Neurosurgical Anaesthesia

A. R. HUNTER

M.D. F.R.C.S.(Glas.) F.F.A.R.C.S. D.A.

Consultant Anaesthetist to
The Royal Infirmary, Manchester,
Stepping Hill Hospital, Stockport and
The Regional Thoracic Centre,
Baguley Hospital, Manchester

BLACKWELL

SCIENTIFIC PUBLICATIONS

OXFORD

NEUROSURGICAL ANAESTHESIA

A. R. HUNTER

M.D. F.R.C.S.(Glas.) F.F.A.R.C.S. D.A.

Consultant Anaesthetist to
The Royal Infirmary, Manchester,
Stepping Hill Hospital, Stockport and
the Regional Thoracic Centre,
Baguley Hospital, Manchester

BLACKWELL
SCIENTIFIC PUBLICATIONS
OXFORD

FIRST PUBLISHED 1964

Printed in Great Britain in the City of Oxford by
THE ALDEN PRESS
and bound by
THE KEMP HALL BINDERY, OXFORD

DEDICATED TO
THE MEMORY OF
G.J.

ACKNOWLEDGEMENTS

The author is grateful to those who have given permission to reproduce illustrations from other works in this book. Figs. 2, 3, 4, and 13 are by permission of Dr R. G. Reid, Department of Neuroradiology, The Royal Infirmary, Manchester; Fig. 5 is from Blackburn & Lawrie: *Textbook of Surgery* by permission of the authors; Fig. 6 is reproduced by permission of the editor of the *Journal of Clinical Investigation* and the authors; Fig. 16 by permission of Medical & Industrial Equipment Ltd., Figs. 28, 29, 30, 32, 34, 36 and 37 are by permission of the Editor of the *Proceedings of the Royal Society of Medicine*; and Fig. 42 by permission of the authors and the editor of the *Lancet*.

PREFACE

This book is an attempt to put on record the various methods of anaesthesia, which in the course of some 20 years' experience of neurosurgical work, have in the author's view, proved their worth. It is perhaps unfortunate that he is unable to offer controlled series of investigations to prove the various points made. It is, however, a regrettable fact, that the cost of a failure on the part of a neurosurgical anaesthetist must be paid by the patient who is returned to bed with his operation uncompleted and it is simply not justifiable to persist with techniques with appreciable failure rates. This means of course that the basis of development has been a process of trial and error. Fortunately the errors have been fairly few but in the course of these years a great deal has been learned by the author about the brain and its response to drugs, and it is hoped that this experience will be of interest to those working in related fields.

It is hoped too that this book will be to some extent a work of reference and for this reason a fairly extensive bibliography of relevant recent work has been included. For like reason no attempt has been made totally to eliminate the duplication of material, especially in the sections which deal with anaesthesia for particular types of intracranial operations and with the disturbances of the vital functions liable to arise in neurosurgery. Perhaps the specialist reader to whom all sections of the book are of interest will be willing to forgive the reduplication for the benefit of those to whom some chapters are of more importance than others. Also some upsets of the vital centres like hypothalamic disturbance and grosser disturbances of lower brain stem function, though they were common enough once upon a time, seem largely to have disappeared from the author's practice. From casual sentences in the papers of others, however, it would seem that they still occasionally occur. For this reason they too have been mentioned.

The author wishes to place on record his tremendous debt to the late Professor Sir Geoffrey Jefferson to whom this book is dedicated and who read one of the earlier versions of the manuscript. But for

his inspiration and kindly encouragement this book would never have seen the light of day. The author is also indebted to to Mr R.T. Johnson, Mr J.M.Potter and Mr J.E.M.Dutton with whom he has been privileged to work as the anaesthetist member of a team. His thanks are also due to the Departments of Medical Illustration at the United Manchester Hospitals and at Wythenshawe Hospital for their help in the preparation of illustrations and for permission to reproduce them and to the various manufacturers and journals who have provided blocks or photographs. Not least he is indebted to Mrs Ivy Allcock who has typed a large proportion of the manuscript.

A.R.HUNTER

October, 1963

INTRODUCTION

The basis of modern neurological surgery was laid by Harvey Cushing. He began his work as a pupil of Halstead at the Johns Hopkins Hospital in Baltimore and later moved to Boston as Professor of Surgery at Harvard University. Following in the footsteps of his teacher, he developed a technique of meticulous and careful operating and case recording which persists with comparatively few modifications wherever neurosurgical operations are performed in the English-speaking world and in many places outside it.

Neurosurgical anaesthesia also had its beginnings in the Cushing clinic. It was then felt that local was the best method of all. Where unconsciousness had to be produced open ether gave satisfactory results. In the 60 years since Cushing began his work there have been changes in anaesthesia and anaesthetic drugs even more far reaching than those in neurosurgery. Thus though the original principles laid down by Cushing remain unchanged, it is necessary to review the agents and techniques now available by the same method of careful recording and assessment of results to consider what is the bearing of all the modern knowledge and skills in anaesthesia on the handling of the neurosurgical patient. Both Halstead and Cushing began their work on the basis of anatomy, physiology and pathology and it is appropriate that the present work should have the same starting point.

CONTENTS

CHAPTER I

THE PHYSIOLOGICAL PROBLEM

THE INTRACRANIAL PRESSURE

The most important consideration in relation to anaesthesia for neurosurgery is its effect on the intracranial pressure. The reason for this will become apparent later, but for the time being let it suffice to say that fully half the operations of neurosurgery will either be rendered impossible or will be performed at considerable prejudice to the patient's ultimate complete recovery if the activities of the anaesthetist raise the intracranial pressure. The factors which determine the pressure within the skull are therefore of supreme importance in cases of this type.

The intracranial pressure is determined by changes in three variables which are, to a greater or lesser extent, interrelated. These are:

1. The circulation of cerebrospinal fluid through the ventricular system and subarachnoid space.
2. The degree of hydration of the brain substance or, in less exact terms, the presence of or absence of cerebral oedema.
3. The state of the circulation in the cerebral arteries, veins and capillaries, and particularly the cerebral blood flow.

THE CEREBROSPINAL FLUID

The cerebrospinal fluid is formed by the choroid plexuses in the lateral, third and fourth ventricles. It can be regarded as a selective filtrate of the blood plasma though some substances, e.g. bile pigment, never reach it; other components are present in different concentrations in the two fluids. Thus, the bicarbonate and phosphate content of the cerebrospinal fluid is lower as is the concentration of the glucose. The amount of sodium in both fluids is about the same, while the chloride is more concentrated than in the plasma. There is therefore good ground for assuming that the barrier between the

plasma and the cerebrospinal fluid is selective and there is histological evidence to support this conception. Thus the glomerulus of the kidney, which is the organ which characteristically produces a protein-free filtrate of the plasma, is covered by a flat endothelium. The cells of the choroid plexuses, on the other hand, are cubical in shape and resemble much more closely the active cells of a secreting gland. The whole problem is, however, complex and its full bearing on the care of the neurosurgical patient has not as yet been elucidated.

THE CONSTITUENTS OF THE CEREBROSPINAL FLUID

The normal constituents of the cerebrospinal fluid are set out in Table 1. These are of course subject to alteration in disease states. Most of these conditions interest mainly those concerned in the

TABLE 1

Some constituents of the cerebrospinal fluid and blood—modified from *Documenta Geigy*

	Cerebrospinal fluid	*Blood*
Protein	24–40 mg/100 ml	6.0–7.4 g/100 ml
Urea	10–30 mg/100 ml	19–32 mg/100 ml
Glucose	45–80 mg/100 ml	75–91 mg/100 ml
Sodium	141 ±6 mEq/l	134–154 mEq/l
Potassium	2.96 ±0.45 mEq/l	4.1 ±0.3 mEq/l
Calcium	2.45 ±0.45 mEq/l	2.2 ±2.6 mEq/l*
Chloride	115–135 mEq/l	100–107 mEq/l
Bicarbonate	24 mEq/l	24 mEq/l

* Ionized calcium.

diagnosis of disease and in fact the changes are largely related to changes in the cell count. Rise in the protein content, however, fairly regularly signifies that active disease or tumour is in contact with the cerebrospinal fluid somewhere. If the clinical picture suggests disease affecting the spinal cord the lesion is likely to be a tumour blocking the subarachnoid space in whole or in part. If the symptoms relate to the posterior cranial fossa an acoustic neuroma is likely to be responsible. If, however, the cause of the trouble is likely to be above the tentorium cerebelli, it may very well be a vascular meningioma.

THE CEREBROSPINAL FLUID CIRCULATION

The secreted cerebrospinal fluid passes from the lateral ventricles through the foramina of Monro to the third ventricle and thence by the iter or aqueduct of Sylvius to the fourth ventricle. It escapes into the general subarachnoid space through the foramina of Luschka and Magendie which lie in the roof of the last named cavity under the overhang of the cerebellar hemispheres. Most of the cerebrospinal fluid then passes upwards in the subarachnoid space of the posterior

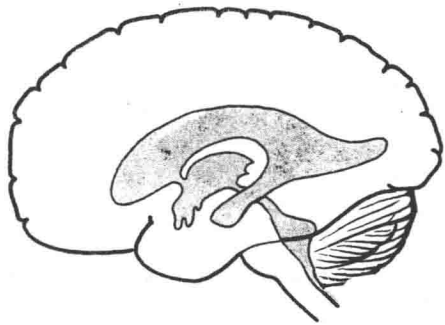

FIG. 1. The ventricular system of the brain.

fossa, either by way of the anteriorly placed cisterna pontis or by the cisterna ambiens. It then gains access to the supra tentorial compartment of the skull. Part of the fluid then travels directly upwards over the cerebral hemispheres in the sulci to the arachnoid granulations alongside the superior longitudinal sinus. The remainder passes through the cisterna chiasmatis and finds its way to the same granulations either by passing round the genu of the corpus callosum into the space between the cerebral hemispheres or out along the Sylvian fissure. The bulk of the cerebrospinal fluid is absorbed into the blood stream through the arachnoid granulations or Pacchionian bodies. Direct absorption into the veins probably occurs also. The forces producing absorption are not known for certain but the sponge-like structure of the granulations suggests a unidirectional valve. The remainder of the cerebrospinal fluid passes down alongside the spinal cord. Some is absorbed into the bloodstream around the nerve roots and some escapes along the dural sleeves extra-spinally.

The normal total amount of the cerebrospinal fluid is about 150 ml in an adult. Of this, some 20–25 ml is contained in the lateral ventricles and the remainder distributed throughout the subarachnoid space around the brain and spinal cord.

THE CEREBROSPINAL FLUID CIRCULATION IN INTRACRANIAL DISEASE

One of the earliest effects of intracranial tumours or for that matter, of any local change in the brain bulk, is the production of deformity of the ventricular system. Almost without exception, tumours in the cerebral hemispheres press on the ipsi-lateral ventricle and flatten it. In addition the whole ventricular system is often bodily displaced and compressed. The result is that ultimately the amount of cerebrospinal fluid in the ventricles is greatly reduced and the cavities may become almost slit-like. Under such circumstances the total amount of ventricular cerebrospinal fluid may be reduced to as little as 5 to 10 ml. The reduction in volume and therefore the additional access obtainable by ventricular tap in these cases, especially if it is done on the same side as the tumour, is small and the anaesthetist must take special pains not to make it even less by increasing the brain bulk.

By contrast tumours growing adjacent to the narrower parts of the cerebrospinal fluid pathway obstruct them and give rise to a gross dilatation of the lateral ventricles which may come to contain 100 to 150 ml or even more cerebrospinal fluid. This dilatation of the ventricular system can occur in a remarkably short time and rapidly growing lesions like cerebellar abscesses may, if they press on the aqueduct of Sylvius, produce obvious hydrocephalus within a week. With such large lateral ventricles the surgeon is able to obtain a very considerable reduction in the intracranial tension by ventricular tap and removal of fluid. In such cases therefore it is permissible to use a technique of anaesthesia which may add a little to the intracranial pressure provided it offers other advantages.

VENTRICULOGRAPHY

It is sometimes necessary where the exact localization of an intracranial tumour is in doubt, to demonstrate by radiological means the type of deformity of the ventricular system which it has produced. Two methods are currently employed for this purpose. A small quantity of cerebrospinal fluid may be removed and an equal volume (1–2 ml) of myodil, a radio-opaque oil which is less heavy and irritant

than lipiodol, injected into the ventricle. This material has some irritant effect on the meninges but the rise in intracranial pressure it produces is usually too small to be of any serious significance. The essential drawback to this type of ventriculography is that not more than a small part of the ventricular system can be made visible at one time. It is, however, most valuable for demonstrating lesions of the third and fourth ventricles and of the iter of Sylvius.

The other material which is used to render the ventricles visible on a radiograph is air. For this purpose it is necessary to remove some of the cerebrospinal fluid in the lateral ventricles and to replace it with enough air to allow an accurate estimate of the tumour site. The amount of air put in must always be less than the amount of fluid removed. This procedure is almost invariably followed by a marked increase in the intracranial pressure. Indeed, so great is this rise that it is unsafe to induce general anaesthesia in a previously compressed patient for many hours after air ventriculography unless ventricles have first been tapped and the intracranial pressure lowered to safer levels. It is very often necessary to tap the ventricle yet again as a first stage in the operation, so marked and persistent can be the rise in pressure after air ventriculography. Where the intracranial pressure is normal air can be introduced into the lumbar theca; it will then, if the patient is sitting up, pass up into the ventricles and outline them (lumbar encephalography).

DISTURBANCES OF CEREBROSPINAL FLUID FORMATION AND ABSORPTION

The rate of cerebrospinal fluid formation and absorption is unlikely to alter significantly during the short time for which a neurosurgical operation lasts. It is, however, almost certain that the induction of hypotension or hypothermia must be associated with a reduction in the rate of cerebrospinal fluid formation. It may well be too that some of the reduction in intracranial pressure brought about by the administration of hypertonic solutions is due to changes in the dynamics of the cerebrospinal fluid as well as to alterations in the hydration of the brain [7]. Finally, it is possible, though by no means certain, that some of the rise in intracranial pressure which follows ventriculography and encephalography is due to a failure of cerebrospinal fluid absorption because air bubbles block the access of the cerebrospinal fluid to the Pacchionian granulations where it would normally be absorbed.

The whole question of the physiology of the cerebrospinal fluid has gone into the melting pot again in the last few years [1]. Protein is added everywhere throughout the nervous system where there is contact with the circulation. The function of the Pacchionian granulations is not primarily fluid absorption but protein absorption. The purpose of the choroid plexuses is to provide the bulk of the fluid; they do not seem to add any more protein than any other part of the nervous system in contact with cerebrospinal fluid. These ideas call for some alteration in the current views concerning the etiology and pathogenesis of hydrocephalus but until their meaning has been more fully worked out it is probably more satisfactory to think in terms of the older ideas of Weed [2] who regarded cerebrospinal fluid as secreted by the choroid plexuses, circulated through the cerebrospinal fluid pathways and absorbed by the Pacchionian granulations, with the reservation that this is almost certainly an oversimplification.

THE BRAIN BULK

The second factor to be considered in the pressure within the skull is the brain bulk. There are two aspects of this, the brain itself and its blood flow. In considering the first of these it is convenient to regard the brain substance as a sponge with pores of colloidal size surrounded by a semi-permeable membrane through which water alone may find its way with any facility. The total size of the brain will therefore be governed by the amount of hydration necessary to keep it in approximate osmostic equilibrium with the plasma of the blood. If the blood is diluted by the entry of large amounts of water a corresponding dilution will occur within the brain substance with an accompanying increase in its bulk. If, on the other hand, the osmotic pressure of the plasma is raised as it will be by the intravascular injection of concentrated solutions or by simple dehydration, the brain volume will be reduced because the brain loses water to regain its previous osmotic state.

Regular use is made of these facts in neurosurgery. When an intracranial tumour has raised the pressure within the skull to dangerous levels it is possible to obtain in some cases temporary remission of this pressure by the intravenous injection of 75–150 ml of a 50 per cent solution of sucrose [3]. This substance can, however, cause renal tubular damage and is now little used. Concentrated solutions of serum protein (3 or 4 times concentrated) have also been used to

dehydrate the brain [4]. So also have hypertonic solutions of Dextran [5]. These last have, however, proved singularly disappointing because the active polysaccharide subsequently seeps into injured brain and makes oedema more persistent than it would otherwise have been.

Unfortunately, for some reason which is not quite clear, the response of the brain exposed by the surgeon to the injection of these hypertonic solutions was somewhat disappointing, and they were of comparatively little value in improving a situation in which a tight and dry brain was making an operation difficult.

A 30 per cent solution of urea, 'lyophilized' in 10 per cent invert sugar, succeeds where the older hypertonic solutions failed [6, 7]. The intravenous injection of this fluid in a dose of up to 90 g is stated to reduce the bulk of the brain markedly [8]. Indeed it is so active that some fear that it may occasionally cause permanent damage. Where, however, the ensuing electrolyte changes have been followed it seems that, provided the patient was not dehydrated beforehand, no serious upset is likely to occur [9].

Urea succeeds where other dehydrating agents fail because its solutions do not merely increase the plasma osmotic pressure. The active substance also acts as a potent diuretic. In consequence the overall effect does not end with the withdrawal of water from the brain into the plasma. Urea also promotes a markedly increased excretion of the water from the tissues to the exterior. Its effects are therefore much more permanent. In spite of this there is a stage early in its action in which the primary effect is an expansion of the plasma volume. This is very often indicated by a small rise in blood pressure. Increased bleeding from blood vessels which have dilated to accommodate the increased volume regularly occurs and this can be a nuisance during an operation. Further this artificially increased plasma volume must temporarily mask the adverse effects of blood loss on blood pressure. Later, however, when the excess urea has gone from the body taking with it the water required for its excretion the plasma volume is reduced below its normal level and fall in blood pressure is then to be anticipated, often to a lower level than the known blood volume deficit might seem to indicate.

It has been suggested that urea might be very persistent in its effects in hypothermia and might even be responsible for the onset of ventricular fibrillation in hyperventilated patients [10, 11]. These conclusions, however, are based on work on dogs whose temperatures