Mood disorder as a specific complication of stroke

Alan J Carson

Robert Robinson published a fascinating study demonstrating that experimentally induced strokes in rats led to alteration in cerebral metabolism of catecholamines that correlated with behavioural changes in the rats that mimicked depression. Folstein’s data appeared to be an early example of translational research and were widely disseminated as they appeared to link laboratory based neurobiology with clinical practice. Tantalisingly it seemed to offer a human model for studying the anatomy of depression. Appearing, as it did, contemporaneously with the development of cerebral imaging techniques, this was the impetus researchers had needed. Over the next 2 decades, 143 reports were made on this topic. Sadly, the theory of anatomical location of brain lesions as a simplistic explanation for mood disorder did not stand up to scrutiny. It was perhaps too good to be true; a salient reminder of the need for confirmation in humans of findings from animal models.

In critical analysis the paper itself has stood the test of time. We now know that 33% of stroke patients suffer from depression (95% CI 29% to 36%). We now know that this depression leads to increased disability and probably increased mortality. Most importantly, we now know that antidepressants are effective in treating it. Countless patients round the world are benefitting from this knowledge and that is an impact that any researcher can be proud of.

REFERENCES
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