1. PT is a patient stabilized on chronic phenytoin therapy. She has just been
diagnosed with rheumatoid arthritis and her physician would like to start her on
high dose aspirin therapy. However, the physician is concerned about a possible
drug interaction with aspirin. You find in your pocket reference that high dose
aspirin is known to displace phenytoin from its plasma protein binding sites.
Describe (as you would to the physician) the clinical relevance of this interaction
and your therapeutic recommendations.

2. A phenytoin patient has a plasma concentration of 10mg/L at 300mg/day and
25mg/L at 400mg/day. Using graph paper, determine the Km and Vmax as well
as the dose needed to produce a concentration of 15mg/L.

3. E. A., (37y, 70 kg, male), had been taking 300mg/day of phenytoin; however, his
dose was increased to 350 mg/day because his reported plasma concentration was
only 8 mg/L. Now his reported plasma phenytoin concentration is 20 mg/L. Both
of the reported plasma concentrations represent steady-state level. Calculate a
new daily dose of phenytoin that will result in a steady state level of 15 mg/L
(Salt factor = 0.92).

4. C.S., a 36-year-old, 70-kg woman, is to be given carbamazepine as an
anticonvulsant agent. Calculate a daily dose that will produce an average steady-
state plasma concentration of 6 mg/L.

5. O.S., a 48-year-old, 59 kg male patient, received a living-related renal transplant
1 ½ years ago. He is currently receiving cyclosporine 300 mg orally as the
solution BID and, in addition, prednisone 30 mg/day and azathioprine 75 mg/day.
O.S.’s current serum creatinine is 2.1 mg/dL and his steady-state cyclosporine
trough concentration is 590 µg/L. The physician has ruled out the possibility of
rejection and feels that the recent rise in serum creatinine is due to cyclosporine
toxicity. What questions would one ask O.S. and how would one adjust his
cyclosporine regimen in order to achieve a new steady-state cyclosporine
concentration of approximately 200 µg/L?