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Review

Vision therapy: Occlusion, prisms, filters, and vestibular exercises for mild traumatic brain injury

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ABSTRACT

A number of treatment approaches have been advocated for persistent visual complaints following mild traumatic brain injury. These include devices such as binasal occlusion, yoked prisms, vertical prisms, and filters, as well as vestibular training. We discuss the rationale and the evidence for each of these approaches. Binasal occlusion has been advocated for visual motion sensitivity, but it is not clear why this should help, and there is no good evidence for its symptomatic efficacy. Base-in prisms can help manage convergence insufficiency, but there are few data on their efficacy. Midline shift is an unproven concept, and while the yoked prisms advocated for its treatment may have some effect on egocentric neglect, their use in mild traumatic brain injury is more questionable. A wide variety of posttraumatic symptoms have been attributed to vertical heterophoria, but this is an unproven concept and there are no controlled data on the use of vertical prisms for mild traumatic brain injury symptoms. Filters could plausibly ameliorate light intolerance but studies are lacking. Better evidence is emerging for the effects of vestibular therapy, with a few randomized controlled trials that included blinded assessments and appropriate statistical analyses. Without more substantial evidence, the use of many of these techniques cannot be recommended and should be regarded as unproven and in some cases implausible.

Traumatic brain injury is a common problem across the age spectrum.[25] While most postconcussion symptoms resolve within 3–12 months after the injury, a minority of patients has persistent symptoms,[16] which can include a variety of painful, sensory, cognitive, and emotional problems.[6] There is increasing awareness that this long list of potential complaints includes visual symptoms such as light sensitivity, blurry vision, double vision, and trouble reading.[4,31,54,58] Reflecting

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this diversity of symptoms, patients with mild traumatic brain injury (mTBI) are commonly treated with a variety of modalities, to which vision therapy is being added more frequently.[75]

Vision therapy has been defined as "a sequence of neurosensory and neuromuscular activities to develop, rehabilitate, and enhance visual skills and processing".[3] These activities include ocular motor training, which involves a series of sessions of eye movement exercises,[78] as well as the use of devices such as lenses, prisms, occluders, and filters.[3] Also, vestibular therapy is a related approach that uses eye as well as balance exercises, but with a different motivation, being directed at promoting adaptation or habituation in the vestibular system. We have discussed the rationale and evidence behind ocular motor training for mTBI patients in a recent review.[7] Here, we survey the use of devices and vestibular habituation in patients with persistent postconcussive symptoms.

1. Binasal occlusion

Binasal occlusion refers to the placing of opaque tape on the inner third of lenses (Fig. 1) and began as a therapy for esotropic strabismus. A number of anecdotal case reports later claimed that binasal occlusion also improved visually provoked dizziness, balance problems, sensitivity to busy environments, and eye pain induced by eye movements in patients with mTBI.[28,29,56]

The first cohort study assessed 10 hospitalized patients with ill-defined traumatic brain injury and 10 healthy controls.[50] Compared with the controls, the patients at baseline complained of illusory motion and had reduced acuity, poor convergence, accommodation, and pursuit, and exophoria at far and near. Base-in prisms with binasal occlusion increased the amplitude of the P1 component of binocular visual evoked potentials (VEPs), though effects on latency were not mentioned. There was no report about the effects of binasal occlusion on symptoms or signs.

Another group reported 2 studies. The first involved 10 subjects who met criteria for mTBI, 1–10 years after injury, and who also had visual motion sensitivity.[18] It compared them to 10 healthy subjects. The study examined the effect of binasal occlusion without prisms on the binocular full-field (central 15°) VEP. They replicated the finding that it increased VEP amplitude in those with mTBI, with no effect on latency, which was normal at baseline. Subjects replied to an informal query that they had reduced nausea and disorientation and a sense of better fixation, walking, and grasping.

Fig. 1 – Example of binasal occlusion using frosted tape.

The second study examined 15 subjects who met criteria for mTBI occurring a mean of 7 years before the investigation (range 1 to 27 years) and who also had visual motion sensitivity.[84] They were compared with 20 healthy subjects. Treatment was binasal occlusion with or without small (2-diopter) base-in prisms. The main finding was an increase in the VEP amplitude, which contrasted with a decrease in healthy subjects, but no change in latency. Subjective impressions suggested less illusory motion when viewing a checkerboard, "more comfortable" walking, and better grasp in the 5 subjects who complained of impaired depth perception. However, without a randomized, blinded placebo-controlled design, it is impossible to have confidence in these impressions. Without a control group of mTBI patients who did not have visual motion sensitivity, we also do not know if the results are related to that specific symptom.

Why might binasal occlusion work? Some of the proffered explanations[17] are directed more at explaining its effect on VEP amplitudes than its effect on symptoms. These explanations include a) reduced activity in the "ambient system",[50] b) reduced need of patients with visual motion sensitivity to suppress peripheral visual motion, which leads in turn to increased responsivity of the central field,[18] and c) reduced distraction by peripheral motion, which enhances attention to the central field.[17]

To explain symptomatic benefit, some suggest that binasal occlusion may simply provide a stable frame of reference in the visual field.[28] If so, monocular nasal occlusion should also work, or perhaps just having a visible line on the lens. These have not been tried. Along these lines, the increases in VEP amplitude were not replicated in 2 subjects using just trial frames, which could provide such a nonocclusive visual reference, but the effect on symptoms was not described.[18] A second suggestion is that visual motion sensitivity reflects inadequate filtering of motion in the peripheral field, and that binasal occlusion reduces peripheral motion information.[17] Alternatively, reducing peripheral motion may help if visual motion sensitivity results from abnormal motion processing, either in the magnocellular pathway or in cortical regions responsible for distinguishing object motion from self-motion, such as area V6a.[17]

Is there any support for these anatomic speculations? Selective lesions of the magnocellular pathway are hard to come by, but there are proposals that this pathway is affected in early glaucoma.[85] This may account for impaired motion perception in this setting,[70] yet patients with glaucoma do not complain of motion sensitivity. Parietal lesions can also cause a variety of motion perception deficits,[10,82] yet dizziness induced by visual motion is not a reported symptom. It has to be acknowledged, however, that some studies of visual vertigo excluded subjects with clinical or imaging evidence of lesions,[33] and an older report of visual vertigo included a few subjects with cerebellar or brainstem lesions, though not cortical ones.[14]

It should be stressed that binasal occlusion is not the same as binasal field loss. Because the occlusion is fixed to the glasses but the eyes are free to move, the device only occludes the binasal fields when the eyes are looking straight ahead. In side gaze, the effect is closer to monocular occlusion of the adducting eye with relatively full field in the abducting eye. Nevertheless, this may still be relevant if the effects of binasal occlusion stem somehow from reduced inputs to binocular cortical cells.[50] Of course, if this were the explanation of its benefit then monocular occlusion should also work, but this has not been shown.

Visual motion sensitivity typically refers to visually-induced symptoms of dizziness and imbalance. It is not clear that this is due to abnormal motion processing in the visual periphery. For one, subjects with visually-induced dizziness also complain of this with static patterns. For another, their symptoms can be provoked by motion confined to the central field, as with scrolling on smart phones or computer screens. Such attribution is also at odds with current concepts of visually induced dizziness. These propose that a triggering event such as a vestibular insult leads to activation of high-risk postural control strategies and a shift to greater reliance on visual cues, which is problematic when visual data are ambiguous about self versus environmental motion.[15,33,55]

The work on binasal occlusion is hampered by its focus on binocular VEP amplitudes. First, it is not clear how VEP amplitude is related to the symptoms experienced by patients. Second, it is not known what changes in amplitude mean, in either the mTBI patient at baseline or their response to interventions. Some have argued that reduced VEP amplitudes in mTBI reflect dysfunction in the anterior visual pathways.[27] This would be more convincing if interocular asymmetries in monocular VEPs were reported or if there were corroborating evidence, from optical coherence tomography for example. Cerebral damage to the geniculostriate pathway could also degrade VEPs but should lead to hemifield defects. It should be noted that changes in VEP amplitude could reflect other cognitive processes besides vision, being modulated by arousal and attention, for example.[23,47]

2. Prisms and occluders for convergence insufficiency

Convergence insufficiency is relatively common after mTBI. A meta-analysis concluded that it occurs in 37% of such patients.[44] However, its diagnosis requires some caution. Reliance on questionnaires such as the Convergence Insufficiency Symptom Survey[61] may substantially overestimate its prevalence compared with objective measures of binocular alignment.[72,80] Convergence insufficiency is a plausible cause of blur or binocular horizontal diplopia when viewing near but not far objects, but is also blamed for more nebulous symptoms provoked by near work, such as headaches, eye strain, and poor concentration and understanding when reading.[19,21]

As with any form of binocular misalignment, the visual effects of the misalignment can be reduced by occluding one eye or using prisms to compensate for the misalignment. There has been surprisingly little study of the use of base-in prisms for convergence insufficiency.[66] One study gave 29 adults glasses with and without prisms in a randomized order and found a greater reduction in scores on the Convergence Insufficiency Symptom Survey with 3 weeks of prism use.[77] A follow-up report of the same data noted that the scores on the survey did not predict benefit, but objective measures of ocular alignment did.[53] A second study randomly assigned 72 children to use prisms or placebo reading glasses and found similar reductions in the Convergence Insufficiency Symptom Survey scores in both groups.[65] Neither of these studies were directed at patients with traumatic brain injury, though.

Both occlusion and prisms can be viewed as forms of "strategic compensation" for convergence insufficiency. They do not fix the underlying problem but allow one to achieve the same visual goal (i.e., single vision at near) by another means. An alternate "remedial" approach is to try to improve convergence insufficiency through exercises. These have been reviewed elsewhere.[7,66] Essentially, while there is evidence that 12 weeks of office-based exercises can improve idiopathic convergence insufficiency in children[20] and young adults,[67] there is as yet no convincing evidence for similar benefit with the posttraumatic variant.

3. Yoked prisms for midline shift

Midline shift syndrome is a deviation of the location where a subject believes their midline to be. It is said to create postural changes during gait and believed to reflect a mismatch between visual and proprioceptive information about the body's center of mass.[52] Although this explanation can be challenged, as a phenomenon such a shift is reminiscent of egocentric hemineglect from right parietal lesions, which is associated with an ipsilateral deviation of the estimate of "straight ahead" in the dark.[39] Also, subjects will past-point toward the side of a unilateral vestibular lesion when their eyes are closed.[12] It is seldom stated how or why a similar spatial deviation should arise in mTBI. As these subjects by definition do not have gross cerebral changes on brain imaging, perhaps the most reasonable speculation at present is vestibular dysfunction. The latter is not uncommon in mTBI,[1] though it likely affects otolithic more than semicircular canal signals.[41] Posttraumatic otolith damage is frequently bilateral, but can be unilateral,[9] and certain types of impact may generate asymmetric vestibular effects.[74] Whether the presence and direction of such vestibular asymmetries correlate with midline shift syndrome has yet to be investigated.

Yoked prisms are prisms placed in both lenses that deviate the image by the same amount in the same direction (e.g. base-in in one eye, base-out in the other). These can generate either perceptual or postural effects while the prisms are worn or after-effects following removal of the prisms. Horizontal yoked prisms have been used to reduce egocentric left hemineglect, where they improve manual straight-ahead pointing and postural imbalances.[48,59,71,79] Horizontal or vertical yoked prisms have also been used to treat abnormalities of body posture, but not without controversy. Careful studies of postural after-effects with short-term use in healthy subjects have produced mixed results.[45,76]

Yoked prisms have also been used to try to counteract midline shift, mainly through effects evident while the prisms

are worn, rather than analyzing for after-effects when they are removed. In a first study, 30 patients with strokes were compared with 30 healthy controls.[51] The locations of the strokes were not described beyond their lateralization nor were their clinical signs, such as hemiparesis, hemifield defects, or hemineglect. Over half of the stroke patients were said to have midline shift, though this is not actually clear in the results. Wearing yoked prisms reduced this shift and moved weight bearing toward the hemiparetic side (whether the latter is a good thing can be debated) in about 80% of trials, whereas wearing prisms in the reverse direction had a similar effect in 25% of trials. It is not stated whether subjects or examiners were blinded as to which prism was being worn on a given trial.

A second study reported on 36 subjects with imbalance and "possible midline shift syndrome", without specific criteria.[52] Ten had had a concussion and the rest a variety of lesions, including Chiari malformation, Lyme disease, multiple sclerosis, stroke, brain tumor, and essential tremor. They had their gait analyzed for sway on a pressure-sensitive mat, first without and then with yoked prisms in place. Prisms had their base opposed to the direction of weight shift, and the magnitude of prisms was individualized. The study reported a reduction in the balance shift of about 2-3 points, on a scale of 0-40. They inferred from this a reduced risk of falls, though the latter was not actually measured, and no data or references were given on the link between fall risk and either their variables in general or a 2-3 point reduction in particular. Other deficiencies of this study include its unblinded protocol and the lack of a comparison intervention (such as reversing the direction of the prisms, as in their first study). It was not stated whether any effects on balance persisted with use in daily life.

4. Vertical prisms for vertical heterophoria syndrome

Vertical heterophoria syndrome refers to a small vertical misalignment of the eyes, which has been hypothesized to arise from utricular or brainstem damage.[24] This is not implausible given that otolithic damage can occur with head trauma,[41] and skew deviation is one sign of unilateral utricular dysfunction.[13] The effort required to fuse the image is said to be a source of eyestrain and fixation instability, with resulting symptoms of blur, transient diplopia, and dizziness. It is claimed that these in turn lead to a range of secondary problems such as anxiety, headache, light sensitivity, and feeling overwhelmed in crowds: in essence, many of the varied symptoms of the postconcussive state. Of course, if the strain to fuse a vertical phoria were the cause of these complaints, they should resolve with patching one eye. To our knowledge, this has not been demonstrated. Furthermore, eye movement studies have shown that vertical dysconjugacy during natural viewing is no different between patients with mTBI and healthy subjects.[64]

There are 2 retrospective reports of vertical prism use from the same group. The first report reviewed 43 patients with persistent postconcussive symptoms after traumatic brain injury with undefined criteria and severity, at a mean of 3.6 years after trauma.[24] The diagnosis of vertical heterophoria was based not on objective measures of vertical ocular misalignment but on whether a vertical prism empirically reduced symptoms that included not just double vision, difficulty reading, or blurred vision, but also dizziness, light sensitivity, headache, and anxiety, all of which were considered potential manifestations of binocular visual dysfunction. Forty patients were excluded for various reasons. In the 43 reported, there were improvements on an unvalidated[1] questionnaire about those symptoms. The second study was very similar.[60] It reviewed 36 patients with postconcussive symptoms, again diagnosed with vertical heterophoria by symptomatic response to prisms and again evaluated by questionnaires on postconcussive symptoms. They claimed an 80% reduction of such symptoms, but the circular logic of the approach of these two studies is evident; patients selected for a positive response are then evaluated for a positive response. Their other limitations are also apparent: both were open-label, uncontrolled retrospective surveys of subjective outcomes.

5. Light filters

Light intolerance—a.k.a. light sensitivity, photophobia—is a common posttraumatic symptom.[11] Also, while there is still controversy surrounding the nature of posttraumatic headaches,[63,73] such headaches can have migrainous features, including light intolerance.[83] It is reasonable to speculate that reducing the intensity of light with the use of filters may alleviate these symptoms in mTBI.

Studies of filters in atraumatic migraine may be relevant to mTBI. FL-41 lenses were designed to reduce the blue-green spectrum of light that is thought to be responsible for symptoms evoked by fluorescent lighting. A study of 20 children with migraine found that 4 months of FL-41 lens use reduced headache frequency by a factor of 4, whereas blue-tinted lenses had no effect.[30] While the cause of light intolerance in mTBI is not yet clear, migrainous light intolerance may originate in intrinsically photosensitive retinal ganglion cells, whose output modulates thalamocortical neurons.[49] A study of 37 subjects with chronic migraine used a crossover design to evaluate the efficacy of filters designed to attenuate light around 480 nm, the peak sensitivity of the melanopsin in these retinal ganglion cells.[37] This was compared with the efficacy of a sham filter blocking light around 620 nm. Both filters reduced headache impact test scores, and subjects were equally likely to choose to keep either lens after completion of the study. While this equivalency might suggest a placebo effect, the authors suggested that the sham filter might have inadvertently affected melanopsin function too.

What about mTBI? One study looked at 7 patients with undefined mTBI and photophobia, with time since injury ranging from 5 months to 10 years.[38] Contrast sensitivity was measured using the Pelli-Robson chart with and without 3 different filters, the latter in random order. While contrast sensitivity without filters was similar between these patients and 7 healthy subjects, some filters increased contrast

[1] A later report performed a validation study in 29 stroke patients.[69]

sensitivity in the patients, but not in the controls, and led to faster reading times. Use of filters at home after completion of the study was said to be associated with reduced light intolerance and fewer headaches, but this was not rigorously assessed.

A pilot study reported on 12 subjects who met criteria for mTBI and complained of photophobia, at a mean of 5 years posttrauma.[26] It compared the effects of a few minutes of use of red, blue, neutral density, or individualized filters, as well as a "no lens" condition, in random order. There was little effect of lenses on measures of reading performance or VEPs. While the abstract claimed subjective improvement in 11 patients, neither the methods nor the results described how this was assessed. Curiously, effects on light intolerance were not mentioned. The authors speculated that more prolonged use in a larger sample might be needed to clarify benefit.

On the other hand, a retrospective review of 62 young adults with mTBI found that 40% had a delayed improvement in light intolerance after a year.[81] They noted that improvement was twice as likely in individual who did *not* wear tinted lenses. This suggested that either (a) those whose light intolerance improved were less likely to continue wearing lenses, or (b) wearing tinted lenses inhibited long-term adaptive changes that reduced light intolerance. Other risk factors for persistent light intolerance in this study included dry eyes and migraine.

Given the above, the benefits of filters in mTBI have to be considered unproven at present. While it is plausible that they may have a symptomatic role in reducing light intolerance, the last study raised a concern that they impede long-term improvement, which needs examination in a prospective randomized study. It would be helpful if future studies of filters concentrated on proving their efficacy for the specific symptom of light sensitivity, which can be measured in mTBI.[11]

6. Vestibular therapy

Prior reviews have noted the dubious quality of the evidence for ocular motor training in mTBI and other neurologic conditions.[5,7,8,62] How does this compare with that for other postconcussive treatments? Vestibular therapy is of particular interest because dizziness is one of the somatic symptoms in the diagnostic criteria for postconcussive syndrome.[42,43] There is substantial work showing that vestibular therapy is effective for patients with unilateral vestibular dysfunction.[35] However, there are much less data on its efficacy in mTBI,[46] as is generally the case for all forms of exercise therapy in this setting.[57]

There are two retrospective reports. One studied 84 patients with undefined mTBI and unstated time since injury, who had individualized vestibular home exercises of varying duration.[2] This found improvement on dizziness questionnaires as well as on measures of gait and balance, including dynamic computerized posturography, though some measures improved only in children. A second study was directed at a specific comparison of neck versus vestibular home exercises in a select group.[34] This evaluated 48 soldiers who had dizziness and met defined criteria for mTBI. Critically, subjects were included if they had signs suggesting problems with cervical proprioception and excluded if they had signs of central or peripheral vestibular dysfunction. Subjective resolution of dizziness for at least 2 weeks was the only outcome measure. This occurred in 22 of the 26 who had neck exercises but in only 4 of the 22 who had vestibular therapy, and improvement was more likely with treatment in the first year.

There are two prospective studies without control observations. One followed 58 military personnel who met criteria for mTBI and who were referred for dizziness of unstated duration.[36] All subjects had otolaryngologic and neurologic evaluations, vestibular testing, and magnetic resonance imaging. Subjects fell into 3 defined categories: positional vertigo ($n = 16$), migraine-associated dizziness ($n = 24$), and spatial disorientation ($n = 11$). After 6 to 8 weeks of individualized exercises, there was purported improvement on tests of the vestibulo-ocular reflex in 84% of those with migraine-associated dizziness but only 27% of those with spatial disorientation. These results were not given in detail. Return to work took longer for those with spatial disorientation than those with migraine-associated dizziness (16 versus 3.8 weeks).

A second prospective study without a control intervention followed 82 soldiers with undefined blast-induced mTBI who had disequilibrium, vertigo or positional vertigo of unstated time since injury.[32] All had the same 12-week exercise program, twice a week with the therapist as well as at home daily. They were evaluated on dynamic posturography, measures of gait, and standardized visuovestibular tests. Compared with baseline, subjects improved by 4 weeks on measures such as dynamic visual acuity and posturography and by 12 weeks on measures of gait.

There are three randomized controlled trials of vestibular therapy in mTBI. One exists only in abstract form.[22] This trial randomly assigned 38 pediatric patients recruited within 2 weeks of concussion to either daily home exercises or rest. There were no differences after 5 or 10 days of treatment on daily scores of headache, balance, and dizziness severity. Without a more detailed publication, it is not clear what to make of these results.

A second trial recruited 30 young people with undefined sports-related concussions, ranging from 1 week to 9 months postinjury, with more than 10 days of headaches, neck pain, or dizziness.[68] Subjects were randomly assigned, half to 8 weeks of individualized cervical and vestibular therapy and half to rest with range of motion exercises. The primary outcome was time to medical clearance by a blinded physician. This was achieved within 8 weeks by 11 of the 15 treated patients but only 1 of the 14 in the control group, though 3 in this last group dropped out. A blinded therapist also judged secondary outcomes that included both symptom ratings and signs on exam, but the analysis of these measures focused on the difference between those who were cleared and those who were not, rather than comparing the treatment and placebo groups.

A third trial studied 65 patients with undefined mTBI at a mean of 3 months postinjury, who had a positive Romberg sign, endorsed dizziness on the Rivermead postconcussion questionnaire, and scored more than 16 on the Dizziness Handicap Inventory.[40] Subjects were randomly assigned to

8 weeks of biweekly group-based vestibular therapy with individualized home exercises or to no therapy other than optional psycho-educational group sessions. Evaluations were done immediately after the 8 weeks and 2 months later. The primary outcome measure was the score on the Dizziness Handicap Inventory, with secondary measures being scores on standardized questionnaires about mobility, balance, anxiety, and postconcussion symptoms. The assessor was blinded. A linear mixed model analysis showed improvement on the primary and some but not all secondary outcomes immediately after treatment. While the gains in the treatment group were maintained at 2 months, the control group had also improved to a similar level by that point. The conclusion was that vestibular therapy speeded recovery but did not affect the ultimate outcome.

On the basis of these 3 randomized controlled trials, one is tempted to speculate that early treatment has modest effects because of natural recovery, but later treatment may help in those who have not yet recovered. These studies, however, are still few in number and their outcome variables limited; hence, the results cannot be regarded as conclusive.

7. Conclusions

Evidence for the use of the reviewed visual therapies for post-mTBI symptoms is limited, and the logic of some is questionable. Explanations of the effects of binasal occlusion are not plausible, and there is no evidence that they provide symptomatic relief. Prisms have a logical role in managing convergence insufficiency, but there are no data clarifying their utility specifically in the posttraumatic state. Midline shift may occur with egocentric hemineglect or vestibular dysfunction, but it is less clear why or if this occurs after mTBI. It is also not clear whether its proposed treatment with -yoked prisms has any effects on balance that translate to a meaningful difference in daily life. Vertical heterophoria syndrome remains an unproven construct, and circular reasoning confounds the retrospective reports of its treatment. It is reasonable to expect that light intolerance and possibly headaches may benefit symptomatically from the use of light filters, but at present their benefits remain unproven, as do their effects on the long-term prognosis of these symptoms. The number of studies of vestibular therapy for mTBI is small, but they show a movement toward desirable properties for clinical trials. These include prospective design, diagnostic and inclusion criteria, larger samples, random assignment to treatment and control groups, blinded evaluators, assessment of symptoms relevant to the patient, objective measures of balance function that can be linked to those symptoms, and appropriate statistical analyses. More work of such quality is needed and should serve as a role model for studies of other vision therapy in mTBI.

8. Method of Literature Search

We accessed MEDLINE through PubMed and also Google Scholar. Search terms included “filter, prism, occlusion, binasal, vestibular therapy, vestibular habituation” in conjunction with “head trauma, mild traumatic brain injury, concussion” for all years available. All articles on therapy cited in the reference lists of the resulting articles were retrieved and included in the review. We did not discover any articles that had not been translated into English. To be comprehensive, articles on therapy were included if they described and provided data on trials with groups of patients of any size, whether retrospective or prospective, uncontrolled, or controlled, with the shortcomings of each study described in the review. Anecdotal reports of single subjects were disregarded, unless of historical interest, as were opinion pieces that did not provide any data.

9. Disclosures

Declarations of interest: none.

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