Heroin and malignant coprolalia in Tourette's syndrome / Comment
Marcelo L Berthier; Victor M Campos; Jaime Kulisevsky; Juan A Valero
The Journal of Neuropsychiatry and Clinical Neurosciences; Winter 2003; 15, 1; Health & Medical Complete pg. 116

LETTERS

could not perform the actual series of steps.

Comment
Acaculia commonly results from disturbances in the left parietal cortex but can result from abnormalities in prefrontal and other locations.\(^\text{3}\) Infarction in the territory of the left tuberothalamic artery can produce acaculia from involvement of the ventral lateral and dorsomedial thalamic nuclei with sparing of other nuclei,\(^\text{4}\) similar to the findings in our patient.

We analyzed our patient’s acaculia. There was no evidence of alexia or agraphia for numbers or other specific numerical problems. He could perform most simple mathematical operations, but he had difficulty in performing complex or multistep operations. Along with his initial difficulty with serial reversal and mental control tasks, his examination suggested a form of acaculia stemming from difficulty with working memory. Recent research has documented the role of working memory in performing multistep, complex calculations,\(^\text{5}\) and lesions in the left thalamus, as well as dorsolateral frontal regions, could impair working memory.

Mario F. Mendez, M.D., Ph.D.
Nora C. Papasian, M.D.
Gerald T.H. Lim, M.D.
Departments of Neurology and Psychiatry & Biobehavioral Sciences, University of California at Los Angeles, VA Greater Los Angeles Healthcare Center, Los Angeles, CA

References

Heroin and Malignant Coprolalia in Tourette’s Syndrome

SIR: Coprolalia, the involuntary emission of socially unacceptable or obscene sounds, words, and utterances, is a typical symptom of Tourette’s syndrome (TS).\(^\text{3}\) Rarely, coprolalia can adopt a malignant character because of the uncontrollable emission of obscenities and reactivity to multiple pharmacological agents. We report the emergence of malignant coprolalia and motor tic exacerbation in a woman with TS in association with chronic heroin abuse.

Case Report
The patient was a 37-year-old woman in whom repetitive phonic tics (“ih”) had developed when she was 9. Phonic tics had a fluctuating course thereafter, and at the age of 15 she developed eye movements, mouth opening, neck jerks, bilateral shoulder shrugging, abdominal tensing, touching rituals, and mild self-injurious behavior. The patient rarely pronounced obscene words, and she added letters and changed the accentuation of words to camouflage their obscene content. She also had obsessive-compulsive behavior and borderline personality disorder. She received haloperidol for 3 years with partial control of tics. At the age of 25 she began to smoke heroin once a week to alleviate puerperal dysphoria. Three months later, she experienced an uncontrollable increase in motor tics and began to emit, for the first time, loud phonic tics (cough) and obscenities.

Obscene words and phrases erupted in a distinctly louder tone and with a lower pitch and more obvious hoarseness than the patient used in a normal conversation, were unsuppressible voluntarily, and occurred very frequently (about 10 words or phrases per minute). She was admitted to an inpatient detoxification program for 6 months, during which she stopped smoking heroin. However, coprolalia and motor tics did not improve, and trials of haloperidol, sulpiride, clonazepam, tetramazaine, risperidone, and olanzapine were ineffective. An otolaryngological examination revealed Reinke’s edema due to vocal abuse, and microsurgical decortication of the vocal cords was performed. During the surgical procedure, 30 units of botulinum toxin were injected in the left vocal cord,\(^\text{3}\) but this treatment failed to suppress coprolalia. She continued to abstain from illicit drugs, but 6 months later she smoked a heroin mixture and was readmitted in a tic status (incessant coprolalia and violent motor tics) that required sedation with a propofol infusion. The patient’s condition partially improved, and she was discharged on a regime of sulpiride 600 mg/day and clonazepam 4 mg/day without adequate tic control.

Comment
Converging evidence suggests overlapping biological mechanisms between TS and associated drug abuse.\(^\text{3}\)\(^\text{4}\) Motor tics and vocalizations in TS are thought to be the result of disinhibition of the dopaminergic system.\(^\text{1,2}\) Coprolalia has

116

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
been associated with increased metabolic activity of the left language-related corticostriatal network (perisylvian cortex, cingulate gyrus, basal ganglia, thalamus), which is modulated by dopaminergic projections from the ventral tegmental area (VTA).\(^3\) The mesolimbic system also has a key role in the acquisition of addictive behaviors,\(^4,5\) and PET studies in opiate addict males during heroin injections or exposure to heroin-related visual cues showed activation of reward circuitry, including the VTA and its target regions (dorsal raphe, nucleus accumbens, extended amygdala, insula, dorsolateral prefrontal cortex, and cingulate gyrus),\(^4\) which themselves are also critical for the clinical expression of tics and impulsive-compulsive behaviors in TS.\(^3\) Thus, uncontrollable coprolalia, motor tic exacerbation, neuroleptic refractoriness, and relapse to heroin-seeking behavior in our patient might be due to opiate-induced long-term changes in mesocorticolimbic systems.\(^3,5\)

Based on the evidence from previous cases reporting temporary exacerbation or recurrence of tic symptoms after cocaine use\(^2\) and from the long-term consequences in our case even after heroin discontinuation, it seems that having TS entails an inherent vulnerability for addictive, impulsive, and compulsive behaviors (reward deficiency syndrome)\(^6\) and exacerbation of motor-phonic tics after illicit drug exposure. Individuals with TS should be advised about the devastating effect that heroin and other illicit drugs may have on the severity and long-term evolution of tic symptoms.

\[\text{References}\]

2. Cardoso FE, Jankovic J: Cocaine-induced movement disorders. Mov Disord 1993; 8:175-178

\[\text{On Bresch's Glossary of Eponyms}\]

\[\text{SIR:}\] Mark Twain famously wrote, "The reports of my death have been greatly exaggerated." Equally exaggerated is David Bresch's claim\(^1\) that "no one has ever composed a glossary of psychiatry's numerous eponyms." After many years of work, in 1999 we published a book on the subject.\(^2\) In writing this book we used the principal medical and psychiatric dictionaries in English, French, German, and Spanish, as well as varied bibliographical sources and the main biomedical (MEDLINE, Embase, etc.) and genetic (OMIM) databases. We also traveled to the main Spanish librar-