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Poststroke aphasia : epidemiology, pathophysiology and treatment.

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Aphasia, the loss or impairment of language caused by brain damage, is one of the most devastating cognitive impairments of stroke. Aphasia is present in 21-38% of acute stroke patients and is associated with high short- and long-term morbidity, mortality and expenditure. Recovery from aphasia is possible even in severe cases. While speech-language therapy remains the mainstay treatment of aphasia, the effectiveness of conventional therapies has not been conclusively proved. This has motivated attempts to integrate knowledge from several domains in an effort to plan more rational therapies and to introduce other therapeutic strategies, including the use of intensive language therapy and pharmacological agents. Several placebo-controlled trials suggest that piracetam is effective in recovery from aphasia when started soon after the stroke, but its efficacy vanishes in patients with chronic aphasia. Drugs acting on catecholamine systems (bromocriptine, dexamfetamine) have shown varying degrees of efficacy in case series, open-label studies and placebo-controlled trials. Bromocriptine is useful in acute and chronic aphasias, but its beneficial action appears restricted to nonfluent aphasias with reduced initiation of spontaneous verbal messages. Dexamfetamine improves language function in subacute aphasia and the beneficial effect is maintained in the long term, but its use is restricted to highly selected samples. Pharmacological agents operating on the cholinergic system (e.g. donepezil) have shown promise. Data from single-case studies, case series and an open-label study suggest that donepezil may have beneficial effects on chronic poststroke aphasia. Preliminary evidence suggests that donepezil is well tolerated and its efficacy is maintained in the long term. Randomised controlled trials of donepezil and other cholinergic agents in poststroke aphasia are warranted.

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