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What Sir William Battle Found: Observations Beyond his Sign

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Eponyms remind us of a singular finding and, incidentally, honor the physicians who figured it out for us, typically without benefit of the diagnostic tools available today. As they say, an eponym is here to stay when it no longer presumes first-hand knowledge of the scientist's work. However, "eponymization" may reduce the entire body of an academic practitioner's work to a single observation. One such example may be the Battle sign. Few know he was a surgeon or that the eponym has nothing to do with battle trauma.

William Henry Battle (1855–1936) became known by his eponym, which indicates a retroauricular ecchymosis resulting from a basal skull fracture (Fig. 1). Battle also earned two other eponyms, *Battle incision*, which is used in appendectomies, and *Battle operation*, for femoral hernia repair. However, his major contribution was in traumatic brain injury (TBI), and few are aware of his key lectures entitled "Three Lectures on Some Points Relating to Injuries to the Head" [1–3]. He delivered these lectures at the Royal College of Surgeons of England. At the time, he was Hunterian Professor of Surgery and Pathology in the Royal College of Surgeons and Assistant Surgeon to the Royal Free Hospital.

His first lecture involved the description and explanation of bloody, watery discharge from the ear and external bruises following TBI. The second lecture discussed cranial nerve involvement with basal skull fractures. The third lecture focused on changes in temperature and the rise of temperature, which he noted, for the first time, was a red flag for intracranial contusions. What can be learned from these observations today?

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Battle's Observations

Battle's lectures [1–3] were published in three subsequent issues of the *British Medical Journal* (Fig. 2). The first lecture showed the position of fractures involving the middle fossa and some that were unaccompanied by bleeding from the ear (Fig. 3). This lecture concerned the sign that became one of his best-known eponyms in TBI. In his description of mastoid ecchymosis, Battle found that the bruise became apparent 3 to 14 days after the traumatic head injury, and his subsequent autopsy research discovered that extravasated blood could move from the suboccipital region to the more superficial tissue:

> *I must ask you to consider this sign somewhat fully, for I consider it under certain circumstances, to be a most important indication that the posterior fossa of the skull is of the fracture [2].*

In his personal series, he noted a large proportion of patients dying from injury to the brain (more than half of the total number), and he felt it necessary to comment that the 11 cases in which fracture of the base was strictly limited to the posterior fossa also had a contused brain, with hemorrhage as the cause of death. He emphasized that hemorrhaging from the ears was the most important sign of fracture of the middle fossa, particularly when bleeding was copious and sustained:

> *In the main these observations confirm the opinion that immediate profuse and continued bleeding from the ear is an important sign of fracture of the middle fossa, and of a fracture passing through the tympanum associated with a rupture of the membrana tympani; also that that fracture has lacerated some of the vascular channels round the petrous bone.*

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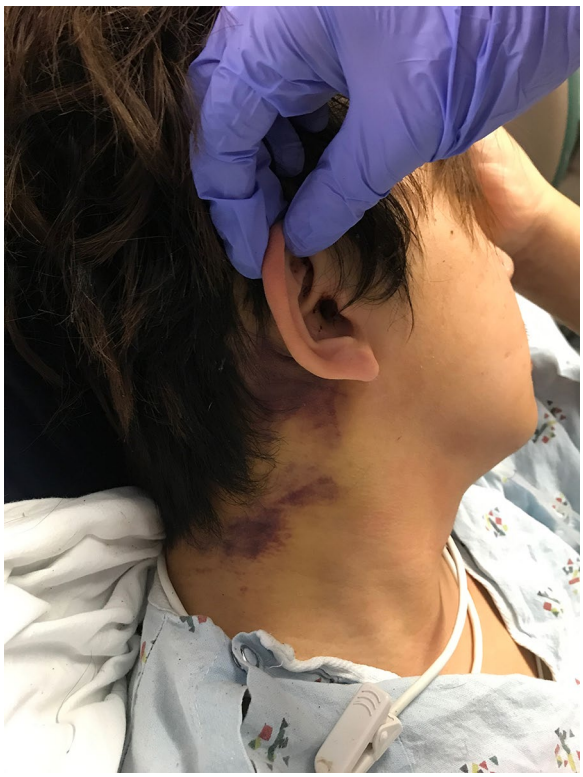


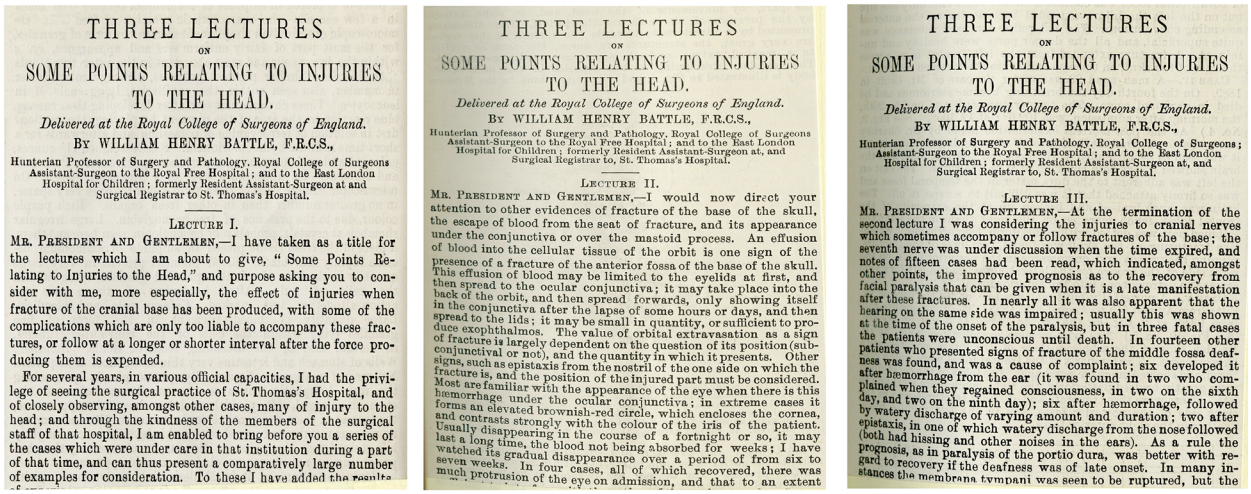
Fig. 1 Battle sign in patient with traumatic brain injury

He also noted hemorrhage from the eyes, and he thought this might have possibly escaped from the lachrymal canals. The patients who presented this symptom were both severely injured. At autopsy, one had a fracture in the posterior and middle fossa in the side opposite to the eye with the hemorrhage.

However, in the second lecture, Battle more clearly described signs of fracture of the anterior fossa, where effusion blood was limited to the eyelid at first and then spread to the ocular conjunctiva and the retroorbital ecchymosis. He emphasized that watery discharge appeared later and said the following:

> *I am of the opinion that more reliance should be placed on the chemical analysis of watery discharges. If these show on examination the presence of a large amount of chlorides, and give the sugar reaction to Fehling's solution, then we may say that there is a fracture and communication with the subarachnoid space through a tear in the tubular prolongation of the membranes which surround the facial and auditory nerves; and this applies although there be some albumen from admixture with serum, blood, or secretion from the ear [2].*

Of his three lectures, the third lecture has been infrequently cited; however, it has important new observations. In this lecture, Battle described temperatures following cases of injury to the brain. When there was only fracture of the posterior fossa, he found no evidence of temperature changes; temperatures remained normal or were mildly decreased. However, a later spike in temperature was associated with a fatal outcome and often preceded the patient lapsing into a coma. These temperature charts were shown in great detail (a representative one is Fig. 4), and he emphasized that temperature could be an important sign of contusions to the frontal and temporal lobes. He concluded the following:



THREE LECTURES
ON
SOME POINTS RELATING TO INJURIES TO THE HEAD.
Delivered at the Royal College of Surgeons of England.
BY WILLIAM HENRY BATTLE, F.R.C.S.,
Hunterian Professor of Surgery and Pathology, Royal College of Surgeons; Assistant-Surgeon to the Royal Free Hospital; and to the East London Hospital for Children; formerly Resident Assistant-Surgeon at, and Surgical Registrar to, St. Thomas's Hospital.

LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—I have taken as a title for the lectures which I am about to give, "Some Points Relating to Injuries to the Head," and purpose asking you to consider with me, more especially, the effect of injuries when fracture of the cranial base has been produced, with some of the complications which are only too liable to accompany these fractures, or follow at a longer or shorter interval after the force producing them is expended.

For several years, in various official capacities, I had the privilege of seeing the surgical practice of St. Thomas's Hospital, and of closely observing, amongst other cases, many of injury to the head; and through the kindness of the members of the surgical staff of that hospital, I am enabled to bring before you a series of the cases which were under care in that institution during a part of that time, and can thus present a comparatively large number of examples for consideration. To these I have added the results

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LECTURE II.

MR. PRESIDENT AND GENTLEMEN,—I would now direct your attention to other evidences of fracture of the base of the skull, the escape of blood from the seat of fracture, and its appearance under the conjunctiva or over the mastoid process. An effusion of blood into the cellular tissue of the orbit is one sign of the presence of a fracture of the anterior fossa of the base of the skull. This effusion of blood may be limited to the eyelids at first, and then spread to the ocular conjunctiva; it may take place into the back of the orbit, and then spread forwards, only showing itself in the conjunctiva after the lapse of some hours or days, and then spread to the lids; it may be small in quantity, or sufficient to produce exophthalmos. The value of orbital extravasation as a sign of fracture is largely dependent on the question of its position (subconjunctival or not), and the quantity in which it presents. Other signs, such as epistaxis from the nostril of the one side on which the fracture is, and the position of the injured part must be considered. Most are familiar with the appearance of the eye when there is this hæmorrhage under the ocular conjunctiva; in extreme cases it forms an elevated brownish-red circle, which encloses the cornea, and contrasts strongly with the colour of the iris of the patient. Usually disappearing in the course of a fortnight or so, it may last a long time, the blood not being absorbed for weeks; I have watched its gradual disappearance over a period of from six to seven weeks. In four cases, all of which recovered, there was much protrusion of the eye on admission, and that to an extent

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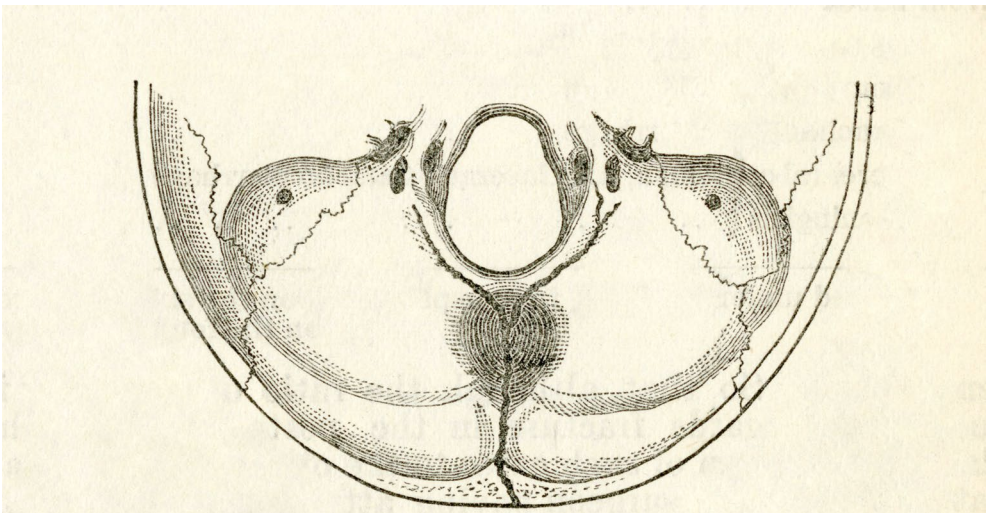
LECTURE III.

MR. PRESIDENT AND GENTLEMEN,—At the termination of the second lecture I was considering the injuries to cranial nerves which sometimes accompany or follow fractures of the base; the seventh nerve was under discussion when the time expired, and notes of fifteen cases had been read, which indicated, amongst other points, the improved prognosis as to the recovery from facial paralysis that can be given when it is a late manifestation after these fractures. In nearly all it was also apparent that the hearing on the same side was impaired; usually this was shown at the time of the onset of the paralysis, but in three fatal cases the patients were unconscious until death. In fourteen other patients who presented signs of fracture of the middle fossa deafness was found, and was a cause of complaint; six developed it after hæmorrhage from the ear (it was found in two who complained when they regained consciousness, in two on the sixth day, and two on the ninth day); six after hæmorrhage, followed by watery discharge of varying amount and duration; two after epistaxis, in one of which watery discharge from the nose followed (both had hissing and other noises in the ears). As a rule the prognosis, as in paralysis of the portio dura, was better with regard to recovery if the deafness was of late onset. In many instances the membrana tympani was seen to be ruptured, but the

Fig. 2 The three lectures by Battle (1890)



Fracture in the posterior fossa of the skull. (After a sketch by Dr. Richardson of the bones in his case.)

Fig. 3 Middle fossa fractures

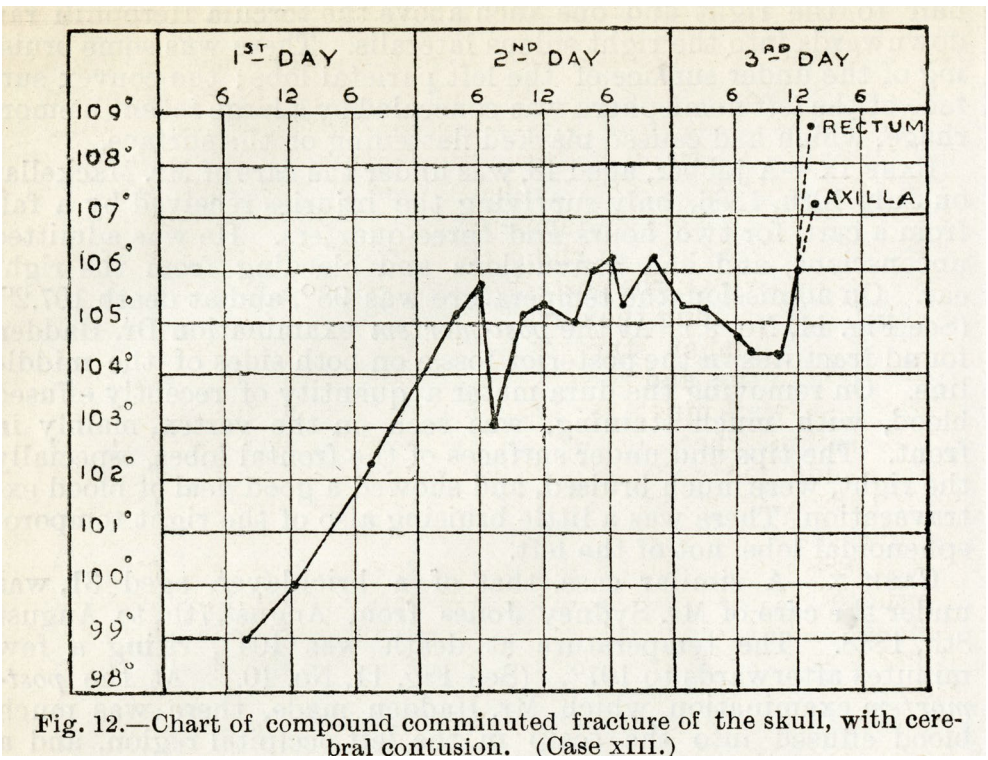


Fig. 12.—Chart of compound comminuted fracture of the skull, with cerebral contusion. (Case XIII.)

Fig. 4 Temperature chart in patient with a developing brain contusion

I would submit to your judgment the following propositions, which express my ideas somewhat differently. If within three days after an injury to the head a patient passes from a condition of unconsciousness or semi-consciousness to one of increasing coma, or after a period of return of consciousness a similar condition of coma supervenes and with the coma there be a rise of temperature of or above 103°, the condition is the result of cerebral contusion with some laceration.... Rarely do we find acute meningitis developing and running its course to a fatal termination within 24 hours, with coma and unequal pupils [3].

He specifically noted that in the single case in which meningitis was diagnosed after TBI, the patient had no rise in temperature.

Man of Many Parts

Battle made more observations than the ones that have survived as eponyms. As a surgeon, he collected records of many patients with traumatic head injury and continually searched for early clinical signs predictive of deterioration. Remarkably, he managed to synthesize this information in the late 1800s without the confirmatory tests available today, relying exclusively on the physician's detailed examination. The Battle sign is typically overlooked because it requires flipping the earlobe. Later, the ecchymosis may descend further down the neck, and only then is it noted.

Secondary deterioration after traumatic contusions is well known in the modern world of neurointensive care. We have forgotten to observe temperature courses. Knowing that hyperthermia occurs in the setting of cerebral vasospasm, new persistent spikes weeks after a traumatic head injury could certainly indicate the presence of meningitis or, after craniotomy, the appearance of an epidural empyema. But an early rise in temperature seems to indicate more than a skull fracture and increased probability of a contusional lesion. In their retrospective study, Meythaler and Stinson [4] noted an increased risk of "neurogenic fever" among patients with severe TBI who had experienced either diffuse axonal or frontal lobe contusions. Thompson et al. [5] noted similar findings. Because the frontal lobe has no major thermoregulatory centers, frontal lobe injury possibly indicates hypothalamic injury. Skull fracture does not independently predict development of fever; however, it has previously been associated with other indicators of hypothalamic injury following TBI, such as diabetes insipidus. Additionally, patients with TBI are at risk of secondary injury from fever; every 1-°C rise in body temperature substantially increases the metabolic rate. This burdens the stressed energy reserves of the severely brain-injured, catabolic patient.

When the central nervous system effects of hyperthermia following both global cerebral ischemia and fluid-percussion trauma were examined in rodent models, hyperthermia significantly increases mortality and cellular damage [6, 7]. This temperature course should be differentiated from dysautonomic storming, another important overlooked observation that came more than 75 years later [8].

William Battle was an astute clinical observer, and we should be grateful for his observations, including the subtle ones that we seem to have forgotten. He frequently closed his lectures with a few words on management:

When we regard the large number of patients who die from cerebral injury with hemorrhage from the part of the brain lacerated, the question presents itself most strongly for answer as to the advisability of using the trephine more often in an attempt to relieve them.... The operator must be guided by his knowledge of the effect of these injuries on the brain substance, and recollect that the fatal hemorrhage will usually be over that part of the brain directly opposite to the part struck, and not immediately underneath it.

That statement rings true today.

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