20.1 Introduction

Many uninformed neurosurgeons have regarded surgery for post-traumatic intracranial hematomas as unrewarding. This pessimism is based on the belief that outcome is determined principally by the magnitude of the initial injury and, therefore, frequently remains poor despite optimal surgery. In fact, management of post-traumatic epidural hematoma is one of the most ‘cost-effective’ of neurosurgical procedures in terms of quality of life and years preserved (Pickard et al., 1990).

It is particularly in those with moderate head injury that hematoma management may make the difference between survival with permanent disability and a good outcome. Intracranial hematoma is by far the most common cause of secondary deterioration after all head injuries and constitutes over 70% of the causes of death in patients who ‘talk and die’ (Reilly et al., 1975; Rose, Valtonen and Jennett, 1976). Emergency surgery for post-traumatic intracranial hematomas may be among the most difficult procedures performed by neurosurgeons because of the frequency of complications such as heavy bleeding and brain swelling yet, because these operations frequently occur at night, it is too often the less experienced surgeons who are delegated to do them. Similarly, decisions regarding removal of hematomas, particularly contusions and intracerebral hematomas, may be extremely difficult, especially so when surgery is prophylactic and intended to prevent deterioration. There have been major changes in the patterns of management of intracranial hematoma in recent years, and this chapter has in mind neurosurgeons in training and aims to provide some guidelines for surgical management.

20.2 Post-traumatic lesions on CT

CT scanning may reveal a number of patterns of intracranial hematoma often associated with cerebral swelling:

- small, scattered hemorrhages associated with diffuse injury;
- isolated, single or multiple deep cerebral hematomas;
- intraventricular hemorrhage;
- subdural or extradural ‘extra-axial’ hematomas.

Each of these, particularly intraventricular hemorrhage, may occur with diffuse axonal injury, ischemic cell damage, contusion or any of the other types of hemorrhage.

Large hematomas occur most frequently at the frontal and temporal poles, often associated with extensive contusions and subdural hematoma (‘the exploded pole’). Swelling around these severe parenchymal injuries may increase in the days following injury and ICP may become more difficult to control.

Marshall has proposed a reproducible and quantifiable ‘CT classification’ for head injury, which is now widely accepted as a method of stratifying head injury in addition to the Glasgow Coma Scale (Marshall et al., 1991; Table 20.1).

Although brain swelling may occur in relation to hematomas, the degree is quite variable and need not relate to the size of the hematoma. Indeed, swelling of a hemisphere or of the whole brain may also occur without CT evidence of bleeding.

Temporal lobe hematomas are prone to causing brain-stem compression at low ICP and with little midline shift.

20.3 Indications for evacuation of intracranial hematomas

Craniotomy is not required in all patients with mass lesions due to severe closed head injury. In the Traumatic Coma Data Bank series only 37% of comatose patients underwent surgery for removal of intracranial hematomas (Eisenberg et al., 1991).
CT is being used earlier and more often in patients who sustain head injuries, even in those with mild injuries, partly because of the medicolegal implications of failure to detect an intracranial hematoma. In consequence, patients with minimal or no neurological signs or symptoms are often found to have intracranial mass lesions. Neurosurgeons must then weigh up the risks and benefits of conservative treatment, knowing that the majority of such lesions will resolve spontaneously, yet a relatively small number will develop raised ICP and secondary brain damage which could have been prevented by prophylactic surgery.

At least 25% of patients with intracranial mass lesions will show clinical or radiological worsening in the first 2–3 days after the injury. This is particularly likely in patients with cerebral contusions, where swelling is the norm rather than the exception.

Recently, several studies have provided guidelines for the management of extradural and subdural hematomas in asymptomatic patients (Bullock, Smith and Van Dellen, 1985; Knuckey, Gelbard and Epstein, 1989; Hamilton and Wallace, 1992; Pozzati and Tognetti, 1986; Mathew et al., 1993). Management decisions in individual patients must take into account a number of factors, such as extracranial injuries, the age of the patient and the presence of associated intracerebral contusions or hemisphere swelling. Unfortunately, such management decisions are even more difficult in patients with intraparenchymal lesions such as contusions and intracerebral hematomas.

20.3.1 GUIDELINES FOR INITIAL CONSERVATIVE THERAPY

Non-operative therapy should only be considered:

- in patients who are fully conscious;
- when the extra-axial mass lesion is the single dominant lesion, i.e. there should not be multiple contusions or potentially significant contralateral mass lesions (which may be preventing midline shift);
- when there are no features of mass effect such as midline shift greater than 3 mm, or basal cistern effacement (Bullock and Teasdale, 1991).

In the conscious patient with an acute subdural hematoma where points 1–3 above are fulfilled and the lesion is less than 10 mm at its thickest point, conservative therapy has been shown to be successful in most cases (Mathew et al., 1993). The subdural hematoma will usually resorb within 1 month, although there are occasional instances of chronic subdural hematoma formation.

Similarly, deep-seated interhemispheric or tentorial subdural hematomas and small extradural hematomas in a stable, conscious patient may not need surgical evacuation.

Indications for surgical removal are:

- presence of a mass lesion >40 ml;
- in the conscious, communicating, non-ventilated patient:
  - decline in conscious state;
  - development of focal signs;
  - severe and especially worsening headache, nausea or vomiting;
- in the unconscious, non-communicating, ventilated patient:
  - decline in neurological state; (this may only be indicated by the development of brain-stem signs);
  - increase in ICP, e.g. >25 mmHg;

Either of these developments should lead to an urgent CT scan.

- increase in hematoma size on CT scan (Galbraith and Teasdale, 1981).

20.3.2 INTRACEREBRAL HEMATOMAS AND FOCAL DEFICIT

There has been debate about the benefit of removing an intracerebral hematoma causing a focal deficit but
without clinical effects of raised pressure or significant midline shift. Experimental intracerebral hemorrhage produces ischemia in the surrounding brain, apparently due to blood constituents as well as compression (Jenkins et al., 1990). However, clot removal does not relieve the ischemic neuronal damage, since this has already occurred as a result of the clot. Removal should therefore be based on evidence of harmful mass effect. When mass effect is present, removal has been reported to lead to a more rapid resolution of neurological deficits (Levinthal and Stern, 1977).

### 20.4 Techniques for craniotomy

#### 20.4.1 Preoperative Preparation

In general, cranial surgery should not be performed until a stable blood pressure and adequate lung function, confirmed by blood gas analysis, have been achieved (Chapter 17).

However, in a patient who is comatose, particularly if there has been a documented deterioration in conscious level or development of focal signs, removal of the hematoma is a matter of great urgency. The patient should be intubated and hyperventilated, if this has not already been done, and mannitol 1 g/kg should be given immediately as the patient is taken to the operating theater.

Prior to, or during the preparation for craniotomy, the following ‘checklist’ should be completed:

1. blood to laboratory for:
   - (a) cross matching (2 units of whole blood);
   - (b) coagulation studies
     - (i) prothrombin index
     - (ii) partial thromboplastin time
     - (iii) platelet count;
   - (c) blood gas analysis;
   - (d) routine full blood count and electrolytes;
2. X-rays of chest and cervical spine (or keep cervical spine in collar);
3. consent for surgery;
4. Foley catheter in bladder;
5. two large bore peripheral i.v. lines, or one peripheral and one central line (maintaining CVP > 5 cmH₂O);
6. arterial catheter;
7. protection of both eyes from fluids and pressure;
8. adequately secured cuffed endotracheal tube.

The head should be placed on a horseshoe or doughnut headrest, turned to place the operative side uppermost and slightly elevated above the level of the heart. A sandbag placed beneath the ipsilateral shoulder makes turning the head easier. Pressure points should be carefully padded. Unless deterioration is rapid, the scalp should be shaved and prepared with povidone-iodine as for any other intracranial procedure. The drapes can be stapled into place to prevent them becoming dislodged if the head has to be turned or moved during the procedure. Antibiotics, anti-convulsants and mannitol are used as required. Strict attention to anesthetic techniques is vital to avoid hypercarbia and further elevation of intracranial pressure.

#### 20.4.2 Exploratory Burrholes

With the wider availability of CT, the necessity for exploratory burrholes is declining. They will be required only very rarely in hospitals with a CT scanner; however, they may be life-saving in rural locations where transfer to a CT-equipped facility may involve long delays.

The use of exploratory burrholes implies that confirmation of the position or even the presence of a hematoma is lacking. It is therefore important to have access to the whole head and to explore all likely sites bilaterally before discarding the diagnosis of intracranial hematoma. The extent of this exploration will be determined at least in part by the experience and skills of the operator, given that this will frequently be a non-neurosurgeon working in less than ideal circumstances. It should always be possible to obtain neurosurgical advice by telephone and this should be encouraged. Specific guidelines for the management of head injury in remote locations in Australia have been established by the Neurosurgical Society of Australasia.

(a) Technique

The patient is placed supine on a horseshoe or doughnut headrest. The whole head is shaved, prepared and draped to allow access to both frontal, parietal and temporal areas. The site for the initial burrhole is determined according to the most likely site of the suspected hematoma. If a dilated pupil is present, it will usually be ipsilateral to the hematoma. The next most valuable localizing feature is a hemiparesis, which will usually be contralateral to a hematoma. If a fracture is present it is most likely to overlie an extradural hematoma. It must be stressed that none of these signs is absolute and if no hematoma is found on the suspected side, the other side should be explored in all cases.

Unless a fracture is present in a different location, the first burrhole should be temporal, as this is the site of most extradural hematomas. It is frequently written that a burrhole placed as little as 2 mm away from the edge of an extradural hematoma will fail to identify it, and this tends to discourage the inexperienced doctor in a difficult situation. The temporal burrhole is not
intended to be placed in any position to that degree of accuracy. It should ideally be just above the zygoma and approximately one finger breadth in front of the tragus. The head is turned away from the side of exploration if the cervical spine is normal or the patient is placed in the lateral position in a suitable collar if there may be a cervical spine fracture.

A vertical scalp incision is made as shown in Figure 20.1. The scalpel is used to cut directly through the temporalis muscle down to the skull. To prevent injury to the facial nerve the inferior limit of the incision is the zygomatic arch. Bleeding will be encountered from the superficial temporal artery but this can be easily controlled with diathermy or ligation after a retractor is placed. The peristeum and temporalis muscle are scraped off the temporal bone with a sharp periosteal elevator and a self-retaining retractor is inserted to hold back the muscle. A burrhole is drilled, using the perforator until an irregular wobbling is felt and there is a perforation through the central part of the hole that can be palpated with forceps (Figure 20.2). Through this small hole, the surgeon may see either dura or extradural blood, but for a confident diagnosis the hole needs to then be enlarged with the burr to complete the hole, but only if the perforator has breached the inner table. The temporal bone is usually quite thin (2–3 mm), whereas the bone in the frontal and parietal areas may be more than 10 mm thick. If an extradural hematoma is present, the hole will need to be enlarged to allow removal of the clot. For an experienced operator in a well-equipped site this will usually involve a craniotomy; for an inexperienced operator or when facilities are limited a craniectomy is often more appropriate. If no abnormality is found at the temporal burrhole, further burrholes are performed in the ipsilateral frontal and posterior parietal areas. The technique is the same but the bone will be thicker. The scalp incisions should be made in such a way that they can easily be converted into a ‘question-mark’-type flap (Figure 20.3).

If all three burrholes on one side are negative, the process should be repeated on the opposite side. Opinions regarding opening of the dura vary. It is very difficult to determine whether a significant intradural hematoma exists without first opening the dura. The presence of blue, bulging dura seen through a burrhole is a very poor diagnostic feature. Following
trauma, the brain is often swollen and frequently there is a thin rim of acute subdural blood which is of little significance. Inexperienced operators performing exploratory burrholes for the first time will often mistake this for a significant acute subdural hematoma and may be dissuaded from looking elsewhere, thereby missing a more important hematoma. We believe it is better to complete the standard six burrholes to exclude an extradural hematoma before opening the dura in search of an acute subdural hematoma. The dura is opened by gently incising the outer layers with a number 15 blade, picking these up with a sharp hook to lift the dura off the brain before completing the incision. The dural edges can then be picked up with forceps and a cruciate incision can be completed. The dural edges are diathermied, taking care to avoid any underlying cerebral vessels. A thin layer (2–3 mm) of blood is common and should not be mistaken for a significant acute subdural hematoma. If a thicker layer of blood is encountered, it should be exposed by performing a craniotomy or craniectomy, depending on the circumstances.

20.4.3 EXTRADURAL HEMATOMA

(a) Craniectomy

Although craniotomy is preferable when skills and conditions permit, craniectomy is often used as a means of extending the exposure after exploratory burrholes. Initial evacuation should be performed rapidly through the craniectomy in order to relieve brain-stem pressure, then either a formal craniotomy or a more extensive craniectomy may be performed, the hematoma evacuated and the bleeding point secured.

The technique is simple. A rongeur is used to nibble bone away and expose the entire hematoma. When the bone is very thick, it is sometimes easier to make several burrholes and nibble the bone away between them. The dura will have already been separated from the inner table of the skull by the hematoma and is not prone to injury. At the margins of the hematoma the dura will be adherent to the bone and should not be stripped any further. If there is a bleeding point on the dura it should be coagulated with bipolar diathermy or, if this is not available, unipolar diathermy may be used on a low setting. Bleeding from the edges of the wound may be troublesome and difficult to stop with diathermy. It is best to pack a little Surgicell or Avitene (postage-stamp size) under the bone edge and then ‘hitch up’ the dura to surrounding pericranium with fine sutures (Figure 20.4). It is however, important not to traumatize the underlying brain with these sutures, and this may be achieved by first lifting the dura off the brain with forceps, or opening the dura to allow air or saline to lift the dura from the brain. The bone fragments should be left out, and the wound closed in layers with a suction drain in the subgaleal (extradural) space for 12 hours. One should not be deterred by the amount of bone that is removed since an elective cranioplasty is easily performed when the patient has recovered.

(b) Craniotomy

When the site of the hematoma has been determined preoperatively by CT scan, the operation of choice will usually be a craniotomy. It is helpful to transpose the position of the hematoma on to the CT scout film, as unusual gantry angles may be confusing (Figure 20.5). Most extradural hematomas are temporal and can be adequately removed through the standard ‘question mark’ trauma scalp flap (Figure 20.3). More posterior hematomas may be approached through an inverted horseshoe flap (Figure 20.6). Frontal and subfrontal hematomas should be exposed through a bicoronal flap (Figure 20.7) to avoid an unsightly midline forehead scar.

The patient is positioned with the head in a horseshoe headrest and turned to bring the operative site uppermost. (A three-pin headrest may be used if the location of the hematoma is established.) The scalp is shaved and prepared with povidone-iodine. Depending on the urgency of the situation, the whole scalp flap can be turned down initially, or a small incision can be made and a burrhole craniectomy performed to allow rapid decompression, before completing the craniotomy. In most cases it is more efficient to quickly turn down a myocutaneous scalp flap followed by a free bone flap. The use of a high-
speed drill system permits this to be done just as quickly in skilled hands as hand-drilling a single burrhole. A small hole is drilled in the thin temporal bone or elsewhere depending on the location of the hematoma. A craniotome cutting attachment is then used to complete the craniotomy. The risk of dural damage is small, especially in the presence of an extradural hematoma. The medial edge of the bone flap is taken no closer than 2 cm from the midline to avoid injury to the superior sagittal sinus and its draining veins. Unless the CT dictates otherwise, the inferior edge of the bone flap is taken well down into the temporal region to allow clear access to the middle fossa.

After removing the bone flap, the hematoma is sucked and irrigated away. Bleeding dural arteries and veins

**Figure 20.5** The extradural hematoma seen in the axial scans in (a) has been measured and transposed on to the corresponding scout film (b). This may be of considerable assistance in planning the site for craniotomy.
are then coagulated with bipolar diathermy. Bleeding bone edges are controlled with bone wax. Hemostatic agents such as Surgicell or Avitene can be used around the margins of the craniotomy to control venous bleeding, which often seeps out from just beneath the bone edge. The addition of dural ‘hitching’ sutures also helps to control this bleeding and prevent recurrent hematoma formation.

The bone flap is replaced and fixed with mini-plates or stainless-steel wire if the brain is not swollen, and suction drains are used in the extradural and subgaleal spaces for 12 hours. The muscle and galea are closed with Vicryl and the skin with staples. In patients who have sustained a significant primary injury (that is all who were in coma), an ICP monitor is inserted at the end of the procedure. When brain swelling occurs the dura becomes tense and it should always be opened to exclude an underlying subdural hematoma.

20.4.4 ACUTE SUBDURAL HEMATOMA

The treatment of acute subdural hematoma follows the same principles as extradural hematoma, although the prognosis is much worse (60% die or survive with a bad outcome – Chapter 18). The affected area is exposed by craniectomy or preferably craniotomy as described for extradural hematoma, taking care to use all means available to reduce ICP first (see Chapter 18). The dura is opened using a sharp hook and scalpel, revealing dark, clotted blood under pressure. For the inexperienced, it is preferable to make several slits in the dura without joining them together (Figure 20.8) as this will minimize herniation of the brain, which can be dramatic and may even prevent closure of the scalp. If a dural flap is used, it should be based medially to avoid damage to the superior sagittal sinus and the draining veins running into it (Figure 20.9). The hematoma is removed by suction and irrigation, taking care not to use suction beyond the limits of the craniotomy in areas that are not directly visible and accessible. Often there will be no obvious bleeding point. Bleeding from a contused cortical

Figure 20.6 An ‘inverted horseshoe’ flap may be more appropriate than a ‘question mark’ flap for hematomas that are positioned more posteriorly.

Figure 20.7 A bicoronal or ‘aneurysm’ flap for a frontal hematoma avoids a visible forehead scar.

Figure 20.8 To minimize brain herniation an acute subdural hematoma may be evacuated through several small dural incisions rather than one large dural flap.
surface can be controlled with bipolar diathermy or Gelfoam. This may not be easy, especially if the brain is swelling rapidly.

An attempt should always be made to identify a parasagittal venous bleeder, if no cortical source of bleeding is found. The medial cortex should be very gently depressed, while irrigating parasagitally. Raising the venous pressure may reveal the bleeder. This is best done by asking the anesthetist to increase inflation pressure for three or four breaths to 40 cmH₂O. If a bleeding parasagittal vein is found, it should be coagulated with bipolar diathermy. If the vein is avulsed from the sagittal sinus, bleeding may be very difficult to arrest, requiring application of ‘ligaclips’ or a muscle patch for tamponade. Failure to seek a parasagittal bleeding source (present in 40–50% of subdural hematomas) may account for the high incidence of recurrent hematomas in some series (Richards and Hoff, 1974).

Contused and necrotic brain is best removed with suction and gentle irrigation, with consideration of partial frontal or temporal lobectomy when the damage is severe. This should not be undertaken by non-neurosurgeons, who should only remove damaged brain.

After removal of the hematoma the dura is closed. This may require a duroplasty, using an insert of temporalis fascia, pericranium or other dural substitute such as lyophilized bovine dura or woven collagen. Commercial forms of human lyophilized dura are no longer available in many countries because of the known cases of transmitted Creutzfeldt–Jakob disease (Will and Matthews, 1982; Simpson et al., 1996).

It is generally preferable to replace the bone flap, as the advantages of decompression in this situation are outweighed by the disadvantages of cerebral herniation through the wound with consequent venous and even arterial compromise. The bone may be secured with mini-plates or stainless-steel wire unless brain swelling is present, in which case the bone is best left ‘free’ and secured only by a few pericranial sutures to allow expansion. A suction drain is placed in the subgaleal space with or without another in the extradural space (if the dura has been closed). The scalp is closed in two layers and an ICP monitor is used routinely.

20.4.5 POSTERIOR FOSSA HEMATOMAS

Suspicion of a posterior fossa hematoma should mandate rapid transfer to a neurosurgical facility, or a neurosurgical retrieval team (Chapter 15). The major concerns are rapid onset of CSF obstruction and brain-stem compression, and air embolism during surgery due to venous sinus injury. The patient is positioned prone with the head fixed in a three-pin headrest. The head should be slightly elevated to reduce venous bleeding but not so much as to promote air embolism. A midline incision and standard suboccipital craniectomy are performed. It is important to continue the decompression through the rim of the foramen magnum, as postoperative swelling in the posterior fossa may cause death by brain-stem compression. If the trauma has already involved a sinus, bleeding should be controlled by tamponade with muscle, Gelfoam, Surgicel or similar substances while carefully elevating the head. An esophageal stethoscope and end-tidal CO₂ measurements should be used to detect any air embolism. Many extradural hematomas extend both above and below the level of the tentorium, overlying the transverse sinus. In these cases, a bridge of bone should be left over the sinus to allow the dura to be tented up and tamponade bleeding sites.

20.5 Specific surgical problems

20.5.1 INTRAVENTRICULAR HEMORRHAGE

Intraventricular hemorrhage usually occurs in the setting of a severe diffuse injury with other CT evidence of parenchymal injury. In about 40% of cases there is no evidence of an intraparenchymal source of the bleeding, although there will often be an injury to the corpus callosum or septum pellucidum (Sato et al., 1987). Hydrocephalus is uncommon (Christie, Marks and Liddington, 1988; Jayakumar et al., 1990) but when it is present, raised ICP should be treated by ventricular drainage. Mortality is high in most series but tends to reflect the GCS on admission and hence the severity of the primary injury (Chapter 10).
20.5.2 INTRACEREBRAL HEMATOMAS

Some 88% of patients with intracranial hematomas after trauma were found to have raised ICP postoperatively (Miller et al., 1981). When control of ICP by all available medical means fails then removal of contusions may be a highly effective form of ICP control, especially when accompanied by a large craniotomy and duroplasty. A swollen and contused temporal pole may cause early tentorial herniation and brain-stem compression, even at surprisingly low ICP in the sedated, ventilated patient.

20.5.3 SURGICAL DECOMPRESSION FOR BRAIN SWELLING

Decompression by large craniotomy has been advocated in several older studies when other methods of controlling ICP have failed (Kjellberg and Prieto, 1971; Ransohoff et al., 1971, Britt and Hamilton, 1978). Although all these older clinical (Cooper, Rovit and Ransohoff, 1976; Venes and Collins, 1975), and experimental studies (Cooper et al., 1979) suggested no benefit, and indeed worsening of brain edema following craniectomy, there are many more recent anecdotal experiences in many neurosurgical centers where decompressive craniectomy has been successful. Gaab et al. (1990) proposed indications for large unilateral or bilateral fronto-temporo-parietal decompression in selected trauma patients. This was considered when medical treatment had failed in patients under 40 years, with initial GCS of 7 or more and evidence of clinical deterioration, but without the signs of brain-stem failure. In their report, 30 of their 37 carefully selected patients made good recoveries or had only moderate disability. Alexander, Ball and Laster reported on the application of the more limited surgical approach used by Cushing. In patients who did not respond to medical treatment, they performed a subtemporal decompression, leaving dura open and resecting the anterior temporal lobe if it was damaged. (Alexander, Ball and Laster, 1987).

One of the well known hazards of craniotomy for brain swelling is herniation through the craniotomy. Hence, large craniotomies have generally been advocated. Bifrontal craniotomies should be made so that the inferior margin is cut low across the supraorbital margin, and they should extend well posterior so that the brain can expand forward and not herniate against the bony margin. For effective decompression the dural envelope must be enlarged. The dura should be opened widely, hinged medially and the falx and sagittal sinus divided as low as possible taking care to preserve draining veins whenever possible. An augmentation duroplasty is used to cover the brain.

At the Medical College of Virginia, a wide decompressive craniectomy with duraplasty and bone-flap removal is performed for ICP control under the following circumstances:

- ICP control not achieved with maximal therapy using mannitol, ventricular drainage, pressors, moderate hyperventilation ($P_{\text{a}CO_2} = 32 \pm 2\text{ mmHg}$) and moderate hypothermia ($32 \pm 2^\circ\text{C}$);
- pupils not fixed and dilated;
- preserved brain-stem responses and central conduction time on evoked potential testing.

Decompression is used before barbiturate therapy, in view of the propensity for barbiturate therapy to lower CPP and worsen outcome (Figure 20.10).

20.5.4 LOBECTOMY FOR BRAIN SWELLING

Surgery for removal of swollen and contused brain is more controversial. It frequently does not control ICP and more than 80% of such patients may still have raised ICP postoperatively (Miller et al., 1981). It is therefore better to control ICP by medical means initially, removing contusions only when this treatment has failed (Miller, 1992). However, as noted earlier, a swollen and contused temporal pole may cause tentorial herniation and brain-stem compression at quite low ICP and, in the sedated, ventilated patient, the only (late) sign of this may be pupillary dilatation.

Removal of contused and swollen brain to control intracranial pressure may be beneficial when other means of control have failed. Litofsky et al. (1994) found this to be so in patients under 40 years with higher initial GCS scores. In this small retrospective report 55% of patients had a ‘good’ or ‘moderately disabled’ outcome and there was no increase in vegetative survivors. Lobectomy aimed to remove damaged brain and to avoid eloquent areas (Figure 20.11).

20.5.5 INTRAOPERATIVE BRAIN SWELLING

As noted above, removal of acute hematomas may be followed by severe brain herniation. The brain herniating through the craniotomy may occlude surface veins and even rupture against the bony–dural margins. This is most commonly seen with acute subdural hematomas and may be anticipated when the preoperative CT scan shows brain swelling in addition to the hematoma (Bullock and Teasdale, 1991). There are several potentially remediable causes that must be considered urgently before concluding that the swelling is simply a reflection of a non-survivable injury. The surgeon should quickly establish with the anesthetist that ventilation has not become impaired (e.g. by pneumothorax, endotra-
cheal tube dislodgment or occlusion) and that the patient is hyperventilated. Secondly, a hidden hematoma, either intracerebral or beyond the craniotomy margin, may have developed. An intracerebral hematoma is possible if there is a cerebral contusion or ‘exploded pole’. Acute hydrocephalus is uncommon but should be excluded by attempting to cannulate the ipsilateral frontal horn. Intraoperative ultrasound is valuable in seeking and excluding these events. Contralateral hematomas occasionally develop or enlarge after removal of the major hematoma. This possibility should be considered particularly if there is a contralateral fracture or small hematoma on the preoperative CT, but indeed has been reported if neither of these features is present.

20.5.6 MANAGEMENT OF DIFFUSE INTRAOPERATIVE BLEEDING

(a) Prevention

No matter how urgent the need to evacuate a hematoma, it is imperative that a fixed surgical routine be followed in order to avoid excessive bleeding. After marking out the large craniotomy flap required to evacuate an acute subdural hematoma or intracerebral contusions, a burr hole should be made and the hematoma should be decompressed acutely. This allows the surgeon to open the remainder of the craniotomy in a controlled fashion, securing the bleeding points in the scalp with diathermy, scalp clips or hemostats. Bleeding points in the temporalis

Figure 20.10 A 22-year-old male was ‘car surfing’ when he fell from the top of the car, which was travelling at about 25 km/h. GCS on admission was 10. CT scan showed bifrontal contusions, obliteration of the basal cisterns and an occipital fracture. He was intubated, ventilated and monitored. ICP stayed above 30 mmHg, despite medical treatment, excluding barbiturate therapy. CT scans showed increase in the size of the frontal contusions. A bifrontal decompressive craniotomy without lobectomy was performed. He recovered with a mild short-term memory impairment. (a) Preoperative scan showing bifrontal contusions and a ‘tight’ brain. (b) Scan 2 weeks after decompressive craniotomy. (c) Following replacement of the bone flap 1 month later.
muscle are coagulated. Several units of blood may be lost from badly secured scalp edges.

(b) Management

Profuse and diffuse bleeding from brain and meningeal surfaces almost always indicates a significant coagulopathy. Blood should be sent intraoperatively for coagulation studies, and a transfusion of fresh frozen plasma and/or whole blood should be given while awaiting the results of these studies. Diffuse hemorrhage may be accompanied by massive brain swelling and herniation through the craniotomy. The surgeon is then faced with the prospect of worsening the bleeding by performing a temporal or frontal lobectomy in order to control the brain swelling. A management checklist should be followed before proceeding with lobectomy.

1. Check the coagulation studies.
2. Transfuse blood and fresh frozen plasma as appropriate, usually 6–10 units.
3. Consider intraoperative ultrasound to detect the development of an intraoperative hematoma, either intracerebral, epidural or subdural on the same or opposite side.
4. Optimize cerebral perfusion pressure. Sometimes increasing CPP intraoperatively to a mean of 80 or 90 mmHg will help to control brain swelling by promoting vasoconstriction if the patient has intact autoregulation. Clearly, however, it is equally possible that this maneuver will worsen the brain swelling if the patient does not autoregulate. (These patients almost always die.)
5. Give thiopental 250 mg–1 g in incremental doses, to suppress any cerebral electrical activity (Chapter 19). CPP must be maintained at about 70 mmHg, using vasopressor agents if necessary.
6. Give more mannitol to a dose of 1 g/kg and lower the $P_{a}CO_2$ to approximately 28 mmHg by increasing ventilation.
7. Occasionally, massive brain swelling may be caused by an intraoperative pneumothorax, which raises inflation and ventilatory pressures and hence intracranial venous pressure. The chest should, therefore be auscultated and, if necessary, X-rayed, during the surgery.

It is usually advisable to wait 15 minutes to assess the effect of these measures before proceeding. If there is no improvement, or if bleeding is ongoing, it is usually necessary to perform a lobectomy. It is important to emphasize that, for any chance of success in this desperate situation, the surgeon and a highly skilled anesthesiologist must work closely together throughout and necessary assistants should be called in.

The best course of action is to perform a swift and generous lobectomy where the damage is greatest, avoiding where possible the speech areas in the

Figure 20.11  A 19-year-old pedestrian was struck by a car travelling at high speed. GCS on admission was 3. CT scan showed a basal ganglionic hematoma with 1 cm midline shift. ICP was 26 mmHg. The clot was partially removed by craniotomy but ICP rose to 30–40 mmHg over the next 24 h, despite medical therapy, excluding barbiturates. A frontal lobectomy was performed and ICP and CPP were well controlled thereafter. (a) CT on admission showing a deep intracerebral hematoma with 1 cm midline shift. (b) Following partial frontal lobectomy.
dominant hemisphere. It may be necessary to remove bone very rapidly by extending the craniotomy with a craniotome or rongeur in order to obtain sufficient access for this. Hemostatic adjuncts may include thrombin-soaked gel foam, Surgicel, and peroxidesoaked cotton wool patties.

20.5.7 CRANIOBASAL SKULL FRACTURE

Penetrating open wounds of the skull are not within the provenance of this book, but craniobasal skull fractures may be internally compound and will be briefly considered. The presence of a dural fistula is indicated by intracranial air, cerebrospinal fluid rhinorrhea or otorrhea or later by meningitis. An intracranial aerocele may expand if the dural tear acts as a one-way valve (North, 1971). Cerebrospinal fluid leakage may occur early, or after some days. It may cease spontaneously, but this may not in itself indicate satisfactory dural healing and the risk of late meningitis may still exist.

We recommend operative repair when:

- CSF leakage persists for 7–10 days;
- CSF leakage ceases, but recurs after 7–10 days;
- there is clinical evidence of a large dural defect indicated by a large aerocele, or by escape of brain tissue through the nostrils;
- meningitis or brain abscess develop after trauma at any time;
- there is radiological evidence suggesting that natural dural repair is unlikely, e.g. a basal skull fracture involving the paranasal sinuses, wide separation of bone edges, a spike of bone or a possible cerebral hernia into the nasal cavity (Reilly and Simpson, 1995).

The incidence of meningitis after cerebrospinal fluid rhinorrhea associated with the Le Fort III fractures appears to be lower than that associated with a vault fracture (O'Brien and Reade, 1984). Indeed, CSF leakage often ceases once the midface fracture is reduced, but the indications for dural repair remain as outlined above. Whether prophylactic antibiotics are protective against meningitis continues to be debated (Brown, 1993; Working Party Report, 1994). Although there is no strong evidence favoring prophylactic antibiotics it is our policy to use intravenous metronidazole, co-trimoxazole and ampicillin if there is evidence of a fistula. These are continued until CSF leakage ceases or for several days after surgical repair is undertaken.

(a) Timing of transcranial repair

The transcranial repair of a dural fistula is usually undertaken electively and therefore does not affect the early acute management of the patient with severe head injury. Acute transcranial repair is often very difficult, because the brain is ‘tight’ and resists retraction and the repair may therefore be inadequate or impossible. If there are facial fractures, a one-stage combined transcranial repair and internal fixation of the facial fractures may be undertaken with the plastic surgery team, usually 5–10 days after injury. Recently, well-localized fractures involving the sphenoid and/or ethmoid sinuses have been approached subcranially via an ethmoidectomy or transnasally aided by endoscopy. This has the merit of not requiring brain retraction and may be performed within days of injury. It is also less likely to cause anosmia when olfaction is still present (Raveh, Redli and Markwalder, 1984).

20.5.8 COAGULOPATHY IN PATIENTS WITH HEAD INJURY

Coagulopathy develops in one quarter to one third of patients who undergo surgery for removal of an intracranial hematoma (Bullock et al., 1990; Stein et al., 1992). In many, this may be mild and self-limiting, but in others it may be sufficiently severe to cause death on the operating table. It is consequently mandatory to determine the coagulation status in every patient who is considered for surgical evacuation of a traumatic intracranial hematoma. Frequently, this will need to be performed as an emergency. Full evaluation should include a prothrombin index, partial thromboplastin time and a platelet count. A template bleeding time, if available, can be used to assess bleeding time. The increasing frequency of coagulation disorders in patients with intracranial hematoma has many causes, but the most important are:

- prior medication with warfarin, heparin or aspirin;
- chronic alcoholism: excess bleeding in alcoholics may be related to reduced levels of coagulation factors; an ethanol-induced decrease in platelet function and nutritional factors may contribute;
- disseminated intravascular coagulation (DIC) due to thromboplastin activator substances derived from brain or from sites of multiple trauma (Simpson, Speed and Blumbergs, 1991);
- coagulopathy associated with massive blood transfusion to replace blood loss from abdominal or limb injuries;
- a pre-existing coagulation problem, e.g. von Willebrand’s disease, thrombocytopenia or hemophilia.
These patients may be predisposed to develop large intracranial hematomas with relatively minor cranial injuries. It is imperative, therefore, to obtain a history from relatives as early as possible, so that relevant pre-existing diseases are known prior to surgery. A marked hyperemic response is frequently encountered in the injured brain, scalp and meninges, and relatively minor abnormalities of coagulation, which might be acceptable for abdominal or orthopedic surgery, can make emergency neurosurgery extremely hazardous. The management goal in such patients should be complete normalization of the hematological parameters by replacing the appropriate factors (see below). Hematological and blood transfusion services should be aware that platelets or fresh frozen plasma may be needed in patients with persistent severe intracranial bleeding, even when coagulation parameters are only mildly abnormal – or indeed normal, in patients with qualitative platelet function disorders.

(a) Qualitative platelet disorders

Chronic aspirin therapy, which is increasingly prevalent in the general population, and chronic alcoholism are the two most common causes of coagulopathy after neurotrauma. When the platelet count is marginally depressed, as in chronic alcoholism, and platelet function is also significantly compromised, the coagulation problem can be particularly severe. Unfortunately, qualitative platelet disorders may be undetectable by standard tests such as bleeding time. More complex measures such as glass bead platelet adhesion tests may be needed and these are not routinely available in the emergency situation that often confronts neurotrauma patients.

20.6 References


