**Case:** A 55kg 16-year-old male with presumed idiopathic kyphoscoliosis was brought to our hospital for elective posterior spinal fusion of the T3-L3 levels. In the preoperative area, an unremarkable focused history and physical exam were done, and an intravenous line was established. He was transported to the operating room where standard ASA monitors were applied. General anesthesia was induced with 100mg lidocaine, 210mg propofol, and 2mg hydromorphone. After tracheal intubation, a total intravenous anesthesia technique was chosen for maintenance, and another intravenous line and an arterial line were established. The patient was maintained on propofol and remifentanil infusions. An infusion of nicardipine was also used to maintain mean arterial pressures at approximately 60-65mmHg.

The patient was set up for multi-modality monitoring consisting of somatosensory evoked potentials, transcranial motor evoked potentials and spontaneous electromyography. No pre-positioning, supine baselines were obtained, as this is standard practice for patients with idiopathic scoliosis without other significant medical issues. Evoked potential baselines were obtained following positioning of the patient prone. It was at this point that the patient was found to have no recordable upper or lower SSEPs or MEP. A surgical pause was requested to troubleshoot the signals to rule out technical factors and/or confirm some pathologic findings. After ruling out all technical causes a change on the standard signal recording scale from 10 millisecond/division to 30 millisecond/division showed very prolonged signal latency. This represented an unusual set of responses from an adolescent with idiopathic scoliosis that typically have very robust responses with normal latencies.

**Discussion:** No technical problems were identified. Neuromonitoring findings showed extremely prolonged motor evoked potential latencies, delayed conduction, reduced effectiveness of stimulus synchrony, and severely attenuated amplitude. Given these findings, the patient appeared to have an undiagnosed peripheral polyneuropathy affecting all extremities, cause otherwise unknown.

After further investigation and more careful examination of the lower extremities, the patient was noted to have bilateral high arched feet and decreased lower extremity musculature, which are physical signs of Charcot Marie Tooth. Postoperatively, discussion with the patient's family revealed a failure to mention that the patient's brother was diagnosed with CMT.

**Summary:** Given the neuromonitoring findings, physical exam findings, and a positive family history of CMT, it is presumed that our patient likely has scoliosis secondary to a neuromuscular cause, likely CMT. A multidisciplinary team approach was essential in arriving to this conclusion. Neuromonitoring analysis prompted further investigation and a team-based approach led us to suspect a diagnosis of CMT in this patient.