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Alwyn Lishman's contribution to the neuropsychiatry of head injury (traumatic brain injury); two key papers

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ABSTRACT

Introduction: Alwyn Lishman appreciated that if we are to understand the psychological consequences of cerebral disorder we must study the interaction between organic disease and psychological processes.

Methods: We have reviewed Lishman's two major publications on the neuropsychiatry of head injury, published in 1968 and 1988, and considered their conclusions in the light of current knowledge.

Results: In his 1968 paper on the psychiatric sequelae of open head injuries sustained in World War II Lishman demonstrated associations between the type of psychiatric sequelae and the location of the injury. He also found that those with "somatic complaints", such as fatigue or sensitivity to light, showed less evidence of organic injury. In his 1988 paper, he attempted to explain why a mild head injury may be followed by long-lasting symptoms. He suggested that in the absence of complications early, organic, symptoms (physiogenesis) should recover quickly. However, this healthy recovery could be jeopardised by psychological factors (psychogenesis), resulting in long-lasting symptoms. This model of physiogenesis and psychogenesis remains relevant today.

Conclusions: The ideas Lishman developed in these two papers were the basis for his huge contribution to the field of neuropsychiatry, and remain relevant today.

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Introduction

Alwyn Lishman's interest in neuropsychiatry can probably be dated back to his national service, in the 1950s, when he worked at the Military Hospital for Head Injuries in Oxford. There he worked with one of his early mentors Ritchie Russell, a neurologist, who had been officer in charge of the hospital during WWII.

Russell had introduced the idea that the duration of post-traumatic amnesia (PTA) was a useful measure of closed head injury severity (Russell, 1932). Russell went on to show that the duration of PTA could differentiate organic from non-organic sequelae of closed head injury (Russell & Smith, 1961). That study found that while the prevalence

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of neurological symptoms, such as dysphasia or motor problems, was clearly associated with length of PTA, for symptoms such as headache, dizziness sans vertigo and anxiety, it was not.

Lishman appears to have been stimulated by these findings. For his MD thesis, which was in turn the basis of his 1968 paper (see below), he looked at the organic basis for psychiatric sequelae of head injuries. The ideas set in train by that study were the basis for one of his great contributions to neuropsychiatry: that we need to appreciate the interaction between organic and psychological factors to understand the psychological consequences of cerebral disorder (Lishman, 1978). This theme dominates the two key papers he wrote (Lishman, 1968, 1988) on the neuropsychiatric sequelae of head injury.

Brain damage in relation to psychiatric disability after head injury 1968

In this paper, Lishman highlights that research to date had failed to significantly define the extent to which psychiatric disability relates to physical brain damage. He mentions that from the early 1900s and through the Second World War there had remained limitations to objectively assess the degree of injury, even with techniques available at the time; psychological, radiographic, electroencephalographic and operation records. He also observes that most studies had previously focused on psychogenic rather than physiogenic factors, but that these were inherently challenging to separate. While clinical syndromes such as frontal lobe syndrome and temporal lobe sequelae had already been described, research studies lacked standardisation of procedures and brain injury data and sampling. It is interesting that these are issues we grapple with today (Raymont & Thayanandan, 2020) and are compounded by the understanding that TBIs are not only acute events, but trigger a progressive process of multiple, parallel, interacting, and interdependent cascades of biological reactions (Wang et al., 2018) and that it remains unclear if TBI is a risk factor for heterogeneous pathologic processes or contributes to a common pathologic mechanism (Perry et al., 2016).

This paper focuses on 1,024 patients with head injuries that were part of the Oxford Head Injury Bureau recruited in WWII and followed up by Ritchie Russell. Eight hundred twenty-nine of the sample had sustained penetrating TBIs, but 141 were excluded because of inadequate follow up, leaving a final sample of 670. Patients with all severity of TBI were followed up for five years via annual questionnaires, plus information from relatives, employers, GPs, social agencies and the Ministry of Pensions & Pension Boards. A four-grade index of brain injury (BI) was used which incorporated depth of damage based on X-rays and operation records, and location of tissue destroyed. One strength of this study is that all psychiatric symptoms were included, with no attempt to assess the aetiology, which fed into a “comprehensive estimate of psychiatric disability” rated as mild, moderate or severe. General intellectual disability was also graded as mild or severe. The drawbacks are that the follow up was not psychiatry-focused and largely consisted of document review.

There are four sub-investigations in this paper. The first looks at brain injury measures, allowing for military rank (as a proxy for IQ), age at injury and extent of physical and intellectual disability. The data show consistent statistically significant positive correlations between depth of penetration and total brain tissue destroyed and psychiatric disability, which supports the hypothesis that brain damage could contribute to the

causation of psychiatric disability after head injury. It is recognised that psychiatric disability was measured rather broadly, but that the definition of brain injury was precise for its time. The quantity of brain tissue destroyed (not just the depth of damage) appears to be related to psychiatric disability, but Lishman suggests that this may just reflect the severity of the initial impact rather than diffuse effects. As a way of addressing this, in the second sub-investigation, Lishman goes on to assess PTA and post-traumatic epilepsy (PTE).

PTA was used as an indicator of TBI severity. PTE occurs most frequently in penetrating TBIs and has a significant psychological impact (Smith, 1961). Lishman reports that in those with a PTA lasting less than an hour there were less cases with higher psychiatric disability, whereas the reverse was true for those with PTA of more than 24 hours. But for those who had PTA between 1 and 24 hours there was no clear direction of correlation. Lishman suggests that while these results confirm the importance of PTA as a prognostic factor, the variability could reflect diffuse injury effects. PTE also correlated with the degree of BI, but to a lesser degree, with epilepsy of early rather than late-onset after injury being mainly responsible for the association with psychiatric disability.

The third and fourth sub-investigations look at how location and extent of penetrating injuries ($n = 345$) correlate with psychiatric disability, and significantly, its components. Psychiatric disability was found to be more closely related to left than to right hemisphere damage and was related to extent of BI especially in the left hemisphere. Temporal lobe BI (especially on the left side) was more associated with psychiatric disability than frontal, parietal or occipital injuries, although there was some correlation with right frontal and left parietal lobe BI. Brain injuries which led to dysphasia, sensory motor or visual field defects were significantly related to psychiatric disability, although the association between left temporal lobe BI and psychiatric disability persisted even after excluding these cases. Lishman comments that these data provide substantial evidence that focal, rather than diffuse, BI produce differing levels of psychiatric sequelae. The analysis focusing on individual symptoms is treated with more caution given the mode of data collection, but of the 144 injuries followed by severe disability, intellectual disorders were more common with left hemisphere damage, while affective disorders, behavioural disorders and somatic complaints were more frequent after right hemisphere damage. Parietal and temporal lobe damage was associated with intellectual disorder, frontal lobe damage with affective disorders.

Lishman was alert to Russell and Smith's (1961) observation, in those with closed head injuries, that headache, dizziness sans vertigo and anxiety, did not correlate with brain injury severity (see Introduction above). Lishman observed a similar effect in his series of open head injuries (Lishman, 1968). Those with "somatic complaints" (such as fatigue, dizziness and sensitivity to noise), showed less evidence of organic injury. And this tended also to be true for complaints of anxiety, depression and difficulty concentrating. And he writes "There is an obvious alignment between the group of somatic complaints and all other symptoms which show least evidence of an organic aetiology". This observation was therefore relevant to the aetiology of long-term complaints after mild head injury; the subject of his 1988 paper (see below).

Lishman's, 1968 paper was ground-breaking, addressing as it does the impact of BI location and extent on subsequent psychiatric symptoms. It is telling though, that one

of the main criticisms of research into psychiatric sequelae of TBI today remains the low standardisation of diagnosis and study methodology (Phipps et al., 2020).

Physiogenesis and psychogenesis in the “Post-concussional syndrome” 1988

In his 1988 essay paper, Lishman introduced a model, physiogenesis and psychogenesis, to explain why some after a mild head injury (mild traumatic brain injury; mTBI) develop long-lasting symptoms, often labelled as the “post-concussion/al syndrome” (PCS).

The paper starts with a review of aetiological factors relevant to PCS. Several studies had shown that symptoms suggesting injury to brain or the vestibular system were common in the first few days after an mTBI. It seemed likely that cerebral dysfunction/injury to the head explained early symptoms such as diplopia, slowed information processing, fatigue, headache and dizziness.

On the other hand, the longer the time post-injury the patients were studied, the more likely it was that psychological effects were identified as explaining the symptoms.

And there was evidence that the symptoms themselves changed over time. Lidvall et al. (1974) had shown that in the days post-injury headache was frequently reported, but then, over the course of a few weeks, became much less prevalent. On the other hand, anxiety tended to increase in frequency over those first few weeks post-injury.

Based on these observations Lishman proposed that in the hours and days post-injury cerebral dysfunction and vestibular injury commonly led to symptoms that were, to start with, firmly organic in origin (physiogenesis). Central to these were headache, dizziness and fatigue.

Lishman suggested that if the patient was untroubled by these early, organic, symptoms or by other matters, then a full recovery, over the course of a few days or weeks, was likely to take place. However, obstacles of a psychological nature could hinder the natural process of recovery (psychogenesis). Such obstacles included having a vulnerable constitution, worrying unduly or being under stress. In the presence of such adverse psychological effects, the patient was at risk of developing persistent symptoms.

Lishman’s model whereby persistent symptoms are the result of adverse psychological effects, heavily based on anxiety, interfering with the healthy and expected recovery of early organic symptoms, is central to many attempts since 1988 to understand why some develop long-term problems after an mTBI (Potter & Brown, 2012). The model has been modified to allow for psychophysiological effects and poor motivation (Jacobson, 1995). In 2011 Silverberg and Iverson revisited physiogenesis and psychogenesis and found that, by and large, the model remained a valid account of the aetiology of PCS. However, recent research had shown that psychological effects from the outset appeared able to predict symptoms. Vicious circles, with anxiety driving somatic and cognitive symptoms which in turn increase anxiety, are now understood to play a part (King, 2003).

How does physiogenesis and psychogenesis measure up when it comes to recent studies of neuroimaging and psychosocial factors in those with mTBI? The model is based on the premise that there is early cerebral dysfunction. This is different for example, from Mittenberg et al. (1992), who suggested that expectations could be sufficient to explain early symptoms. However, support for early cerebral dysfunction

comes from MRI studies which show that changes in white matter tract diffusion imaging are to be found in many following mTBI (Wallace et al., 2018).

However, as anticipated by Lishman, evidence of cerebral injury is not a good predictor of outcome. The most sensitive neuroimaging marker of cerebral lesions, shear haemorrhages found on susceptibility-weighted MRI imaging (SWI), generally does not predict outcome, although Einarsen et al. (2019) found a small effect. Diffusion tensor imaging (DTI) or diffusion kurtosis imaging (DKI), and other measures of white matter integrity such as fractional anisotropy (FA), generally do a little better at predicting outcome (Karlsen et al., 2019; Yuh et al., 2014), particularly if done early post-injury (Richter et al., 2021). But Stenberg et al. (2021) found that if a measure of pre-injury mental ability (Vocabulary) was taken into account, much if not all of the ability of low fractional anisotropy early post injury to predict the outcome, was lost.

This last finding is consistent with Lishman's model and consistent with studies that find that psychosocial factors are better at predicting outcome after mTBI than neuroimaging markers of brain injury (Wäljas et al., 2015). The systematic review of Silverberg et al. (2015) found that while the literature on neuroimaging predictors of outcome after mTBI was somewhat inconsistent, studies of psychosocial predictors of outcome routinely found effects. This conclusion is supported by van der Naalt et al. (2017) who found that emotional distress and maladaptive coping early post-injury, as well as pre-injury mental health problems and education, were important predictors of outcome at six months following mTBI.

In our experience, patients after an mTBI often find physiogenesis and psychogenesis a helpful way to understand their chronic symptoms. The model does not deny that there has been any cerebral or vestibular injury, nor does it suggest that the outlook is necessarily bleak because of persistent damage. The model allows for the possibility that psychological treatment may improve outcome (Potter et al., 2016).

Conclusion

The ideas that Alwyn Lishman developed in these two major papers on the neuropsychiatric sequelae of head injury, were fundamental to his understanding of the psychological sequelae of cerebral disorder, regardless of the diagnosis of the cerebral disorder. His studies of head injury were, therefore, the foundation for his huge contribution to the field of neuropsychiatry.

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