

Reply to “Nonconvulsive status epilepticus in Creutzfeldt-Jakob disease”

We very much welcome the comments of Dr Fernández-Torre on our review ‘EEG in Creutzfeldt-Jakob disease’ (Wieser et al., 2006). We actually agree with Dr Fernández-Torre’s elucidations regarding the difficulty of interpretation of determined EEG anomalies in Creutzfeldt-Jakob disease and we would like to stress that it was not our intention to suggest that Fernández-Torre et al. (2004) might have ‘misunderstood EEG alterations as ictal epileptiform activity when they seemed to be PSWC’ in their paper. On the contrary, the phrase ‘as noted in the study of Fernández-Torre et al., 2004’ was meant to pay deference to the authors for emphasizing these difficulties in their paper.

Heinz Gregor Wieser
Kaspar Schindler
Dominik Zumsteg

*Department of Epileptology and EEG, University Hospital,
Frauenklinikstrasse 26, Zürich 8091, Switzerland
E-mail address: hgwepi@neuro.unizh.ch*

1388-2457/\$30.00 © 2006 International Federation of Clinical Neurophysiology. Published by Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.clinph.2006.05.005

The frequency of ulnar to median nerve anastomosis (Marinacci communication)

We read with interest the article by Meenakshi-Sundaram et al. (2003) published in this journal but would like to comment on some points.

The authors claimed that the prevalence of Marinacci anastomosis (ulnar-to-median anastomosis) is unknown but it is probably very rare (Amoiridis, 1992; Amoiridis and Vlachonikolis, 2003; Kimura et al., 1976; Resende et al., 2000). To our knowledge, until the publication of the paper under discussion, the presence of a Marinacci anastomosis had been clearly established in only 4 cases in the world literature (Kimura et al., 1983; Marinacci, 1964; Stancic et al., 2000; Streib, 1976).

The authors describe their methods indefinitely writing that ‘when unexpected and/or unexplained change in amplitude of CMAP between the proximal and the distal site of stimulation was noted, the following technique was used. The ulnar nerve was stimulated over the wrist and below the elbow, and CMAPs were obtained from the APB’. They mean apparently, that recordings over APB on ulnar nerve stimulation at wrist and elbow were initiated, when the CMAP over APB was less than 80% on median elbow

than wrist stimulation. The latter can occur occasionally as a result of submaximal stimulation of median nerve at elbow when surface stimulating electrodes are used (deep nerve location). Subsequently, stimulus spread to the median on ulnar nerve stimulation at elbow can perfectly imitate a Marinacci anastomosis.

The authors believe that an initial negativity of the CMAP over APB on ulnar stimulation using surface electrodes for CMAP recordings proves that this potential is generated in this muscle. This is not totally true. The initial negativity indicates only that a motor point of an excited muscle is vertically under the recording electrode, but it can be located in another muscle layer (Amoiridis and Schols, 1997; Amoiridis and Vlachonikolis, 2003; Amoiridis et al., 1996; Magistris and Truffert, 1997). The authors report that ‘Care was taken to avoid volume conducted responses with initial positivity of the CMAP’ but they do not explain how. We suppose they have moved their recording electrodes, until a potential with an initial negativity was recorded, maybe over the deep head of flexor pollicis brevis or above the end point region of FDI.

Furthermore, the authors think that F-waves over APB which are obtained by ulnar nerve stimulation below elbow can be used as an evidence for an ulnar to median nerve anastomosis. After reading their publication, we could obtain them in a series of 10 consequent subjects without Marinacci anastomosis. These F-waves originate in ulnar nerve innervated thenar muscles (for example, the deep head of flexor pollicis brevis).

The main criterion of a Marinacci anastomosis, that is a higher CMAP over APB on elbow than on wrist stimulation of the ulnar nerve, appears in the results, instead of the methods in the publication of Meenakshi-Sundaram et al. (2003). According to this criterion there is no Marinacci anastomosis in their cases 1 (right arm) and 2 (right arm too) of Table 1 in their publication.

Additionally, the authors have recorded a higher CMAP on proximal than on distal stimulation of the ulnar nerve over ADM, which they cannot explain. They claim that Streib reported a similar observation in his patient but data from Streib contradict this (Streib, 1976).

Positive criteria for a forearm anastomosis between median and ulnar nerve or vice versa can be caused by stimulus spread to adjacent nerve at elbow (Amoiridis, 1994). Stimulus spread to median nerve imitates a Marinacci and stimulus spread to ulnar nerve a Martin-Gruber anastomosis. Neither collision nor pharmacological blocks can differentiate between anastomosis and stimulus spread (Amoiridis et al., 1998). Only near nerve stimulation by means of right-to-the tip Teflon-insulated electrodes enables supramaximal nerve stimulation without stimulus spread to the adjacent nerve. However, this kind of stimulating electrodes should be used in studies of anomalous innervation, only if the positive criteria for the occurrence of a fiber crossing between ulnar and median nerve do not disappear, when