



Probabilistic classification learning in Tourette syndrome

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Received 31 May 2001; received in revised form 10 October 2001; accepted 10 October 2001

Abstract

Tourette syndrome (TS) is characterised by stereotyped involuntary movements, called tics. Some evidence suggests that structural and functional abnormalities of the basal ganglia may explain these motor symptoms. In this study, the probabilistic classification learning (PCL) test was used to evaluate basal ganglia functions in 10 children with less severe tics (Yale Global Tic Severity Scale (YGTSS) scores < 30) and in 10 children with more severe symptoms (YGTSS score > 30). In the PCL task, participants are asked to decide whether different combinations of four geometric forms (cues) predict rainy or sunny weather. Each cue is probabilistically related to a weather outcome, and feedback is provided after each decision. After completion of the probabilistic stimulus-response learning procedure, subjects received a transfer test to assess explicit knowledge about the cues. The children with TS exhibited impaired learning in the PCL task in comparison with the 20 healthy control subjects. This impairment was more pronounced in the TS patients with severe symptoms, and there was a significant negative relationship between the final classification performance and the YGTSS scores. The patients showed normal learning in the transfer test. These results suggest that the neostriatal habit learning system, which may play a central role in the acquisition of probabilistic associations, is dysfunctional in TS, especially in the case of more severe motor symptoms. The classification performance and the severity of tics were independent of the explicit knowledge obtained during the test. © 2002 Published by Elsevier Science Ltd.

Keywords: Tourette syndrome; Probabilistic classification learning; Basal ganglia; Memory; Tics

1. Introduction

Tourette syndrome (TS) is characterised by involuntary, rapid, non-rhythmic skeletal movements and sounds. Simple vocal tics include throat clearing, whistling, snorting, barking, growling, whereas complex vocal tics consist of words and phrases sometimes with obscene or aggressive content. In a similar vein, simple motor tics can be observed in the form of eye blinking, grimaces and jerks, whereas complex motor tics involve stereotyped facial expression, grooming, touching, hopping, banging and so on [8]. Involuntary and non-purposeful muscle activity in TS suggest a primary disturbance in motor control, although there is also a marked impairment in affective and cognitive regulation [6]. In addition, TS shows a great degree of comorbidity with attention-deficit hyperactivity disorder (ADHD) and obsessive-compulsive disorder (OCD) in which intrusive and non-voluntary thoughts and emotions are clearly present [7].

Despite an extensive research effort, the neuronal basis of TS remains to be elusive. Consistently with the widespread hypothesis of subcortical dysfunction, studies using structural magnetic resonance imaging (MRI) found decreased volume of the left basal ganglia [30,37]. However, the specificity of this structural abnormality is called into question by the studies reporting abnormal size of the corpus callosum and enlarged right lateral ventricle [15,31]. In addition, the magnitude of volumetric abnormalities is small (about 5%) and these are not consistently replicated [26]. Regional cerebral blood flow measurements using single photon emission tomography (SPECT) and positron emission tomography (PET) revealed even more heterogeneous results: abnormal blood flow was found not only in the basal ganglia, but in widespread cortical regions including the lateral prefrontal cortex, anterior cingulate, premotor and supplementary motor areas, parietal and temporal cortex [5,27,34,38]. Based on these findings, suggesting the dysfunction of a large-scale neuronal network, one can expect that several domains of neuropsychological functioning may be affected in TS. Although evidence suggests that attention, executive functions, visuospatial integration and visuospatial functions are impaired in patients with TS, some authors

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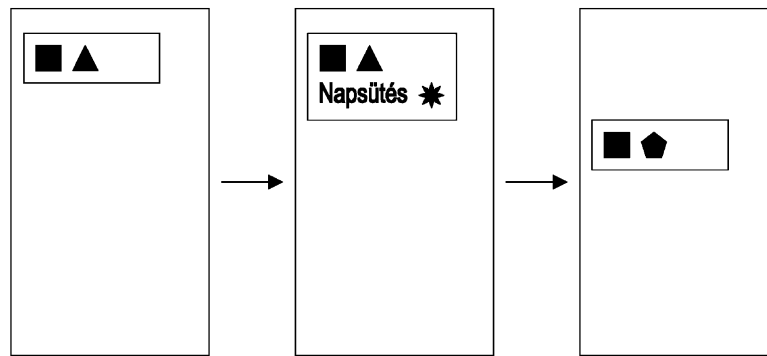


Fig. 1. The PCL procedure. First, subjects are presented with a stimulus (first frame) and are asked to decide whether the stimulus predicts sunny or rainy weather outcome. After the decision, the window moves downward and verbal (Hungarian words) and symbolic feedbacks appear (second frame). Additionally, the experimenter provides the correct answer orally. Finally, the window moves again and the next stimulus is shown (third frame).

reported only mildly affected or normal performances in several neuropsychological tests [3,11,12,29,36,40]. It has been also shown that compromised cognitive functions are present more frequently in TS patients with severe symptoms and with comorbid ADHD/OCD [14,28]. The main conclusion from the data reviewed above can be that, given the uncertainties of neuroimaging and neuropsychological studies, the central role of basal ganglia impairment in TS awaits further confirmation.

Recently, the probabilistic classification learning (PCL) test has been introduced as a candidate tool for the investigation of parallel memory systems, including learning functions related to the basal ganglia [13,17–20,32,33]. In the PCL task, participants are asked to decide whether combinations of different geometric forms (cues) predict good or bad weather. After each decision, the subject receives a feedback about the correctness of the prediction (Fig. 1). The essence of the test is that each cue is probabilistically related to a particular weather outcome. For example, cue A predicts sunny weather with a high probability, whereas cue B frequently signs rainy weather. It must be emphasised that the cue–outcome associations are not absolute: in a small proportion of trials shape A means rain and shape B means sunshine. The probabilistic nature of cue–outcome relationships disrupts the natural tendency to consciously memorise the categorisation rule, particularly in early phases of learning [13]. The assumption that participants may learn cue–outcome associations implicitly is supported by the finding that amnesic patients with damage to the medio-temporal or diencephalic structures show a normal rate of learning in the PCL task despite their severe explicit memory impairment. Indeed, these patients are unable to consciously recall details about the PCL task and fail to explicitly define the meaning of cues [18,19,33]. In contrast, individuals with basal ganglia disorders such as Huntington's and Parkinson's disease exhibit severe dysfunctions in early phases of the PCL, suggesting that these subcortical structures play a central role in the acquisition of probabilistic stimulus–response associations [19,20].

Therefore, if basal ganglia disturbances are characteristic features of TS, the patients will show impairment in the PCL task. In addition, if explicit memory and executive functions are relatively spared in TS, the patients will perform normally in the transfer test, evaluating explicit knowledge related to the materials presented during the PCL task. These hypotheses were tested in 20 highly functioning children with TS. Inclusion of young participants without a long history of medication helps avoid artefacts due to the possible progression of disease or the long-term effects of antipsychotics.

2. Methods

2.1. Participants

Twenty children (8 males, 12 females) with DSM-IV diagnosis of TS and 20 healthy volunteers (8 males, 12 females) participated in the study [2]. All subjects lived with their families and attended regular schools. The group of children with TS was divided into patients with severe and less severe motor symptoms, which was rated with the Yale Global Tic Severity Scale (YGTSS) [22]. Patients who scored less than 30 on the YGTSS were classified as less severely affected ($n = 10$, mean YGTSS score: 20.8 (S.D. = 7.4)), whereas children who had higher scores than 30 were classified as more severely affected ($n = 10$, mean YGTSS score: 48.2 (S.D. = 17.7)). The criterion of severity was chosen because it was close to the median of the YGTSS scores measured in the whole TS group (31.5). The mean age was 12.3 years (S.D. = 2.2) in the control group, 12.4 years (S.D. = 2.5) in the less severely affected TS group, and 12.9 years (S.D. = 3.7) in the more severely affected TS group. A one-way analysis of variance (ANOVA) revealed no significant differences in age ($P > 0.8$).

The mean IQ, as estimated from the Raven progressive matrices test (RPMT) [24], was 108.1 (S.D. = 10.9) in the control participants, 106.7 (S.D. = 12.3) in the TS patients

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