Zahid Hussain Khan *Editor*

Challenging Topics in Neuroanesthesia and Neurocritical Care

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Foreword

While training as a young intensive care consultant, I was always amazed at how little emphasis was placed on the well-being of the lump of gray matter sitting in our heads called the brain. Numerous ICU conferences elaborated on the exciting topics of ARDS and ventilation, diagnosis and mechanisms of sepsis, oscillators and jet ventilation, and ECMO. We discussed heart disease and physiology, pulmonary mechanics, dialysis, sepsis, and various other thought-provoking areas. But there existed a general lack of awareness that the point of all this was to ensure that the brain was maintained in an adequately working state. The impact of diseases and treatments on the brain were largely ignored. When the brain was directly impacted by disease such as following traumatic brain injury (TBI), stroke, or SAH, most clinicians took a relatively nihilistic view that as outcomes could not be significantly impacted by hospital management and as most patients survived who made it to hospital (although the level of functioning was largely ignored), there was little interest in pursuing research to improve outcomes for these patients. In many instances, this was because the brain was largely a closed book to most of us. While cardiac imaging was advancing in leaps and bounds, there was little insight into the working of the brain and even less into how to investigate and monitor cerebral activity.

Very few intensive care centers had taken any interest in neurocritical care at this time, and patients were often managed in general ICUs or neurosurgical HDUs and not necessarily by intensivists. A few specialized centers were emerging, however, notably Massachusetts General Hospital and Addenbrooke's Hospital in Cambridge, and these would eventually become the leaders in the push for a more brain-focused approach to the critically ill. Their efforts were pivotal in initiating the long process of undertaking and collating research in order to determine what did and, as importantly, what therapies didn't improve outcomes post ABI. Furthermore, a closer look at outcome measures would be necessary, as mortality alone in the setting of major neurological deficits was recognized as insufficient and issues of quality of life would have to be looked into.

Over the past 25 years, momentum has steadily grown, and a number of important papers have been produced demonstrating positive outcomes from a range of interventions including improving cerebral hemodynamics, cerebral vasospasm, and intracranial hypertension, to name a few. Furthermore, the Brain Trauma Foundation published their guidelines on the management of TBI and, although light on evidence, was able to provide a baseline from

which further research could be undertaken. As such, a PubMed search today will reveal a plethora of papers in the field of neurocritical care reflecting a massive increase in interest and funding in this field and setting the stage for new and exciting developments in the future.

With this newfound interest in all things brain, the number of books published on the topic has increased exponentially. Many seem to follow the time-honored tradition of approaching the topic broadly with a reference to basic physiology, general cerebral monitoring and management, and a few chapters on specific topics. As such, most books provide an overview of management for the ABI population as a whole but may be a little short on specifics when it comes to managing the individual patients or when specific problems develop which may be a little out of the ordinary.

In *Challenging Topics in Neuroanesthesia and Neurocritical Care*, Zahid Khan has attempted to delve into specific issues that are faced by the practicing clinician on a daily but also occasional basis. With many years of experience in neuroanesthesia and neurocritical care, Khan has chosen topics that may not readily be found in the average neuroICU textbook. And while many of the chapters cover topics broadly reviewed by other titles, Khan attempts to deal with specific issues confronting the neuroanesthetist and neurointensivist. For example, the chapters on airway and pain management, common but often overlooked issues, are reviewed in detail. A number of chapters are reserved for discussing anesthetic techniques in a variety of complex situations including posterior fossa surgery, pituitary surgery, and awake craniotomies. And newer therapies such as the use of inhalatory sedation in neurointensive care are also reviewed. And the final chapter on neuroprotection, a rapidly expanding and very promising area of research, concludes the reviews.

It is hoped that by dealing with specific topics, this book will provide an alternative source of information filling the gap in knowledge and improving clinicians' ability to manage patients with complex neurocritical issues. Many topics covered may be controversial and lacking in widespread evidence to guide management. As such, the title *Challenging Topics in Neuroanesthesia and Neurocritical Care* seems an apt choice.

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Preface

An overwhelming interest did exist with me when I started as a young anesthesiologist catering for neuro-cases more than two decades back. This interest could hardly have arisen but for a conflux of circumstances and an influx of patients, some of them exceedingly rare which I had the opportunity to report later.

Iran has the highest number of road accidents in the world, and as such, we have a large number of head injury and spinal cord injury patients landing in our emergency departments with compromised cardiovascular status requiring urgent resuscitative and airway management strategies. I thought of it and the fate of these patients by day and nursed it by night. It became an overpowering, absorbing passion with me, and I resolved to take up the task of writing a book with the aim of addressing the most challenging issues in neuroanesthesia and neurocritical care which I thought would be of help in the overall management of these cases. I had been thinking over it most deeply and most intently. Head injury has been a continual and ephemeral problem commonly felt but less acknowledged. For such a book, I needed to muster all my resources and get the help of celebrities in the field. A search provided me an egress to some of my old friends and scholars in the field.

Head injuries take a massive toll of our younger generation, and some of these cases are wheeled into the neurointensive care units with diffuse axonal injuries and with a Glasgow Coma Scale score of 6 or 7 or even below that. Ischemic injuries have been observed in more than 90% of patients dying as a result of head injury. Systemic insults, such as hypotension and hypoxia, are frequent in the early posttraumatic period as well as in the intensive care setting. Thus, these patients not only need an intensive care but in fact an extravigilant care; otherwise, they would pass away without pain or suffering like a clock someone had forgotten to wind.

Prognostic values of traumatic brain injury (TBI), brain tissue oxygenation in TBI, intracranial compliance, TBI and management, and biomarkers in TBI have been dealt with exquisite detail. Likewise, blood glucose concentration management, paroxysmal autonomic instability with dystonia, perioperative stroke, subarachnoid hemorrhage, postoperative hematoma, deep brain stimulation, and diagnosis of brain death have all be dealt with in sufficient detail. Apart from these, there are some highly important chapters on airway and pain management and anesthesia in different clinical and challenging scenarios, related to the central and peripheral nervous system.

The book covers areas about brain monitoring in severe head trauma. These chapters would provide the clinician an insight whether jugular bulb oxyhemoglobin saturation (SjO_2)-guided therapy provides better outcomes than intracranial pressure (ICP)- and cerebral perfusion pressure (CPP)-guided therapy in clinical practice based on expert opinion or based on randomized, clinical trials. SjO_2 monitoring is considered to be appropriate for detecting global cerebral ischemia. Low SjO_2 values indicate a higher extraction of oxygen and thus are indicative of ischemia. But as there are different types of tissue hypoxia, SjO_2 monitoring will not be able to detect all causes of tissue hypoxia, and furthermore, tissue hypoxia may be present despite normal or increased venous saturation values. The use of invasive monitoring system should be justified by a hierarchy of evidence. Jugular desaturation implies that cerebral blood flow (CBF) is inadequate for the metabolic needs of the brain, and SjO_2 values below 50% are a medical emergency. However, SjO_2 cannot always be relied upon if it is one sided, and bilateral SjO_2 monitoring should be applied if the SjO_2 data are inconsistent with other physiological variables. Currently, ICP- and CPP-guided therapies are the main therapeutic modalities in the treatment of severe head injuries. The underlying aim is to ensure that cerebral blood flow (CBF) and oxygenation are adequate to meet the metabolic demands.

Continuous monitoring of partial pressure of brain tissue oxygen ($PbrO_2$) that provides an additional information on the local oxygen status of the injured brain has also been described as a safe method of brain monitoring. Low $PbrO_2$ recordings reflect hypoxia of the relatively undamaged tissue in the first 24 h after head injury and thus could serve as a useful tool of monitoring in expert hands. In some studies, a correlation between low $PbrO_2$ and normal or even high SjO_2 has not been found. In intensive care management, it is imperative that a focus is made not only on increasing CPP but, more specifically, on attempts to increase brain tissue oxygen levels.

Early detection of impending cerebral ischemia in comatose patients is the cornerstone in obviating secondary insults to the brain that has received a trauma or injury. Several researchers have upheld the notion that the brain tissue PO_2 ($PtiO_2$) would be adequate if the CPP is maintained at 60 mmHg. Increasing the CPP above 60 mmHg would not further improve $PtiO_2$. Hyperventilation should preferably be carefully monitored by SjO_2 and $PtiO_2$ monitoring to prevent hypocapnic-induced cerebrovascular constriction and cerebral ischemia. The modifications in $PaCO_2$ have served to be of prognostic value in treating head injury patients and help as therapeutic strategies in controlling ICP and adjusting CBF to meet metabolic needs. The transcranial Doppler (TCD) measures the change in CBF during $PaCO_2$ variations and to test CO_2 cerebrovascular reactivity. This aspect has been adequately covered in one of the chapters.

Since head injury patients or those undergoing neurosurgical operations or monitoring present a plethora of problems, it is recommended that the different indices are monitored and optimized. These aspects are fully covered and dilated upon in the different chapters of the book. Some recommend continuous infusion of norepinephrine if the CPP is lower than 80 mmHg and ICP higher than 20 mmHg and/or jugular venous oxygen saturation ($SvjO_2$) is

equal to or less than 55%. Of course, under such circumstances, it is mandatory that the blood volume is optimized first. This treatment modality is used with the explicit purpose to stabilize CPP between 80 and 100 mmHg so as to optimize ICP and CPP. However, when CPP is compromised between 65 and 80 mmHg but ICP remains lower than 20 mmHg and $SvjO_2$ higher than 55%, no therapeutic intervention is needed. The first chapter of the book tackles in sufficient detail the intracranial compliance which is defined as the change in cerebrospinal fluid volume per unit change in pressure, which is not constant but increases as pressure rises. The lower limit of autoregulation represents the CPP level (about 40 mmHg in normal subjects) below which cerebral vasodilation and reduction in cerebrovascular resistance (CVR) can no longer compensate adequately for the decreasing CPP.

The lower limit of autoregulation is best defined by serial TCD monitoring, such as measurements of SjO_2. The authors fully highlight these domains in their respective chapters.

In the chapters on anesthetic management, controversial issues have been brought to the limelight and adequately addressed which would serve as a useful reference book for the practicing anesthesiologists and neurointensivists.

Each author has fortified his/her discourse with all the arguments and evidence that human art and wit would devise, and this is clearly evident in all the three parts of the book. I am grateful to all the authors who most willingly opted to serve as contributors and presented their scholastic works for this book. By writing these exemplary chapters, the neuro-patients would be debtor to you so long as they live and would be paying you their gratitude so long as they are breathing, and until that moment, they take the last sigh of their life. If anything would increase my happiness and delight, it would be perceiving that the baby would have outgrown its set of caps or in other words the book would have received a general applause from its readers.

All the reviews presented herein would help the neurointensivists, neuroanesthesiologists, emergency medicine specialists, pulmonologists, and airway care specialists in taking the required and appropriate steps when such challenging cases are encountered.

I also avail of this opportunity in extending my profound gratitude to the Springer publications and the team involved in the publication of this book who helped me at every step through their suggestions and advices. It was indeed very rewarding working with the Springer publishers and its highly diligent team.

I owe a great deal to my father who has always been the driving force for all my academic pursuits, to my spouse who has always been a source of help and encouragement, and to my patients who taught me a lot the intricacies of this discipline through their illnesses and eventual outcomes.

I am also grateful to Professor Hayden White for having spared the time in writing a foreword for my book.

Tehran, Iran Zahid Hussain Khan, MD, FCCM

Contents

Part I Traumatic and Spinal Cord Injury

1 Intracranial Compliance, Traumatic Brain Injury and Management 3
Zahid Hussain Khan and Pooya Kalani

2 Advanced Monitoring in Neurocritical Care: Brain Tissue Oxygen Pressure 27
Rafael Badenes and Armando Maruenda

3 Predictors of Outcome in Traumatic Brain Injury 43
Angels Lozano and Rafael Badenes

4 Brain Death: Understanding the Process of Brain Death Declaration Through Real-Life Case Scenarios 55
Abhijit Lele and Michael Souter

5 Paroxysmal Sympathetic Hyperactivity Following Acute Acquired Brain Injury 69
Parmod Kumar Bithal and Keshav Goyal

6 Spinal Cord Injury 83
K.H. Kevin Luk and Michael J. Souter

7 Aneurysmal Subarachnoid Hemorrhage 97
Pratik V. Patel and Michael J. Souter

Part II Airway and Pain Management

8 Airway Management in Neurosurgical Procedures (Along with Ventilation Management) 109
Isabel Gracia, Anna M. López, and Ricard Valero

9 A New Airway Assessment Classification for Edentulous Patients and Its Potential Role in Neurocritical Care 123
Zahid Hussain Khan and Ramooz Hussain Khan

10 Airway Management in Neurocritical Care 129
Mohammad Jalili

11 Postoperative Pain Management After Craniotomy 139
Ramani Vijayan and Loh Pui San

Part III Neuroanesthesia and Neurocritical Care

12 Anesthesia for Awake Craniotomy 149
Luca Titi, Shaun E. Gruenbaum, and Federico Bilotta

13 TIVA for Neurosurgery 155
Andreia Martins Costa and Francisco Lobo

14 Anesthetic Challenges in Pediatric Craniofacial Surgeries. ... 167
Lucy Chan

15 Acromegalic Patients: Do They Pose Airway Problems? 179
Zahid Hussain Khan and Razmeh Hussain Khan

16 Airway and Fluid Management of Patients in a Prone Position 187
Mathieu Asselin and Orlando Hung

17 Anaesthetic Considerations in Posterior Fossa Surgery 203
Anju Grewal, Nidhi Bhatia, and Sandeep Kundra

18 Anesthesia for Pituitary Surgery..... 213
Fauzia Khan and Faraz Shafiq

19 Venous Air Embolism in Neurosurgical Patients..... 229
S.K. Malhotra

20 Blood Glucose Concentration Management in Neuro-Patients...... 239
Federico Bilotta and Elisabetta Stazi

21 Anaesthesia for Deep Brain Stimulation...... 249
Carolyn Yim Chue Wai

22 Intraoperative Magnetic Resonance Imaging 253
Pui-San Loh and Ramani Vijayan

23 Sedation in Neurocritical Units 259
Rafael Badenes and Mario De Fez

24 Transcranial Doppler and Transcranial Color-Coded Duplex Sonography..... 275
Chiara Robba and Andrea Rigamonti

25 The Conundrum of Postoperative Hematoma in Intracranial Surgery...... 291
Zahid Hussain Khan and Shahid Nisar Khalid

26 Neuroprotection in Neuroanesthesia and Neurocritical Care..... 303
Shaun E. Gruenbaum and Federico Bilotta

27 Sepsis and Septic Shock 317
Piero Ceriana

Index..... 329

Part I

Traumatic and Spinal Cord Injury

Intracranial Compliance, Traumatic Brain Injury and Management

1

Zahid Hussain Khan and Pooya Kalani

1.1 Introduction

Traumatic brain injury (TBI) has been reported to be 50,000 annually in the mean age group of 35 year in the United States alone [1]. Survivors live with the TBI related disabilities. In the contemporary literature, a 20% reduction in mortality has been reported, and this could be attributed to an improvement of our knowledge of pathophysiology of TBI and an advancement in our management strategies. The last decade has witnessed giant strides in monitoring, critical care techniques, indications and timing of surgery, which had an overall favorable impact on the mortality.

The brain is an organ that is exquisitely sensitive to hypoxemia, episodes of hypotension and alterations in the blood pH. TBI ushers in a cascade of events that bring in a change in the body hemostasis which if not corrected urgently can end up in more profound damage to the brain that would be difficult to treat.

The major causes of head injury include road vehicle accidents, falls from heights and sports, etc. Traditionally, head injury has been divided into the primary injury and the secondary injury. The initial assault or impact damage once inflicted is hard to reverse, that, it causes inevitable destruction depending upon the severity of the injury but is preventable if safety measures are adopted and rules of driving and other sports abided.

The impact damage can appear in the form of contusions or lacerations, or else appear as epidural, subdural or intracerebral hematomas when bleeding occurs in the contusions or else several contusions coalesce together because of bleeding occurring within them. The secondary brain damage occurs within minutes to days after the initial insult in the form of hypoxia, hypercarbia, brain edema, brain shift, ischemia and infection which are preventable to some extent, and this aspect is the cornerstone in the management of TBI.

1.2 Intracranial Compliance/ Elastance

The brain is besieged by the rigid skull and an increase in intracranial pressure (ICP) may reduce cerebral perfusion pressure (CPP), and demolish cerebral blood flow (CBF) leading to cerebral ischemia.

The craniospinal axis is essentially a partially closed box containing both viscous and elastic elements. The elastic or its inverse the compliant properties of the container will ascertain as to how much volume can be added to it before the ICP shows a rise.

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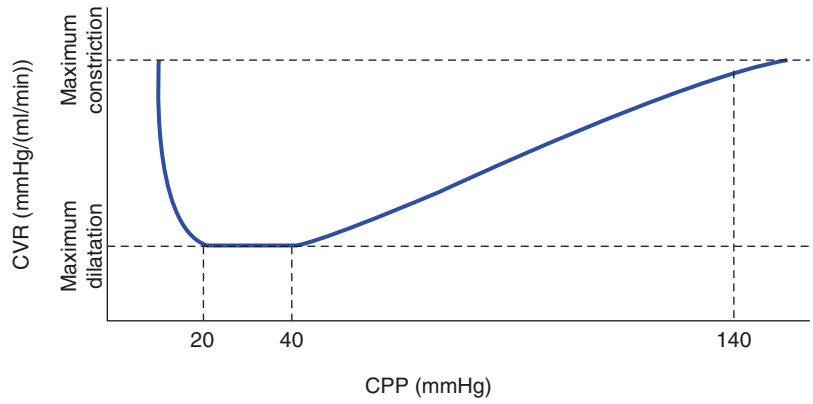

Fig. 1.1 Auto-regulatory relationship between vascular resistance (CVR) and CPP. *CVR* main cerebrovascular resistance, *CPP* cerebral perfusion pressure

Intracranial compliance (dP/dV) is the inverse of compliance. Elastance is sometimes referred to as the volume-pressure response (VPR).

$$\text{Compliance} = \frac{1}{Elastance} = \frac{1}{VPR}$$

Cerebral compliance literally expresses the capability to buffer an intracranial volume increase while buffering a rise in ICP. The auto-regulatory response to any variation in CPP influences the cerebral blood volume (CBV) which is an important determinant of intracranial compliance [2]. The ability of the intracranial compartment to compensate to added volume is an important factor in the development of raised ICP after TBI [3]. Intracranial compliance or its inverse elastance is considered to be index of the volume buffering capability of the brain, and a reduced compliance will eventually lead to increased ICP [4]. Variations in CPP have significant influence upon cerebrovascular resistance (CVR) and on CBV which regulate a constant cerebral blood flow (CBF) (Fig. 1.1).

$$CVR = \frac{MAP - ICP}{CBF}$$

Different methods have been developed to measure cerebral compliance.

Marmarou [2] provided a full mathematical description of the craniospinal volume-pressure correlation and also found a mathematical model of the CSF system for general solution of the CSF pressure. He described nonlinear volume–pressure relationship as a straight line segment relating the logarithm of pressure to volume.

It can be determined as a monoexponential relationship between volume and pressure.

Relationship has been described quantitatively by a pressure-volume index (PVI) which is the notional volume required to raise ICP tenfold. In fact, the slope of pressure-volume relationship determines this index (Fig. 1.2).

PVI can be calculated by the underlying formula:

$$PVI = \frac{dV}{\log \frac{P_0}{P_m}}$$

dV = Volume = milliliters
P_0 = initial pressure
P_m = final pressure

Ordinarily, the PVI measures are obtained by repeated withdrawal and injections of 2 ml and the average PVI is calculated from multiple injections. In ICP rising, fluid injection is not performed and PVI is obtained only from withdrawal of fluid.

Miller and colleagues [5, 6] introduced another craniospinal volume pressure relationship parameter, the volume pressure response (VPR). It is calculated from the ICP response resulting from a rapid bolus injection of saline into the CSF space, as a direct measure, not of compliance, but of its inverse elastance.

The fundamental principles of raised ICP were developed by Monro [7] and Kellie [8]. But

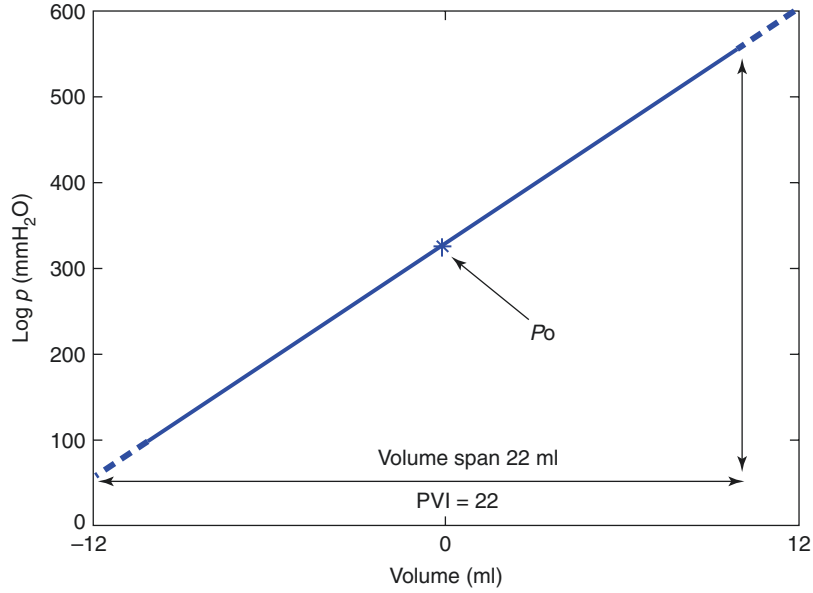

Fig. 1.2 These methods are based upon the manual injection from the CSF space of the patient while measuring the ICP before and after the volume change. PVI is defined as the volume of CSF that would have to be infused to raise the ICP ten-fold. Normal PVI is 26 ml. Less than 13 ml is considered to indicate reduced volume buffering capacity. VPR is defined as the change in ICP with the infusion of 1 ml of CSF. Normal VPR is less than 2 mmHg and greater than 5 ml is considered to indicate reduced volume buffering capacity

this doctrine did not take into account the CSF as a component of the cranial component. The concept of reciprocal volume changes between blood and CSF was introduced by Burrows and was later extended in the early twentieth century [9, 10] to allow for reciprocal changes in all the craniospinal constituents.

The brain floats in the CSF which has an important role. Pascal's principle describes that the transmission of fluid pressure exerted anywhere in a confined incompressible fluid is transmitted equally in all directions throughout so that the pressure variations remain the same. According to this law, all gradients of the ICP within the CNS are equilibrated. Marmarou's mathematical model introduced interrelationship of the static and dynamic process of formation, storage and absorption mechanisms of CSF. Also, Davson [11] has shown the relationship between CSF pressure and cerebral venous pressure. As a result, the steady-state ICP equation developed:

ICP = CSF formation rate × [CSF outflow resistance + venous pressure (pressure in sagittal sinus)].

The production of CSF is balanced by its storage and reabsorption via the sagittal sinus.

Marmarou has extended this hypothesis with mathematical model:

$$R_0 = t_2 * \frac{P_0}{(PVI)\log\left(\left(\frac{P_2}{P_1}\right) * \frac{(P_p - P_0)}{(P_2 - P_0)}\right)}$$

V_0 = Single volume injection
P_0 = Starting pressure
Pp = the peak pressure resulting from bonus volume injection
P_2 = the pressure point on the return trajectory at time T_2 (T_2 = 2 min post injection)
R_0 = out flow resistance

The formula is derivation of an equation for CSF out flow resistance based on a bolus injection technique.

After traumatic brain injury, CSF out flow resistance is one possible parameter of raised ICP.

Mamarou's study has shown that the role of CSF in ICP raising was only about 30%. Therefore, jugular bulb pressure was measured by Marmarou in 1993 [12] and it determines that ICP elevation relates to venous outflow pressure.

Gray and Rosner [13] determined the role of vascular factors in craniospinal compliance.

It revealed that the PVI could be regarded complex function of CPP, reinforcing the idea that the direction of the CPP-PVI relationship