Neurological Signs: Echo Phenomena

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To cite:
Larner AJ.
ACNR 2015;15(3):16.

A number of echo phenomena are described in the neurological literature,[1] some of which are briefly considered here.

Echophenomena/Imitation behaviour

Much acquired human social behaviour is imitative in origin, both adaptive and maladaptive, but in neurological practice the term "imitation behaviour" is reserved for the reproduction by the patient of the examiner's words or gestures without preliminary instruction to do so ("naive imitation behaviour") or even despite explicit instruction not to do so ("obstinate imitation behaviour").[2] The term echophenomena has sometimes been used interchangeably with imitation behaviour.

To be labelled as such, the behaviours must be consistent and, as implied in the "obstinate" terminology, have a compulsive quality to them. Echophenomena may be accompanied by frontal release signs and utilisation behaviour (another reflection of environmental dependency), and are usually attributed to frontal lobe dysfunction, though have been associated on occasion with either basal ganglia or thalamic lesions, and exceptionally with parietal lesions.

Kahlbaum's 1874 description of catatonia included the symptoms of echophenomena, and echolalia and echopraxia feature amongst the symptoms listed in the criteria for catatonia in DSM-5. Obstinate imitation behaviour has been reported to distinguish frontotemporal dementia from Alzheimer's disease,[3] but I think this is likely to be a specific (few false positives) but not very sensitive (many false negatives) sign.

Echolalia

Echolalia is the involuntary repetition of an interviewer's speech utterances (as opposed to the voluntary mickey-taking which characterises an irritating game typical of childhood, but sometimes indulged in by adults). As well as frontal lobe lesions, catatonia, and dementia syndromes, echolalia may also be encountered in children with autism, in Tourette syndrome,[4] and rarely as an ictal phenomenon, possibly with a left supplementary motor area origin.[5]

Echolalia may also occur in certain aphasia syndromes, for example in transcortical sensory aphasia, a fluent aphasia with well-preserved repetition skills. The aphasia of Alzheimer's disease has sometimes been likened to transcortical sensory aphasia, and a "mixed transcortical aphasia" with echolalia has been reported in Creutzfeldt-Jakob disease.

In contrast, "effortful echolalia" has been reported in left medial frontal lobe infarction including the supplementary motor area with a non-fluent output typical of transcortical motor aphasia.[6]

In "dynamic aphasia" speech output is characterised by a difficulty in initiation, with the phenomenon of "incorporational echolalia" when the patient uses the examiner's question to help to form an answer. This has sometimes been conceptualised as a form of transcortical motor aphasia, and may sometimes be seen in progressive supranuclear palsy.

Echopraxia

Echopraxia is the involuntary repetition of an interviewer's movements or gestures. As with echolalia, this may be seen in frontal lobe disorders, catatonia, Tourette syndrome,[4] and rarely as an ictal phenomenon.[5] A mechanism for echopraxia in schizophrenia has been postulated which invokes activity in mirror neurons providing representation to the inferior frontal gyrus and motor cortex which becomes an executed movement due to decreased inhibition and/or increased arousal.[7]

Echolocation

An entirely separate echophenomenon is echolocation.

Visiting the Liverpool Asylum for the Blind in 1805, the American chemist Benjamin Silliman (1779-1864) reported:

"How ... can we account for the acuteness of hearing which enabled a particular blind man, by means of the echo produced by his whistling, to decide when he was approaching any object of some magnitude ...".[8]

Echolocation is the comparison of outgoing sound pulses with the returning echoes in order to navigate or hunt. Though echolocation is most familiar (and studied) in bats and dolphins,[9] some blind individuals have developed the ability to use self-generated sounds, such as tongue clicks or finger snaps, as a form of sensory substitution to perceive the environment (Youtube has some informative videos). Sighted individuals can also be trained to do this.

A possible answer to Silliman's question has been provided by a functional MR imaging study of two blind echolocators which found that calcarine ("visual") but not auditory cortex was activated when the subjects listened to recordings of echolocation clicks and echoes, suggesting a possible role for cross-modal brain plasticity in the development of this faculty of compensatory enhancement.[10] It would be interesting to learn if this was also the case in sighted individuals trained to echolocate.

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