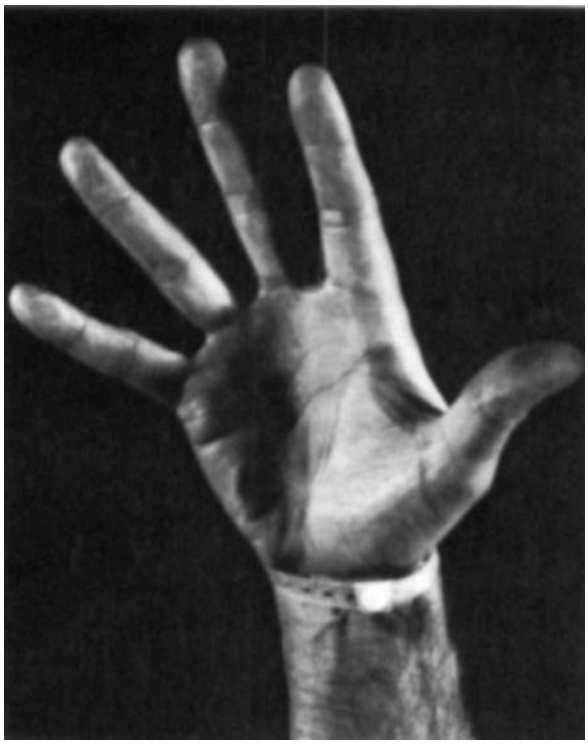

Ulnar neuropathy due to a lesion at the canal of Guyon. Note the wrinkling of the palm due to contraction of the palmaris brevis.

compression usually spares the abductor digiti quinti brevis, allowing abduction of the little finger.

In ulnar nerve lesions of the hand at the pisohamate ligament, voluntary abduction of the little finger is accompanied by wrinkling of the ulnar side of the palm due to simultaneous contraction of the palmaris brevis. This allows visual confirmation that the ulnar nerve lesion is distal to the origin of the anterior terminal division of the nerve (Figure). Even when the abductor digiti quinti brevis is weakened, digital compression of the ulnar nerve against the pisiform bone produces contraction of the palmaris brevis with wrinkling of the palm [2], again affording visual confirmation of the distal location of the site of ulnar nerve dysfunction. This sign is not present in the more common ulnar nerve lesions above the wrist, such as those occurring at the elbow.

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Chloramphenicol-Induced Phenytoin Intoxication

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It is well known that phenytoin (DPH) metabolism may be affected by various medications. Warfarin, disulfiram, and isoniazid may all elevate blood levels of DPH as well as prolong its half-life [2]. We recently observed a woman who developed DPH intoxication during treatment of meningitis with chloramphenicol.

A 30-year-old woman with a history of multiple shunting procedures for pseudotumor cerebri developed recurrent *Escherichia coli* meningitis. She was taking 300 mg of DPH and 750 mg of Mysoline daily for a seizure disorder. No nystagmus or ataxia was noted. The DPH level was 14 μg/ml and liver chemistry tests were normal. She was treated with chloramphenicol, 1 gm intravenously every 4 hours, for a total of 21 days. Ten days after admission she complained of nausea and vomiting, and 6 days later she had a seizure. Examination revealed marked horizontal and vertical nystagmus as well as appendicular and truncal ataxia. The DPH level was 33.9 μg/ml, and liver chemistry determinations remained normal. The DPH was discontinued for 3 days and she improved symptomatically; she was able to be discharged from the hospital in 3 days. On follow-up examination two weeks later she was asymptomatic.

DPH is parahydroxylated by the hepatic microsomal enzyme system, and because it is loosely bound to the microsomes, its metabolism is more likely to be inhibited than stimulated [2]. Chloramphenicol also undergoes hepatic biotransformation, by a different enzyme system than that for DPH, and it has been shown to have an inhibitory effect of noncompetitive nature on the microsomal system of mouse livers [2]. Ballek et al [1] reported a case of chloramphenicol-induced DPH intoxication; their patient's DPH level increased from 7 to 24 μg/ml following the addition of chloramphenicol.

Physicians should be aware of this interaction and make the appropriate dosage adjustments for DPH when drugs are added which affect its metabolism.

References

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