We report on what we believe to be a clinical illustration of function-based cerebral plasticity.

At the age of 4 years, a developmentally delayed boy suffered a fall which resulted in a supracondylar fracture of his right humerus. The fracture went unnoticed and consolidated with a 120° endorotation of the humerus. The boy presented to us at 6 years of age. The clinical diagnosis of Angelman syndrome was confirmed by the cytogenetic demonstration of a chromosome 15q11–13 deletion using the GABR3 and SNRPN probes. In this condition, patients typically tend to walk with their elbows flexed [1]. On examination, this patient has resumed manipulation skills similar to most patients with Angelman syndrome despite his right arm deformity. However when he walks, while he has an uplifted, flexed posture of the left arm, he has a downward flexion attitude of his right arm. This automatico-voluntary dissociation is consistent with the idea that so-called ‘associated reactions’ such as upper-limb posture during ambulation depend on different pathways than those implicated in voluntary movements [2]. The resumption of right upper-limb dexterity also suggests a cortical reorganisation through functional connections which may reflect novel corticospinal projections [3], transposing kinematic invariants such as those demonstrated for the command of complex limb movements [4]. A similar adaptation of the central nervous system to an alteration in the peripheral system subserving motor behaviour has recently been demonstrated by the incorporation of exogenous material in the schema of the hand as coded by neurons in the caudal postcentral gyrus of macaque monkeys trained to use a rake [5]. Our case provides an illustration that what has been called ‘normal movements in atypical populations’ [6] can develop relatively late even in a context of congenitally impaired central nervous system like in Angelman syndrome.

References