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The role of mirror neurons in speech perception and action word semantics

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The discovery of mirror neurons in the macaque monkey has ignited intense interest in motor theories of cognition, including speech and language. Here we examine two such claims, that the perception of speech sounds critically depends on motor representations of speech gestures (the motor theory of speech perception) and that the representation of action-related semantic knowledge critically depends on motor representations involved in performing actions. We conclude that there is strong evidence *against* the claim that speech perception critically depends on the motor system and that there is no conclusive evidence in support of the view that the motor system supports action semantics. We propose instead that motor-related activity during perceptual processes stem from spreading activation in sensory-motor networks that are critical for speech and language production.

Keywords: Mirror neurons; Speech perception; Action semantics; Aphasia.

Mirror neurons, cells originally found in the cortical motor system of monkeys (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992; Gallese, Fadiga, Fogassi, & Rizzolatti, 1996), have come to be associated with two ideas in speech/language processing. One is the hypothesis that the perception of speech *sounds* is critically dependent on the activation of motor programs underlying speech gestures (Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Liberman & Mattingly, 1985). The other is the hypothesis that the representation of body-related, action word *meaning* is, in part, dependent on motor representations of body-related actions

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(Pulvermuller, 2001). Although these ideas are related in that they are both a form of motor theory to some extent, they are logically independent—the validity of one hypothesis is neither supported nor refuted by the validity of the other. The goal of the present paper is to critically review the evidence for these two hypotheses and their connection to mirror neurons as well as to provide an alternative account of apparent motor involvement in perceptual and conceptual semantic processing (see (Toni, de Lange, Noordzij, & Hagoort, 2008) for an excellent discussion of these issues in a similar spirit). Specifically, it is proposed that motor systems are activated not because they are the basis of perceptual "understanding" but rather because the reverse relation holds, that sensory/conceptual systems can inform action. This is true at the articulatory-phonetic level where acoustic feedback control of speech production has been convincingly demonstrated and at the conceptual semantic level where strong associations are no doubt formed between conceptual representations of actions and specific motor plans. Thus, hearing the word *kiss* activates motor lip systems not because you need lips to understand *kiss* (c.f., actions that humans cannot perform such as those of animals, *fly*, *slither*, *echolocate*, of plants, *bloom*, *root*, and of inanimate matter, *implode*, *evaporate, etc.*) but because activation of the meaning of *kiss* causes spreading activation to motor systems controlling lip-related actions due to prior association.

MIRROR NEURONS AND THE MOTOR THEORY OF ACTION UNDERSTANDING

Here we provide a brief review of mirror neurons and their proposed role in action understanding generally. This provides the foundation for considering the role of mirror neurons in speech/language processes. For more detailed discussion see (Hickok, 2009a).

Mirror neurons, which fire both during action execution (e.g., grasping) and during action observation, were originally discovered in area F5 of the macaque monkey (di Pellegrino et al., 1992; Gallese et al., 1996). Other types of cells in F5 also have sensory-motor response properties; for example, some respond both during grasping and during passive viewing of objects (Rizzolatti et al., 1988). However, only mirror neurons have been widely interpreted as supporting perceptual recognition functions ("action understanding"). F5 cells that respond to visually presented objects are not considered by most theorists to support "object understanding", but instead as a means for sensory information to access a "motor vocabulary" that is represented in F5 (Nelissen, Luppino, Vanduffel, Rizzolatti, & Orban, 2005; Rizzolatti et al., 1988; Figure 1).

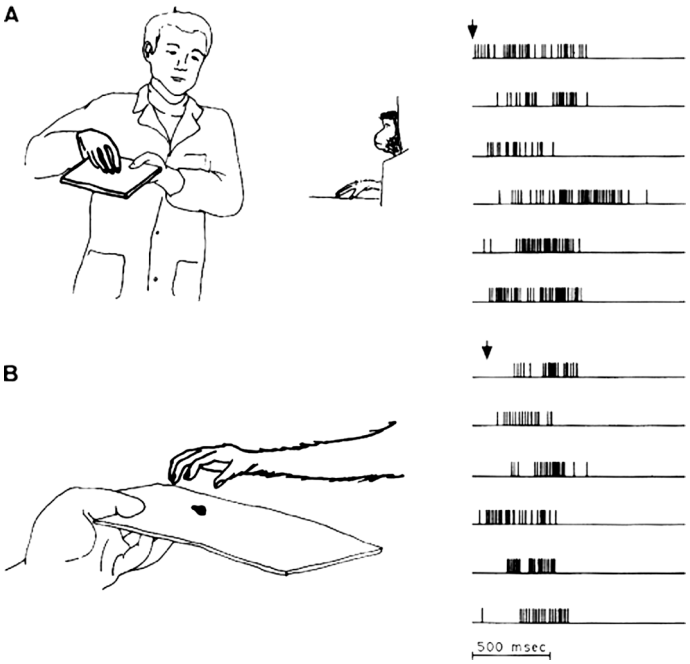

Figure 1. Example of a mirror neuron in monkey area F5 during action observation (A) and action execution (B). Arrows denote the onset of movement. Figure from Rizzolatti and Fabbri Destro (2008).

Mirror neurons have subsequently been discovered in portions of the monkey parietal cortex (Gallese, Fogassi, Fadiga, & Rizzolati, 2002) as well as primary motor cortex (Tkach, Reimer, & Hatsopoulos, 2007). The latter finding is problematic for the action understanding interpretation of mirror neuron function because it is consistent with a less interesting interpretation, namely that "mirror" responses reflect motor preparation (early mirror neuron experiments attempted to rule out this possibility by showing that primary motor cortex did not contain mirror neurons (Gallese et al., 1996)).

The hypothesis that mirror neurons support action understanding has never been directly tested in monkeys. Available data only provide evidence for an *association* between action observation and neurophysiological responses in the motor system of monkeys. A direct test of the theory would involve functionally disrupting the mirror system and demonstrating an impairment in action understanding. Although it is possible to produce deficits in action execution by disrupting function in regions containing mirror neurons (Fogassi et al., 2001), no studies have reported on the perceptual effects of this critical manipulation.

MIRROR NEURONS IN HUMANS?

The existence of mirror neurons in humans has been inferred on the basis of a variety of findings. One is the demonstration that viewing an action can result in motor potentiation in the viewer: the amplitude of the electromyographic (EMG) response in distal muscles induced by motor cortex stimulation via transcranial magnetic stimulation (TMS) is enhanced by action observation (Fadiga, Fogassi, Pavesi, & Rizzolatti, 1995). Another source of evidence for the existence of mirror neurons in humans comes from functional imaging studies that have found motor-related activation during the perception of action (Decety et al., 1997; Grezes, Costes, & Decety, 1998; Iacoboni et al., 1999; Koski, Iacoboni, Dubeau, Woods, & Mazziotta, 2003; Koski et al., 2002). A final source of evidence comes from neuropsychology, apraxia in particular. Apraxia is a disorder of voluntary movement that is associated with fronto-parietal lesions (Heilman & Gonzalez Rothi, 1993). A subset of apraxic patients also have pantomime recognition deficits (Heilman, Rothi, & Valenstein, 1982), which has been taken as evidence for overlap in the neural systems supporting action execution and action understanding (Gallese et al., 1996). Some studies of apraxic patients have linked action recognition deficits to the left posterior inferior frontal gyrus (Pazzaglia, Smania, Corato, & Aglioti, 2008), which is a candidate for a homologue of monkey area F5 (Rizzolatti & Craighero, 2004).

While this appears to be an impressive collection of evidence, none of it is particularly compelling (Hickok, 2009a). The TMS/EMG studies, as well as the functional imaging studies demonstrate correlation not causation, and further, do not rule out a motor preparation or motor priming interpretation of the findings. The neuropsychological evidence is potentially more convincing. However, there are several problems with this literature. One is that mirror neurons in monkeys do not respond to pantomimed actions (Gallese et al., 1996), thus studies of pantomime recognition deficits in humans are, by definition, not investigating mirror neurons as they are understood in the monkey (i.e., where detailed neurophysiological data and relevant control experiments are available). One can make the assumption that mirror neurons in humans have evolved to support pantomime recognition, but this assumption requires empirical justification. Another problem is that the same neuropsychological literature attests to dissociations between action execution and action understanding (Mahon & Caramazza, 2008), thus providing evidence *against* the mirror neuron-based claim. A third problem is that some studies implicate posterior parietal areas as the critical region associated with pantomime recognition deficits (Buxbaum, Kyle, Grossman, & Coslett, 2007; Buxbaum, Kyle, & Menon, 2005). While parietal regions are considered part of the mirror system (Rizzolatti & Craighero, 2004), this result would indicate that damage to the

motor "core" of the system in the frontal lobe may not produce the predicted action understanding deficits. Finally, it is currently a matter of debate whether mirror neurons exist at all in humans (Dinstein, 2008; Dinstein, Hasson, Rubin, & Heeger, 2007; Lingnau, Gesierich, & Caramazza, 2009).

Thus, while it is widely claimed that mirror neurons are the basis for action understanding, there is in fact very little evidence to support this hypothesis, and indeed important evidence against it (Hickok, 2009a; Mahon & Caramazza, 2008).

MOTOR THEORIES OF SPEECH SOUND PERCEPTION

The motor theory of speech perception holds that speech sounds are not recognised on the basis of auditory representations, but on the basis of the motor representations that underlie speech gestures (Liberman & Mattingly, 1985; Liberman et al., 1967). The motor theory of speech perception has few remaining supporters within the field of speech perception (Galantucci, Fowler, & Turvey, 2006), but owing to its resonance with mirror neurons, has become quite popular outside the field (Fadiga & Craighero, 2006; Rizzolatti & Arbib, 1998). For example, Fadiga and Craighero (2006) note, "Liberman's intuition...that the ultimate constituents of speech are not sounds but articulatory gestures...seems to us a good way to consider speech processing in the more general context of action recognition" (p. 489). Indeed, the motor theory of speech perception was noted in the earliest empirical mirror neuron papers and thus served both as the cognitive foundation for the generalisation of the mirror neuron theory of action understanding to human speech, and arguably as indirect evidence for the validity of the mirror neuron theory of action understanding itself:

> ... the mechanism matching action observation and execution ... is very similar to that proposed by Liberman and his colleagues for speech perception .... According to this theory, the objects of speech perception are not to be found in the sounds, but in the phonetic gesture of the speaker, represented in the brain as invariant motor commands .... Considering the homology between monkey F5 and human Broca's area, one is tempted to speculate that neurons with properties similar to that of monkey 'mirror neurons', but coding phonetic gestures, should exist in human Broca's area and should represent the neurophysiological substrate for speech perception. (Gallese et al., 1996, p. 607)

Motor areas involved in speech production have been found to activate also during the perception of speech sounds (Wilson, Saygin, Sereno, & Iacoboni, 2004)—although not always in Broca's area (Figure 2)—which is consistent with the motor theory of speech perception. However, like

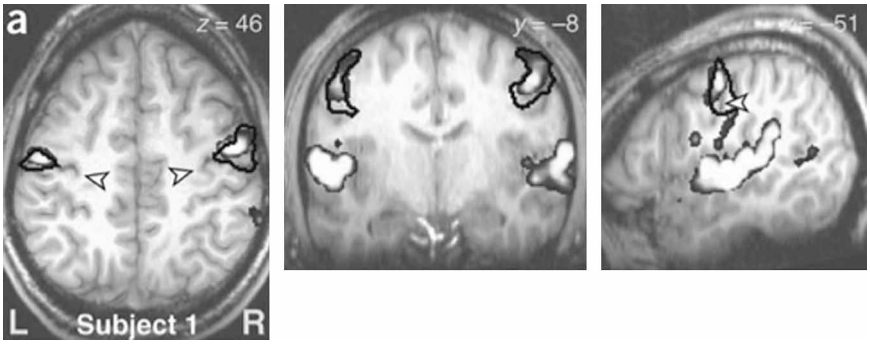

Figure 2. Activation of motor speech areas during speech perception. Black outlines show regions activated during speech production whereas white blobs indicate activation during speech perception. The largest effect in this study was found in premotor cortex, dorsal to Broca's area. Figure from Wilson et al. (2004).

physiological recordings of mirror neurons themselves, functional activations can only show correlations, not causation. For this we need neuropsychological evidence.

MOTOR THEORY OF SPEECH PERCEPTION: EVIDENCE FROM APHASIA

But is there any evidence in support of a motor theory of speech perception? There is certainly good behavioural evidence for the existence of a tight connection between speech perception and speech production (see recent review in Galantucci et al., 2006), and there are numerous demonstrations that neural activity in the motor and perceptual speech systems are tightly coupled (Hickok, Buchsbaum, Humphries, & Muftuler, 2003; Wilson et al., 2004; Zatorre, Evans, Meyer, & Gjedde, 1992), but neuropsychological evidence clearly indicates that any strong version of a motor theory of speech perception is incorrect (Hickok, 2009a; Lotto, Hickok, & Holt, 2009).

Motor theories of speech perception predict that disruption of the motor speech system should cause concomitant deficits in speech perception. This is not the case. Patients with Broca's aphasia often have very large lesions involving the left motor speech system and can have very severe speech production deficits (Naeser, Palumbo, Helm-Estabrooks, Stiassny-Eder, & Albert, 1989), yet are quite capable of processing speech sounds as evidenced by their preserved word-level comprehension (Damasio, 1992; Goodglass, 1993; Goodglass & Kaplan, 1983; Hillis, 2007). If the neural system supporting speech production were critical to speech recognition, Broca's aphasia should not exist.

Relatively preserved speech perception ability following left frontal damage has also been demonstrated using discrimination tasks in which pairs of words or non-words are presented auditorily and aphasics are asked to judge whether the two items are the same or different (different items typically differ by a single phonetic feature). One study reports an average accuracy at 95% or above in six Broca's aphasics for word and non-word versions of such a task (Blumstein, Baker, & Goodglass, 1977). In another paper involving eight Broca's aphasics, accuracy is not reported numerically but is presented in graph form (Baker, Blumsteim, & Goodglass, 1981) from which an average accuracy level across all discrimination types (voicing feature, place feature, and voice + place) of approximately 95% can be calculated. Even on the most difficult discrimination condition, place contrasts, average accuracy is approximately 85% correct. But this value underestimates discrimination performance because percent correct is contaminated with response bias. When this value is corrected for response bias by calculating d', one finds a value of 4.8 which indicates excellent discrimination performance (zero = chance discrimination and d' values can be interpreted in a manner similar to z-scores with integer units representing standard deviations above zero). Figure 3 presents place-contrast discrimination performance in the Baker et al. study in both percent correct and A′ units for Broca's aphasics. A′ is a measure of the area under the ROC curve and therefore approximates proportion correct once bias is corrected. Note the near ceiling performance.

Some classic large-scale studies on the speech perception abilities of aphasics are sometimes cited as evidence for the role of frontal/motor systems in speech perception. For example, Miceli, Gainotti, Caltagirone, and Masullo (1980) studied 69 aphasics and categorised them into two

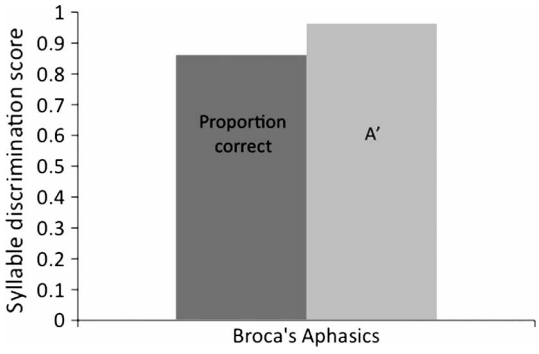

Figure 3. Discrimination performance in a group of Broca's aphasics from (Baker et al., 1981) using both proportion correct and A′ measures. A′ is a response bias corrected measure of area under the ROC curve and approximates proportion correct. d' value for these same data was 4.8. Note that proportion correct underestimates discrimination ability and that Broca's patients are performing near ceiling. Performance values were estimated from graph in Baker et al. (1981).

groups, those with phonemic output disorders (phonemic errors in speech production) and those without. Patients *with* a phonemic output disorder made more errors than patients *without* a phonemic output disorder on a same–different, non-word syllable discrimination task. This may be taken as evidence for a relation between speech production and speech perception. However, there are two problems with such an interpretation. First, the group of patients with a phonemic output disorder included both fluent and non-fluent patients. The former group could have lesions that completely spare the mirror system and judging from other data, fluent patients such as Wernicke's aphasics tend to perform more poorly on syllable discrimination than non-fluent Broca's aphasics (Baker et al., 1981; Blumstein et al., 1977). Thus, the correlation between phonemic output disorder and syllable discrimination could be driven by patients with damage to *sensory*, not motor systems. A second problem is that even in the more severely impaired group, performance on average is quite good. Miceli et al. reported their data in such a way that d' measures could be calculated: patients with a phonemic output disorder had an average d' of 3.34 and an A′ of 0.90. In sum, lesion studies show that aphasics with damage to frontal motor-related structures largely retain the ability to perceive speech sounds.

MOTOR THEORY OF SPEECH PERCEPTION: FURTHER NEUROPSYCHOLOGICAL, DEVELOPMENTAL, AND COMPARATIVE EVIDENCE

Additional evidence comes from several sources. One is acute deactivation of the entire left hemisphere in patients undergoing sodium amobarbitol (Wada) procedures. This is a revealing subject population because (i) there is no opportunity for plastic reorganisation to compensate for disrupted tissue and (ii) the procedure produces a complete mutism with left hemisphere deactivation in most patients. In a recent study, anaesthesia of the left hemisphere produced complete speech arrest (mutism) yet left speech sound perception proportionately intact (phonemic error rate $< 10\%$ in a four alternative forced choice word-to-picture matching task) (Hickok et al., 2008; Figure 4). Importantly, this pattern holds even though fine phonetic discrimination is required for successful comprehension (i.e., phonemic distractors were included in the choice arrays; Hickok et al., 2008).[1]

[1] This result also argues for bilateral organization of the speech perception system in auditory regions because left hemisphere deactivation, which included not only the motor speech system but also left auditory regions (Hickok & Poeppel, 2000, 2004, 2007). Note, one cannot appeal to bilateral organization of a motor speech perception system because the motor speech systems was functionally deactivated in patients with left anesthesia; i.e., they were completely mute at testing. See also lesion evidence in text.

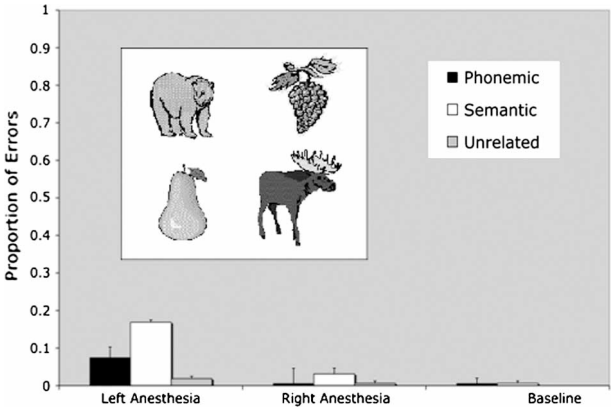

Figure 4. Proportion of errors in an auditory word-to-picture matching task in patients under going Wada procedures. Response cards included the matching target (e.g., BEAR), a minimal pair phonemic foil (e.g., PEAR), a semantic foil (e.g., MOOSE), and unrelated foil (e.g., GRAPES). The number of errors of each type are plotted for left anesthesia, right anesthesia, and baseline. Subjects were completely mute during the performance of the task with left anesthesia indicating that even with complete deactivation of the motor speech system patients make fewer than 10% phonemic errors and many of these may have resulted from deactivation of left hemisphere auditory systems.

Another source of evidence comes from bilateral lesions. Although the functional disruption to speech production indicates that unilateral lesions are sufficient to interrupt motor speech function severely, one may still wish to argue that only bilateral lesions to the human "mirror system" may cause the predicted severe speech perception deficits. Yet, bilateral lesions to Broca's area, argued to be the core of the human mirror system (Craighero, Metta, Sandini, & Fadiga, 2007), do not cause word level speech recognition deficits (e.g., see Case 3 in Levine & Mohr, 1979). Further, bilateral lesions to the cortex of the anterior operculum and foot of the precentral gyrus can cause anarthria, that is, loss of voluntary muscle control of speech, yet these lesions too do not cause speech recognition deficits (Weller, 1993).

Still further evidence comes from observations that motor speech capacity is not required for speech perception ability. The failure of a child to develop motor speech, as a result of a congenital anarthria (Lenneberg, 1962) or an acquired anarthria secondary to bilateral anterior operculum lesions (Christen et al., 2000) does not preclude the development of normal receptive speech (Christen et al., 2000; Lenneberg, 1962). A similar point can be made developmentally. Babies develop sophisticated speech perception abilities including the capacity to make fine distinctions and perceive speech categorically as early as one-month of age, well before they develop the ability to produce speech (Eimas, Siqueland, Jusczyk, & Vigorito, 1971).

Finally, species without the capacity to develop speech (e.g., chinchillas) can nonetheless be trained to perceive subtle speech–sound discriminations in a manner characteristic of human listeners, that is, categorically (Kuhl & Miller, 1975).

In short, disruption of a number of levels of the motor-speech system, including its complete deactivation or complete failure to develop, does not preclude the ability to make subtle speech sound discriminations in perception. These facts render a strong version of the motor theory untenable.

PROBLEMS WITH RECENT STUDIES CLAIMED TO SUPPORT A MOTOR BASIS FOR SPEECH PERCEPTION

Several recent studies have been claimed to support the view that the motor system supports speech perception. Careful examination of these studies either reveals that alternative explanations are readily available, or that the studies are flawed.

One TMS study has reported a decline in the perception of meaningless syllables in noise as a result of stimulation to motor cortex. Meister, Wilson, Deblieck, Wu, and Iacoboni (2007) found that stimulation of premotor cortex resulted in a decline in syllable identification (subjects indicated via button press which of three syllables they heard) from 78.9% at baseline to 70.6% during stimulation. A similar colour-based task was not affected by stimulation to the same site. The problem with this study is the use of a syllable identification task. The keys were unlabelled; subjects had been trained on the mapping between syllables and keys prior to testing (S. M. Wilson, personal communication, August 12, 2009). First, signal detection analyses were not employed; it is unclear whether significant differences would have held up in d′ units. Second, in order to correctly perform this task, the subject had to perceive the auditorily presented speech syllable and then match the heard syllable to an internal representation of a previously learned mapping between a syllable and a key press. It is unclear whether the stimulation affected the perception of the heard syllable (a speech perception effect) or interrupted the internal representation of the matching response (an effect that does not implicate speech perception). It is relevant that syllable identification tasks (indicating which syllable one heard) are generally harder than syllable discrimination tasks (indicating whether two syllables are the same or different) (Baker et al., 1981). This may result from the fact that discrimination tasks entail comparisons within the auditory modality whereas identification tasks entail matching between an auditory signal and a non-auditory stimulus. Because Meister et al. used a syllable

identification task rather than a discrimination task, it is impossible to link the performance decline to the auditory perceptual system.[2]

In another recent study (D'Ausilio et al., 2009), TMS was applied to the lip or tongue areas of M1 while participants were asked to identify speech sounds that either involved prominent lip articulation, [b] and [p], or prominent tongue articulation, [d] or [t]. They found a double-dissociation: relative to a non-stimulation baseline, participants were faster to indicate that they heard a lip-related sound when TMS was applied to motor lip areas, and faster to indicate that they heard a tongue-related sound when TMS was applied to motor tongue areas. Several points are relevant. One is that response times, like percent correct measures, are sensitive to response biases (McQueen & Sereno, 2005). It is therefore unclear whether stimulation modulated perception or response bias.[3] Second, note that stimulation did not *disrupt* perception (the critical test) but rather facilitated response time. Such an effect does not require a motor theory explanation. For example, even if we assume that the manipulation affected perception and not bias, stimulation of lip (or tongue) cortex may prime the acoustic correlates of these sounds in auditory cortex via sensory-motor associations (e.g., forward models), which may facilitate perception in auditory areas. This explanation holds that motor systems can *modulate* acoustic perception of speech in a top down fashion, much like lexical context can (Hickok, 2009b).

In yet another recent TMS study (Mottonen & Watkins, 2009), subjects were asked to perform syllable identification (which of two syllables did you hear?) and syllable discrimination (are the two syllables you just heard same or different) tasks. The stimuli were place or voice onset time continua and the design followed a standard categorical perception (CP) experimental design. These tasks were performed either pre-TMS or after TMS was applied to the lip area of left motor cortex. I will focus here on the discrimination task for reasons discussed above. The critical condition was when the subjects discriminated a lip-related sound (/ba/ or /pa/) from a non-lip-related sound (/da/ or /ga/). The authors report that discrimination across a category boundary was less accurate after TMS to motor lip areas than before. Specifically, for the /ba/-/da/ stimuli, the mean proportion of "different responses" to cross category (i.e., physically different) syllables was 0.73 pre-TMS and 0.58 post-TMS; similar findings are reported for

[2] A similar color-matching task was used as a control and showed no effect of stimulation. The authors argue that this result rules out post-perceptual task effect. However, this interpretation relies on pure insertion logic (i.e., that there is no interaction between stimulus type and task), which may not be valid (Friston et al., 1996).

[3] For example, if my lips are tingling I may be more likely to guess a lip-related sound when I am presented with a partially ambiguous stimulus. Perhaps clever subjects developed a response strategy for ambiguous stimuli based on sensory effects of the stimulation!

the /pa/-/ta/ stimuli. Discriminations that did not involve lip-related sounds (/ka/-/ga/ or /da/-/ga/) were not affected by stimulation, nor was lip-related speech sound discrimination affected by motor hand area stimulation. Although this at first appears to be solid evidence that stimulation of motor cortex can modulate speech perception, the result is in fact uninterpretable because only proportion of "different responses" to different stimuli were reported. These values are only meaningful if we know the proportion of "different responses" to identical stimuli (which affords a signal detection-type analysis): a subject who is 100% correct on different trials (/ba/-/da/) and 100% wrong on same trials (/ba/-/ba/) is clearly not discriminating, but rather just saying "different" to every stimulus. Thus, depending on the responses to same trials in this study, pre- and post-TMS discrimination could be identical or even the reverse of what is reported. One simply can't determine from only half the data.

Finally, in an interesting neuropsychological study, Moineau et al. (2005) tested three aphasic groups (Broca's, Wernicke's, & anomic), right hemisphere non-aphasics (RHD), and control subjects on a word comprehension test. The test involved two listening conditions, clear speech and degraded speech (low-pass filtered and temporally compressed), and utilised picture-word verification: subjects heard a word and saw a picture and indicated whether it matched (named) the picture or mismatched.

In the clear speech condition only the Wernicke's patients showed any deficits on the comprehension task. In the degraded speech condition all subjects performed more poorly as expected but now the Broca's patients performed statistically as poorly as the Wernicke's patients and both Broca's and Wernicke's performed worse than controls and RHD patients (Broca's did not differ from anomic aphasics, but Wernicke's did). In other words, single word comprehension deficits in Broca's aphasia appear to be "uncovered" by presenting speech in an acoustically degraded form, and under these conditions they look as bad as Wernicke's aphasics.

But again there is a flaw in the analysis. Recall that the task in Moineau et al. is to detect matches and reject mismatches. Unfortunately Moineau et al. did not calculate bias-adjusted scores (d′ or A′) in their analysis. Instead they simply took the proportion correct in the match and mismatch trials to calculate accuracy scores. This of course could lead to biased, possibly invalid results. In fact, when they looked at accuracy as a function of "congruence" (whether it was a match or mismatch condition) they reported that Broca's and Wernicke's patients have opposite biases! Wernicke's and control subjects performed better on the congruent trials (they tended towards "yes" responses) and Broca's and RHD subjects performed better on the incongruent trials (they tended towards "no" responses). These group differences in response bias suggest that the overall findings are indeed themselves biased.

In summary, while there is evidence favouring the view that there is a tight connection between speech perception and speech production (Doupe & Kuhl, 1999; Galantucci et al., 2006; Hickok & Poeppel, 2007), there is no evidence in support of a strong version of the motor theory of speech perception and good evidence against it. In fact, available evidence suggests that perception has a much stronger influence on production than the inverse, and that the auditory system (bilaterally) comprises the basis for speech perception (Hickok & Poeppel, 2007). The motor system at most may have a weak modulatory role on auditory perception of speech.

An alternative to the mirror neuron/motor theory of speech perception is a sensory theory of speech production (Guenther, Hampson, & Johnson, 1998; Hickok & Poeppel, 2000, 2004, 2007). There is strong evidence from the effects of altered auditory feedback on speech production that perceptual systems play an important role in speech production (Burnett, Freedland, Larson, & Hain, 1998; Burnett, Senner, & Larson, 1997; Houde & Jordan, 1998; Waldstein, 1989). Lesion evidence also supports this view: damage to left auditory regions (dorsal superior temporal gyrus) produces phonemic-level speech production deficits (Damasio & Damasio, 1980; Hickok, 2000; Hickok et al., 2000).

Recent discussions have framed these auditory-motor interactions in the context of motor control networks in which auditory speech representations form the target for motor speech acts and both overt speech feedback and internal models (such as forward models which are used to make predictions about the sensory consequences of an action) support the control processes necessary to achieve these auditory goals (Guenther et al., 1998; Hickok & Saberi, in press; Houde & Jordan, 1998; van Wassenhove, Grant, & Poeppel, 2005; Figure 5). Clearly, these processes require extensive auditory-motor interaction, which we have argued is supported by the dorsal auditory stream (Hickok, Okada, & Serences, 2009; Hickok & Poeppel, 2000, 2004, 2007). This proposal shares with motor theories the view that there is a tight connection between speech perception and speech production, only the direction of primary influence is reversed. This association can explain the fact that motor speech systems are often activated during speech perception tasks (i.e., via spreading activation in associated circuits) and also provides a pathway for the modulatory feedback influence of motor systems on perceptual systems.

In sum, there is unequivocal evidence for auditory influence on motor control of speech production, as well as unequivocal evidence that motor speech systems are not necessary for speech perception. Thus strong variants of motor theories of speech perception are incorrect and apparent motor involvement in speech perception can be explained via auditory-motor associations that operate primarily in the service of speech production.

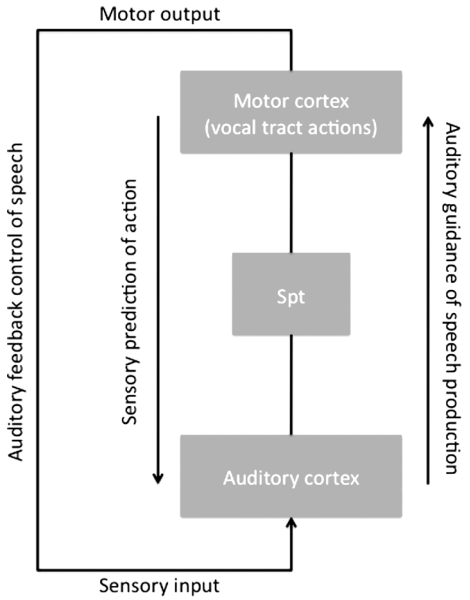

Figure 5. Schematic model of sensory-motor integration functions. Sensory representations of speech in auditory areas serve as targets for motor control processes providing feed-forward guidance for example in the repetition of non-words where a novel sequence of phonemes must be represented acoustically and then reproduced motorically. An internal motor-to-sensory circuit (forward model) would generate sensory predictions for motor speech actions, which would be compared to overt self-generated speech. The internal forward model may also be a mechanism for motor modulation (priming) of sensory processes. Adapted from Hickok and Saberi (in press).

MOTOR THEORIES OF ACTION SEMANTICS

Motor-related theories of action semantics are not new. Wernicke argued explicitly that the motor associations of concepts are stored in motor cortex,

> . . .the memory images of a bell. . .are deposited in the cortex and located according to the sensory organs. These would then include the acoustic imagery aroused by the sound of the bell, visual imagery established by means of form and color, tactile imagery acquired by cutaneous sensation, and finally, motor imagery gained by exploratory movements of the fingers and eyes. (Wernicke, 1885–1886/1977, p. 179)

> Close association between these various memory images has been established by repeated experience of the essential features of bells. As a final result, arousal of each individual image is adequate for awakening the concept as a

> whole. In this way a functional unit is achieved. Such units form the concept of the object, in this case a bell. (Wernicke, 1885–1886/1977, p. 179)[4]

> Since these different sensations occur simultaneously, their memory traces remain associated with each other. In this way every tangible object is related to an acquired association of memory traces of different senses, and this association is stronger the more frequently the object is perceived by our senses. We have in this manner arrived at the anatomical substrate for what psychology has long called a "concept". (Wernicke, 1900, p. 30), quoted in (Gage & Hickok, 2005)

Modern authors have expressed similar views over the last several decades (Damasio, 1989; Pulvermuller, 1996; Tranel, Adolphs, Damasio, & Damasio, 2001). For example, Pulvermuller promotes a position that is effectively indistinguishable from Wernicke's:

> Associative learning may play a role in the acquisition of meaning of *content words* (nouns, verbs, adjectives) in particular if their meaning is concrete and well imaginable. For example, if the meaning of the word *fish* is being acquired, the learner may sometimes be exposed to stimuli of various modalities related to the meaning. He or she may see, smell, taste, touch, or catch a fish while hearing the word form. These multimodal perceptions and actions are related to neuronal activity in various cortical regions. Thus, neurons in various sensory and motor cortices become active together with the perisylvian assembly representing the word form. According to the Hebbian logic, the frequently coactivated neurons strengthen their connections and develop into a higher-order assembly representing the phonological word form together with its meaning. (Pulvermuller, 1996, p. 319)

Much recent research within this framework has focused on action concepts, in particular whether motor cortex is involved in the representation and/or processing of action knowledge. This work has uncovered a wide and impressive range of evidence that appears to support this position (e.g., see Pulvermuller, 2005). Here we will consider a sample of such findings from neuroimaging, TMS, and neuropsychology.

Neuroimaging

A number of studies have reported motor-related activation associated with processing action words and sentences, which appears to be somatotopically organised in some cases (Aziz-Zadeh, Wilson, Rizzolatti, & Iacoboni, 2006; Hauk, Johnsrude, & Pulvermuller, 2004; Kemmerer, Castillo, Talavage,

[4] Wernicke's ideas on concept formation invoke Hebbian principles decades before Hebb (Gage & Hickok, 2005).

Patterson, & Wiley, 2008; Pulvermuller, Harle, & Hummel, 2001; Tettamanti et al., 2005). For example, when subjects process (e.g., read) a word such as *kick*, motor areas corresponding to leg movements are activated, whereas for a word such as *kiss*, lip areas are activated. The problem with this type of result is that it cannot determine whether motor cortex is activating because it is part of the semantic representation of the words (the usual claim) or simply because there is an association between the word meanings and the actions they refer to. That is, the meaning of *kick* may be stored independently of motor cortex, but because this meaning is associatively linked to kicking actions, access to the meaning of *kick* results in spreading activation within brain networks to which it is associatively linked, including the leg region of motor cortex.

A recent study by Hauk, Davis, Kherif, and Pulvermuller (2008a) attempted to tease apart these two possibilities by looking for brain regions that showed word frequency effects in action vs. non-action-related words. The logic here is that word frequency effects should only be evident in areas that support lexical-level processes rather than purely motor processes. They found indeed that distinct areas could be identified which were correlated with frequency for action vs. non-action words. However, the dominant frequency effect for action words was not found in motor cortex, but in the posterior temporal lobe suggesting that motor cortex is not a critical site for the storage of lexical-level action word information.

Transcranial magnetic stimulation (TMS)

TMS has been used in two ways to assess the role of motor cortex in action word processing. In one approach, stimulation is applied to motor cortex to generate motor evoked potentials (MEPs) in distal muscles. MEPs are then used as the dependent measure. Using this approach, Buccino et al. (2005) found that listening to hand-related sentences modulated MEPs in hand muscles, whereas foot-related sentences modulated MEPs in foot muscles, thus providing evidence for somatotopically organised motor cortex activity during the processing of action-related language stimuli. Another approach involves stimulation to motor cortex and assessing the behavioural effects on action word processing. For example, Pulvermuller, Hauk, Nikulin, and Ilmoniemi (2005) stimulated motor cortex for hand or leg areas while subjects performed a lexical decision task. TMS to hand areas led to faster reaction times to hand-related words (e.g., pick) than leg-related words (e.g., kick), whereas the reverse held for TMS to leg areas.

The proposed conclusion from these studies is that "the motor system [stores] action-related aspects of word meaning" (Pulvermuller et al., 2005, p. 797). But as with the functional imaging results, there is another possible explanation, namely that activation of a motor-related concept, which may

be stored outside the motor system, can associatively activate (or prime) related motor systems and vise versa. Thus, the motor system is activated when processing action words or sentences not because it is coding the meaning of those concepts but because the meanings are *associated* with motor actions.

Associative priming is not restricted or unique to the motor system, of course. For example, if a soccer fan were presented with the word *kick*, we may be able to document priming not only of specific motor representations such as kicking movements, but also specific kinds of sensory representations such as the visual representation of a soccer ball or the image of their favourite soccer player. No one would be particularly surprised, for example, if the word *kick*, led to faster recognition times for images of David Beckham among soccer fans. Does this mean that neural systems supporting the recognition of David Beckham are an integral part of the concept KICK? No, it just means that there is an associative relation between the representation of the word *kick* and the representation of David Beckham such that one can prime the other. We suggest that the motor cortex activity observed in functional imaging and TMS studies of action word processing is of the same nature.

Proponents of the motor theory of action semantics have acknowledged the ambiguity of their findings, at least for neuroimaging studies. Hauk et al. write, "previous neuroimaging results have not been able to distinguish between activation evoked by elementary recognition processes (accessing the meaning of the word 'hammer', for example) and 'epiphenomenal' post-recognition processes, such as deliberate imagery (e.g., imagining using a hammer) or 'post-understanding translation'" (Hauk et al., 2008a, p. 1856). Attempts to disambiguate these results have relied on studies of the timing of the motor activation (Hauk, Shtyrov, & Pulvermuller, 2008b) or frequency effects in action word processing (see above; Hauk et al., 2008a). But there is a much more straightforward way to distinguish between an (epiphenomenal) associative priming explanation and the motor-semantics explanation of motor cortex involvement in action word processing, namely, train subjects on a paired-associate learning task where the stimulus is non-action-related and the response is a specific action, then assess whether the non-action stimulus elicits a motor response upon subsequent passive presentation. For example, one could train subjects to make a leg movement upon seeing a picture of a cloud and an arm movement upon seeing a picture of the Empire State Building. Prior to training, one would not expect cloud perception to elicit a motor response. After training, however, activation of the leg area may occur upon seeing a cloud if the associative priming account is correct. Since leg movements are presumably not a component of the semantic representation of clouds, such a result would demonstrate that simple association can explain motor activation during action word processing.

There is already evidence to this effect. From classical conditioning paradigms we know that if you repeatedly pair a tone with a puff of air to the eye (eliciting an eye blink), pretty soon the tone all by itself will elicit the motor response. A more recent result using the TMS/MEP paradigm shows a similar effect. Catmur, Walsh, and Heyes (2007) found that MEPs in index finger muscles were enhanced when watching index finger movements, but not little-finger movements, and vise versa. They then trained subjects to make little-finger movements when they viewed index finger movements and to make index finger movements when they viewed little-finger movements. After training the MEP pattern reversed: watching index finger movements modulated little-finger MEPs and vise versa. This shows that motor cortex activity is dependent on paired-associate learning as suggested above. Presumably subjects did not misunderstand index finger movements for little-finger movements indicating further that sensory-motor priming effects can dissociate from action understanding.

Neuropsychological studies

Neuropsychology provides a potentially less ambiguous approach to determining the role of motor cortex in the representation of action knowledge. If action knowledge is critically dependent on motor systems, then damage to the relevant motor systems should result in circumscribed deficits in action knowledge processing.

It is well-known that damage to left frontal regions can produce a deficit in *naming* that is more severe for actions than objects (Hillis, Tuffiash, Wityk, & Barker, 2002; Tranel et al., 2001) suggesting an association between action word processing and motor-related cortices. However, if the reported frontal-related, action naming deficit results from a disruption of action *knowledge*, as opposed to some speech output-specific process, one would expect to see impairment also on the comprehension side. This prediction appears not to hold in stroke patients. In a study that examined naming of actions and objects as well as comprehension of action and object words (Hillis et al., 2002), 23 out of 33 patients were impaired in naming actions, and 11 of these were also impaired in the comprehension of action words. Impairment on both the naming and comprehension tasks was associated only with infarcts or hypoperfusion of the left superior temporal gyrus not motor-related areas. Conversely, patients with naming deficits and *spared* comprehension had lesions that involved precentral gyrus (i.e., motor areas), and of the patients who had frontal lesions and more difficulty naming actions than objects, all of them performed well on the action word comprehension task. This suggests that the association between action naming and left frontal lesions is not a result of disruption to action knowledge systems.

Another study (Saygin, Wilson, Dronkers, & Bates, 2004) took a different approach to studying action comprehension deficits in stroke patients. Patients viewed pictures of actions with the object of the action removed, such as a boy licking an ice cream cone, but with no ice cream cone in his hand. The task was to pick the matching object out of an array of two pictured objects that included the target (ice cream cone) and a distractor. A matched verbal version of the task was also administered in which subjects read sentence fragments ("He is licking the _____") and had to pick the correct picture, as above. The results showed (i) performance on the two task was completely uncorrelated (after the one outlier patient was removed) and (ii) that deficits on the pictorial task were correlated with lesions in the inferior frontal gyrus, whereas deficits on the linguistic task were correlated with a different distribution of lesions that did not involve the frontal lobe but was more posterior and inferior involving the superior temporal gyrus and inferior parietal lobe. Although this result might be interpreted to indicate that the inferior frontal gyrus supports the comprehension of actions, it also shows quite clearly that the source of the deficits is not action *knowledge*: if action *knowledge* systems were impaired, then one would not expect modality effects. Instead, what appears to be disrupted in these patients are those (modality-dependent) systems that *access* action knowledge (or perhaps those systems involved in making complex inferences from partial information).

Another study (Neininger & Pulvermuller, 2003) examined the effects of *right* frontal lesions ($n = 12$) and versus *right* inferior temporo-occipital lesions ($n = 6$) on lexical decisions to action-related verbs vs. object-related nouns. The authors report that more errors were made on verbs than nouns among the frontal patients, and the reverse pattern held for the temporo-occipital patients. So performance on the two types of stimuli dissociate, but does this indicate that the impairment on action-related verb stimuli reflect disruption to (i) motor cortex, and/or (ii) action *knowledge* systems? The answer is *no* on both counts. First, because the distribution of the lesions in the frontal patients extended beyond motor cortex in every case (eight had involvement of the temporal lobe, seven had parietal lobe involvement, and all 12 had damage to the basal ganglia), there is no way to pinpoint the source of the deficit to motor cortex. Second, given Saygin et al.'s demonstration that "action processing" can be disrupted in a modality-dependent fashion (indicating a deficit in *access* rather than *representation*), there is no way to know whether the effect is specific to linguistic stimuli. Further, it is not obvious how well a lexical decision task assesses the integrity of conceptual knowledge, rather than some linguistic-specific operation. Finally, a TMS study by the same group (Pulvermuller et al., 2005) reported that stimulation of right motor cortex did not lead to any

effect on lexical decision responses in direct contradiction to their lesion result.

Tranel, Kemmerer, Adolphs, Damasio, and Damasio (2003) report a large group study that sought to identify the location(s) of lesions that resulted in deficits in conceptual knowledge for actions. Conceptual knowledge was assessed using non-linguistic tasks that required subjects to make inferences based on static images depicting actions (e.g., decide which of two depicted actions would make a louder sound, dealing cards or shuffling cards) and to judge which of three static pictures of actions doesn't belong with the other two (e.g., peeling and apple, peeling a banana, lifting a lid off of a pot). Lesion analyses revealed that patients with deficits on these tasks had lesions involving left premotor/prefrontal regions, left parietal regions, and/or a left posterior temporal region. From this we can infer that these regions are involved in some capacity in the performance of these tasks. But given that the tasks involve not only access to action-related conceptual knowledge but also making inferences (what sound does that action make, what action does that static image represent), comparative processes, response selection, etc., and given that the performance of these patients on difficulty-matched non-action tasks was not assessed, it is not clear whether deficits on these tasks reflect impairments of conceptual knowledge or impairment on some task-specific processes.

A case study reported by Kemmerer and Tranel (2003) provides a possible example of a deficit in action knowledge processing associated with a frontal lesion. Patient JP performed poorly on a range verbal and non-verbal tests of action processing including naming, comprehension, and a picture comparison test. Conversely, JP performed quite well on a set of tests involving locative prepositions. JPs lesion involved all of Broca's region (pars opercularis, pars triangularis) as well as more anterior portions of the inferior frontal gyrus (pars orbitalis) and more superior regions including premotor cortex. One issue that needs to be addressed is whether the source of the deficit indeed comes from disruption of "motor" representations. The involvement of premotor cortex makes this a possibility, but it is also very possible that the deficit stems from damage to the inferior frontal gyrus (IFG). Given the wide range of functions ascribed to various portions of the IFG, it is not clear whether the deficit reflects damage to motor cortex. It will be instructive to learn whether similar patients are found with this pattern of deficit.

A range of data from other neurological conditions has been brought to bear on the question of the neural basis of action knowledge/processing in recent years. Deficits in processing action-related stimuli have been reported in primary progressive aphasia (Hillis, Tuffiash, & Caramazza, 2002), Parkinson's disease (PD; Boulenger et al., 2008), and motor neuron disease (amyotrophic lateral sclerosis (ALS); Bak, O'Donovan, Xuereb, Boniface, &

Hodges, 2001) among others. PD and ALS are particularly interesting because they clearly disrupt (different aspects of) the motor system.

ALS severely affects motor neurons and until recently hadn't been thought to affect higher-order processes such as language. However, Bak et al. (2001) reported on six ALS patients who had language impairments, and in both naming and comprehension, verbs were more affected than nouns. Such a finding might be compelling evidence for the involvement of motor systems in action verb processing except for the fact that the pathology in these cases was not restricted to motor cortex. In fact, a consistent finding in these cases was the involvement of Brodmann areas 44 & 45, which have been implicated in a number of functions including working memory (Rogalsky, Matchin, & Hickok, 2008; Smith, Jonides, & Koeppe, 1996), syntactic processing (Caplan, Alpert, Waters, & Olivieri, 2000; Grodzinsky, 2006), and cognitive control (Novick, Trueswell, & Thompson-Schill, 2005), among others. Further, the ALS patients studied by Bak et al. were a subset of patients who presented with a dementia/aphasia variant of the disease, further calling into question whether it is the motor involvement that resulted in the action word processing deficit.

Boulenger et al. (2008) studied noun versus verb processing abilities in a group of PD patients using a masked identity-priming paradigm. The task was lexical decision. The authors report that on medication, PD patients showed priming for both nouns and verbs, whereas off medication, i.e., when their basal ganglia circuit disorder is more pronounced, PD patients only showed priming for nouns. They conclude that this provides "compelling evidence that processing lexico-semantic information about action words depends on the integrity of the motor system" (p. 743). The problem with this conclusion is that PD is known to have effects well beyond the motor system including higher-order cognitive dysfunction, a fact noted by Boulenger et al. It is therefore not possible to conclude that the verb processing effect is attributable to motor system dysfunction. Further, the pattern of reaction times in the on- versus off-medication assessments was less than straightforward. While it is true that off-medication response times were faster to nouns when they were primed compared to when they were not, it is also true that response times to nouns in the unprimed condition were substantially slower than to unprimed verbs. In the on-medication assessment response times were roughly equivalent for unprimed nouns and verbs. So one might just as easily argue that increased motor impairment (no medication condition) causes *noun* processing deficits! The results are clearly less compelling than the authors suggest.

In summary, the functional imaging and TMS findings regarding motor cortex involvement in action processing can be explained in terms of a simple associative priming mechanism and while neuropsychological data clearly demonstrate that action- and object-related processing are dissociable, the

available data do not allow one to conclusively link action processing deficits to motor cortex, and instead appear to implicate higher-order systems, perhaps in the inferior frontal gyrus. It is possible that the relative difficulty of processing verb stimuli and/or task demands drive many of these effects: a recent case study of a frontotemporal dementia patient (frontal variant) showed that deficits in naming and comprehending verbs relative to nouns assessed using static depictions of actions (pictures) disappeared when videotaped actions were used for assessment (d′Honincthun & Pillon, 2008). The authors suggest that the noun–verb dissociation is due more to different demands on executive resources between noun and verb tasks than to any action verb processing deficit per se.

Finally, it is important to consider how much (or little) explanatory power one gets from a motor account of action knowledge. First, as hinted at in the introduction, motor accounts of action concepts can only apply to a small range of actions, namely those that can be executed by the human motor system. We therefore need some mechanism for representing concepts that humans cannot perform (e.g., *fly*, *hover, tail-wag*, *locomote-via-jet-propulsion*), that some humans cannot perform (e.g., *juggle, swim, play-the-sousaphone*), that don't involve the motor system (e.g., *think*, *grow*, *bleed, sleep, compare*), and for any number of non-motoric verbs (e.g., *digitise, melt, erupt*). If we have a mechanism for representing these concepts such that we can understand *fly* or *juggle* or *hover* even if we cannot perform such actions, then why do we have to invoke the motor system as a necessary component of concepts such as *kick* or *kiss*? Second, even for actions that the human motor system can execute, motor programs often fail to make the relevant conceptual distinctions. Consider *walk* versus *hike*, which are distinguishable in terms of their goals not their motor actions. In fact, motor actions and action concepts double-dissociate. The same motor act of grasping a pitcher full of water, lifting it, and then rotating the arm would be used for any number of actions including *fill*, *re-fill*, *pour*, *spill* (if the water didn't make it in the glass), and so on. Conversely, one can perform the action *fill* (as in, *to fill a glass*) using any number of motor actions including pouring from a pitcher, turning on a faucet, dipping in a lake, setting a glass in the rain, or even asking your waiter to do it for you (Pinker, 1989). Thus, even for many action concepts that are executable by humans, motor actions provide little explanatory power.

A typical response to this line of argument is that motor information is only part of a broader representation and that motor information augments action concepts. This may be better way to think about it (Mahon & Caramazza, 2008). For example, personal experience with ballet dancing may result in a greater appreciation of the dance during the observation of a ballet. An experienced dancer may recognise mistakes more easily or better predict the outcome of a movement. But to say that motor knowledge

augments the concept with idiosyncratic personal information is a much more modest claim than that which is typically promoted in this literature.

CONCLUSIONS

Despite the widespread enthusiasm for motor theories of speech perception and action semantics, and despite the glut of empirical reports claiming to support such views, there is, in fact, little evidence in support of these ideas. There is strong evidence against motor theories of speech perception in the form of neuropsychological syndromes in which motor speech systems can be virtually obliterated without concomitant effects on speech perception and the direct demonstration that destruction of the proposed human "mirror system" does not result in speech recognition deficits even when fine phonetic distinctions are required for accurate recognition. Likewise motor theories of action semantics don't fare much better: Functional imaging and TMS data can be explained readily in terms of simple associative priming and what lesion evidence exists is either ambiguous or suggests that the source of action processing deficits is not at the level of action *knowledge* but instead is modality or task specific and/or involves higher-order cognitive systems.

Conversely, much evidence exists showing that auditory systems play an important role in speech production. It is puzzling that this empirically well-established sensory-motor integration effect receives far less attention than the role of motor systems in perception.

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