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Refractoriness and the healthy brain: A behavioural study on semantic access

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ABSTRACT

While many behavioural studies on refractory phenomena in lexical/semantic access have focused on the mechanisms involved in the oral production of names, comprehension tasks have been almost exclusively used in neuropsychological studies on brain damaged patients. We report the results of two experiments on healthy participants conducted by means of speeded word to picture matching tasks. They assess the effects of the same variables examined in the study of refractory access dysphasic patients: semantic distance and word frequency (experiment 1) and presentation rate and serial position effects (experiment 2). Semantic access patients usually show little effect of word frequency but a large semantic distance effect. However, critical in characterising the syndrome as 'refractory', effects of presentation rate and serial position should also be present. The experiments involved the use of a deadline response procedure. The critical manipulation was the absence of a Response Stimulus Interval (RSI) in the fast presentation rate conditions; slower presentation rates involved 1 s RSI. With these manipulations the typical behavioural pattern of performance provided by semantic access dysphasic patients was reproduced. Semantic distance effects were more powerful than word frequency effects (experiment1). Presentation rate effects were found and, most important for a "refractory" account of the effects, a serial position effect was obtained (experiment 2). These results provide the first evidence of such a broad range of refractory effects at the same time in comprehension tasks in healthy subjects and support a purely semantic account for the locus of refractoriness. Moreover, error analysis showed a predominance of perseverative errors with subsequent representations of the same target, supporting a failure of cognitive control mechanisms as the cause of refractory behaviour. The findings are discussed in the light of current models of lexical and semantic processing.

1. Introduction

A number of types of semantic memory impairment can be caused by neurological diseases. Two major contrasting forms are those resulting in degradation of semantic representations (Warrington, 1975), as in the case of semantic dementia (Hodges, Patterson, Oxbury, & Funnel, 1992; Mummery et al., 1999; Snowden, Goulding, & Neary, 1989), and those resulting in difficulties in accessing semantic representations (Warrington & Cipolotti, 1996; Warrington & McCarthy, 1983; Warrington & McCarthy, 1987; Warrington & Shallice, 1979).

A number of criteria have been held to be useful to assess whether a semantic impairment is due to the degradation of the semantic representations or to a problem in accessing (as in access dysphasia) mostly intact stored representations (Warrington & McCarthy, 1983; Warrington &

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McCarthy, 1987; but see Rapp & Caramazza, 1993 for a criticism on this position; Warrington & Cipolotti, 1996). For instance, in the Warrington & Cipolotti study, patients held to be of an access type, were found to be inconsistent in their performance on individual items. They were also only weakly affected by the frequency of the target word to be retrieved. On the other hand, the semantic distance between the target and the distractors presented played an important role in the impairment of these patients: they had greater problems in selecting between semantically related stimuli than more distant ones. In general, semantic distance effects were found to be much larger in these patients than effects of word frequency (which were rarely significant). Moreover the rate of presentation of the stimuli significantly influenced the performance of semantic access patients: patients performed better when the interval between presentations (Response Stimulus Interval, 'RSI') was longer. Finally, repeated presentations of the same set of target stimuli led to a progressive deterioration in their performance (see also Cipolotti & Warrington, 1995; Forde & Humphreys, 1995; Forde & Humphreys, 1997; Warrington & Crutch, 2004 for similar findings).

On the other hand, when the complementary pattern of performance is found, this is held to arise from degradation of semantic representations themselves: such patients are consistent in their likelihood of retrieving concepts; they are strongly affected by word frequency and much less by semantic distance; they are not influenced by the rate of presentation and their performance tends not to deteriorate in time (Warrington & Cipolotti, 1996).

1.1. Refractory behaviour in patient populations

The cause of a specific "semantic access" deficit has been linked to abnormal refractoriness within the semantic system. In this context, refractoriness is defined as "the reduction of the ability to utilize the system for a certain period of time following activation" (Warrington & McCarthy, 1983, p. 874). In this formulation, refractoriness is assumed to be a normal neural state which is abnormally prolonged in these patients and this could potentially explain all the effects linked to the typical semantic access pattern of impairment. If, following an initial successful accessing of the meaning, the target representation falls into an abnormally prolonged refractory state, the faster the presentation rate of the stimuli, the higher the probability of dysfunctional access (presentation rate effect). Moreover, if the duration of refractoriness exceeds the interval between two of the same stimuli of a set in a series, further attempts to access the same concept will lead to a decrease in the probability that concept will be correctly accessed (serial position effect). Furthermore, many computational models of the lexical system assume that when a given target is activated, some activation spreads to representations of neighbouring concepts. If this also happens in the context of the abnormal refractoriness of the system, then concepts that are semantically related to the previously accessed one will be more difficult to access, while unrelated concepts will still be relatively easily accessed (semantic distance effects). Finally, the weakness of the word frequency effect could be explained by the high frequency concepts being assumed to have richer and more interrelated representations in which more synapses are involved. In this situation, refractoriness would affect the synapses of high frequency concepts more than those of low frequency ones. This effect would therefore work against the normal frequency effect (Crutch & Warrington, 2005).

Genuine refractory behaviour is indicated by a sensitivity to temporal factors such as the rate of presentation and especially the serial position effect. Indeed semantic distance effects have also been reported in the absence of a clearly refractory symptom pattern (e.g. Gotts, Incisa Della Rocchetta, & Cipolotti, 2002; Warrington & Leff, 2000 in the context of lexical access; Crutch and Warrington (2005) in the context of degradation syndromes; Warrington & Cipolotti, 1996; Campanella, Mondani, Skrap, & Shallice, 2009 in the context of semantic access problems).

However in these cases the problem is generally attributed to a deficit occurring outside the semantic system itself. By contrast, in all refractory semantic access syndromes reported in the neuropsychological literature, the semantic system itself has been presumed to be the locus of the refractory behaviour. For example in Warrington and Cipolotti (1996) and in Forde and Humphreys (1997) the locus of damage was held to be directly within the semantic system, since the performance of the patients was unimpaired in all presemantic tasks such as visuoperceptive matching tasks. In other studies, the semantic system was held to be indirectly influenced through the failure of cognitive selection mechanism (Forde & Humphreys, 1997; Forde & Humphreys, 2007; Jefferies, Baker, Doran, & Ralph, 2007; Schnur, Schwartz, Brecher, & Hodgson, 2006; Wilshire & McCarthy, 2002) or through the putative breakdown of neuromodulatory systems controlling physiological synaptic depression dynamics (Gotts & Plaut, 2002).

According to the 'frontal selection' account of Jefferies and Lambon Ralph (2006), refractory behaviour in semantic access difficulties is explained by inadequate functioning of a selection mechanism, held to be in the lateral inferior prefrontal cortex, which is used by the cognitive system to resolve the competition between coactivated semantic competitors during highly demanding tasks (see e.g. Badre & Wagner, 2007). Therefore the competition could arise within the semantic system itself, but be modulated by the action of an external system (LIPFC), which acts as an active selection mechanism.

On the other hand, a more 'automatic' account of the resolution of semantic access conflict is given by the 'neuromodulation' account (Gotts et al., 2002). According to this position, efficient access to concepts is supported by a number of neuromodulatory systems acting to minimize the effects of physiological refractory processes which are also operating in the healthy brain. In particular it has been suggested that acetylcholine reduces the probability of transmitter release in presynaptic neurons while, at the same time, it blocks the adaptation of post synaptic cells to the repetitive firing (firing rate adaptation) which occurs after repeated stimulation of the same synapse, so making the synapse more efficient and functional for a longer time (e.g. Hasselmo & Bower, 1992; Tsodyks & Markram, 1997).