MECHANICS OF PUPIL BLOCK*†

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PUPIL block is widely regarded as one of the factors in the initiation of closed-angle glaucoma (Barkan 1953, 1954; Chandler 1952, 1955). Whilst the occurrence of pupil block is an established fact, statements as to its mechanism are uniformly vague; *e.g.* both Barkan and Chandler consider it to be produced by contact between the iris and lens, Sugar (1964) states that the posteriorly directed vector of force exerted by the iris sphincter is responsible, and Lowe (1966) gives an explanation of pupil block (caused by mydriatics) that depends on an incorrect resolution of forces. The mechanics of pupil block are explained below in accordance with clinical observation.

The forces involved in pupil block cannot withstand a rigorous mathematical analysis since this involves the concepts of rigid bodies and mathematical points. Such treatment does, nonetheless, provide an idea of factors involved, and reasoning along these lines leads to interesting results. Initially, certain basic facts can be established:

(1) During contraction of the sphincter muscle it is constantly attempting to reduce the area of the pupil. This force of contraction is thus resolved into a net force acting not in the direction of the iris plane but towards the axis of the pupil and thus at right angles to the antero-posterior lens axis (Fig. 1).

(2) Since there is a variable degree of iris bombé in eyes with a shallow anterior chamber, the dilating force of the dilator muscle must be the sum of the number of forces acting as shown in Fig. 2 (*a*). These can be resolved in both magnitude and direction by the line drawn straight from A to B. Thus the dilating force of the dilator muscle can be represented by line AB acting from the point of iris/lens contact to the iris insertion (Fig. 2*b*).

(3) Contraction of the sphincter results in stretching of iris tissue. This induces a contrary force tending to resist extension which acts in the same sense and direction as that due to the dilator muscle in Fig. 2.

FIG. 1.—The force resulting from contraction of the sphincter muscle acts at right-angles to the antero-posterior lens axis.

FIG. 2.—The force resulting from contraction of the dilator muscle acts from the point of iris-lens contact to the iris insertion (A to B).

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FIG. 3.—A force F has a component $F \cos u$ in a direction u° to its line of action.

(4) A force F acting in the direction shown (Fig. 3) has a component $F \cos u$ in a direction u° to its initial line of action. Also $\cos 0^\circ = 1$ and $\cos 90^\circ = 0$, i.e. the larger the angle the smaller its cosine and the smaller the component of the resolved force (Avery and Nelkon, 1954).

There are thus three forces involved, each requiring consideration:

(1) **FORCE DUE TO SPHINCTER CONTRACTION S .**—This is a force acting at right angles to the antero-posterior lens axis. The component of this force “pulling” the iris back onto the lens is directly proportional to pupil dilatation. In Fig. 4, p is the angle between the line of action of force S and the normal, at the point of iris/lens contact. The posterior component of S along this normal, pulling the iris back, is $S \cos p$. It can be seen that in extreme miosis p is an angle approaching 90° and thus $S \cos p$ is quantitatively a minute force. Conversely, as dilatation proceeds, the angle p decreases, $S \cos p$ increases, and the effect of the sphincter in causing pupil block increases as the pupil dilates.

FIG. 4.—The pupil-blocking force of the sphincter increases as the pupil dilates (angle p decreases, $\cos p$ increases).

If it is assumed that the anterior surface of the lens is a segment of a sphere of radius 10 mm. (Duke-Elder and Wybar, 1961), then $S \cos p$ can be given a rough quantitative value. Thus, at 1 mm. pupil dilatation, angle $p =$ approximately 87° and $\cos p = 0.05$. Only 1/20th of S is effective in blocking the pupil. Again, at 4 mm. dilatation, $\cos p$ is 0.2 and thus 1/5th of S is effective as a blocking force. Even at 6 mm. dilatation only approximately 1/3rd is available.

In considering the magnitude of $S \cos a$, the following factors must also be taken into consideration:

(a) **Lens Position.**—In Fig. 5a (opposite), the pupil is dilated X mm. (say). The posterior component of S is $S \cos p$. Fig. 5b shows the same degree of dilatation, but the lens is more anteriorly situated, and it can be seen that angle p does not change; thus a forward position of the lens would not increase pupil block if this were due solely to sphincter contraction. It is also apparent that increase in the sagittal diameter of the lens due to growth, and forward position due to accommodation will have no effect on $S \cos p$ for any one state of dilatation and will not increase pupil block.

(b) **Tonus of the Autonomic Nervous System.**—This will have the effect of increasing or decreasing S depending on excitation or inhibition and $S \cos p$ will vary proportionately.

FIG. 5.—Lens advancement does not alter the pupil-blocking force of the sphincter (angle p unchanged).

(c) *Parasympathomimetic Drugs*.— S will be increased and so, too, $S \cos p$ by a direct action of the drug on the motor end plates, or by choline esterase inhibition.

(d) *Parasympatholytic Drugs*.— S is decreased and similarly $S \cos p$, since the motor end plates are blocked.

(2) **FORCE DUE TO DILATOR CONTRACTION D** .—This is a force acting as shown in Fig. 2. In Fig. 6, q is the angle between the line of action of force D and the normal at the point of iris/lens contact; the posterior component of D along this normal pulling the iris back is $D \cos q$. It can be seen that in extreme miosis q is an angle less than 90° but that as dilatation occurs q increases, *i.e.* the greater the dilatation the smaller is the posterior component $D \cos q$ in causing pupil block. A point is reached with a high degree of dilatation when $D \cos q$ is negative, *i.e.* actually pulling the iris away from the lens. Assigning a rough quantitative value to $D \cos q$ is more difficult than is the case with the sphincter because angle q is subject to numerous variables. However, if the dimensions shown in Fig. 7 are assumed, *i.e.* that the lens is a segment of the sphere of radius 10 mm., the chord of the segment being 9 mm. long (Collins, 1890), and that the line of action of D is in the direction shown, then at 1 mm. dilatation $\cos q = 0.21$, *i.e.* nearly 1/4th of D is effective as a blocking force. Corresponding values for 4 and 6 mm. are 0.15 and 0.07 respectively. It should be emphasized that these values are only inserted to give an idea of how $D \cos q$ and $S \cos p$ vary with pupil dilatation. They do not indicate absolute values.

FIG. 6.—The pupil-blocking force of the dilator decreases as the pupil dilates (angle q increases, $\cos q$ decreases).

FIG. 7.—Dimensions of lens used for calculation.

As with $S \cos p$, the following factors must also be taken into consideration when assigning a value to $D \cos q$:

(a) *Lens Position.*—In Fig. 8 (a) the pupil is dilated, say, Y mm. and the posterior component of D is $D \cos q$. Fig. 8 (b) shows the same degree of dilatation but the lens is more anteriorly situated and it can be seen that angle q decreases. Thus, the forward position of the lens increases the pupil-blocking force due to the dilator muscle. Similarly, the effect of accommodation and growth of the lens will be also to increase pupil block.

FIG. 8.—Lens advancement increases the pupil-blocking force of the dilator (angle q decreases, $\cos q$ increases).

(b) *Tonus of the Autonomic Nervous System.*—This will increase or decrease D depending on inhibition or excitation, and $D \cos q$ will vary proportionately.

(c) *Sympathomimetic Drugs.*— D and $D \cos q$ are increased by direct action of the drug on the motor end plates.

(d) *Sympatholytic Drugs.*— D and $D \cos q$ are directly inhibited.

(3) *FORCE DUE TO STRETCHING OF IRIS TISSUE E .*—This force is proportional to the degree of miosis. Since it acts in the same sense and direction as force D , the factors which affect $D \cos q$ will also affect $E \cos q$, i.e. $\cos q$ will decrease with dilatation but increase with miosis. Autonomic tone and autonomic drugs will affect E indirectly in that miosis decreases and mydriasis increases angle q . Also iris stretch is greater in miosis and consequently so is force E .

The magnitude of force $(D + E)$ relative to S also needs consideration. Fig. 9 shows conditions operative at any degree of dilatation. D , E , and S are acting in the directions shown, the angle between their directions of action being c . Now in a steady state $(D + E)$ must equal $S \cos c$. (If this were not so then either miosis or mydriasis would occur depending on whether $S \cos c$ was greater or less than $D + E$). Since c is a small angle, this means that $D + E$ must nearly equal S . As dilatation occurs, E automatically decreases, but since $S = D + E$ (approximately) then D more nearly equals S . This is true for any degree of dilatation however produced. Again, if S is decreased, then so also must the sum $(D + E)$; conversely, if S is increased, then so must $(D + E)$ increase.

FIG. 9.—The force resulting from dilator contraction (D) and iris stretch (E) is approximately equal to that of sphincter contraction (S)—angle c is small, $\cos c$ is nearly 1.

Discussion

The proximate cause of primary closed-angle glaucoma is occlusion of a narrow angle by iris tissue which may be the bunched-up tissue of a dilated pupil, or the billowing iris of iris bombé, or a combination of the two. The main anatomical differences between an eye subject to closed-angle attacks and that of chronic simple glaucoma are a narrow angle and an anteriorly placed lens in the former (Sugar, 1964). Since a narrow angle by itself does not cause closed-angle glaucoma but only predisposes to this condition, then the anteriorly-placed lens must be of some significance.

In an eye with a deep anterior chamber the iris lies astride the lens. If these two structures are in contact the posterior component of S must be active in "pulling" the iris onto the lens. Since the lens is not anteriorly placed, the component of $D+E$ must be small (Fig. 8a). In spite of this blocking force pupil block and bombé do not occur—the iris plane in eyes with deep anterior chambers being typically flat. The eye predisposed to closed-angle glaucoma, however, has an anteriorly-placed lens and, whilst this does not affect the posterior component of S (Fig. 5b), the component of $D+E$ is increased. This added force, as it were, tips the scales in favour of pupil block so that iris bombé occurs and with it closure of a narrow angle.

Lowe (1964, 1966) observes that, in eyes with intact irides and shallow anterior chambers, dilatation with parasympatholytic drugs is an infrequent cause of closed-angle glaucoma, but that sympathomimetic drugs are highly dangerous. This can be explained as follows:

A parasympatholytic drug directly inhibits the force S , but $(D+E)$ retain their normal values.

Conversely, a sympathomimetic drug augments force D but leaves S unaffected. Thus the total force available for causing pupil block is greater in the latter than in the former. Hence, bombé and angle closure more readily occur. Conversely, a rise in tension is more likely to occur during provocative testing with a sympathomimetic drug than if a parasympatholytic drug is used.

Marchesani's syndrome, because of a small spherical lens and relatively lax zonule, predisposes to pupil block glaucoma. Treatment of this glaucoma with "miotics" results in a rise in tension whilst dilatation with cycloplegic drugs reverses this effect (Chandler, 1964). A parasympathomimetic drug, in these circumstances, produces the following effects:

- (1) The sphincter muscle is stimulated to contract and S increases.
- (2) Miosis decreases $S \cos p$ (Fig. 4) but increases $(D+E) \cos q$ (Fig. 6).
- (3) Lens advancement (loosening of a lax zonule) leaves $S \cos p$ unaffected (Fig. 5) but increases $(D+E) \cos q$ (Fig. 8).

All these factors combine to increase the pupil-blocking force and indirectly a rise in tension. A parasympatholytic drug, however, induces the following:

- (1) The sphincter is directly inhibited and thus S decreases.
- (2) Dilatation increases $S \cos p$ but $(D+E) \cos q$ decreases.
- (3) Lens retraction (tightening of a lax zonule) leaves $S \cos p$ unaffected but decreases $(D+E) \cos q$.

This combination of effects decreases the pupil-blocking force and thus lowers the tension. Dilatation with sympathomimetic drugs might not produce this result since:

- (1) The dilator muscle is directly stimulated and D increases.
- (2) Dilatation increases $S \cos p$, but $(D+E) \cos q$ decreases.
- (3) There is no stimulus to lens retraction (ciliary muscle unaffected). Therefore $(D+E) \cos q$ retains its previous high value.

This combination of events may well exacerbate the pupillary block.

On entering a darkened room the normal pupil dilates. The mechanism of this dilatation is relaxation of the sphincter pupillae and augmentation of the dilator muscle (Lowenstein and Loewenfeld, 1962). The effect of this on the pupil-blocking force is to decrease S and increase D but, since dilatation occurs, $S \cos p$ increases and $(D+E) \cos q$ will, depending on the degree of dilatation, remain a force of some magnitude. Herein lies an explanation of the dangers of mid-dilatation as a potent cause of pupil block, iris bombé, and angle closure, viz: $S \cos p$ is now becoming an appreciable force, $(D+E) \cos q$ is by no means entirely extinguished, and thus pupil block is around its maximum value. Further, the iris is more lax, bombé more readily occurs, and with it closure of a narrow angle. The darkroom test of Seidel as a provocative test in closed-angle glaucoma depends on some such mechanism as this.

The light reflex involving a contraction of the pupil in response to incident light is mediated largely by parasympathetic activity (Lowenstein and Loewenfeld, 1962), i.e. sphincter contraction and an increase in force S ; the accompanying miosis increases angle p , i.e. decreases $\cos p$. Hence, $S \cos p$ will not be at its maximum value. Since S has increased so also must the sum $(D+E)$; further angle q (Fig. 6) decreases, i.e. $\cos q$ increases. However, this sequence of events does not cause sufficient bombé to produce angle closure; indeed an attack of sub-acute angle-closure glaucoma can be quietly terminated by looking at a bright light. Why then should the reading provocative test (Higgitt and Smith, 1955) produce a positive result at all, since here the pupil also contracts? Accommodation is associated with miosis and retraction of the iris root (Burian and Allen, 1955). Miosis due to sphincter contraction increases S but the component $S \cos p$ will be lowered since angle p increases (Fig. 4). Since S increases, the sum $(D+E)$ increases too, as does $\cos q$ (iris root retracted and pupil miosed, Fig. 6). In addition, accommodation is associated with a change in the radius of curvature of the anterior lens surface from approximately 12 to 5 mm. (Fincham, 1937); this will increase $\cos p$ and decrease $\cos q$, a combination of events that produces a greater pupil-blocking force than the light reflex and leads to iris bombé and angle closure.

Consequently, statements that pupil block is due to the posteriorly-directed vector of force exerted by the sphincter muscle cannot withstand analysis. Throughout all the pupil-blocking situations mentioned, force $(D+E) \cos q$ is of some moment and has to be taken into consideration. Hitherto the treatment of an eye subject to angle-closure attacks has been surgical, and if outflow facility is not impaired then a peripheral iridectomy is preferred. If for some reason an iridectomy is not performed, miotics in the form of parasympathomimetic agents are prescribed.

These have the effect of increasing force S and also the component $(D+E) \cos q$. Sympatholytic agents, however, will leave S unaffected, while angle p (Fig. 4) increases (miosis) and component $S \cos p$ decreases. Similarly, the dilator is inhibited and the component $D \cos q$ (Fig. 6) is decreased. The sum of the total blocking forces would thus be less and iris bombé and closure of a narrow angle less probable. Unfortunately, drugs of this nature have hitherto received little attention in ophthalmology. Dibenzamine hydrochloride (Christensen and Swan, 1949) is too irritating for topical use. Priscol was introduced (Leydecker, 1954) as a provocative test in chronic simple glaucoma, the rationale being an increase in aqueous production by this drug. Opilon (6 acetoxymethylthymoxy ethyldimethylamine) used topically (Pau, 1955) is not irritating but produces a paralytic ptosis and chemosis. This latter exhibits adrenergic alpha receptor blocking activity only

(Birmingham and Szolcsányi, 1965); a non-toxic drug of this nature would appear to be ideal for producing miosis in an eye subject to closed-angle attacks.

Summary

The forces involved in pupil block are described and their variation with physiological and pharmacological situations considered. Reasons are given to indicate the importance of dilator muscle contraction and iris stretch in the initiation of closed-angle attacks.

The non-surgical treatment of closed-angle glaucoma is discussed.

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