

Karl Pribram

*Commentary on 'Synaesthesia' by
Ramachandran and Hubbard*

Ramachandran and Hubbard's superb article on 'Synaesthesia: A window into perception, thought and language' (2001) fills a gap in our understanding of a phenomenon that many of my students have asked me to fill. Up to now I have failed to have a satisfactory answer. It really does no good to say that somewhere in the brain or brain stem 'representations' must be able to get together. The evidence presented that cortical connectivity is involved (though perhaps not exclusively) provides an entrée into the process that raises important issues.

In my James Arthur lecture on the Evolution of the Human Brain at the American Museum of Natural History in New York (Pribram, 1971a) I pose the following possible dilemma: my research with nonhuman primates showed corticocortical connections to be unimportant in all of the behaviours that I studied. Rather, the connections with the subjacent basal ganglia were intimately involved (see also, Pribram, 1972; 1986). By contrast, human clinical neurology depended heavily on corticocortical disconnection syndromes as explanations for disturbances of language and other cognitive processes.

The dilemma would not have occurred if the human clinical data had been reliable and if the nonhuman primates had been able to use language and display evidence of other human-like cognitive processes (and if they did, as for example in some of the work with chimpanzees, I would be allowed to do brain surgery on them). As to the reliability of the human postmortem clinical data, when I had a conversation with Norm Geschwind asking whether his disconnection syndromes could involve reciprocal (cortex to basal ganglia to cortex) subcortical paths, he unhesitatingly answered 'of course'.

Thus the current research using tomographic imaging techniques showing 'cross wiring' at the cortical level is most helpful — although the possibility still exists that the cross wiring occurs by way of the basal ganglia.

However, the mechanisms for cross-wiring proposed by Ramachandran and Hubbard present us with a paradox. The proposal is that there might be either a loss of inhibitory connections or a failure in pruning excess connectivity. The

Correspondence: Karl Pribram, PO Box 679, Warrenton, VA 20188, USA.

paradox consists of the fact that the synaesthesias they report are often highly specific and in many cases unidirectional (e.g. from auditory to visual). There would have to be some process in addition to loss of inhibitor connections or of failure of pruning to account for such specificity.

The paradox is resolved if we assume that synesthesia and the derivatives discussed in the paper are due to an *increase in inhibitory connectivity*. In well known instances neural inhibition leads to *differentiation* of behaviour, *not its suppression*. The most obvious case is the pyramidal and extrapyramidal tracts. When severed, the Babinsky and Gonda signs return and the extremities become spastic. Inhibitory control by input from cortex ‘sculpts’ the process to allow differentiation of behaviour. Another example is the corpus callosum which increases markedly in size from nonhuman to human brains. Much of its action must be inhibitory because of the ipsilateral sensory input to each hemisphere (1/3 of the somatosensory input is ipsilateral). Event related potential studies have shown that during maturation the ipsilateral response progressively decrements (Pribram, 1971b, Chapter 19). Further, frontal lobe function has routinely been thought of as inhibitory: disconnection through lobotomy produces changes in behaviour interpreted as due to disinhibition.

In short, I suggest that synaesthesia and its related processes are due to the propensity of the human brain to allow differentiation of experience and action. Synaesthesia thus becomes a specialization such as face recognition, hand recognition and the like. The responsible corticocortical connectivity is inhibitory, not excitatory.

The import for philosophy of this conjecture is that we are too prone to emphasize the associative aspects of cortical activity. Of course associative processes are important. My by now infamous suggestion that a process similar to the one that is used to produce fMRIs (that is a quantum holographic-like process) deals with associativity in a most fundamental sense. But associativity via corticocortical connections (rather than by a sweep from intralaminar thalamic origin) harks back to Flechsig’s association cortex and Viennese philosophy. Vienna was during this period heavily in league with English associationism, witness the fact that Wittgenstein could move so readily between Vienna and Cambridge. It is time neuroscience rid itself of these antiquated constraints.

Enough. Ramachandran and Hubbard have provided a major contribution to our understanding of brain function in synaesthesia and in processes they speculate might be related such as the origin of language and metaphor. My proposal for an additional possible ‘mechanism’ results from their own stimulating ideas. I look forward to reading their response.

References

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