EDITORIAL

OF PILLS AND POTIONS

It would seem, if drug information sheets are to be believed, that practically every systemically administered drug has some potential ocular side effect. But are these dangers real or perceived? Given the vast quantities of pills and potions consumed, serious ocular adverse reactions would appear relatively rare. Undoubtedly, severe ocular reactions may occasionally result from systemic drug therapy: few who witnessed the oculomucocutaneous syndrome induced by the beta-blocker practolol could forget its devastating effects upon the eye. Indeed, the severity of these complications contributed, at least in part, to its final withdrawal in 1976. Fortunately, most drug-induced ocular problems are mild in comparison, the value of treatment far outweighing the potential risks. The frequency of ocular adverse reactions to systemic drug therapy varies considerably. Some effects are an almost inevitable consequence of therapy. For example, studies have shown that corneal deposits occur in approximately 98% of patients taking amiodarone,[1] and yet only a small percentage of affected individuals experience any visual symptoms, which are always mild, reversible and do not compromise the visual acuity. In contrast, adverse reactions to other drugs (e.g. corticosteroid-induced cataracts) are probably idiosyncratic, affecting only susceptible individuals. Furthermore, adverse reactions to many pharmaceutical agents probably only occur when toxic plasma concentrations are achieved; indeed, the onset of ocular problems may be the first indication of systemic toxicity.[2] In this respect, both the ocular motility problems induced by phenytoin and the visual disturbances associated with digoxin occur, in the majority of cases, when the normal plasma concentrations are exceeded.[3,4]

The development of the ubiquitous cataract has been attributed to systemic administration of a wide variety of agents. Again, the formation of posterior lens opacities following the use of systemic corticosteroids in susceptible individuals is immutable. However, whilst lenticular deposits may occur following systemic treatment with gold or chlorpromazine,[5,6] I doubt whether many ophthalmologists can recall having to remove a lens because of them. Similarly, although Bar *et al.*[7] in 1983 reported the development of presenile cataracts in two patients receiving phenytoin for the treatment of epilepsy, I am unaware of any further reports which substantiate this association.

Certain pharmaceutical agents are capable of inducing abnormalities in more than one ocular tissue. One agent which can involve both the anterior and posterior segments of the eye is tamoxifen. In 1978, Kaiser-Kupfer and Lippman[8] reported both corneal and retinal changes associated with tamoxifen therapy. These findings have been confirmed by others[9,10] and, whilst the corneal changes appear to be of no consequence, the retinal changes may result in a loss of visual acuity. In the initial report, the patients were all receiving very high doses of tamoxifen; subsequent studies have reported changes on lower dose regimes.[10] It is of interest to note that two British studies failed to find any evidence of retinal toxicity.[11,12] In this issue, Tang and her colleagues report the results of a comprehensive study into the prevalence of retinal toxicity associated with the administration of tamoxifen in patients with breast cancer. They found retinal changes in only 3 of 274 patients studied. No changes were observed in any patient receiving tamoxifen for less than 3 years or where the cumulative dose was less than 23.7 g. Moreover, visual acuity was not affected in the 3 patients who developed detectable retinal deposits. The results of this study add considerable support to the argument that serious retinal changes associated with tamoxifen, when used in low dosage, are rare and that regular screening for tamoxifen retinopathy is unwarranted.

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