

## Editorial: Mechanisms in glaucoma

All phakic eyes, in which iris and lens are apposed, generate pupil block. Iris bombé of varying degree is one consequence, and, because of this, peripheral iris moves closer to cornea, so that the first stage of angle closure—iridocorneal contact—becomes more probable. It is a necessary consequence too that pupil block occurs in eyes with deep anterior chambers, but that fact is of little clinical consequence, because iris bowing, sufficient to touch the cornea, cannot occur. Neither, it is argued, does an acute reduction in anterior chamber depth cause closed angle glaucoma, because acute reductions in depth are assumed rarely to happen spontaneously. Arguments like these have produced the present concept of primary glaucomas as representing 2 essentially different disease processes. At one extreme is closed-angle glaucoma, at the other open-angle glaucoma, with a dual mechanism operating in a very few mixed glaucomas. The article by Mr Mapstone in this issue of the *BJO* gives reason to question these views.

Experiments are described in which phenylephrine drops produced no significant change in anterior chamber depth whereas pilocarpine drops did. If the 2 drugs were instilled simultaneously, the change in depth was greater than the change produced by pilocarpine alone. But it they were instilled into an eye that had had an iridectomy an opposite change was produced, and the anterior chamber became deeper. During a positive provocative test too the anterior chamber became shallower, whereas during the resolution of the induced acute glaucoma it deepened.

These results cannot be attributed to a change in lens shape alone but must be a result of an interaction between a change in lens shape and the effects of the pupil block force. They also show that, far from being static, anterior chamber depth can undergo rapid

transient change.

One implication of these observations is that inferences made from gonioscopic appearance alone are of limited value. It can be asserted that an eye with an angle that is gonioscopically narrow may get closed-angle glaucoma, but it cannot be asserted that an eye with an angle that is not narrow will not, because acute reversible shallowing may occur. Measurement or gonioscopic assessment of the anterior chamber depth would miss this possibility. It is a necessary consequence too that eyes with medium or even wide angles can get acute closed-angle glaucoma.

There is also another implication. If an anterior chamber can develop intermittent shallowing and intermittent closure, the intraocular pressure may transiently increase. As an isolated event this may not be clinically significant, but the cumulative effect of repeated pressure increases can damage the trabecular meshwork and produce an ocular hypertension or 'open'-angle glaucoma. But the clinical appearance of the eye suggests a disease process that is wholly unrelated to an angle-closing mechanism. Many cases of primary glaucoma may simply represent the effects of one mechanism (angle closure) which can produce a spectrum of disease. At one end of the spectrum are eyes in which an angle-closing mechanism is operating in anterior chambers which are shallow all the time. This group merges imperceptibly into cases at the other end of the spectrum, that is, eyes with deeper anterior chambers which become shallow only some of the time. The mechanism of glaucoma production is essentially the same, but the clinical presentation is so different that there appear to be 2 distinct clinical entities with a different pathogenesis.



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