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Effects of hydrocephalus and ventriculoperitoneal shunting on afferent and efferent connections of the feline sensorimotor cortex

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Background

Previous reports have suggested that connectivity of the cerebral cortex may be irreversibly altered by hydrocephalus, but very few studies have examined this possibility directly. Therefore, an axonal tracer study was initiated in a feline model of infantile hydrocephalus that was amenable to ventricular shunting.

Materials and methods

Obstructive hydrocephalus was induced in 10-day old kittens by intracisternal injections of kaolin; saline-injected animals served as controls. Hydrocephalic kittens received low-pressure ventriculoperitoneal (VP) shunts 10–14 days post-kaolin. Unilateral injections of wheat-germ agglutinin-conjugated horseradish peroxidase (HRP) were made into the sensorimotor cortex (Brodmann's areas 4, 6 and 3) in hydrocephalic animals (n = 5) at 9–15 days post-kaolin to represent the pre-shunted condition. Shunted animals (n = 5) received HRP injections at 1, 2 and 4 weeks post-shunt. Control animals (n = 5) received HRP injections at set time-points between 12–47 days of age to broadly match the sacrifice times of the shunted animals.

Results

VP shunting reversed all behavioral abnormalities, including spastic movements, extensor rigidity, and lethargy. Reduction of anterograde and retrograde HRP labeling was most profound within the contralateral cortex and portions of the midbrain, and considerable in the thalamic relay nuclei, especially the ventrolateral nucleus. Labeling within cell bodies of the ventral tegmental area (VTA) decreased greatly in untreated hydrocephalus. Untreated hydrocephalus reduced retrograde labeling in the locus coeruleus but did not affect the raphe nucleus. Shunting increased both anterograde and retrograde labeling of contralateral motor cortex to near normal. Thalamic relay nuclei recovered retrograde labeling, but anterograde labeling did not return to control levels. Shunting restored cellular label to the VTA and locus coeruleus. Surprisingly, recovery of labeling occurred as early as 7 days after shunting, suggesting that functional deficits in axoplasmic flow, rather than structural deterioration of axons, were responsible for at least some of the changes associated with untreated hydrocephalus.

Conclusion

Collectively, these data suggest that (1) cortical connectivity involving both afferent and efferent pathways was impaired in untreated hydrocephalic animals, and (2) shunting improved both cortical afferent and efferent connectivity, but (3) complete re-establishment of the cortical efferent pathways did not occur. Dysfunction in cortical pathways, if permanent, could be responsible for many of the motor and cognitive deficits seen clinically in afflicted hydrocephalic children.