I feel that some apology is due for venturing to comment upon cases which one has not oneself observed. Dr. Holmes's cases are, however, recorded with so much perspicuity and discrimination that one's confidence in the accuracy of his observations is at least as complete as if the opportunity of making them had occurred to oneself. They are, moreover, in the highest degree interesting and suggestive.

An analysis of the conditions described indicates that the function of vision in so far as central visual acuteness goes was, in some cases at least, unimpaired; that the reflex pupillary mechanism was not interfered with and that colour vision and stereoscopic vision were normal. In short, the lesions caused no defect in central (or foveal) vision. Even projection in the case of foveal images was apparently not essentially different from normal though the normal facility of bringing the retinal image of any object engaging attention on to the fovea was more or less conspicuously absent. When to this is added the fact that, so far as is stated, peripheral visual acuteness and peripheral colour vision did not show any abnormality, one may conclude that cone vision and cone reflexes were not disturbed by the lesions which caused the interesting complex of symptoms. Yet all these symptoms were indicative of very serious impairment of visual functions.

One question which suggests itself is: Can the symptoms be ascribed to the more or less complete damage done to cortical areas, directly associated with, but anatomically separated from, the visual centres? Without taking into consideration the most prominently characteristic class of symptoms, viz., what he so well summarises as "disturbances of orientation and localisation by sight, inability to estimate absolute and relative distances and failure to recognize relative lengths and sizes," there are observations recorded, the explanation of which may, no doubt, be found in cortical damage. For instance, visual retentiveness was found in one case to be very defective.

On the other hand, many of the facts elicited by the subjective examination of the cases appear to point less to central than to more
sub-cortical lesions, though both may have contributed in different degrees in the different cases to the functional impairments which they revealed.

Normal projection is characterised by the following: (1) A central image, on uniconal fixation, is projected along a line which passes from the object to the eye to meet and thus to form a continuation of the axis of vision. (2) On binocular fixation, the projected central images of the two eyes are superimposed, whether the two foveal images are those of the same or different objects and therefore whether the eye axes are parallel, convergent, or divergent. (3) This projection takes place along a line which meets, somewhere, usually at its midpoint, the line joining the centres of rotation of the two eyes. (4) A peripheral retinal image is projected in the direction of the line which passes from its object to the eye, i.e., it has an angular separation from the axis of vision equal to that of the change of direction which would be required to bring the fovea to bear upon the object. When the image of any eccentric object falls to the right or left of the retina of each eye or above or below, it is seen singly. The images are then said to occupy "identical" points on the two retinae. The identity is only strictly true for objects at a great distance or for objects lying on what is called the horopter. The theory of identical points need not, however, be considered here. It is of no practical importance.

The point of physiological importance is that for the accurate central fixation of an eccentric object both eyes have to move in response to an innervation which is always equally distributed over the two eyes, whether or not the change in the directions of their axes which this calls for, is of exactly the same angular amount. And the change in direction is effected with ease and rapidity and without giving rise to the appearance of movement of any objects in the field of fixation.

When an external object forms its image to the right of the fovea in one eye and to the left of the fovea of the other eye the objects appear double. Except where the images happen to be too eccentric the object can be fixed binocularly. This also takes place in response to an equally distributed innervation over both eyes and again also whether the angular change in the direction of fixation in each eye is the same, or different.

Now, while central projection, pathologically, actually, and physiologically (under certain conditions) apparently, can be faulty, or in other words fail to correspond to the laws of normal projection, peripheral projection, relatively to central projection, remains invariable. That is to say that whether or not the central projection is true, the projection of peripheral images maintains the same angular relationship to the foveal projection.

It is not difficult to show that the faulty projection which
characterises an oculo-motor paresis is entirely comparable to normal projection in that it is proportionate to the effort made to alter the line of fixation, and the same is true with respect to unocular physiological faulty projection (vide *Edin. Med. Ji.*, June, 1913).

Projection takes place therefore in strict accordance with the degree of innervation which is required to alter the lines of fixation. Any departure from the normal effort of innervation required for this affects projection. Normally, therefore, projection comes to correspond, accurately and consistently, with the position of the eyes; and this relationship is established by the normal degrees of innervation habitually called for to effect fixation, whether in like directed excursions or in convergence of the axes of vision.

If it were not for the existence, under pathological conditions, of faulty projection it would be difficult to recognize the factor of innervation as the basis of normal projection.

Another class of cases which show, not only the influence of innervation, but also that the normal associations between the line of projection and the axis of vision may be lost, is met with in long standing strabismus. Operation in case of convergent stabismus for instance, whether by tenotomy or advancement or both combined, occasionally gives rise to diplopia where none existed before operation. The diplopia, however, is often not in accordance with the relative positions of the two eyes. Thus, although some convergence may still remain after operation, the patient will be found to have crossed diplopia. His previous projection had, therefore, come to be one which corresponded to parallelism of the axes instead of to convergence, and consequently the operative diminution in the degree of convergence has had the same effect as if the axes were actually now divergent. The same phenomena are presented, *mutatis mutandis*, after advancement for divergent strabismus.

We must, therefore, recognize, in connection with the movements of the eyes to effect fixation of any object, a sense (unconscious, no doubt) of the effort required to do so. This I have always referred to as the sense of innervation (vide *Edin. Med. Ji.*, June, 1913).

But, although the visual impression resulting from the retinal image of any external object elicits consciousness of the presence of that object it apparently does not necessarily at the same time cause the consciousness of its position in space. This, I think, may be inferred from the phenomena exhibited by Dr. Holmes's cases. In them the sense of innervation seems to have been impaired or abrogated. Dr. Holmes seems to ascribe this to a "disturbance of visual attention." But even when attention was directed to eccentric objects his patients were unable to fix, or had great difficulty in fixing them, in addition to having little or no clear notion as to their positions in space.
It is no doubt the same sense of nerve effort required to pass from the fixation of one point in space to another that creates the consciousness of the shape and size of an object whose image does not lie wholly on the fovea. No doubt such a process as determining the midpoint of a line, or the centre of a circle, can be done with the maximum degree of accuracy by "running the eye" over the different parts of these figures. A very great and quite astonishing degree of accuracy can, however, be found to exist, without any change in the position of the eyes. The effort need in fact not be made, and yet there is a consciousness of what the effort would have to be, a sense of what changes of innervation would be called for.

The sense of innervation in the sphere of function of the oculo-motor muscles is in fact closely allied to the so-called muscular sense of the skeletal muscles. In some respects they are different, particularly in regard to the end-organs in each case which in the one are in the retina and in the other in the muscle itself. Intramuscular end-organs would indeed serve no useful purpose in the case of an oculo-motor muscle. In many respects they are similar. Not only are we conscious of the effort required to bring the muscles into the state of activity required for the performance of a definite action, but we can gauge accurately what would be the effort required for the same action without making it. Both for the innervation sense and the muscular sense, in this potential form, habit is no doubt what establishes their accuracy and perfection.

It is interesting, therefore, to note that, in some, at least, of the cases recorded there was apraxia. Further, the relatively smaller interference with habitual movements was well exemplified. For instance, movements to order were executed with greater difficulty than walking or conveying food on a fork to the mouth. The explanation of this is, no doubt, that the more firmly a habit, whether good or bad, is established, the more is the innervation called for capable of becoming independent of a higher control. The effect of habit is indeed very well seen in different eye movements (vide Edin. Med. Jl., November, 1912.)

In one case in which there was no apraxia the patient could move his eyes in convergence when his own finger was approximated to his eyes, but was unable to do so in order to fix any external object brought close to the eyes. Presumably this would also have been found possible if the patient's own finger had been passively approached to his eyes. This is not stated, but may probably be inferred from the observation that the passive jerking of his own hand towards his eyes caused blinking, which was otherwise not elicited. Something, therefore, in addition to the retinal impressions, whose position the patient became conscious of through
another sense than sight, was necessary to evoke the responses which would normally be got through ocular impressions alone. Convergence, however, on his own finger, though it occurred, did not do so constantly. But this does not detract from the significance of the phenomenon, as even under normal conditions it is not easy to converge on one's finger without seeing it.

Two other points which characterized the cases recorded call for consideration, viz: the absence of blinking and the want of appreciation of the movement of objects lying outside the line of vision. They are perhaps more difficult to explain. Obviously we have to deal, as regards the first, with the abolition of a reflex. Reflex blinking was observed to be absent in all the cases except one, with regard to which no reference is made to this point. Of this one particular case, moreover, it is stated that "the disturbances of visual orientation were slighter than in the others, and they diminished considerably while he was under observation." It is possible, therefore, that the loss of this reflex may not have occurred in this case. Yet it is unfortunate that the record of the case does not clear this up. One would expect, in view of the similarity exhibited otherwise in the symptoms, and because of the absence of reflex blinking, in the other case which is stated to have improved while under observation, that reflex blinking must have been absent or impaired in this case as well.

The reflex mechanism which leads to blinking is therefore found to be defective, some part of its entity being interfered with by the same lesions which cause the defects in orientation and localization. That is to say that the peripheral retinal impressions which, though perceived, do not admit of being transferred by appropriate eye movements to the foveae, are also prevented in some way from eliciting the involuntary protecting closure of the lids.

This suggests that the fixation movements of the eyes are, to some extent at least, also reflex in their character. Certain it is that while there is no difficulty in moving the eyes in similarly directed association without the stimulus of different objects on which to direct them, convergent movements can only readily take place when they are induced by the stimuli of hetero-lateral retinal impressions. Thus we find that in some of Dr. Holmes's cases it is stated that all the movements of the eyes were normal except convergence.

With the evidence indicative of an intact connection of the peripheral cones, viz.: unimpaired vision and colour vision and pupillary reflex, it seems not an unreasonable assumption to make that the end-organs for the orientation and localization reflex are the retinal rods. There is good reason for believing that the rods are not colour-sense end-organs, and some reason for assuming that they play only a subordinate part, if any, in connection with the
form-sense. On the other hand, the normally acute sense of movement at the periphery of the field of vision, when compared with that of the peripheral form-sense, is suggestive of the rods being end-organs for movement. And the sense of movement is evidently closely allied to that of orientation and localization. It is interesting to note in this connection that, in three of the cases, an inability to recognize the movements of peripherally placed objects was observed.

In regard to another function with which there is sufficiently good reason to associate the rods, viz: retinal adaptation, there is no reference in Dr. Holmes's paper from which any conclusion as to the integrity of this function could be arrived at. It is unfortunate that no examination of the light-sense seems to have been made, although the observations are otherwise so complete and so discriminating.

I have long held the view, arrived at from purely speculative considerations, that the rods might possibly be orientation end-organs (vide Ophthalm. Review, Vol. IX, 1890, pp. 136-139). I have consequently been on the look-out for any evidence pointing in this direction. The symptoms which Dr. Holmes has elicited in the cases described in his paper appear to do so. In any case they very clearly demonstrate that lesions causing hemianopia, the site of which is well known, do not cause orientation defects. A particularly beautiful illustration of this occurred in his third case, in which there was a temporary left hemianopia and in which "when vision to the left of the fixation point had recovered, it was observed that he could localize the position of objects in space better to this side, which had been blind, than to his right."

Notwithstanding the fact that in most of the cases the difficulty of making convergent movements in the interest of fixation is referred to, there is no note of diplopia having been complained of. Presumably it was not a symptom that spontaneously obtruded itself on the attention of the patients. It is not stated if any attempts were made to elicit it.

It does not seem improbable that the greater or less impairment of the sense of orientation would at the same time cause an absence of the symptom of diplopia. This point should, however, receive consideration in other similar cases which may be observed now that attention has been called to traumatic orientation defects.

Another point which may be briefly referred to is the question as to the presumable infrequency of orientation defects.

Although in my service at the Second Scottish General Hospital I have seen many cases of hemianopia from war wounds in which all degrees and complexities have been represented, I have never observed any which were complicated by even a trace of the symptoms indicative of orientation defects. I conclude from this
that they cannot be a frequent accompaniment of traumatic hemianopia.

On the other hand, these symptoms may quite well have been missed in other cases which have caused impairments other than distinctively visual ones, and such cases do not to any extent come within the purview of the ophthalmic surgeon. Orientation defects may quite well be less infrequent than the absence of recorded cases hitherto would suggest. That remains to be seen. But granting that they are infrequent, and this I think is most likely, it is possible that a cause for this may be found in the orientation centres and their direct connections, which must surely be represented on both sides of the brain, being, unlike the visual centres, intimately interrelated, so that one alone may suffice to carry on the function when the other has been thrown out of gear. This possibility, which was suggested to me by a distinguished neurologist, is supported by the fact that there was not shown to be any markedly one-sided character in the orientation difficulties in any of Dr. Holmes's cases, unless the observation to which I have already referred, where orientation appeared to be less imperfect in the area of a recovered hemianopia than in the opposite half of the field of vision, can be taken as indicating that there may be exceptions to this rule. In any case, this is another point which future observations may clear up.

I make no attempt at suggesting a localization of the lesion which leads to the particularly interesting functional interferences discovered by Dr. Holmes. I do not even offer any explanation as to whether the symptoms point to a cortical or a sub-cortical interference with the orientation reflex, or to both. In some cases one would suppose that the cortex was not involved or was not the main area whose function was disturbed, but I do not feel that my knowledge of cerebral anatomy is sufficiently intimate to analyse this aspect of the matter. My main object has been to attempt to trace a connection between localization and cognate disorders, and reflexes which it is suggested have their normal starting point from the retinal rods.

In the October number of this journal, Dr. Holmes concludes his paper with a fuller analysis of the cases and a discussion of the position of the lesions which may be held to cause orientation defects.

The above comments on the cases themselves were made without further knowledge of Dr. Holmes's views than could be gathered from the first portion of his article, and were in the hands of the Editor before the concluding portion appeared. A few explanatory supplementary remarks are suggested by the views expressed by Dr. Holmes.
The only material way in which his views seem to me to differ from my own, lies in his apparent adherence, to some extent, to the old “nativistic” conceptions regarding certain ocular phenomena. He says: “We know that as a result of certain cerebral lesions the locality of a tactile stimulus can no longer be recognized, though there may be no affection of either the threshold of sensibility or the quality of the sensations evoked; the local signs of the cutaneous surface then no longer furnish the necessary information to consciousness. Similarly, the cerebral wounds in these cases affected the local sign functions of the retinae, and the patients consequently became unable to project correctly, and arrange in their proper relations in space, the images which excited vision, and to recognize the actual spatial relations of objects seen and their respective sizes and lengths.” Unless what he here calls “the local sign” arouses consciousness in dissimilar ways in the two cases, the analogy is complete. It appears, therefore, somewhat enigmatical that Dr. Holmes should seem to infer that a radical distinction is called for to explain, on the one hand, disturbances which take place in bidimensional space, and, on the other, in space of three dimensions. He says: “In discussing these errors we can conveniently, on physiological grounds, deal separately with those made in the coronal plane, and those due to disturbance of the perception of distance.” And the reason for doing so appears in a subsequent sentence: “But retinal stimuli alone suggest only the magnitudes and the relative positions of objects in a bidimensional plane. To obtain a correct knowledge of the locality of the objects in relation to ourselves, afferent impressions from the ocular and neck muscles and tendons, which can inform us of the direction of the visual axes and the position of our heads, are also necessary.” Surely both are equally referable to a stimulus conveyed to (presumably) the angular gyrus, and started by a retinal image. A similar distinction is also drawn between inability to appreciate the absolute and the relative positions of objects. It is difficult to see why the one should not necessarily entail the other.

When Dr. Holmes talks about impulses from the oculo-motor muscles, as being provided by extra-retinal afferents, he obviously has in view something in connection with the muscles which he supposes exists apart from the retinal stimuli. But it has to be remembered that it is impossible with closed lids to tell for certain what is the position of the eyes. No doubt it is possible, without fixing any object, to rotate the eyes in the same direction, but apart from the consciousness of making this voluntary movement, there is no consciousness of the exact position of the eyes. Convergent movements, if possible at all under similar conditions, evoke no consciousness of convergence.

As the matter appears to me, the afferent path in all cases starts
from some retinal end-organ, and the impulse which thus results in, let us say, the angular gyrus, produces in some way a consciousness of the position of the object whose image falls on a particular part of the retina. A lesion occurring at any part of this afferent tract, or involving the angular gyrus itself, must therefore impair orientation. When the heteronymous images on the two retinas, which normally induce either an active or a potential convergence, fail to do so, one of the chief factors which contribute to the sense of distance, in the case at least of objects which are not far removed from the eye, is lost. If the lesion does not happen to be central, but only involves some sub-cortical portion of the afferent tract, it must still remain possible to converge on an object, whose distance is more or less correctly appreciated otherwise than through its retinal image, e.g., the patient's own finger. In this connection the following observation by Dr. Holmes is interesting: "Though Case 4 accommodated for his own finger accurately, and Case 2 could do so occasionally though not constantly, the other cases in which the test was made failed even when, as under such conditions, they were aware of the nearness of the object."

Dr. Holmes shows some tendency to mix up convergence and accommodation. It is certainly only convergence or the sense of convergence innervation which has any bearing upon distance estimation. It would be extremely disturbing if a hypermetrope or a myope were to judge distance differently when looking at objects with and without his optical correction. On the other hand, prisms which call for greater or less convergence than corresponds to the distance of an object looked at, alter the apparent size, and, more or less consciously, the apparent distance of the object.

I am the less able to understand how Dr. Holmes arrives at the views on which I have commented, as he very clearly states, in referring to the angular gyrus: "It may be consequently assumed that one function of this part of the brain is the reflex adjustments of the eyes to peripheral stimuli, or the co-ordination and integration of the afferent impressions which, through these efferent centres, evoke appropriate ocular movements in response to retinal excitation."

I have some difficulty also in accepting Dr. Holmes's view that the behaviour of his patients indicated "a local disturbance of visual attention." There may, no doubt, have been evidence of some general slackness of attention. But I see no reason to ascribe the symptoms which indicated a local disturbance of attention to anything else but the orientation difficulties. These difficulties might surely explain why: "attention lacked its normal spontaneity and facility in directing itself to new objects." Even normally, peripheral objects are to a great extent unnoticed unless attention is directed to them, when, so far as their localization goes, that is
always perfect, even if no direct fixation of them is made. That this is so is evident from it being possible rapidly to alter the line of fixation so as to bear accurately upon them without any preliminary searching movement. I do not suggest that “visual inattention” is to be regarded as never having any pathological significance, merely that this is an unnecessary assumption where orientation defects exist. Dr. Holmes states in fact that: “visual inattention often occurs as a unilateral phenomenon as a result of parietal and lateral occipital injuries when spatial orientation is not affected.” If, as Dr. Holmes infers, visual inattention is to be accorded a place in the symptom-complex of orientation disturbance, this fact may be looked upon as suggestive of the possible close interdependence of the orientation centres on the two sides of the brain to which I have referred above.

With most of the other valuable comments which Dr. Holmes makes in his analysis of the symptoms presented by his cases, I find my own views in more or less complete accordance. One of his points deserves, I think, more prominence than he gives it. He explains the fact that reading was generally less difficult than might have been expected from the inability that there was to count objects in the field of vision, by noting that: “the appropriate movements of the eyes from left to right, which had been acquired by long training and fixed by habit, were less affected than their voluntary deviations to objects that were at the moment outside central vision.” There can be little doubt that in acquired orientation defects, “habit” must have a modifying effect on the functional impairment as it has in other disturbances implicating the oculo-motor muscles. Its effect is noticeable, e.g., in the increase in the area of diplopia on downward fixation in abducens paresis. I have pointed out too (vide Trans. Ophth. Soc., 1901) that in the peculiar, not altogether rare, condition of convergence spasm, the circumstance that the convergence, though always in excess of binocular fixation, is diminished to either side of the field of fixation, is due to the persistence of the habit established by the necessity, when reading, of maintaining fixation of words on a plane near to and parallel with the eyes. When this subtle variation of convergent effort can assert itself under conditions which interfere with reading, it is not difficult to understand that running the eyes along a printed line should be possible even when localization and orientation are seriously impaired.

The concluding paragraph of Dr. Holmes’s communication is as follows: “The angular gyri contain centres from which the movements of the eyes can be elicited, and it may be to damage of these portions of the cortex that the disturbance of ocular fixation, the failure to accommodate near objects, and the absence of the blinking reflex were due, in so far as they were not a direct result of the
defective recognition of the position of the object in space, and especially of its distance.” The cases show, I think, that the statement would be more complete if after “damage of these portions of the cortex” there were added, “or of their afferent fibre connections from the retinal end-organs.”

The last clause of the paragraph I have quoted seems to indicate that Dr. Holmes assumes there is some other independent cortical centre which has to do with the recognition of the positions of objects in space. This may be so; yet if the potential response of the eye-movements centre is interfered with, either by lesion of the centre itself or of the afferent path associated with that centre, this would surely be enough to account for the symptoms. In cases of oculo-motor paralysis, although projection may be faulty, there is no change in the relative localization of external objects. This is evidently because the voluntary oculo-motor centre with its afferent tract are not involved in the lesion causing the paralysis. The reflex and voluntary innervations are normal, but the response to efferent stimuli is defective. Consequently, projection is no longer in accordance with the position of the eye, as from established habit it normally is.

JOTTINGS ON LOCAL ANAESTHESIA
IN OPHTHALMIC SURGERY*

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I was suddenly summoned by telephone by a Suffolk doctor to go over and see a patient with exophthalmic goitre, whose left eye had become seriously affected; if I found it necessary to remove it, what preparation for the anaesthetic should I like? I replied I would come and remove the eye if necessary and that no preparation for an anaesthetic was necessary. I went; a glance was sufficient to condemn the globe, and with perfect ease and freedom from pain I proceeded to remove it under local anaesthesia. The doctor concerned, an able man, told me he had thought it necessary to remove the eye during my absence and was prepared to do it; in fact, he had called in a colleague to give a general anaesthetic for the purpose, but the colleague was so concerned at the patient’s condition that he declined to administer any such, and she was left for my return.

A few weeks later I saw for a colleague a woman with a good-