Intrathecal baclofen withdrawal

Douglas AF, Weiner HL, Schwartz DR. Prolonged intrathecal baclofen withdrawal syndrome. Case report and discussion of current therapeutic management. J Neurosurg. 2005 Jun;102(6):1133-6. Review. PubMed PMID: 16028775.

Intrathecal baclofen withdrawal results in down-regulation of GABA-B receptors that cannot be compensated by PO baclofen or small doses of ITB. Interruption of ITB therapy may occur as a result of: empty pump reservoir, pump battery failure, end of pump life (Synchromed II pumps shut off automatically after 5 years, audible alarms give ample warning to replace the pump), catheter migration/breakage/kinking/disconnection/occlusion, programming error, catheter tip granuloma (less common with ITB than with opioid, or stiff-man syndrome (antibodies to glutamic acid decarboxylase which reduces available endogenous GABA). If it is necessary to electively (or semi-electively) remove a pump system (e.g. for infection), the optimal scenario is to gradually taper the drug by reprogramming the pump and/or by filling the reservoir with solution of decreased baclofen concentration. The severity of withdrawal syndrome depends on dose of drug used (increased with higher dose) and duration of therapy (increased with longer therapy). Syndromes with abrupt discontinuation of ITB: mild withdrawal symptoms: return of spasticity and rigidity, tachycardia, piloerection (goosebumps) & pruritus (pruritus without a rash is very suggestive of ITB withdrawal) more significant withdrawal symptoms: seizures & hallucinations. severe withdrawal symptoms (estimated incidence: 3–5%)78: increased rebound spasticity, rigidity, fever, labile BP, and reduced level of consciousness. If untreated, the severe syndrome can progress over 24–72 hours to rhabdomyolysis (with elevated creatine phosphokinase (CPK) and transaminase), hepatic and renal failure, DIC, and occasionally death.