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Thursday

Processing of acoustically degraded emotional prosody in primary progressive aphasia and Alzheimer's disease

Jess Jiang, Mai-Carmen Requena-Komuro, Elia Benhamou, Jeremy Johnson, Harri Sivasathiaseelan, Anthipa Chokesuwattanaskul, Lucy Russell, Jason Warren, Chris Hardy

State of the art: Accurately interpreting a speaker's nonverbal emotional vocal signals is often essential for successful communication. In the noisy conditions of daily life, this presents the brain with a difficult computational challenge that may be vulnerable to neurodegenerative pathologies. However, processing of acoustically degraded prosody has not been studied in these diseases.
Methodology: Here we addressed this issue in patients representing all major syndromes of primary progressive aphasia (PPA) (N=33) and Alzheimer's disease (AD) (N=18) versus healthy age-matched controls (N=24). As a model paradigm for the degraded 'noisy' speech signals of everyday life, we used noise-vocoding: digital division of the speech signal into a variable number of frequency channels constituted from amplitude-modulated white noise (fewer channels convey less spectrotemporal detail and reduce intelligibility). We assessed the impact of noise-vocoding on recognition of three canonical prosodic emotions (anger, surprise, sadness) at three noise-vocoding levels (6, 12, 18 channels) using cost and information transfer analysis.
Results: Compared with healthy controls, all patient groups were impaired recognising prosodic emotions in clear speech, while patients with AD and logopenic variant PPA showed a disproportionate cost for recognition of the noise-vocoded emotion of sadness. AD patients displayed a significantly higher degree of confusion than did healthy controls for all noise-vocoded stimuli.
Conclusion: Our findings open a window on a dimension of emotional communication that has been relatively overlooked in dementia, and suggest a novel candidate paradigm for investigating and quantifying this systematically.

Conflicts of interest

N/A