Congenital deficiency of abduction, associated with impairment of adduction, retraction movements, contraction of the palpebral fissure, and oblique movements of the eye / by Alexander Duane.

CONGENITAL DEFICIENCY OF ABDUCTION, ASSOCIATED WITH IMPAIRMENT OF ADDUCTION, RETRACTION MOVEMENTS, CONTRACTION OF THE PALPEBRAL FISSURE, AND OBLIQUE MOVEMENTS OF THE EYE.

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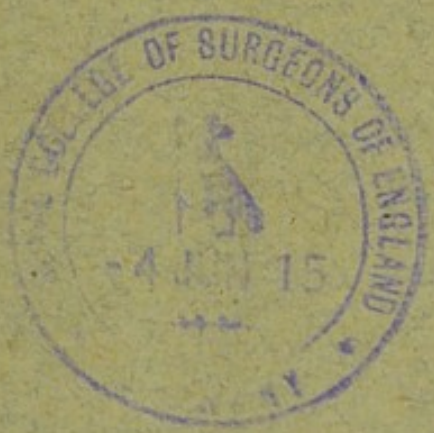

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CONGENITAL DEFICIENCY OF ABDUCTION, ASSOCIATED WITH IMPAIRMENT OF ADDUCTION, RETRACTION MOVEMENTS, CONTRACTION OF THE PALPEBRAL FISSURE AND OBLIQUE MOVEMENTS OF THE EYE.

By Dr. ALEXANDER DUANE, New York.

ATTENTION has been repeatedly called to a special class of congenital deficiency of movement of the eye characterized by all or some of the following peculiarities:

1. Complete, or less often partial, absence of outward movement in the affected eye.

2. Partial, or rarely complete, deficiency of movement inward of the affected eye.

3. Retraction of the affected eye into the orbit when it is adducted.

4. A sharply oblique movement of the affected eye either up and in or down and in when it is adducted.

5. Partial closure of the eyelids (pseudo-ptosis) of the affected eye when it is adducted.

6. Paresis, or at least marked deficiency of convergence, the affected eye remaining fixed in the primary position while the sound eye is converging.

This condition has been described more or less adequately by various authors, Collins,[15] Sinclair,[9] Türk,[10, 14] Bahr,[11] Friedenwald, [6, 12] and others; also, except so far as concerns the retraction, by Kunn.[7]

The first complete account, however, was given by Wolff,[16] who described the symptoms with more accuracy

and fulness than had been done before, and gave also a well considered presentation of the different theories that have been offered to explain the retraction. A subsequent paper by Axenfeld and Schürenberg [23] is a valuable contribution to the pathology of the condition. Continuing the work done by these two authors and by Wolff, Evans [27] gave a résumé of 27 cases, including 2 of his own, together with an excellent description of the symptoms and objective appearances.

Most of these authors have considered the condition mainly with reference to the retraction. But this, although an extremely frequent symptom, is not invariably present, and when present is sometimes but very little marked even when the other symptoms are prominent and typical. Hence it is not an absolutely essential feature of the syndrome—its absence, as Axenfeld and Schürenberg pointed out, being due to special causal conditions.

It is the aim of the present paper to collate all of Evans's cases with cases that have occurred in the author's practice and with others that are accessible in literature or in the case-books of his colleagues. In this connection the author wishes to express his sincere thanks to a number of fellow ophthalmologists, who in response to his inquiries kindly took the time and trouble to furnish him with the case-histories cited below.

Not strictly belonging in this category, but usually cited as an instance of retraction movements, is Heuck's case.*

Male, eighteen. Right eye almost immobile; it is directed 32° down, and is so firmly fixed that even under narcosis it cannot be drawn up with forceps. Enophthalmus slight in primary position; some retraction on any energetic attempt to move the eye. When attempt is made to look down and in, vertical meridian rotates to right. No spontaneous diplopia. In looking in different directions, moves head instead of eyes.

Dissection showed that the superior and external recti were attached too far back. Internal and inferior recti not examined.

* This is Case 1 of Evans's list, but Evans's description is inaccurate, applying apparently not to this case but to another one, illustrating a different kind of congenital anomaly.

The following are authentic cases of the condition in question:

Case 1. HIRSCHBERG, J.[1] [Case 5 of Kunn's list (7).]
Case 2. PFLÜGER.[2] [Case 10 of Kunn's list (7).]
Case 3. PFLÜGER.[3] [Case 11 of Kunn's list (7).]
Case 4. GRAEFE, A.[4] [Case 20 of Kunn's list (7).]
Case 5. STILLING, J.[5] [Case 2 of Evans's synopsis (27).]
Cases 6 to 9. FRIEDENWALD, H.[6, 12]
Case 10. KUNN, C.[7]; also described by WOLFF[16] and DUANE.[19] [Case 69 of Kunn's list and Case 18 of Evans's list.]
Cases 11, 12. KUNN, C.[7] Also described by WOLFF.[16] [Cases 70, 71, of Kunn's and Cases 19, 20, of Evans's list.]
Case 13. KUNN, C.[7] [Case 73 of Kunn's list.]
Case 14. RAIA, V. L.[8]
Case 15. PARKER, reported by SINCLAIR.[9] [Case 3 of Evans's list.]
Cases 16, 17. NETTLESHIP, E., reported by SINCLAIR.[9] [Cases 4 and 5 of Evans's list.]
Cases 18, 19. MACLEHOSE, reported by SINCLAIR.[9] [Cases 6 and 7 of Evans's list.]
Cases 20, 21. TÜRK.[10] [Cases 8 and 9 of Evans's list.]
Case 22. BAHR.[11] [Case 14 of Evans's list.]
Case 23. BRAUNSCHWEIG.[13] Reported by AXENFELD and SCHÜRENBERG.[23] [Case 12 of Evans's list.]
Case 24. TÜRK.[14] [Case 10 of Evans's list.]
Case 25. COLLINS, E. T.[15] [Case 11 of Evans's list.]
Case 26. INOUYE.[20] Reported by AXENFELD and SCHÜRENBERG.[23] [Case 15 of Evans's list.]
Cases 27, 28. WOLFF, J.[16] [Cases 16 and 17 of Evans's list.]
Case 29. ALLING, A. N.[17] [Case 22 of Evans's list.]
Case 30. KNAPP, H.[18] [Case 21 of Evans's list.]
Case 31. EATON, F. B.[21]
Cases 32, 33, 34. AXENFELD, T., and SCHÜRENBERG, E.[23] [Cases 23, 24, and 25 of Evans's list.]
Case 35. ZUR NEDDEN.[25]
Cases 36, 37. EVANS, J. J.[27]
Case 38. SPULER, R.[28]

CASE 39.—WEEKS, JOHN E.[32] Male; twenty. No spontaneous diplopia. Holds head directed 10° to the right and

about 5° down. In primary position there is esophoria, and L enophthalmus of 1.5*mm*. L palpebral fissure 8*mm*. L eye can move out 10° beyond middle line; power of outward movement of equal amount when eye is raised, and only 5° when eye is lowered. In moving up and out, L rises above the R, and in moving down and out goes below the R. In moving straight out it protrudes 0.75*mm* and its palpebral fissure widens 0.5*mm*. L can move straight in 17°, and in so doing does not turn up or down, but it retracts 3.5*mm*, and the palpebral fissure contracts to 3*mm*. In looking up and to the right, L rises higher than R and scarcely goes in at all; in looking down and to the right, L goes below R and goes inward about 17°. Movements of R normal and even extensive except outward, where they are slightly limited. [R palpebral fissure appears to narrow somewhat in adduction and also slightly in abduction.] Convergence = 0. Homonymous diplopia in looking to left; crossed diplopia in looking to right; vertical diplopia above and below the horizontal plane. Each eye tested separately shows following rotations:

	R	L
Out	45°	10°
In	60°	17°
Up	35°	27°
Down	65°	55°

Pupils and accommodation normal. V, R $\frac{20}{20}$ + (E); L —0.25 —0.25^c, 85°, $\frac{20}{20}$ —.

Case 40.—De Schweinitz, G. E.[33] Edward, F. G., aged thirty; defect of muscles always present. Symptoms: headache, pain in the eyes, and much wrinkling of the brow. General health good. No other members of the family similarly affected.

R + 2.75 ⊂ + .50^c axis 105°, 6/6.
L + 3.00 ⊂ + .75^c axis 80°, 6/5.

With this glass, the spherical being reduced .75, vision after mydriasis was normal in each eye. With head in the primary position, the Maddox rod showed at 6*m* esophoria 5°, right hyperphoria 9°. Typical retraction movements of R eye, with paresis of R external rectus. R eye sinks in a number of *mm* and goes slightly up, and the lids half close, reducing the normal width of the palpebral fissure fully 50 per cent. when adduction is attempted or on movement up and in and down and in. In the right

field there is homonymous lateral diplopia, which disappears gradually as the test-object is moved to the centre, and is gone when it reaches slightly to the left of the centre of the field. When the object is moved to the left lower field there is vertical diplopia, the right image being lower and to the left of the left image. These measurements were the same whether the patient looked through the correcting glasses or without them. The rotations measured on a perimeter were as follows :

R eye	L eye.
Out, 10°	Average normal rotations
Down and out, 35°	in all directions.
Up and out, 15°	
Up, 40°	
Down, 45°	
Down and in, 30°	
In, 20°	
Up and in, 45°	

CASE 41.—CARPENTER, JOHN T.[30] Female, aged forty. Diplopia since earliest recollection ; noticed when looking to left very severe nervous reflex disturbances, car-sickness, etc. Holds head directed to left, and tipped toward right shoulder.* With head directed straight forward, L extreme enophthalmus and L convergent squint of 6°; L palpebral fissure same size as R. L can be abducted *2mm* beyond median line, and abducted still more when above or below the horizontal plane ; in abduction, eye comes forward, but width of palpebral fissure does not change. Adduction L subnormal (cornea does not reach inner canthus) ; eyeball at same time becomes more retracted and moves obliquely up ; palpebral fissure does not narrow. Movements of L eye straight up nearly normal ; down, somewhat restricted. Movements of R normal. Convergence near-point *9cm* from root of nose. Homonymous diplopia increasing greatly in looking to left ; vertical diplopia everywhere except in right upper field where there is single vision. When R eye is screened and L is made to fix, R shows a pronounced secondary deviation, especially in looking to the left and below. R + 0.25 cyl. 90°, $\frac{6}{6}$; L + 0.25 + 0.25 cyl. 90°, $\frac{6}{6}$.

CASE 42.—ALLING, A. N.[31] Female, aged seventeen. Congenital paralysis L externus. L when adducted retracts. Eye

* Evidently to obviate the vertical diplopia.—A. D.

normal except for My. of 1 D. Case exactly like Alling's earlier recorded case. (See No. 29.)

Case 43.—Thomas, F. G.[30] Female, aged ten. Holds head turned slightly to left. Occasional momentary diplopia and vertigo. With head directed straight forward, orthophoria. L abduction = 0, but in attempted abduction there is slight rotation upward and slight widening of the palpebral fissure. Power of abduction rather greater than when eye is elevated and rather less when it is depressed. L adduction more nearly normal, but, on looking above horizontal, movement is restricted, and at same time eye makes a marked clockwise rotation about the antero-posterior axis ; and there is slight narrowing of the palpebral fissure. In adduction there is a slight tendency of the eye to move obliquely up, this being more marked when the eye is elevated. Restriction in motility of L eye same whether R eye was covered or not. Convergence near-point 20.5*cm* from root of nose. L eye could not be abducted with forceps. In chosen position of head, binocular vision is quite perfect both at distance and near, but in any other position of the head, the left image is at once suppressed. R $\frac{6}{6}$ (+ 1.00 sph. under atropine) ; L (under atropine) + 0.25 sph $\bigcirc$ + 1.00 cyl. 120°, $\frac{6}{6}$. Symptoms entirely relieved by use of glasses, which patient of her own accord uses constantly.

Case 44.—Gradle, H.[34] Female, aged twenty-two. Asthenopia due to refractive error. No spontaneous diplopia. Holds head straight. In primary position orthophoria ; slight enophthalmus L. L abduction = 0 ; adduction 1–2*mm* and as eye turns in, it retracts, turns up, and becomes intorted. Relations of palpebral fissure not noted. Convergence = 0. When R eye is covered and L fixes, L shows a pronounced secondary deviation.

R + 6.00 + 1.00 cyl. 90°, $\frac{20}{30}$; L + 6.00 + 2.00 cyl. 180°, $\frac{20}{80}$.

Cases 45, 46.—Schapringer, A.[36, 37]

Case 47.—Schapringer, A.[38] Female, aged thirty. L enophthalmus in primary position. L abduction = 0 ; adduction restricted and difficult, and associated with slight retraction. R V and refraction normal ; L $\frac{10}{200}$; apparently normal.

The following cases occurred in my own practice:

Case 48.—Duane, A.[19] Bertha S.; eight. Deviation of eyes noticed since birth. Used to hold head to right, and does so still

when looking intently at anything. Left eye cannot move out beyond median line. When eyes are carried horizontally to the right, the left eye suddenly flies upward and becomes buried beneath the upper lid. In associated movements in the lower field, the left eye moves normally with the right. Behind the screen the left eye deviates high up and somewhat in. Deviation of either eye behind the screen about equal. No double images attainable. V : R $\frac{20}{40}$, with + 1.00 D $\frac{20}{30}$; L $\frac{20}{40}$.

Case 49.—Duane, A.[19] Bella S., aged twenty. Headache, unilateral or bilateral, and pain in left eye noted for two years, since she has been using her eyes for close work. Asthenopia. Vertigo slight. No spontaneous diplopia.

Under homatropine, R + 1.25 D $\frac{20}{20}$; L + 1.50 D $\frac{20}{30}$. In the primary position, moderate convergent squint greater for distance than for near. Screen deviation of left eye much more marked than right.

R eye cannot move out beyond the middle line. Movement inward, though varying in amount at different times, restricted (from $\frac{1}{2}$ to $\frac{3}{4}$ of the normal). In looking up and to the right the R. eye can move slightly outward, and if then the test object is carried vertically down, the eye moves down and in. So also, in looking down and to the right, the R eye can move slightly outward, and if then the test object is carried straight up, the eye moves up and in.

In looking to the left there is no retraction of R eye, nor any tendency to an oblique movement upward or downward, but R palpebral fissure contracts from a width of 7 or 8*mm* down to 3*mm*. In looking to the right, L palpebral fissure shows a similar contraction.

In the primary position there is homonymous diplopia, which diminishes as the eyes are carried to the left, and when the eyes are turned about 10° to the left there is single vision and true orthophoria. As the eyes are turned still farther to the left, the orthophoria is succeeded by exophoria, and a little farther on (when the eyes are directed 25° to the left) there is crossed diplopia. Near-point of convergence from 7 to 15*cm*.

Case 50.—N. F. V., a man of thirty-one. L eye has never been able to turn out, and whenever he attempts to turn his eyes to the left has diplopia ; has to turn his head in order to avoid this. No asthenopia, pain, or vertigo.

Under homatropine : R emmetropic, V = $\frac{15}{12}$; L — 0.25, V =

$\frac{15}{15}$. In the primary position, esophoria of 3° to 5° with binocular single vision, and a prism divergence of 3°. As soon as he looks to the left, gets a marked and increasing homonymous diplopia. In looking up and to the right there is crossed diplopia with the image of the right eye higher (left hyperphoria of 8° or 9°). The left eye can move out only a few degrees beyond the middle line; movement is somewhat restricted. The movement up of both eyes is markedly restricted and painful.

MONOCULAR FIELD OF FIXATION.

	R	L
Out.....	35°	12°
In.....	48°	37°
Up.....	12°	13°
Down.....	63°	55°

Near-point of convergence, from 7 to 12*cm.* Little, if any, impairment of projection in either eye.

CASE 51.—Minnie B., aged sixteen. Headaches frequent, especially in the right temple, also a feeling of tension in the eyes after using them for near work. There seems to have always been something obviously wrong in the appearance of the eye, for during the last 9 years they have been repeatedly examined and she has always been asked if she did not "see double." She has, however, never had any spontaneous diplopia. L eye cannot move out at all beyond the middle line. Movement inward less than normal by 1 or 2*mm.* Movements of R eye normal, except that adduction is also somewhat defective. When she looks to the right, L palpebral fissure contracts from a width of 12 or 13*mm* down to 8*mm.* There is no retraction of the eye when she looks to the right, but there are slight, slow, nystagmoid twitchings in both eyes; when she looks to the left there is sometimes a slight lateral and rotary nystagmus. When the eyes are directed a little above the horizontal plane and carried to the right, L eye shoots obliquely up above the right and also diverges. When the eyes are carried up and to the left, L eye can move out a little beyond the middle line.

In the primary position there is a moderate divergent squint, the deviation of the right eye behind the screen being fully three times as great as that of the left (*i. e.*, it takes a prism of over 30° base in before the right eye, and one of only 10° before the left

eye, to abolish the screen movement). When the eyes are turned slightly to the left the divergent squint is replaced by orthophoria, and as the eyes are turned still farther this almost immediately gives place to a convergent squint. Here again the screen deviation of the right eye (secondary deviation) greatly exceeds the primary deviation of the left eye.

In the primary position there is crossed diplopia with the Maddox rod. No hyperphoria in the primary position with the Maddox rod, but when the eyes are carried up and to the right, there is a left hyperphoria of 15°. There is marked paresis of convergence. In following, with both eyes open, an object carried toward the eyes in the middle line, the left eye remains fixed in the mid-position, while the right eye converges on the object. There appears, however, to be some attempt at convergence, for in this case there is a fairly well-marked convergence-reaction of the pupil. This is also the case if the right eye is made to converge on the object and the left eye is covered. If, however, the right eye is covered and the left is made to converge on the object, the right eye diverges almost to the outer canthus and the pupils do not contract.

The patient ordinarily carries her head turned slightly to the left. Projection with the left eye is somewhat faulty, the finger being carried a little beyond the object when she is looking either to the right or to the left.

At first, examination showed under homatropine R+ 2.00 + 0.50 cyl. 100°, L + 0.75 + 2.50 cyl. 80° and got R + 1.50 + 0.50 cyl. 100° ; L + 2.50 cyl. 80°. Troubles continuing and increasing in spite of this prescription, free tenotomy of right externus was performed. This was done because it was evident that what caused her the most trouble was not the homonymous diplopia in looking to the left, but the crossed diplopia in looking to the right. To have attempted to remedy this by advancing the left internus would have been improper, for advancement done on the affected eye in such cases simply increases the retraction and the sense of tension which the patients complain of. Result, six months after operation : Patient holds head 5°–10° to right and 20° when object is at reading distance. When eyes are directed straight forward, esophoria 5°–6°. In looking to the right, crossed diplopia increasing slowly (up to 12°–14° prism) ; *primary and secondary deviations equal ;* abduction R limited 2–3*mm ;* convergence still very deficient (convergence near-point about

80 *cm.*), and in converging with right eye covered, latter flies far out (excessive secondary deviation). In looking up and to right, L shoots high above R ; in looking down and to left, shoots far below R. In looking to right, moderate contraction of L palpebral fissure ; in looking to left, slight contraction of R and slight dilatation of L palpebral fissure.

Immediate effect of operation was relief of feeling of strain, but this returned two months later, and was finally much benefited by changing her glasses to R + 1.75 + 0.75 cyl. 100°, L + 0.75 + 2.50^{c} 80° ; with these V = R $\frac{15}{15}$; L $\frac{15}{30}$.

Case 52.*—D. T. K., male, aged twenty-two. Asthenopia dolens for ten years, partly relieved by glasses. Occasional vertigo after reading; occasional momentary diplopia for distance and also diplopia (vertical) in reading. Holds head directed about 10° to the right. In this position, esophoria of 4° or 5° (prism) and, with red glass, slight homonymous diplopia. With head straight, there is spontaneous diplopia without the red glass, R converging and also being noticeably higher than L. No enophthalmus in primary position and R palpebral fissure = 10*mm.* R cannot move out beyond the middle line; movement outward somewhat greater when eye is directed downward. In abduction, eye does not protrude, but palpebral fissure widens a little. R moves in to about 1*mm* of caruncle and in so doing it moves obliquely up and retracts 4–5*mm*, and its palpebral fissure narrows to 5*mm*. In looking up and to left, R goes nearly straight up and much higher than the L. Movements of L eye straight up and straight down normal. Movements of R normal. Convergence near-point 25–30*cm* from root of nose. On reading holds book slightly to right. With red glass, homonymous diplopia increasing fast in looking to the right; slight vertical diplopia in this part of field. In looking slightly to left, single vision, changing almost at once to crossed diplopia, which increases fast and is accompanied by a rapidly increasing vertical diplopia (with image of right eye below = right diplopia). The vertical diplopia increases greatly in looking up and to the right, and continues, although constantly diminishing, down to and even some distance below the horizontal plane (= spasm R inf. oblique). Very marked secondary deviation of L eye.—*i. e.*, in looking to right, L eye, when R fixes, deviates excessively in; in looking to left, it deviates excessively out. In the patient's

* For the privilege of examining this case I am indebted to Dr. C. H. May.

chosen position of the gaze with head directed slightly to the right, the screen-deviation of the L eye is 4–5° (prism); when R is covered and L fixes, the (secondary) deviation of the R is 10° to 12°. With head straight, primary deviation is 14° (prism), secondary is 20°. So also at 12 inches, with the object in the median line, the deviation of the R eye out behind the screen is 12°, that of the L is 18°; and a similar disproportion between the primary and secondary outward deviation occurs in looking to the left. With Dr. May's correction, R + 1.00 + 0.50 cyl. 70°; L + 1.50 + 0.50 cyl. 60°; V = $\frac{15}{15}$ each.

Case 53.—K. D., female, aged twenty-six. Asthenopia dolens and headache brought on by work at sewing machine. No diplopia noticed. Holds head directed about 10° to the left; in this position there is orthophoria and no enophthalmus, but L palpebral fissure is 9*mm*, while R is 10. L abduction = 0 or at most 2–3*mm;* in attempted abduction it does not come forward, nor does the palpebral fissure widen. Power of abduction not greater in upper or lower field than when eye is directed horizontally. L eye can move in to 1-2*mm* of caruncle; in adduction, it does not move obliquely up or down, but is retracted 2–3*mm*, and palpebral fissure contracts to 7*mm*. When the eye is carried far inward it sometimes makes a marked movement of extorsion (vertical meridian rotates outward). Movements up and down normal. R adduction 1–2*mm* less than normal, and movement is jerky; palpebral fissure remains of same width when eye is abducted. R adduction apparently normal, and there is no retraction, but palpebral fissure contracts to 8*mm*. Convergence near-point 12–17*cm;* sometimes eyes make no effort to converge. In primary position, single vision passing almost at once into homonymous diplopia as eyes are turned to the left, and into crossed diplopia as the eyes are turned to the right. No vertical diplopia. In looking to the left, secondary inward deviation of R eye much greater than primary deviation of L; in looking to right, primary and secondary deviations about equal. R $\frac{20}{20}$—; E. L $\frac{20}{40}$—; — 0.75 cyl. 90°, $\frac{20}{30}$. Ordered R plane, L — 0.75 cyl. 90°.

Case 54.—Schapringer, A.[39] Female, aged twenty-two. Has always had diplopia when she turned her eyes in a certain direction. R and L emmetropic and V normal. R movements normal. L abduction slightly impaired (cornea does not go quite to outer commissure); when she tries to look to right, L eye retracts slightly and goes almost straight upward; palpebral

fissure at same time contracts markedly. When she looks down and to the right, adduction much more extensive. No enophthalmus in primary position. Usually holds head turned to the right and tilted toward left shoulder. If head is directed straight, L strabismus divergens and sursumvergens.

The findings in these cases may be summarized as follows:

OBJECTIVE SYMPTOMS.

(*a*) *Restriction of Abduction.*—In 41 of the cases abduction was altogether or almost altogether absent, while in 12 it was more or less deficient. In one (Case 54) it was only slightly restricted. Even, however, when it was impossible, or at least very difficult, for the patient to abduct the eye in the horizontal plane, it was noted in several instances (Cases 10, 41, 43, 46, 49, 51, 52) that he could abduct it to a notably greater extent when it was directed up or when it was directed down. This fact is significant as indicating the part taken by the superior and inferior obliques in performing abduction—*i. e.*, although the externus is completely inert, the inferior oblique will abduct the eye somewhat, particularly when the eye is elevated, and the superior oblique will abduct it when it is depressed. If the external rectus retain some power so that it will abduct the eye moderately in the horizontal plane, the superior and inferior obliques will be still better able to reinforce it, and in such a case the power of outward movement in the upper and lower fields may be complete. This was seen in Case 11.

It is further to be noted that in spite of the completeness of paralysis of abduction that obtains in most cases, there is rarely, if ever, any contracture of the internus.

(*b*) *Restriction of Adduction.*—Out of 49 cases, adduction was normal in only 2, was diminished slightly in 2, diminished more or less considerably in 37, and absent or practically absent in 8. Moreover, as Kunn pointed out, even when the eye can be adducted pretty far, it cannot maintain adduction well nor keep up fixation when adducted.

(*c*) *Oblique Movements Upward or Downward.*—In 31 cases at least, oblique movements were noted in the affected eye. In 24 the eye when it was adducted moved up and in, in 3 it moved down and in, and in 4 it moved up and in when the eyes were directed somewhat up, and down and in when the eyes were directed downward. The oblique movement was absent in at least 3 cases (45, 49, 53).

This peculiar oblique movement of the eyes may be observed in certain cases of acquired paralysis. It is then usually the expression of the secondary deviation that occurs in the non-paretic eye when the paralyzed eye is used for fixing.

(*d*) *Torsion Movements.*—In 5 cases, when the affected eye was carried in, or up and in, its vertical meridian rotated strongly outward (spasmodic action of inferior oblique). In one case (Case 44), when the attempt was made at adduction the eye went up and rotated inward (spasmodic action of the superior rectus). In Cases 10, 21, and 34 torsion movements, indicating probably spasmodic action of the obliques, took place when attempts were made to abduct the eye.

(*e*) *Retraction Movements.*—Retraction movements were noted as absent in 2 cases (49, 51), very slight in 3, slight in 6,* and more or less well-marked in 32. It may be present, as in Cases 14 and 16, when there is no adduction at all, but the patient simply makes an effort to adduct.

When present, the amount of the retraction varied from 1 to 10*mm.* To determine the amount, it is a good plan, as Weeks suggests, to put a spectacle frame containing a diaphragm on the patient and measure the distance between the diaphragm and the eyeball when the eye is turned in various positions.

Sometimes there is an appearance of retraction when no retraction really exists. Thus, the mere closure of the palpebral fissure, which occurs when the affected eye is adducted, makes the eye look as if it had receded. Again, if we look at the eye from the side and the eye turns inward, it will seem to recede, because the cornea which before was

* In one of these (Case 43) perhaps absent as the only evidence of its presence was the contraction of the palpebral fissure.

projecting, and therefore nearer our plane of measurement, is now turned out of the way. To avoid this error the measurement from the diaphragm in the spectacle frame should be taken to the sclera at the edge of the cornea and not to the cornea itself.

In the same case the amount of retraction may vary greatly from time to time (Case 46).

In at least 2 cases (33, 38), in which retraction was produced by voluntary adduction of the eye, it could not be produced when the eye was drawn inward with forceps.

Not only is there recession of the affected eye when adducted, but it may be sunken when the eyes are looking straight ahead. This was noted in 14 cases, although in 5 the enophthalmus was but slight. In one (Case 41) it was extreme. In 3 cases (52, 53, 54) there was no enophthalmus in the primary position.

It must be further noted that, although very rarely, retraction movements are found in other conditions besides the one here described. Thus, it was present in a case noted by Varese,[24] in which there was absolute paralysis of adduction and but slight restriction of the other movements of the eye, yet, whenever the attempt was made to adduct the eye it was retracted strongly. This case was not unlike Case 54 and may after all have to be classed with our cases. Perhaps also to be classed with them, although this seems doubtful, is a case seen by De Schweinitz[33] in which there was an ordinary convergent squint with only slight limitation of outward movement, but in which there were decided retraction movements with narrowing of the palpebral fissure.

Nor is the condition always congenital. In a case reported by Koerber[26] there was retraction in an atrophic stump whenever an attempt was made to move the eyeball upward. Here the retraction was doubtless due, as Koerber thinks, to the presence of cicatricial bands.

(*f*) *Protraction in Abduction.*—In nine cases it was noted that the affected eye protruded somewhat when abducted. In two of these cases this protraction was very slight. In two cases (52, 53) it was not present.

(*g*) *Narrowing of the Palpebral Fissure.*—Closure of the palpebral fissure when the affected eye was adducted was noted as present in 40 of the cases and as absent in 2. In three of the cases in which it was present it was but slight. It seems a significant fact that it was present in one case (Case 13) at least, when, although adduction itself was impossible, yet contraction of the lids occurred whenever the attempt at adduction was made.

There may be some contraction of the lids even in the primary position. This was so in at least 9 cases. In 2 others the palpebral apertures were alike in the two eyes, and in one case (Case 7) the affected eye was wider open than the other.

In at least 4 cases narrowing of the palpebral fissure was observed in both the affected and the sound eye.

When the eye is abducted the *palpebral fissure often dilates* somewhat. This was noted in fourteen cases, but was not present in Case 53.

This peculiar contraction of the palpebral fissure during associated lateral movements of the eyes occurs in other conditions as well. A collection of such cases with extensive bibliography is given by Wilbrand and Saenger.[22] See also Friedenwald.[6] In conditions except that here described the contraction usually takes place when the eye is abducted. I myself noted one such case in which there was a congenital paralysis of the right inferior oblique, and in which, when the eyes were directed to the right, the right palpebral fissure was *6mm* in width, but when directed to the left was *13mm.*

The narrowing of the palpebral fissure, it must be noted, is evidently not a ptosis but due to *contraction of the orbicularis*, the closure being effected as much by an ascent of the lower lid as by a descent of the upper.

(*h*) *Insufficiency of Convergence.*—In 20 of the cases in which the power of convergence was examined, it was normal or but slightly subnormal in 4, decidedly subnormal or paretic in 7, and nearly or quite absent in 4. The ability to perform convergence, however, appears to depend very much upon how the eyes are directed. If directed away

from the side of the insufficient internus they may converge quite well.

The objective symptoms just described constitute the characteristic features of the syndrome. Other features of more or less significance are the following:

(*i*) *Restriction of Passive Movement.*—In most of the cases in which traction with forceps has been tried, it has been found that there is more or less restriction of passive movement, either outward (4 cases) or inward (2 cases). Such restriction, of course, indicates the existence of an actual obstacle, probably an inelastic band, preventing the movement in a given direction. In one case there was very slight resistance to traction either out or in, and in Case 38 the eye could be moved in either direction, but when released flew back to its original position (indicating probably the presence of a more or less elastic band, opposed to the movement).

(*j*) *Deviation in the Primary Position.*—In the primary position there may be orthophoria (noted in 6 cases), divergence (10 cases), or convergence (18 cases). If there is convergence (esophoria or convergent squint) in the primary position, the eyes will be in equilibrium or there will be orthophoria, when the gaze is directed toward the side of the unaffected eye. If there is exophoria or divergent squint in the primary position, the eyes will have to be turned somewhat to the side of the affected eye before equilibrium is attained. In any event, the range through which equilibrium is maintained is usually a very narrow one, the condition changing quickly into one of convergent squint when the eyes are turned in one direction and into divergent squint when the eyes are turned in the opposite direction.

In a number of cases there is marked hyperphoria or vertical squint in the primary position.

(*k*) *Secondary Deviation of the Sound Eye.*—When the affected eye is made to fix, and the sound eye is covered, the latter usually shows a pronounced secondary deviation (so in 13 of the cases). In 8 of these it is expressly stated that the secondary exceeded the primary deviation, just as

it does in ordinary paralyses. In two cases the primary seemed to equal the secondary deviation, and in one case (Case 24) there was no secondary deviation at all.

(*l*) *Position of the Head.*—In many cases the patient carries the head to one side either to avoid diplopia or possibly to avoid the tension produced on the eye when it was not in the position of equilibrium. When there is a tendency to convergence in the primary position, he will, of course, turn the head toward the inactive externus; if there is a tendency to divergence, he will turn the head the other way.

(*m*) *False Projection.*—False projection appears to be an infrequent and little-marked symptom in this condition.

(*n*) *Nystagmus and Pseudo-nystagmus.*—True nystagmus and slow nystagmoid twitchings may occur in these cases particularly when the eyes are carried toward the limits of their movement.

(*o*) *Relation to Ametropia.*—Most of the cases are hypermetropic, but few extremely so. Nor does there seem to be any undue number of cases of high astigmatism or marked anisometropia.

(*p*) *Frequency in Women.*—Out of 51 cases in which the sex was noted, 31 were women. This preponderance is the more striking since the other congenital motor anomalies are more frequent in males. Thus in Kunn's list, if we except the cases of the anomaly now under consideration, 38 were males and 27 females.

(*q*) *Frequency in the Left Eye.*—Out of 54 cases, the left eye was affected in 38, the right in 11, while 5 were bilateral. In one of the bilateral cases (No. 31), the affection was almost exclusively left-eyed; and in 3 of the left-eyed cases (13, 51, 53), there were evidences of very slight restriction in the right eye.

The comparative infrequency of the bilateral cases is surprising, since other congenital motor anomalies are as a rule bilateral. Thus, excluding cases of the anomaly now under consideration, Kunn's list of congenital defects showed 4 right-sided, 12 left-sided, and 50 bilateral.

That in the unilateral cases the left eye is more prone to be affected than the right, agrees with what we find in con-

genital anomalies in general (see Kunn's statistics above). Possibly analogous is the fact that in unilateral reflex iridoplegia the left eye is far more often affected than the right.

(*r*) *Absence of Involvement of Pupil or Ciliary Muscle.*—In none of these cases is there noted any impairment of accommodation or any derangement of the pupil. Of course, in those cases in which the convergence is weak or abolished the convergence reaction of the pupil will be absent, just as it is in any convergence insufficiency when the eyes are directed at some object within the convergence near-point.

SUBJECTIVE SYMPTOMS.

The subjective symptoms are not very marked. They are:

(*a*) *Imperfect Vision.*—In some, but by no means all of the cases, the affected eye was more or less amblyopic. In 16 the affected eye had normal vision, in 7 the vision was $\frac{20}{40}$ or better, and in 11 less than $\frac{20}{40}$. The poor vision in some cases could be attributed to anisometropia, high hypermetropia or astigmatism, or marked squint.

(*b*) *Asthenopia, headache, and a sense of more or less strain* when the eyes are used, or of tension when they are moved, are also sometimes complained of, and, in some cases at least, are evidently due to the abnormal condition of the eye muscles (see Case 10).

(*c*) *Diplopia.*—Spontaneous diplopia is sometimes present (in at least eight of the cases),* and is occasionally troublesome. Sometimes the patient himself observes that diplopia occurs only when he turns his head in a certain direction (see Cases 41, 50, 54).

If diplopia is not present spontaneously it can almost always be elicited by tests. In the cases that came under my observation, the field of single vision was evidently very narrow (often only a few degrees). When we consider how comparatively good the action of the internus apparently

* It was doubtless present in others, especially of the younger patients, from whom sufficient data were not or could not be elicited.

is, this seems rather surprising. In cases like these, when the abducens is completely powerless we should expect to find, as we do, an homonymous diplopia occupying almost the entire half of the field of vision ; but it is not so obvious why, with a comparatively good internus, we should have almost all the rest of the field occupied by a crossed diplopia and but a narrow strip, therefore, in which true single vision obtains. This fact and the occurrence of an excessive secondary deviation of the other eye when the affected eye fixed an object toward its inner side, indicated that the internus, however effective apparently, was at least relatively weak.

In the area within which the patient sees single, he has true stereoscopic vision, as shown by Hering's test or by the amblyoscope.

(*d*) *Vertigo.*—This seems rare. In Case 10 it may have been of aural origin.

EXPLANATIONS OFFERED TO ACCOUNT FOR THE PHENOMENA.

Various theories have been advanced to account for this singular combination of symptoms. Such theories must explain why, in a given case, we have (*a*) the absence of abduction, (*b*) the restriction of adduction, (*c*) the retraction movements of the eye, (*d*) the oblique movements observed in adduction, (*e*) the peculiar lid movement observed in adduction, and (*f*) the deficiency of convergence. We may add that, whatever the causal condition may be, it must surely be peripheral in site. We cannot imagine any nuclear or neural lesion that would produce all the phenomena above given, nor one that could produce but a portion of them, and yet invariably leave the interior muscles of the eye exempt.

(*a*) *The absence of abduction* in these cases is most probably accounted for by the fact that the external rectus is absent (as in Case 22), or replaced by an elastic or inelastic connective-tissue cord, as was found by dissection to be the condition present in Cases 26, 29, 33, 34, and 36.

The fact that, in spite of this complete absence of abduction, there is yet no contracture of the internus does not seem astonishing, since secondary contracture is usually* absent in congenital paralyses.

(*b*) *Restriction of adduction* may be ascribed to two causes. First, the internus may be normal and normally inserted, but its contraction may be opposed by a more or less inextensible cord replacing the externus. This condition seems to have obtained actually in some of the cases in which the tendon has been exposed by operation, notably in Cases 26, 29, 33, 34, and 36. It may reasonably be inferred to exist in a given case if, on making passive traction with the forceps, we find that there is a marked obstacle to the movement of adduction, the eye rotating in with difficulty or imperfectly (Axenfeld and Schürenberg).

Second, the internus may be abnormally inserted, especially may be inserted too far back. This condition also has been found to exist anatomically, as in Case 36. It may be inferred to exist in any case if on traction with forceps the eye can be adducted fully and fails to retract.

One point must be considered in trying to account for this defect of inward movement. This defect, apparently slight if we consider the movement of the affected eye alone, is really considerable if we consider the movement of the two eyes together. Thus, even though the internus may carry the eye inward until the cornea nearly reaches the caruncle, there may yet be crossed diplopia over the entire inner half of the field of fixation, showing that even in the primary position the affected eye begins to lag behind its fellow. Again, the presence of an excessive secondary deviation of the sound eye shows that, as compared with its fellow, the movement of the affected eye is considerably hampered in making even the beginning of an inward rotation. This, in itself, would indicate that the cause of the defective movement inward is to be found rather in a mechanical obstacle than in an impairment of nerve energy.

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* Not, however, invariably. See Graefe.[4]

(*c*) *To explain the retraction movements* of the eye no less than five theories have been advanced.

Heuck[3] supposed that the tendon of the internus was inserted too far back, so that it acted not only to pull the eye inward but also backward. In his case and also in one observed by Evans (Case 36), this anatomical anomaly was found to be actually present.

Bahr[11] found in one case that the internal rectus had a second slip inserted far back and acting as a retractor, while the main body of the muscle acted as an adductor. Axenfeld and Schürenberg[23] believed that the same anomaly was present in at least one of their cases (Case 33), since forced traction with forceps failed to produce the retraction and tenotomy of the internus did not abolish it. Knapp's case (Case 30) also seemed to present something of the same sort. Treacher Collins[16] supposed that not only was the tendon inserted too far back, but that there was a deficiency of the check ligaments, so that the natural tendency of the eye to recede was unopposed.

Türk's theory, adopted by Wolff,[16] is that the retraction is due simply to the inextensibility of the external rectus. Under ordinary circumstances, as the internus contracts the externus elongates, so that the eye simply rotates about its centre. On the present hypothesis the externus fails to elongate and the internus, therefore, can contract only in case the eyeball recedes.

This explanation doubtless holds for many of the cases, particularly those in which anatomical examination has shown the insertion of the internus to be normal. This also accounts well for the impairment of rotation inward which these cases show. It seems scarcely to account for the retraction observed in cases where there is no power of adduction at all (see Cases 13, 14, 16, 22, 27, 38, 44, also the case of Varese already cited), nor for the cases in which forced adduction by forceps caused no retraction (Cases 33, 38).

Parker,[9] Peschel,[29] and Schapringer think that retraction is caused by the superior and inferior recti. In those cases where the action of the internus is ineffective, Parker sup-

poses that the superior and inferior recti, acting vicariously as adductors, contract very strongly, and in doing so pull the eye back into the orbit. If this hypothesis be true, we must suppose that the two obliques, which would oppose this retraction, are at the same time relaxed. This Peschel admits and further believes—as Spuler[25] had done before him—that the propulsion of the eye, observed when it is rotated outward, is due to the protracting action of the obliques, which in this situation act very strongly as abductors in place of the ineffective externus.

This hypothesis, so far at least as relates to the retraction, seems to be open to serious objection, as there is no reason for believing that the obliques do relax when the eye is adducted; on the contrary, in these cases they seem to act very strongly. To this point we shall return later.

From the diversity of pathological conditions found, and particularly from the great variation shown in the amount of retraction in cases apparently quite similar, it is evident that the cause of the retraction is not the same in all cases. Indeed when the retraction is very marked it is probable that it may be due to several causes combined.

(*d*) To account for the *oblique movements of the eye*, Wolff[18] suggests the following hypothesis:

"As the eyeball recedes into the orbit it displaces the orbital contents, but the optic nerve, having a firm consistency, offers a certain amount of resistance to the backward movement of the eyeball at its point of attachment. Unless this resistance is made just in the plane of the retraction, the effect must be to rotate the cornea either upwards or downwards: upwards, if the point of resistance lies below the point of retraction; downwards, if it lies above."

In answer to this I have elsewhere said[19]:

"The resistance offered by a flexible body like the optic nerve must be slight indeed, and could scarcely be constant in character and direction. The deviation due to it, therefore, would be irregular and subject to sudden variations, and not marked by the uniform steady increase in the upper field, and the uniform steady decrease in the lower field shown in the present case.

"The vertical deflection might be ascribed to an anomalous insertion of the internus, causing it to act partly as an elevator; but a similar objection to that made against Wolff's hypothesis applies here—namely, that a deviation due to this cause would not be essentially greater when the eyes were directed up than when they were directed down.

"Furthermore, neither theory accounts at all for the singular fact that the left eye, in spite of the almost complete inaction of its externus, diverges markedly and progressively as the eyes are elevated, and when elevated can be abducted to quite a considerable degree.*

"On the other hand, all the conditions are explained upon the hypothesis of an *excessive spasmodic contraction of the inferior oblique*. Such a contraction would tend to carry the eye upward, and the more so in proportion as the eye is turned inward. The ability of the inferior oblique to carry the eye upward, moreover, would increase steadily in the upper field of fixation and diminish steadily in the lower field. Again, when the eye was carried upward, and especially when it was carried upward and outward, the abducting action of the inferior oblique would come into play, and if excessive would cause the eye to diverge in spite of the co-existing paralysis of the externus. Furthermore, in upward, and especially in upward and outward directions of the gaze, the abducting action of the inferior oblique, if excessively exerted, would enable the eye, as in this case, to move outward moderately, even in default of any action of the externus.

"The character of the diplopia, and the way in which it increases and decreases, as also the manner in which the field of fixation is restricted, are strictly in accord with this hypothesis.

"The fact that the diplopia and deviation varied in amount from time to time, while always preserving the same character, lends probability to the assumption of its spasmodic origin.

"It may be added that A. Graefe[1] has cited an instance of a complete paralysis of the externus, in which the obliques

* A similar relation obtained in Cases 41, 43, 46, 49, 51, 52.

acting alone were able to produce a moderate amount of abduction of the eye when the latter was directed up and down, but not, of course, when directed horizontally. In a case of incomplete paralysis of the externus, in which the eye could be abducted somewhat while still in the primary position, this compensatory action of the obliques would come much more markedly into play, since the abducting power of the obliques increases with the degree of abduction of the eye. . . .

"The overaction of the obliques in the cases cited is probably *compensatory in character—i. e.*, the abducting action of the obliques is called into play to an excessive degree to replace, as well as may be, the defective abducting action of the externus."

To this argument it may be added that the torsion movements observed in some of the cases (Cases 2, 3, 10, 21, 25, 34, 43, 53) were such as to indicate spasm of the obliques. In one instance (Case 44) the character of the torsion indicated that the straight up-pull here was due to a spasm of the superior rectus.

This belief that the oblique movements are due to spasmodic action of the inferior or superior oblique, probably often combined with spasm of the superior or inferior rectus, has been confirmed in my mind by the observation of a number of cases, not only of the anomaly we are now considering, but also of cases of an entirely different sort in which the same oblique movement occurred and in which it was undoubtedly due to muscular spasm. Thus it would have been impossible to account, upon any hypothesis of anomalous insertion or pressure of an unyielding optic nerve, or indeed any mechanical explanation whatever, for the following case in which the *oblique movement in one eye was relieved by tenotomy of a muscle of the other.*

R. B., a boy of eight, whenever he looked to the right had always had marked upward deviation of the left eye. In the primary position, left hyperphoria of 6° to 10°, increasing when he looked up and to the right to something like 25°. When he looks to the right, the left eye shoots obliquely upward, particularly if the eye is elevated slightly above the horizontal plane.

In looking down and to the right, the right eye seems to go down excessively. Vertical diplopia in the whole right half of the field of fixation. In monocular vision, no special restriction of the movements of either eye in any direction.

Complete tenotomy of the right inferior rectus, producing some over-correction. Now, when he looks up and to the right, the left eye no longer flies up as before.

Nor would the hypothesis of a faulty insertion or of an unyielding optic nerve account for Cases 6, 32, 39, 51, in which the affected eye went up and in when it was directed slightly above the horizontal plane, and down and in when it was directed slightly below the line. In these cases there was evidently a spasmodic action of both the superior and the inferior oblique.

(*e*) No adequate explanation for the peculiar *closure of the palpebral fissure* when the affected eye is directed inward has been offered. It cannot be due to the enophthalmus or sinking of the eye, since it occurs in cases in which retraction is absent, or nearly so (see Cases 37, 49, 51), and on the other hand may be absent when the enophthalmus is extreme (see Case 41). It is probably, as Parker suggests,[9] an example of some peculiar associated movement produced by synergic action of the facial and third nerve.

(*f*) The *insufficiency of convergence*, which is sometimes present, sometimes absent, seems to be due simply to the mechanical hampering of the action of the affected internus. The patient, finding it difficult to make the affected eye move inward as fast as its fellow, gives up the attempt at converging and lets the faulty eye diverge. When, however, the eyes are so diverted as to impose little tension on the inefficient internus, the act of convergence may be performed in almost normal fashion. This would not be the case if the restriction in convergence was due to central causes, *i. e.*, was a true paresis.

Thus in Case 51, when the eyes were directed straight forward the convergence near-point was at 75*cm*; when the eyes were directed a little to the left (limitation of rotation outward of L eye) it was at 7*cm*. It might be argued that here there was no

true convergence-movement, but really a parallel movement of both eyes to the left, the two eyes converging more and more in doing this, simply because the outward movement of the L eye was arrested. But this view was negatived by the fact that, when the eyes were thus directed to the left and an object in line with the L eye was brought straight in toward the latter, not only did the R eye turn to the left to follow the object, but both pupils contracted, showing that the movement of the R eye was really one of convergence. Such a contraction did not take place when the R was following a distant object that was moved from right to left.

TREATMENT.

In view of the pathological condition which probably underlies most of these cases, advancement by increasing the tension, and hence the retraction, would do more harm than good. So it proved in Case 5.

In cases where there is marked inward squint, tenotomy of the internus may help (see Cases 29, 30).

In the opposite sort of case, where the eye tends rather to diverge and is hampered by an inextensible band replacing the externus, an operation for lengthening this band (after the fashion adopted by English operators) might be serviceable. In Case 36,* however, it did no permanent good; nor did tenotomy of the externus in Case 10. Otherwise the proper operation would be that performed in Case 51, viz., tenotomy of the externus of the other eye.

In general an operation is not required, and is to be avoided when possible.

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* The case here described is Case 46, but the discussion refers to Case 45.