve them (Walsh & Hershey, 1993). From a clinical viewpoint, a 'swings and roundabouts' perspective, has potential advantages for the mental well-being of people who experience loss of cortical function through disease or damage. Depression and demoralisation are common consequences of brain damage, but if clinicians could take a more positive orientation to the problems, focusing not just on pathologies, but also on possible preserved or enhanced abilities, then some advantages may accrue to both patients and the families.

IAN H. ROBERTSON, PhD, Department of Psychology, Trinity College, Dublin, Ireland

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