05 - Mood stabilisers in hepatic impairment6,7,80

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Prescribing in hepatic and renal impairment CHAPTER 8 Mood stabilisers in hepatic impairment6,7,80 Recommendations for the use of mood-stabilising medications in hepatic impairment are summarised in Table 8.3. Table 8.3 Mood stabilisers in hepatic impairment. Drug Comments Carbamazepine6,7,80 Extensively hepatically metabolised and potent inducer of CYP450 enzymes (this can cause modest elevations in gamma-glutamyl transferase and alkaline phosphatase, which in themselves are not an indication for stopping6). In chronic stable disease, caution is advised. Associated with hepatitis, cholangitis, cholestatic and hepatocellular jaundice, and hepatic failure (rare). Adverse hepatic effects are most common in the first 2 months of treatment.80 Hepatocellular damage is often associated with a poor outcome. Vulnerability to carbamazepine-induced hepatic damage may be genetically determined.80 Avoid use in acute liver disease. In chronic liver disease reduce starting dose by 50%7 and titrate up slowly, using plasma levels to guide dosage. Stop if liver function tests (LFTs) deteriorate. Lamotrigine28 Manufacturers advise 50% reduction in initial dose, dose escalation and maintenance dose in moderate hepatic impairment and 75% reduction of these parameters in severe hepatic impairment. Discontinue if there is lamotrigine-induced rash (which can be serious). Elevated LFTs and hepatitis reported. Women, children and patients taking valproate appear to be at increased risk of lamotrigine-related hepatotoxicity. Lithium7 Not metabolised so dosage reduction not required as long as renal function is normal. Use serum levels to guide dosage and monitor more frequently if ascites status changes (volume of distribution will change). Asymptomatic and transient LFT abnormalities reported in small proportion of patients on long-term therapy.28 One case of ascites and one of hyperbilirubinaemia reported over many decades of lithium use worldwide. Valproate81 Highly protein bound and hepatically metabolised. Reduce doses and closely monitor LFTs in hepatic impairment. Use plasma levels (measure free levels; total concentrations may appear to be normal) to guide dosage. Contraindicated in severe and/or active hepatic impairment or family history of severe impairment. Impairment of usual metabolic pathway can lead to generation of hepatotoxic metabolites via alternative pathway. Risk of liver toxicity is increased in people with hepatic insufficiency if salicylates are used concomitantly. Associated with elevated LFTs and serious hepatotoxicity including fulminant hepatic failure (sometimes fatal). Mitochondrial disease, learning disability, polypharmacy, metabolic disorders and underlying hepatic disease may be risk

factors. Particularly hepatotoxic in very young children. The greatest risk is in the first 3 months of treatment.

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