Abstract:

**Topic and purpose:** This article recounts the evidence of Newtonian formulation regarding the laterality of visual space in humans.

**Method:** Based on clinical as well as time-resolved experimental data and a new perspective afforded by the discovery of directionality in callosal traffic, I present evidence that the splitting of nasal retinal fibers at chiasm, suggested by Sir Isaac Newton in 1704 to explain singular vision with two eyes, does not take place.

**Results:** Time resolved and clinical data are reviewed indicating the incompatibility of Newton’s formulation of visual representation of hemispace with the data at the following levels; retina, chiasm, lateral geniculate ganglion and temporal and occipital lobes.

It is **concluded** that we see with our major hemispheres as defined by the new circuitry (left, in vast majority of behavioral right handers). Thus, the major hemisphere is the site in which all commands are formulated and in which visual experience takes place.
By most accounts, it was in 1704 when Sir Isaac Newton published his views as to the formation of a single image from the ones held by the two eyes. He conceived of visual space as two halves meeting at the vertical meridian. Each hemispace was assigned to the retina and hemisphere of the opposite laterality (right or left). This arrangement logically demanded the splitting (decussation, at the chiasm) of the fibers belonging to the nasal half of each retina.

After three hundred years and accumulation of critical relevant data, it is time to ask whether Newton was right in the logical (intuitive) answer he then supplied. The oldest observation contradicting Newton’s formulation is the so-called macular sparing (preservation of central vision) after demolition or removal of nondominant hemisphere, as defined below. This phenomenon has long been awaiting an answer not forthcoming from Newton’s foveal splitting theory. The anatomy subserving this remarkable asymmetry, i.e. the fact that the traffic of motor and somatosensory signals between the two hemispheres is 1-way (and in opposite directions) and that macular vision bypasses the callosum, has been discovered recently. Thus, the asymmetry mentioned above reflects the laterality of the controlling hemisphere containing both the command center as well as the site where seeing occurs. The latter is called the major hemisphere which controls the minor hemisphere via the corpus callosum. This relationship is codified in handedness. Elsewhere, I have documented the facts supporting the idea that macular fibers are destined to the major hemisphere as defined above.

Another critical observation inconsistent with Newton’s view of macular representation is the return to normalcy of visual field defect seen in lesions affecting the minor hemisphere if the nondominant hand is used to test the patient who displays such phenomenon (termed spatial neglect). This latter observation is contingent upon the survival of anterior callosum. The third observation is the fact that lesions (tumors) pressing on the chiasm (where the supposed crossing of the fibers from nasal retina occurs) rarely produce what is expected of such an event, i.e. inability to seeing things located at the sides, commonly depicted in textbooks as bitemporal hemianopias. The incidence of bitemporal hemianopsia in the largest series published has ranged from nil to 32 %, raising doubts about Newton’s proposed anatomy, i.e. the splitting of fibers arising from nasal retinas. On the other hand, it is in the chiasmal “neighborhood” syndrome that findings incompatible with Newton’s proposal abound (i.e. in the Traquair’s junctional defects) “raising many unanswered questions”. These incongruous bilateral field defects have often been explained away by resorting to a mysterious item, called the Wilbrand’s knee. No data exist regarding the handedness of patients in whom these observations were made.

Similar incongruities of visual field defects are also seen after ablative surgery of anterior temporal lobes. Here, overstepping or violation of vertical meridians occurred in six out of 18 in one and five out of 22 in another series of temporal lobectomies for surgical treatment of seizures, in addition to occurrences of hemianopias in the same series. Clearly, such occurrences would be unexpected if there were a splitting of the nasal fibers of both eyes prior to their arrival at the lateral geniculate body. In the same vein, Bender’s finding of central visual defect in patients with optic tract lesion is also contradictory to Newton assumption, as is the maintenance of singular visions in those circumstances in which “chiasmal crossing” fails to materialize.
At the cortical level, awareness that something is awry if a lesion occur is keyed to the dominance status of the affected eye. That macular lesions affecting the dominant eye are far more likely to intrude with vision (be symptomatic) than those affecting the nondominant eye. This was documented recently by, Waheed and Laidlaw who studied such visual handicaps in 44 patients with unilateral full thickness macular holes \( (p=0.001) \). In the latter study, seventy eight percent \( (18/23) \) of those who elected to undergo surgery had a hole in their historically dominant eye \( (p = 0.0003) \). Last but not least, seizures induced by photic stimulation are aborted by occlusion of one eye (usually the right, see below). 23, 24

According to 1-Way callosal traffic scheme, after winding its way through the chiasm macular fibers from both eyes reach the major hemisphere. 3 The belief in binocular vision seems to be based on the fact the vision remain uninterrupted when viewing the world one eye at a time. However, this observation must be reconciled with a constant asymmetry (laterality) of the viewing eye when looking at a particular object of regard; i.e. the blocking of the object (viewed ostensibly with both eyes) when the path of the actual viewing eye is obstructed. 25 The result of this version of “hole in the card” or “circle” test indicates that under such circumstances (focusing) viewing occurs monocularly (by the dominant eye). Time-resolved proof of this claim comes from the fact that the reaction time obtained in circumstances inducing binocular rivalry remains the same regardless of the laterality of the alternating image initiating the response. 26 Thus, when we focus we actually are seeing with one eye (the dominant). This interpretation was confirmed in patients with macular holes, who became symptomatic (complained of their vision) after the involvement of the dominant eye. 21 In another study, nine of the twelve patients who underwent “prophylactic laser treatment to fellow eyes of unilateral retinal pigment epithelial tears” had the procedure performed on the left eye, corroborating the asymmetry of the “viewing” eye in “binocular vision.” 27 Although the idea that “binocular vision” may in fact be monocular has been discussed before, 28 the proposed anatomy to solve disparate observations of the past is entirely new.

Neither the Newton’s view fare better in other time-resolved studies relevant to the subject. 3, 5 According to a recent comprehensive review of Poffenberger paradigm, the interhemispheric transfer time (IHTT) from lateralized visual stimulation tests are so small (less than 2 ms) as to be “beyond the capabilities of even the largest callosal fibers” available for such transfers. 29 This is not a small matter as the range of axonal diameter available for such transmissions determine the processing time. 30 However, Fendrich et al did note that “the difference between crossed and uncrossed reaction times was greatest when subjects were responding with their left hand. There was no difference between crossed and uncrossed responses when subjects were responding with their right hand”. Thus, the new understandings help elucidate the time-resolved asymmetry mentioned above as well as that related to vision (Waheed and Laidlaw). 3-5, 6

A question, therefore, arises as to the function of the “visual cortex” in the minor hemisphere. According to the above described circuitry, the sensory function of the nondominant hemisphere is to collect and transfer to the major hemisphere (via the corpus callosum, for conscious apprehension) data arising from the left hemi-body.31-34
In the motor realm, the minor hemisphere carries out the commands arriving from the major, wherein all movements are planned and executed. Thus, moving the nondominant side and sensing from it become bi-hemispheric events. Directionality in callosal traffic in the motor realm (from the major to minor hemisphere) and the abeyance of the excitatory connection (diaschisis, separation shock) consequent to severing of the synapses involved has other visual consequences. These are reflected in the position of the eyes (gaze). Conjugate eye deviation (CED) toward the damaged right hemisphere is the best known of such short lived syndromes. A cortical one-and-a-half syndrome has also been described. In this presentation, the nondominant eye remains motionless while the other eye shows exotropia. This laterality indexed lone abducting eye syndrome (LAE) must be distinguished from the better known CED for what it represents. According to the anatomy under review, both of these laterality indexed syndromes are due to diaschitic paralysis of the left brainstem in lesions affecting the right hemisphere (in right handers). The difference between the two (CED and LAE) is in the larger size of the lesion in LAE, affecting the entire territory of excitatory chain of command which begins in the left hemisphere. Thus, there is a wider diaschitic suppression of the left brainstem in LAE including the left midbrain and medial longitudinal bundle. The latter originates from the right before crossing to the left to yoke the left eye to the right. Selective involvement of a nuclear subgroup has been reported in similar circumstances involving a portion of the facial nucleus on the left and resulting in impaired volitional closure of the left eye. Hence, because of the multiple chains of commands, lesions affecting the dominant hemisphere has bi-hemispheric consequences and do not result in laterality indexed presentations similar to those involving the nondominant hemisphere. 

As revealed in his diagram of 1914, Hugo Liepmann was cognizant of directionality in callosal traffic, explaining the genesis of apraxia ipsilateral to the major hemisphere. However, it took the advent of time-resolved techniques, PET and fMRI to prove the validity of his proposal; i.e. that commands are initiated in the major hemisphere and those related to the left side are in turn transferred to the minor hemisphere for implementation by effectors on the left. Thus, noninvasive measurement of the reaction time of both hands (or the latency of saccades to both sides) is all that is required for ascertaining a person’s laterality of the major hemisphere (motor control). The dominant side of the body is that which is closer to the major hemisphere by an amount commensurate to the IHTT.

Conclusion:
The evidence derived from lesions affecting multiple levels of visual apparatus strongly indicates that Newton’s formulation of the visual sense of space based on chiasmal crossing of fibers from nasal retina is untenable and should be abandoned. Bitemporal hemianopia, the hallmark of optic chiasmal syndrome, falls in the minority of presentations even in traumatic chiasmal lesions with vast majority of patients maintaining a useful visual acuity/central vision in at least one eye. Evidence denotes that macular fibers of both eyes wind their way to the major hemisphere where vision is apprehended. The laterality of vision evidenced above contains an evolutionary advantage by decreasing the reaction time of a person amounting to one callosal transfer time.
References:

4. Hund-Georgiadis M, Zysset S, Weih K, Guthke T, von Cramon DY. Crossed nonaphasia in a dextral with left hemispheric lesions: a functional magnetic resonance imaging study of mirrored brain organization. Stroke. 2001; 32:2703-2707. (Fig.1 and table)
http://stroke.ahajournals.org/cgi/content/abstract/32/11/2703
30. Salmelin R, Forss N, Knuutila J, Hari R. Bilateral activation of the human somatomotor cortex by distal hand movements. Electroencephalogr Clin Neurophysiol. 1995; 95:444-452. (Fig.7 & page 451)
41. Hassan A, Crompton JL, Sandhu A. Traumatic chiasmal syndrome: a series of 19 patients. Clin Experiment Ophthalmol. 2002; 30:273-380. (Table 2, Fig.4)