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APRAXIA: ANOTHER VIEW

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Abstract

A previous article in this journal,[1] offered a view of limb apraxia rooted in a traditional ideational-ideomotor-limb kinetic taxonomy. This note presents an alternative perspective. It argues that apraxia is better understood in terms of breakdown of constituent components of action semantics and spatiotemporal-kinematic control. Such accounts are more compatible with models of CNS functioning. Clinically, because diagnosis is based on recognising underlying breakdowns, the approach enables a focus on what an individual can and cannot do, with direct implications for rehabilitation. This contrasts with earlier practice where diagnosis relied on categorisation of derailments and assignment to apraxia subtypes not supported by neuropsychological or neurophysiological research and where error types claimed to distinguish subtypes showed multiple underlying causes.

Key words: limb apraxia; assessment

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A VIEW OF APRAXIA

A previous exposition of apraxia,[1] offered an account of underlying impairment and accompanying clinical assessment based on a distinction between ideational, ideomotor and limb-kinetic apraxia. This conceptualisation stems from 19th century models of higher cortical motor, language and visual function, revived by Geschwind,[2, 3] in the 1960s. Liepmann,[4] posited a hierarchical model of action control over three discrete levels. At the top, movement formulae ('visual engrams' of the action) provided overall targets. These activated innervatory patterns to stimulate the appropriate muscles. Coordinated contraction of muscles led to execution of the action. Impairment of movement formulae or their disconnection from the innervatory patterns brought about ideational apraxia. Disruption to innervatory patterns resulted in ideomotor apraxia. Limb-kinetic apraxia, assumed to be a disruption to the smooth neural transmission of the motor commands, was even for Liepmann not a full apraxia. It existed between apraxia and paresis only within a broader view of dysfunction.

Liepmann's model proved valuable in studying apraxia. However, despite its survival in several neurology textbooks, the ideational-ideomotor dichotomy has been replaced as a way of understanding and classifying apraxias,[5-8], similar to the disbandment of Broca's vs Wernicke's aphasia or the dichotomy of associative vs apperceptive agnosia,[9] (though these distinctions, too, still persist in some areas of clinical practice). Issues with the underlying theoretical model of apraxia and difficulties in distinguishing between ideational and ideomotor clinically led to a radical rethink, with Goldenberg,[7, p 332] making the plea to 'relegate the dichotomy of ideational and ideomotor to the history of neuropsychology'. Limb-kinetic apraxia should not be thought a pure apraxia, but rather considered a primary sensory-motor dysfunction. In patients with 'limb-kinetic apraxia' conceptual planning is intact, the contextual variability of apraxia is lacking and the clumsiness observed varies depending on fine motor coordination complexity rather than psychomotor complexity. It is confined to distal control and is typically unilateral,[10].

Problems inherent to the ideational -ideomotor (dis)connection model were several. What constituted ‘visual engrams’ and ‘innervatory patterns’, and the relationship between them, was at the very least highly underspecified, at worst misguided. The same applied to how ‘engrams’ and ‘innervation’ might relate to actual brain/CNS function. Clinically, the model forced clinicians to categorise a patient’s problem as either ideational or ideomotor apraxia. As these categories were derived from a theoretical rather than an experimental model many patients showed difficulties that did not conform to the classification. Furthermore, classification rested on observed behaviours (e.g. altered proximity, body part as object, wrong grasp) that might have multiple different underlying causes.

ANOTHER VIEW OF APRAXIA

Alternative perspectives see action planning and control arising from highly interactive, integrated neurophysiological and neuropsychological networks, as opposed to hierarchical discrete level formulations. Accounts have highlighted control networks distributed across the left hemisphere,[11-15], stressing interaction on the one hand with visual and tactile-kinaesthetic perception and feedback, on the other with subcortical, especially extrapyramidal, elements of motor control in initiation, timing, rhythm, force of movements. At their core, alternative accounts see action planning and control as consisting of and dependent on multiple underlying processes. If one could identify these building blocks of control and how they interact, then one could analyse apraxic behaviour in terms of which underlying factor(s) is at fault. This would enable diagnosis of what a person can or cannot do in terms of preservation-impairment of elements of action semantics and spatiotemporal control (see below), rather than assignment to some general category label which provides no specification of the individual’s problem. It would enable rehabilitation to target root causes directly rather than deliver intervention based on broad categories that may or may not apply to the individual and directed at derailments that may have multiple underlying causes.

The quest was therefore to uncover constituents of action planning and control that operated independently of each other within the interactive networks, but that were also compatible with and demonstrated transparent links to underlying neurophysiological processes/networks.

Starting point for building alternative models were systematic observations and testing out of apraxic behaviour to search for components of action that could break down independently of each other,[16-23]. Such studies have highlighted, for instance, individuals who can name objects (so no visual agnosia or aphasia), but are unable to state their use, and/or demonstrate their application. Others, despite recognising usage show an inability to pantomime the action but faultless performance when they handle the object. Another may describe an object and correctly conjecture how it might be employed, but not discern its exact function. Others attempt usage but show wrongly configured grasp, application to the wrong target of the action (e.g. wrong body part, other object), or inappropriate execution (e.g. stirs tea with a dunking motion). Part-actions (e.g. finger, elbow, shoulder extension-flexion) may be possible in isolation but when control of the multiple degrees of freedom across these joints is necessary simultaneously (e.g. to reach, pick up a glass, bring it to the lips) movements may occur incompletely, out of phase, with trial and error attempts, and so forth.

Clinically, too, one would proceed via systematic evaluation across modes of elicitation (e.g. verbal, visual, tactile), contexts of action (e.g. clinic, naturalistic; with-without handling objects) and types of gesture (e.g. transitive-intransitive; meaningful-meaningless), which aspect(s) of action semantics and planning/control is/are impaired. Through this one aims to identify the precise source of difficulty. For example, the source of breakdown in stirring tea may relate to: despite correct naming, not recognising what a spoon is for; not establishing the association cup-spoon; an inability to appropriately grasp and manipulate the spoon; a

contrasts in miming stirring spontaneously compared to imitation, either with and without the tea and spoon present, with and without touching them. Elucidation might contrast stirring paint (which one would not dunk a biscuit in) and spooning sugar (different grasp and plane etc). There are several validated bedside and clinical screening tests that facilitate this,[24, 25].

Debate continues on which underlying components to action control precisely one should seek, but by and large processes have been distilled into elements around action semantics/ conceptual components of action (knowledge of function, manipulation knowledge and mechanical problem solving,[8, 22, 23, 27]) and kinematic, spatiotemporal constituents of control, (coordination of the degrees of freedom across multiple joints and the amplitude, trajectory, orientation/plane, spatial proximity/dimensions and relative phasing/timing of subparts of actions,[8, 13-15, 26]) .

The dissociations between conceptual components of action and spatiotemporal constituents of control in turn have been linked to interacting but dissociable neurophysiological networks providing a neuro-anatomical and neurophysiological basis for this approach,[11-13, 15, 22]. Dorsal-dorsal, dorsal-ventral and ventral neuroanatomical streams have been identified linked to the knowing ‘where’ and ‘how’ (dorsal) and ‘what’ (ventral) of action control, with further subdivision of the dorsal stream into ‘grasp’ and ‘use’ systems,[11, 15].

This more individual approach has more than theoretical relevance. It identifies the root cause(s) of the individual’s difficulty with action, and through this facilitates a more targeted rationale for intervention,[28]. It also opens up the possibility to explore different characteristic profiles of praxic breakdown across neurological disorders,[29].

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