Transcranial magnetic stimulation of the motor cortex was performed in 10 normal subjects and 10 patients with radiographical abnormalities of the corpus callosum. Seven patients had a complete or partial agenesis or hypoplasia of the corpus callosum, two had a thin corpus callosum due to hydrocephalus or white matter degeneration and one had a circumscript contusion lesion of the corpus callosum. The patients served as a clinical model to investigate transcallosal influences on excitatory and inhibitory effects of motor cortex stimulation and to assess the potential diagnostic use of interhemispheric conduction studies and the contribution of interhemispheric interaction on transcranially elicited contralateral excitatory and inhibitory motor responses. Stimulation over one motor cortex suppressed tonic voluntary electromyographic activity in ipsilateral hand muscles in all subjects with preserved anterior half of the trunk of the corpus callosum. Since this suppression was lacking or had a delayed onset latency in patients with absence or abnormalities of the anterior half of the trunk of the corpus callosum it can be concluded that it is due to a transcallosal inhibition (Ti) of the opposite motor cortex mediated by fibres passing through this part of the corpus callosum. In normal subjects Ti had an mean onset latency of 36.1 +/- 3.5 ms (SD) and a duration of 24.5 +/- 3.9 ms. The calculated mean
transcallosal conduction time was 13 ms. The threshold of Ti recorded in muscles ipsilateral to stimulation tended to be higher than the one for eliciting excitatory contralateral motor responses (56 +/- 6% versus 46 +/- 10% maximum stimulator output). Cortical thresholds (at rest) for contralateral excitatory hand motor responses were higher in patients with developmental abnormalities of the corpus callosum than in normals (66 +/- 17% versus 46 +/- 10% maximum stimulator output), which probably reflects also a facilitatory transcallosal interaction of both motor cortices in normals. In contrast, facilitation of cortically elicited motor responses in one hand by strong contraction of the other hand was the same in the patients with agenesis of the corpus callosum and normals, which suggests that this facilitatory spread takes place on a spinal rather than on a cortical level. Central motor latencies and amplitudes of contralateral hand motor responses were the same in patients with developmental abnormalities of the corpus callosum and normals (6.1 +/- 0.7 ms versus 6.3 +/- 0.7 ms and 6.7 +/- 2.4 mV versus 6.6 +/- 2.9 mV) so that callosal transfers do not seem to influence corticospinal conduction properties.