## Hugo Liepmann revisited, this time with numbers

To the Editor: The recent article by Diedrichsen et al. (2003) mentions the presence of ipsilateral corticospinal innervation in humans on several occasions (for example in p. 2416) despite the ubiquitous occurrence of onset asynchrony (of  $\geq 150$  ms) in their controls-leaving the functional status (relevance) of such fibers (if they exist) in limbo. Neither have they committed themselves as to the reason for the onset asynchrony so nicely depicted in Fig. 8 of the article. The reason, I believe, is paucity of clinical perspective in such studies as follows: the essential feature requiring an explanation, not forthcoming from the doctrine of contralateral innervation (followed by the authors), is the asymmetry of signs and symptoms in patients with comparable lesions affecting the major and minor hemispheres. It is only natural to wonder if such an asymmetry is related to the persistent asymmetry of the reaction time documented in the control subjects of their study. Elsewhere I have shown in detail that the two are related and that the link is coded in the (neural) handedness of humans (Derakhshan 2003a,b). Thus the reason for the inescapable onset asynchrony of their subjects is the callosum-length proximity of the command center to the dominant hand of their subjects as compared with the nondominant, which is connected to the same command via the corpus callosum. This nondominant delay is the interhemispheric transfer time, which varies with the "tempo" of the activity; a fact known to musicologists as the melody lead of the right hand (in right-handed pianists) (Vernon 1936). Liepmann depicted this callosally mediated relationship (delay) in two of his articles, carrying the "movement formula" from the major to the minor hemisphere (Liepmann 1900, 1920) (Fig. 1).

In summary, the anatomy I have described (as a clinician) seems to be the explanation of what Hazeltine colleagues have recently characterized as "a major limitation of human perfor-

mance in the ability to initiate *two goal directed behaviors at the same time*" (Hazeltine et al. 2003). Others have referred to the same as *"the impossibility of humans generating multiple independent, unsynchronized parallel action plans for the two hands"* (Laeng and Park 1999) or have simply called it "synchronization error" (Kristeva and Deecke 1979).

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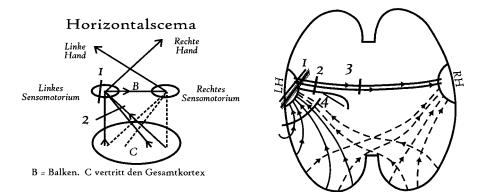


FIG. 1. From articles by Liepmann, written in 1900 (*right*) and 1920 (*left*). Note the direction of the horizontal arrow from the left to right hemisphere. For temporal and neurophysiological implication of this volitional excitatory pathway, see Derakhshan 2003a–c).

Figure: Adopted from Liepmann's 1908 (right) and 1925 (left) papers. Note the direction of the arrow from the left to the right hemisphere in both segments of the drawing, representing directionality of callosal traffic for motor control. The one way callosal traffic scheme represents numerical demonstration of the validity of Liepmann's diagram, using the reaction time (as a probe) in a timing analysis of bimanual coupling. See text for details.

## REPLY

To the Editor: I. Derakshan raises a number of issues in his letter regarding our paper (Diedrichsen et al. 2003). Before turning to the interesting issues regarding hemispheric asymmetries and how they may relate to our study, we wish to point out that his concern about the status of ipsilateral fibers is based on a misunderstanding of the data. Onset asynchronies of 150 ms are by no means "ubiquitous." When we restricted the analysis of the data to the shorter half of the onset asynchronies, the correlation between produced forces remained strongly reduced in callosotomy patients (see Fig. 8). In our opinion, these data are inconsistent with the hypothesis that force coupling is the result of activation from ipsilateral, descending fibers because the neural activity of ipsilateral and contralateral fibers would have strongly overlapped for these responses.

More interesting is I. Derakshan's review of the hypothesis that motor behavior for both hands is mainly controlled by the dominant hemisphere. This hypothesis is supported by a wealth of lesion studies indicating that chronic symptoms of apraxia are usually associated with left-hemisphere damage (for a review, Leiguarda and Marsden 2000). Moreover, neuroimaging (Kim et al. 1993) and TMS (Chen et al. 1997) studies also suggest an asymmetric role for the left hemisphere in motor control.

However, we do not believe that the temporal asynchronies in our study provide evidence for the unilateral control hypothesis as suggested by I. Derakshan. If the temporal asynchronies stemmed from the dominance of the left hemisphere and its producing the motor commands for both hands, then we should have observed a consistent right-hand lead (all of our control participants were right-handed). However, half the participants showed a consistent left-hand lead and the other participants a consistent right-hand lead (p. 2414). Data from other studies also suggest that phase leads during bimanual actions may be a much more complex phenomenon than suggested by the mechanistic explanation offered in I. Derakshan's letter. Temporal lags can be highly influenced by the allocation of attention and other factors (Franz et al. 2002; Swinnen et al. 1996). Moreover, during simple finger tapping movements, no temporal asynchronies are typically observed in healthy controls (e.g., Ivry and Hazeltine 1999). These findings present a challenge to I. Derakshan's simplified version of a unilateral control hypothesis.

While it is likely that asymmetric functions between the two hemispheres emerge at higher levels of the human motor system, the two hands also can exhibit surprising independence of control, for example, during visually guided aiming movements (Diedrichsen et al. 2001, 2004). Elucidation of the interplay between unilateral control and bilateral coordination remains an important goal of further study of the human motor system.

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