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Review

The role of evidence based medicine in neurotrauma

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ABSTRACT

The introduction of evidence based medicine de-emphasised clinical experience and so-called "background information" and stressed the importance of evidence gained from clinical research when making clinical decisions. For many years randomised controlled trials have been seen to be the only way to advance clinical practice, however, applying this methodology in the context of severe trauma can be problematic. In addition, it is increasingly recognised that considerable clinical experience is required in order to critically evaluate the quality of the evidence and the validity of the conclusions as presented. A contemporary example is seen when considering the role of decompressive craniectomy in the management of neurotrauma. Although there is a considerable amount of evidence available attesting to the efficacy of the procedure, considerable clinical expertise is required in order to properly interpret the results of these studies and the implications for clinical practice. Given these limitations the time may have come for a redesign of the traditional pyramid of evidence, to a model that re-emphasises the importance of "background information" such as pathophysiology and acknowledges the role of clinical experience such that the evidence can be critically evaluated in its appropriate context and the subsequent implications for clinical practice be clearly and objectively defined.

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1. Introduction

It is over 20 years since the introduction of evidence based medicine when it was announced that a new paradigm was emerging. This paradigm shift de-emphasised intuition and unsystematic clinical experience when considering clinical decision making and stressed the importance of examining evidence from clinical research [1]. The levels of evidence were stratified according to strength and a pyramid constructed with the meta-analysis of randomised controlled trials placed at the apex and seen to provide the highest level of evidence. Clinical judgement and background information such as pathophysiology were relegated to the base of the pyramid and were deemed to provide the lowest level of evidence (Fig. 1).

For many years a well designed randomised controlled trial published in a reputable medical journal was seen to be the only way to advance clinical practice and there can be no doubt that within the field of neurosurgery there are notable examples. These include the International Subarachnoid Aneurysm Trial (ISAT) investigating the role of coiling *versus* clipping of intracranial aneurysms [2], the Clifton studies investigating the role of hypothermia in traumatic brain injury (TBI) [3,4] and the Medical Research Council Corticosteroid Randomization After Significant Head injury (MRC CRASH) study that investigated the role of steroids in the field of TBI [5]. It is however becoming increasingly apparent that there may be some limitations when attempting to apply this approach across all aspects of clinical practice, not least in the context of neurotrauma where surgery is often performed as a life saving intervention [6,7]. In addition, it is increasingly recognised that considerable clinical experience is required not only to critically evaluate the quality of the evidence but also to assess the validity of the conclusions that are often presented by the investigators. Indeed it has been suggested that even the most rigorous trials have methodological flaws such that the conclusions of many studies are either exaggerated or indeed incorrect [8]. Given these limitations the time may have come for another paradigm shift that re-emphasises the importance of a clear understanding of pathophysiology and acknowledges the role of clinical experience such that the evidence can be critically evaluated in its appropriate context.

A contemporary example of this issue is seen when considering the role of decompressive craniectomy in the management of neurotrauma [9]. There is currently a great deal of evidence available regarding clinical efficacy of the procedure however considerable

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expertise is required in order interpret this evidence and the impact that it should have on clinical practice.

The aim of this narrative review is to highlight the importance of so called "background information" when considering the surgical management of severe TBI and this includes the underlying pathophysiology of the condition and the contemporary medical management. Thereafter the evidence currently available for clinical efficacy will be critically evaluated.

2. Traumatic brain injury: background information

2.1. Pathophysiology

There are two important concepts that require consideration when managing patients with severe TBI. The first is the neuroexcitatory cascade and the second is the Monro–Kellie doctrine.

2.1.1. The neuroexcitatory cascade

Traditionally TBI has been defined as primary, which occurs at the moment of impact, or secondary, which occurs in the minutes, hours and even days thereafter [10]. However it is now becoming increasingly apparent that there is considerable overlap between the two processes due to the heterogeneous nature of the underlying pathophysiology. Areas of ischaemia coexist with mass lesions, contusions and areas with varying degrees of blood–brain barrier disruption, and a substantial amount of cell death that occurs many hours following the initial injury is due to a series of deleterious neurochemical cascade processes that are initiated at the time of injury [11,12]. This concept is well illustrated by the glutamate neuroexcitatory cascade which is one of a number of neuroexcitatory responses that are initiated at the time of injury and can be amplified thereafter by the secondary insults such as systemic hypoxia and hypotension (Fig. 2) [13,14]. In severe trauma the resulting cerebral swelling can further impair cerebral perfusion which in turn can further exacerbate the cellular response.

2.1.2. The Monro–Kellie doctrine

The Monro–Kellie doctrine describes the cranium as a "rigid box" filled with a "nearly incompressible brain" [15,16]. When the brain is injured and starts to swell or there is a mass lesion such as an intracerebral haematoma, initial compensation aimed at maintaining intracranial normotension is made at the expense of blood and cerebrospinal fluid volume (Fig. 3). As the brain becomes progressively more swollen or a mass lesion increases in size these compensatory mechanisms become progressively exhausted, and for incrementally smaller increases in volume there are progressively greater increases in intracranial pressure (ICP) until tonsillar herniation and terminal brainstem compression occurs.

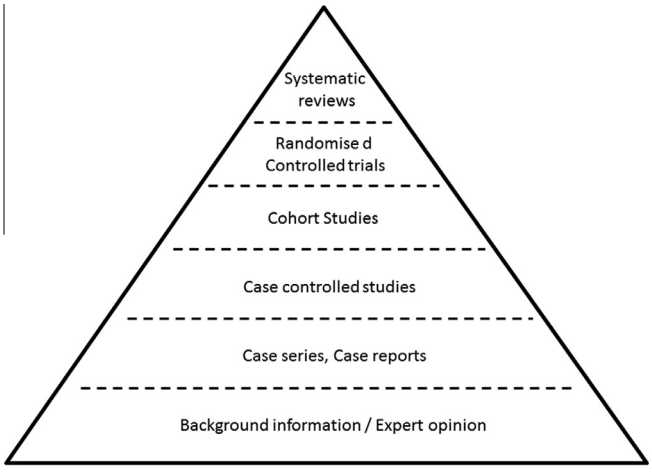

Fig. 1. The traditional pyramid of evidence based medicine.

An appreciation of these concepts is important when considering the limitations of some of the traditional medical therapies that have been used to manage patients with severe TBI.

2.2. Contemporary medical management

The medical management of severe TBI has evolved considerably over recent years. The use of ICP monitoring has become widespread as it provides important information regarding prognosis and allows early identification of those patients who develop surgical lesions requiring evacuation. It also identifies those patients who go on to develop progressive cerebral swelling which would preclude them from early weaning and for whom medical therapy can be targeted in order to optimise cerebral perfusion and minimise secondary insults [17].

The ICP threshold at which these medical therapies are introduced has been set at 20 mmHg and this is because of the strong correlation between outcome and the number of hours the ICP remains above this level [20,21]. These therapies are introduced in a stepwise protocol driven manner and include measures such as head elevation, further sedation and paralysis, cerebrospinal fluid drainage and osmotherapy followed by the judicious use of hyperventilation, barbiturate therapy and hypothermia [17]. However, notwithstanding the prognostic significance of raised ICP, one of the fundamental challenges in neurotrauma has been the inability to demonstrate that the fall in ICP induced by many of these therapies is subsequently translated into an improvement in clinical outcome [3,4,20–23].

For many years patients were routinely hyperventilated, placed in a barbiturate coma, or more recently rendered hypothermic in order to control ICP. The rationale was that by controlling the ICP the clinical outcome was improved, however numerous clinical studies failed to demonstrate that these therapies actually achieved this objective and in certain circumstances may have caused harm [21–24]. On face value these findings would appear counterintuitive; however, studies investigating the effect that these three therapies have on cerebral blood flow indicate a probable reason. There is little doubt that barbiturates and hypothermia have the potential to be neuroprotective due to their influence on many aspects of the cellular response to injury including calcium mediated toxicity, glutamate excitotoxicity, free radical peroxidation and cellular apoptosis [14,25–27]. However the mechanism by which these three therapies produce the often rapid fall in ICP that occurs after their application is predominantly as a result of cerebral vasoconstriction and the subsequent reduction in cerebral blood flow, as demonstrated by a number of studies [28–31].

As demonstrated by the underlying pathophysiology and the compensatory mechanisms that occur when the brain swells, the last thing the swollen injured brain needs is less blood and contemporary medical management aims to optimise cerebral perfusion and tends to reserve therapies such as hyperventilation, barbiturate coma and hypothermia for intractable intracranial hypertension that is unresponsive to the aforementioned medical therapies. It is in this situation that decompressive surgery is often considered.

3. Decompressive craniectomy

By temporarily removing a large section of the calvarium, decompressive craniectomy challenges the Monro–Kellie doctrine by expanding the "rigid box" and providing extra space into which the injured brain can expand. The aims of the procedure are to

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