In 1934, Winston Churchill (1874–1965), the future British prime minister, informed Parliament of the threat of German hostility leading to a European war that could include massive aerial bombing of London. By autumn of 1938 war seemed inevitable, and it was feared that Germany could begin bombardment at any time. A trial nighttime blackout in London was held in August of 1939, and after Germany invaded Poland in early September of that year, blackouts at sunset were mandated with no lights allowed after dark for one-half of a decade. A month later Germany invaded and occupied multiple neighboring countries, prompting the United Kingdom (UK) and France to declare war on Germany. In June of 1940, Germany invaded and began the occupation of France, and by September of that year London was bombarded by the Germans for 57 consecutive nights. More than 1 million London homes were damaged or destroyed and more than 40,000 civilians were killed, approximately one-half in London, and the remainder in other industrial UK cities and ports.15,43,89

The British military’s neurosurgical and neurological response to the challenges of World War II (WWII) resulted in significant progress in the organizational structure, management, and resultant outcome from head injuries, which was of much relevance to future military and civilian trauma management. Head injury research in primates and in vitro modeling of human head injury led to biomechanical theories of brain injury that continue to be influential today. These theories include the critical roles of acceleration/deceleration forces in the production of concussion, and of rotary or angular forces in shear force injuries of the brain.

The dynamic and pioneering Oxford-based British neurosurgeon Sir High Cairns (1896–1952) was personally involved in the conduct of all phases of these uniquely influential neurological and neurosurgical initiatives during WWII. He had the foresight, gained the necessary high-level political and military trust and financial support, and carried out the critical organizational planning and implementation. In the fashion of his neurosurgical mentor Har-
vey Cushing (1869–1939), Cairns insisted upon extensive clinical documentation and outcome analyses, and personally oversaw a multitude of in-depth, well-written publications resulting from these WWII experiences.

Hugh Cairns

Early Career and Neurosurgical Training With Cushing

Hugh William Bell Cairns was born and raised in South Australia, where he finished secondary and medical school at Adelaide, served as a World War I (WWI) physician in the Middle East and France, and came as a Rhodes Scholar to Oxford’s Balliol College in early 1919 (Fig. 1). Handsome, athletic, bright, and outgoing, with a captivating personality, Cairns caught the attention and was an invited guest to the home of his physiology teacher, Sir Charles Sherrington (1857–1952), and the internist Sir William Osler (1849–1919), with whom he had rounded at the Radcliffe Infirmary.

Lady Osler first introduced Cairns to the well-known American neurosurgeon Harvey Cushing (1869–1939). At Oxford, Cairns did a surgical residency and spent a period as anatomy demonstrator at the Radcliffe Infirmary. He married a gifted daughter of A. L. Smith, a well-respected teacher and the Headmaster of Balliol College. In 1921 Cairns became a fellow of the Royal College of Surgeons, went to the London Hospital as house surgeon, and later was appointed as first assistant to the notable general surgeon Sir Henry Souttar (1875–1964). Souttar, who performed some neurosurgical procedures and described the bicoronal incision, was also a founder of the Society of British Neurological Surgeons in 1926. Sherrington, and especially London neurologist George Riddoch (1888–1947), saw distinctive potential in Cairns, who exhibited boundless energy and determination, “qualities necessary to develop modern neurosurgery” in London. Sir Walter Morley Fletcher (1873–1933) of the British Medical Research Council (MRC) and a close friend of Cushing also came to know Cairns.

The above individuals helped Cairns obtain a Rockefeller traveling fellowship to spend a full year of neurosurgical training under Harvey Cushing in Boston (1926–1927). Soon after Cairns’ arrival, Cushing’s astute secretary Ms. Stanton noted, “Cairns here for a year…Runs around everywhere so as to waste no time…Going to be a great success.” In the fall of 1927 Cairns returned to London as one of the first neurosurgeons in that city to be trained in modern methods. In a lengthy letter to Cairns immediately after his training, Cushing emphasized the critical need for neurosurgical specialization to properly advance this demanding field. It was clear that Cairns’ goal in London was to develop a dedicated neurosurgical service emulating the meticulous and painstaking operative techniques, clinic organization, and system of record collection he learned from Cushing.

Sir William MacEwen (1848–1924) of Glasgow, Scotland, and Sir Victor Horsley (1857–1916) of London, England, pioneered neurosurgery in the late 19th and early 20th centuries. Although MacEwen antedated Horsley by 6–7 years in performing successful antiseptic brain operations based on cerebral localization, Horsley is often labeled the “father of neurological surgery…” who pointed the path to the advancement of this newest branch of surgery by physiological experimentation in addition to clinical and pathological contributions…he was the first surgeon to devote the bulk of his time to neurological surgery…about 60%, 40% general surgery.” Cairns, however, is considered the first surgeon in Great Britain to devote himself exclusively to neurosurgery. Geoffrey Jefferson (1886–1961) of Manchester, England, had briefly visited Cushing and is credited with initiating neurological surgery as an actual surgical “specialty” in Great Britain after WWI but performed general surgery as well. Norman M. Dott (1897–1973), of Edinburgh, Scotland, who trained with Cushing several years before Cairns, continued pediatric general surgery in addition to neurosurgery. These modern neurosurgical pioneers and friends shared the same struggles, ideals, and goal of “a self-contained unit with the ambition to raise [neurosurgical] work…to the top level of international ranking.”

Cairns developed a fine reputation at the London Hospital with its first neurological surgery service. This was due to his diligence, perseverance, engaging personality, and surgical results. Cairns was a particular favorite of Cushing, who wrote to the Rockefeller Foundation:

…young Cairns…with all the necessary qualifications for success – ambition, vigorous health, enthusiasm, good train-
Hugh Cairns: an Oxford tale

Cairns subsequently obtained financial support from the Rockefeller Foundation to include surgical equipment and subsidize neurosurgical assistants and trainees. At the London Hospital, Cairns met a young, talented research pathologist named Dorothy Russell (1895–1983), whom he encouraged to study neuropathology. With Cairns’ support she obtained a Rockefeller fellowship to study for several years in Boston and Montreal (1928–1929) and became a leading world authority in neuropathology.5,6 In the next few years Cairns was appointed surgeon to the two principal London neurological hospitals at Maidav Vale (1931–1934) and the National Hospital of Nervous Disease at Queen Square, London (1934–1937).

However, the London Hospital and its senior surgeons would not commit adequate space or facilities to the dedicated and independent neurosurgical department that Cairns believed was necessary. At Queen Square, where Cairns performed only 1 operation, the senior neurologists believed in a “dominance” of neurology over neurosurgery and resisted changes such as granting admission privileges and beds to the surgeons and providing facilities attractive to private patients in the style that Cushing had developed in Boston.43,44,65,82 The Rockefeller Foundation, although well disposed to providing financial support to neurological centers in North America and Europe, realized Cairns’ potential but believed the well-known neurological center at Queen Square held a better chance for success.44

During this period, most of England’s established senior general surgeons remained unconvinced that Cushing’s delicate, overly slow, and plodding techniques with gentle handling of all tissues, and meticulous control of bleeding, resulted in a better outcome.39 Many elder surgeons also believed that the speed of surgery was of prime importance, as anesthetics were more unpredictable and dangerous with prolonged operations. In addition, Cushing had worked with British surgeons during WWI and was known to be a difficult individual at times.28 Finally, few if any English surgeons believed it possible to make an adequate living by specializing in neurological surgery alone. Thus, dabbling as a “ dilettante” in some neurosurgical procedures was considered adequate. 39

In May of 1935, T. E. Lawrence (1888–1935)—better known as “Lawrence of Arabia,” the celebrated British WWI hero, Oxford scholar, author, and authority on Arabia—was fatally injured in a motorcycle accident. Lawrence swerved to avoid an oncoming vehicle, lost control, and pitched forward over the handlebars. Cairns drove about 100 miles to consult on Lawrence, who suffered a severe head injury with skull fracture and survived in a coma for 5–6 days until he died from chest complications. The family allowed Cairns to perform the autopsy and take the brain.43,58,80 The brain, likely in poor condition, was presumed to have been given to Dorothy Russell for study, but no autopsy report is available. Lawrence was not wearing a helmet, as “crash helmets” at that time were predominately worn by those involved in motorcycle rac-

By 1936 Cairns shared his more extensive written proposals with Buzzard, and a scheme was developed to gather support for the plan at the highest levels. Buzzard arranged meetings between Cairns and other influential Oxford University officials who recalled the earlier failed effort.28,43 In 1936 Buzzard was also president of the British Medical Association and the Oxford host of its annual meeting. At the formal British Medical Association reception, it was arranged for Cairns to be introduced by his wife’s mother, a “grande dame of Oxford.”59 At the British Medical Association annual meeting, it was arranged for Cairns to be introduced by his wife’s mother, a “grande dame of Oxford.”28,44 Finally, few if any English surgeons believed it possible to make an adequate living by specializing in neurological surgery alone. Thus, dabbling as a “dilettante” in some neurosurgical procedures was considered adequate.59

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As early as 1934, Cairns began to think about transferring his growing neurosurgical service to Oxford, where there was room for expansion and where he had friends and influence.43,66 In addition to having a beguiling charm and manner, Cairns was politically astute and could be relentless to serve his needs. By the summer of 1935 Cairns had broadened his ideas to include the formation of a high-quality research and clinical medical school and shared these ideas with neurologist and supportive friend Sir E. Farquhar Buzzard (1871–1945), an eminent London neurologist and physician to King George V, who held Osler’s previously held chair as Regius Professor of Medicine at Oxford.28,43 Buzzard had witnessed the failure of similar plans for Oxford by the Rockefeller Foundation in the late 1920s and was determined that this should not happen again.28,44

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World War II Contributions

In 1938 Cairns was appointed advisor to the War Office on the care of head injuries in the armed forces.81 This was not surprising, because since the early 1930s, Cairns had been a civilian consultant neurosurgeon to Queen Alexandra’s Military Hospital at Millbank, a Royal Army Medical Corps (RAMC) facility. Senior military personnel knew Cairns well, liked and respected him, and ap-

J Neurosurg Volume 125 • November 2016 1303
preciated his work. About this time, the British Ministry of Health began serious organization of their Emergency Medical Services (EMS) primarily to provide for possible air-raid casualties. Neurosurgeons and neurologists, some of whom had been involved in WWI, were mobilized for military and civilian designation and assignments. Under the Ministry of Health in 1938, Cairns together with Jefferson divided England between them for civilian EMS neurosurgical organization. In July or August of 1938, in anticipation of a projected steel shortage, Cairns wisely convinced the War Office to procure “all surgical instruments which would be required by army neurosurgical units during the impending war. Consequently, neurosurgical instruments were never in short supply.” “This was in sharp contrast to the situation in other specialties.”

By the fall of 1938, with war seemingly inevitable, it was decided that Oxford was an ideal location for a neurological and neurosurgical military hospital as Cairns and his team were already there. Without significant war-related heavy industry, Oxford was unlikely to be bombed (and never was), yet it was in reasonable proximity to London where many casualties were expected. Cairns asked Charles P. Symonds (1890–1978; pronounced “Simonds”), who was consultant neurologist to the Royal Air Force, and neurologist George Riddoch, a pioneer in the treatment of WWI brain and spinal cord injuries, to assist him in the planning and operation of a military hospital for head injuries at Oxford. Before the end of 1938 Cairns had decided upon the use of St. Hugh’s College for Women near the Radcliffe Infirmary to be the site of Oxford’s Military Hospital for Head Injuries, serving the army and air force. The hospital opened in February 1940 with 50 beds, and that month Cairns, who had been an acting colonel, was appointed as brigadier and the neurosurgical head of the RAMC (Fig. 3). This was a major step in establishing concrete recognition by the British Army Medical Service of neurosurgery as a surgical specialty distinct from general surgery.

The St. Hugh’s Military Hospital for Head Injuries—affectionately nicknamed “the Nutcracker Suite”—had a census that grew to 300, rising to 430 at the height of the battle of Normandy in June of 1944. During WWII, St. Hugh’s treated approximately 13,000 head injuries and became a training ground for a generation of neurosurgeons and neurologists, anesthetists, medical students, neurological nurses, orderlies, and other medical personnel, many of whom had fled the Nazis. Cairns, in the tradition of Cushing, realized much would be learned from this wartime experi-


ence, and insisted that meticulous clinical records be kept, anticipating the necessity of accuracy in forthcoming publications.15,76–78,85 St. Hugh’s was also the base station for the Mobile Neurosurgical Units (MNSUs; see below).

Cairns created two convalescent homes for neurological rehabilitation, a recently introduced and much valued field.19,48 Brigadier George Riddoch, recalling the particular displeasure and hopeless outlook for young soldiers with spinal cord injuries in WWI, induced the British government to establish a special center for these individuals and convinced neurologist/neurosurgeon Ludwig Guttmann (1899–1980) to take charge of this service. Due to Riddoch’s vision, the world’s first “Spinal Injuries Centre” was established at Stoke Mandeville Hospital in Aylesbury, England.11,27,49,55,68

During this period Cairns was also responsible for training the first full-time female neurosurgeon, Diana K. Beck (1902–1956).46 Dr. Beck was highly qualified, having been a fellow of the Royal College of Surgeons of Edinburgh, fellow of the Royal College of Surgeons of England, Surgical Registrar, Demonstrator and Lecturer in Anatomy and Surgical Anatomy at London’s Royal Free Hospital, and Clinical Clerk at Queen Square.43 At age 37 she was appointed House Surgeon to Cairns in January 1939, and completed her neurosurgical training at St. Hugh’s in 1943. A favorite of Cairns, she was a highly motivated, able, and experienced surgeon. Cairns placed her in charge of the introductory course in clinical surgery, and she was also very productive in Dorothy Russell’s laboratory.43,46 Upon leaving Oxford in 1943, Beck was assigned by EMS Director Jefferson in the north of London, and then at Bristol. She was given a senior appointment at London’s Middlesex Hospital (1947), where she was the first woman to head a neurosurgical service, and completed a fine career in neurosurgery until her unexpected death in 1956.46

Motorcycle Crash Helmets

The subsequent bombing of London (1940) brought forth an abundance of severe head injuries in both civilians and the military, especially military dispatch motorcycle riders and pillion passengers.43,64,72,74,88 Cairns had been tracking accidents in military dispatch motorcyclists,14 and he stated,12

My interest in crash helmets arises solely from the fact that during the War, I spent a considerable part of my time treating injured motor-cyclists at the Military Hospital for Head Injuries at Oxford. In other words, it was the segregation of the Army’s head-injury patients in special centres which made possible the prompt recognition of the importance of crash helmets.

Cairns reported that 2279 motorcyclists and associated passengers were killed within the first 21 months of the war (more than 3 per day), and two-thirds of these deaths were army members.12,14 A head injury was present in 92% of motorcycle-related injuries, and although not the sole cause of death, it was clearly a major factor in the majority.15,19 The situation was compounded by blackouts from nightly air-raid bombings and the recklessness of youth. However, Cairns found 7 cases of motorcyclists who were wearing crash helmets and none were fatally injured.14,20

In 1939 Cairns elicited the help of Oxford physicist and motorcycle enthusiast A. H. S. “Hylas” Holbourn (M.A., Edinburgh; D.Phil., Oxford; 1907–1962; Fig. 4), on the study of crash helmets especially as applied to motorcycle accidents.14,20,72 MRC grants helped support this work. Motorcycle helmets were carefully studied after accidents and correlated with radiological and clinical findings as well as outcomes in head-injured patients. This work proved the importance of helmet usage and led to improved helmet design (Figs. 5 and 6).12,14,20

Cairns and Holbourn were influenced by MRC-supported experimental work begun in early 1940 on acceleration concussion by neurologists Denny-Brown and Russell (1941, see below).58 It is likely that Holbourn’s training in physics and earlier research convinced him that angular acceleration from sudden head rotation was most injurious to the brain: “This is the cause of all gross bruises or lacerations of the cortex remote from the site of the blow.”

All the available evidence points to its being also the cause of concussion."20

Holbourn, whose help was acknowledged in Cairns’ 1941 paper on motorcycle crash helmets, apparently shared the above opinion with Cairns, as this paper mentions two helmeted patients: Case V suffered a concussion and post-concussion syndrome, which “crash helmets cannot be expected to prevent”; and for Case VI, unconscious for several days, his helmet showed “marks of violence which were slight, and it is therefore doubtful whether his head received any severe blow...and this suggests a fairly severe head injury from violent rotation of the head, [and]...helmets have the theoretical disadvantages that they increase the diameter of the head and so leverage [the head, increasing]...the likelihood of broken neck and rotational acceleration within the cranium.”14,29 In their joint paper
of 106 head injuries in those wearing helmets, Cairns and Holbourn (1943) concluded that hospitalized, injured motorcyclists who were wearing helmets, as compared with hospitalized motorcyclists who were not, had a significantly reduced percentage of skull fractures, neurological damage, concussion, and prolonged amnesia associated with concussion, and inability to return to duty. They also believed that helmets likely kept a number of motorcyclists from even coming to the hospital after an accident.20

They believed helmets decreased local damage to the scalp and skull, especially from pointed objects, and lessened the numbers of depressed and penetrating skull fractures with associated brain injury: “The so called coup lesion is, in our view, due to the bending of the skull under the blow.”20 The shell of the helmet also functions by “lengthening the blow, that is by spreading it out over a longer interval of time, so that it is not so intense at any particular instant...lengthening reduces not only the local injury but all other effects of the blow.”20 The helmet shell can slide over objects

...instead of stopping more abruptly, as the unprotected head would do owing to its greater coefficient of friction...The blow is also lengthened to some extent by the rotation of the helmet relative to the head...But the main way in which the blow is spread over a longer time is by means of the buffering action of the slings [being stretched] and hatband [compressed]. (bracketed material added by authors)

The effect of the blow lasts all this time, instead of only during the time taken to deform the scalp and skull. They believed the suspension (sling) components on helmets could and should be significantly improved.20

Cairns and Holbourn (1943) concluded that the hard, vulcanized rubber outer shell helmets tended to fracture, resulting in underlying skull fractures being twice as common and an increased incidence of concussion and concussion related amnesia, as compared with those wearing wood pulp outer shell helmets. The wood pulp helmets had an outer shell consisting of compressed wood pulp covered by a thin film of cloth, which tended to crush more on impact. The pulp helmet also had a more secure internal headband liner and suspension system to prevent the rider’s head from coming into contact with the crown of the outer shell (Figs. 5 and 6).12,20

In 1941 Cairns was responsible for making helmets compulsory for army motorcyclists, and by the end of 1942 he clearly had demonstrated a significant decrease in motorcycle deaths.12,48 “This is an example,” he wrote, “of the impelling need for hasty decision in wartime on questions which really should have been worked at thoroughly between the wars.”12 Although foolishly resisted for decades by civilian motorcyclists, in 1973 helmet usage became law in the UK.64,48 Motorcycle helmets have been shown to reduce the risk of head injury by almost 70%, and death by more than 40%.62

Interestingly, the abundance of head injuries in WWII British and Canadian military dispatch motorcyclists also led to the recognition of the necessity to evacuate sizable hemorrhagic coup or contrecoup frontal and temporal cerebral contusions by craniotomy. Associated acute or subacute subdural hematomas may be present as well. These WWII investigations were led by E. Harry Botterell (1906–1997) at the Canadian Neurosurgical Center in Basingstoke, England (personal communication to J. L. Stone by E. Alexander Jr., May 1996; personal communication to J. L. Stone by E. H. Botterell, May 1996).10,15

**Mobile Neurosurgical Units**

In WWI, Cushing (1918) demonstrated improved results with an early, definitive operation; debridement of devitalized brain tissue, in-driven bone fragments, and foreign bodies; dural closure; and primary 2-layer closure of the scalp. Other WWI European surgeons likewise advocated aggressive approaches that led to a lessening of fatal infection and the dreaded complication of fungating herniation of injured brain tissue through a defect in the skull and scalp.23,32,36,57 The postoperative mortality rate in penetrating injuries not receiving adequate early surgical treatment was approximately 50%, and many died before they could be treated.29,31

To enable neurosurgical teams to definitively treat head injuries within 24–48 hours after injury, as Cushing advocated,30,31 Cairns came up with the idea of using MNSUs.15,16 The MSNU vehicles had the appearance of a large ambulance or modified truck (Fig. 7), and the first MNSU was assembled in May of 1940. Almost all of the MNSU staff had been trained at St. Hugh’s, and “the (Cushing) standard of documentation and neurologi-cal examination never slipped, despite the circumstances in which they worked.”100 The MNSUs generally worked in conjunction with a host hospital or temporary casualty clearing station close enough to the front lines for timely general triage and neurosurgical evaluation. The host unit would provide postoperative beds, extra staff, catering, laundry, pathology, radiology, etc.15,16 The MSNU had 1 neurosurgeon, 1 triage neurologist, 1 anesthetist, 2 general RAMC officers, 2 nurse sisters, 4 RAMC orderlies, and 2 RAMC drivers. Each MNSU had its own electrical generator, water supply, tents, 2 heated operating tables,
and operative illumination. There were 2 neurosurgical instrument sets and 1 general use set. The equipment was sufficient to carry out at least 200 cranial operations without replacements.15,81

Of critical importance, Cairns convinced the military authorities that each unit must have its own power suction, electrocautery (diathermy), and lighted brain retractors;16,59,73 “Such luxury army surgeons had never had before… it speaks volumes for the respect and regard in which Cairns was held by the War Office… that he was able to get these things… [and thus] the units were able to obtain hitherto unheard of results in penetrating head wounds.”59

It took only a brief period for the army medical and surgical units to learn how the MNSUs could be optimally interfaced with the existing services. This allowed definitive neurosurgical operation upon brain injuries, usually within about 24 hours of injury (Fig. 8).1,3,15,79 Six active MNSUs were used in France, North Africa, Italy, Northern Europe, India, and Asia, and more than 20,000 patients (80% of soldiers and airmen with head injuries) were treated.15 In Italy 1 unit performed 334 operations in 16 days and another 208 in 15 days.80 About 90% of those with scalp wounds and simple skull fractures returned to their units, and 70% of the brain injured were employable, although few returned to full duty. It was clear that this early deployment of neurosurgical skills resulted in primary healing in 85%, a considerable reduction in infection, morbidity, and mortality rates, and improved the quality of survival.3,15,38,80,81

Of much importance in the final years of the war, the MNSUs were also used in the introduction and critical evaluation of the use of penicillin in injured neurosurgical patients (see below).15,41 MNSU 6 took part in the invasion of Normandy in 1944, and later in WWII the British MNSUs were coordinated with air evacuation of neurosurgical patients.15

The MNSUs are considered the precursor of the mobile army surgical hospital, or MASH, units, and the concept was so successful that it was later used by the US Army in conjunction with helicopter evacuation in Korea, Vietnam, and Desert Storm, decreasing the elapsed time to several hours between injury and definitive debridement.1,81

Howard Florey, Cairns, and Penicillin Trials in the Military

In 1928, the Scottish bacteriologist and physician Alexander Fleming (1881–1955), while examining a Staphylococcus culture plate, noted a halo of inhibited bacterial growth around a contaminant blue-green mold.33,40,50 Fleming concluded the mold (Penicillium notatum) released a substance that inhibited bacterial growth, which he later termed “penicillin.”42 Fleming cultured the original mold but was limited in his ability to purify, concentrate, and produce substantial stable amounts of penicillin to perform adequate clinical trials.42,60

The key physician investigator who would lead this important task was Howard Florey (1898–1968), a meticulous and skilled research professor of pathology at Oxford and an Australian colleague of Cairns. After reading Fleming’s work, in 1938 Florey and coworker Ernst B. Chain (1906–1979) began to investigate the chemical, pharma-

FIG. 8. A: Young man with through-and-through transfrontal gunshot wound upon arrival 16 hours after injury to MNSU 5, Naples, Italy, May 1944. B: Scalp shaved, entry and exit wounds shown. C: Diagrams showing the line of the skin incision and area of brain damage. D: The patient, 10 days after surgery, was still confused with slight right-sided weakness. His bone defect was subsequently repaired and he returned to duty. From Schorstein J: An atlas of head wounds illustrating standard operative technique. Br J Surg 55 (Suppl 1):27–61, 1947. ©British Journal of Surgery Society Ltd. Reproduced with permission of John Wiley & Sons Ltd. on behalf of the BJSS Ltd.
ological, and chemotherapeutic properties of penicillin.\textsuperscript{25} They devised methods to increase the yield of penicillin, demonstrated slight if any toxicity in mice, and reported a 90\% and 100\% success rate in curing mice infected with \textit{Staphylococci} or \textit{Streptococci}, respectively, compared with death in all controls.\textsuperscript{25} While this research was under way, Florey wrote to the MRC for support in September of 1939, “I can get clinical co-operation from Cairns for any products we produce and I have tested on animals.”\textsuperscript{43,65}

In January 1941 Florey approached Cairns with a view to clinical trials in patients with infections at the Radcliffe Infirmary. Cairns quickly directed him to the Nuffield Professor of Medicine, Leslie J. Witts (1898–1982).\textsuperscript{28,43,65} Thus, in February 1941, the first ever systemic usage and clinical trials of penicillin were performed on Professor Witts’ Radcliffe Infirmary patients.\textsuperscript{32} Penicillin was given intravenously in 5 patients with \textit{Staphylococci} and \textit{Streptococci} infections and was also applied locally to 4 cases of eye infection. In all these cases a favorable therapeutic response was obtained, although intravenous administration not uncommonly gave immediate rigors and occasionally fever due to pyrogenic impurities in the penicillin.\textsuperscript{2}

Florey saw the possibilities of the use of penicillin in war surgery, and in 1942 limited amounts of penicillin were sent to the Middle East forces in Cairo, where the results at first were encouraging, and later impressive. In April 1943, penicillin also became available at St. Hugh’s for the treatment of meningitis and the results were striking.\textsuperscript{21} In late May 1943, the War Office sent Cairns and Florey to go together to North Africa and Sicily for 3 months to study potential field usage and penicillin dosage in the treatment of brain injuries, and set up clinical trials (Fig. 9).\textsuperscript{27,65} These trials were largely coordinated with the MNSUs and results sent back to Oxford.\textsuperscript{17,41} Penicillin was used locally (topically) in 23 penetrating brain wounds 3–12 days after injury at the time of postdebridement closure. Almost all wounds were infected with gram-positive organisms and 13 showed purulence. Three patients died, 2 of them from intracranial infection (< 10\% infection mortality rate). The remainder made a satisfactory recovery. Bacteriological results showed that after 2 days of treatment with penicillin, gram-positive cocci were usually no longer found, whereas coliform organisms were invariably present. In several cases \textit{S. aureus} was found to be penicillin resistant, and there was no indication that local installation of penicillin was toxic or caused damage in the low concentrations then available.

Further experiences with penicillin in the treatment of brain abscess, paranasal sinus infection, and meningitis in both military and civilian populations were highly successful.\textsuperscript{13,18,26,27} By the time of the Normandy invasion in June of 1944, mass-produced supplies of penicillin had become available, bolstered by American production of penicillin.\textsuperscript{43,60} Florey was honored for his work on penicillin, knighted in 1944, and shared the Nobel Prize in Physiology or Medicine in 1945 with Fleming and Chain.\textsuperscript{63}

\section*{Brain Injuries Committee and Experimental Acceleration-Induced Concussion}

In early 1940, the MRC established a Brain Injuries Committee of prominent neurologists and neurosurgeons to initiate a timely program of research on brain injury. Several additional members were added in 1941.\textsuperscript{27} The committee chairman was noted neurophysiologist Edgar D. Adrian (1889–1977), who in 1932 shared the Nobel Prize in Medicine or Physiology with Charles Sherrington. Other committee members included neurologists George Riddoch, Charles P. Symonds, Macdonald Critchley (1900–1997), neuropathologist J. G. Greenfield (1884–1958), and neurological surgeons Geoffrey Jefferson, Hugh Cairns, and Norman Dott.\textsuperscript{27} Both Jefferson and Riddoch had significant WWI experience with neurological injuries. In 1941 the Brain Injuries Committee introduced “A Glossary of Psychological Terms Commonly Used in Cases of Head Injury”\textsuperscript{67} in an early but unsuccessful attempt to standardize the usage of descriptive terms related to head injury.

The Brain Injuries Committee supported landmark research by Symonds-trained neurologists Derek Denny-Brown (1901–1981) and W. Ritchie Russell (1903–1980) on acceleration-induced concussion in cats and monkeys.\textsuperscript{35,36,37} These studies concluded that after sufficient impact, the forces of acceleration or deceleration caused...
Concussion (loss of consciousness) or subconcussion (a stunned or dazed condition), each without apparent acute macroscopic or microscopic lesions. However, concussion or subconcussion did not occur if the head was rigidly held when the blow was delivered.37,36

By using a brass pendulum with a broad leading end, the head (usually the occipitoparietal region) was struck and set into motion from the resting position. The forces of deceleration were largely eliminated by the head coming to rest against a soft wool cushion (Fig. 10).36 In all but 1 case, anesthetized animals or decerebrate preparations were used. In these animals a certain degree of intensity of the blow (adequate stimulus) was required with dependence on the rate of increase in velocity (concussion threshold 0–28.4 feet/second in the monkey, 23 feet/second in the cat) before appreciable reversible changes were realized, such as bradycardia; blood pressure drop and then elevation; respiratory cessation, slowing, or irregularity; absent or depressed corneal and pinna reflexes; and unresponsiveness. A brief rise of intracranial pressure did occur when the head was struck, but was not believed high enough or of sufficient duration to account for functional changes.

Denny-Brown and Russell (1941) also found that blows not sufficient to cause concussion still may affect the “vago-glossopharyngeal system,” producing bradycardia, vasomotor, and respiratory changes, along with incomplete loss of consciousness (subconcussion). Transient abolition of the corneal reflex and total loss of reaction (paralytic signs of concussion) were noted in an unanesthetized monkey inadvertently impacted.35,37

Pendulum blows above the concussion threshold were generally necessary to cause focal skull fractures, microscopic or macroscopic injury, cerebral contusions, and brain lacerations. This implied that upon the general background of concussion more severe injuries sequentially developed. They found subpial petechial hemorrhages to occur in the upper segments of the cervical spinal cord and brainstem with increasing forces, which they believed were due to direct rupture of small vessels from localized stress and strain (distortion).

The mode of injury in these experiments was considered akin to that commonly termed “blunt head injury” by clinicians, in that the striking object is so much heavier than the head that “within a few milliseconds...the latter takes up at least the full velocity of the striker on impact.”36

Denny-Brown and Russell believed that cushioning methods used to decrease the blow delay “the transfer of velocity”—that is, “the rate of increase (or decrease) in velocity [acceleration or deceleration]...without diminishing the amount of change.”37 In other words, although the “final velocity and momentum may be the same,” the “slight delay introduced by a helmet” must greatly lessen the damaging velocity change. A monkey fitted with an internally padded, small metal (tobacco can) helmet was subjected to typical concussive and fracture level blows, but neither was observed as in unprotected animals with identical blows. They conjectured that the use of a helmet delayed the blow and protected the brain by decreasing the “fling of the brain” they envisioned with “acceleration concussion.”37

Hylas Holbourn and the Physics of Brain Injury

Hylas Holbourn (see above), as a Research Physicist in Cairns’ Department of Surgery, also performed important investigative work in the Oxford University Laboratory of Physiology in the early 1940s.51 His objective was the understanding of blunt brain injury as determined by the physical properties of the skull and brain, utilizing the primary Newtonian laws of physics and motion. In these studies he acknowledged the help of Cairns, Dorothy Russell, neurosurgeon George F. Rowbotham (1899–1975), and neurologist W. Ritchie Russell. Holbourn constructed 2D gelatin models in the shape of a cross-section of the human brain, which he subjected to sudden rotations or impacts, as might be caused by blows to the head. Utilizing polarized light, he detected the resulting shear strains in the gelatin. He also performed theoretical calculations to study cerebral torque and induced shear strains under various forces (Fig. 11).7,53

Holbourn assumed that a substance with the physical properties of brain tissue (similar to highly incompressible but easily deformed clay-like substances studied in engineering) is more likely to be damaged by rotational “shear strains” during the blow than the linear or translational tensile strains of compression and rarefaction (pulling apart). Shear strain is the sliding of one part of tissue across another, as in the wobbling of jelly or a “deck of cards when deformed from a neat rectangular pile into an oblique-angled pile.”52 He believed rotational forces were the most probable cause of both superficial laceration and general damage to the brain structure, including nerve fibers, neurons and their dendrites, and vascular structures.51-54
Focal skull distortion from impact certainly altered the shape of the brain, but Holbourn believed this was rarely sufficient to cause serious widespread brain damage. He believed that elasticity and distortions (bending) of the skull tend to cause merely local indentations and focal contusional types of injuries and not the diffuse neuronal insult that gives rise to concussion. The uncommon, slower crushing types of injuries to the immobile skull were known to produce generalized distortions and fractures, typically without loss of consciousness unless very extensive.

Other than localized brain injuries due to skull impact and distortion with or without fracture, Holbourn further considered the injurious physical forces or strains delivered to the brain to be mainly those that arose by the change in the velocity of the head caused by a blow. He divided these into velocity changes in a straight line, i.e., “linear (or translational) acceleration,” and an infinite number of rotary axis velocity changes, i.e., “rotary or angular acceleration.” The linear forces he felt produced small compression or rarefaction strains with minimal or no injurious effect, while rotational acceleration forces he believed to be many times greater, and a significant cause of severe shear strains such as in the temporoparietal and high convexity surfaces.

Holbourn’s work showed that almost every injury to the mobile head results in acceleration and deceleration, plus rotational strains. The momentum must be sufficient and the rate of change sufficiently rapid to result in brain damage. Similarly, if the movement of the head is suddenly arrested by impact against a solid object, the momentum or inertia of the brain mass causes continued movement. Thus, acceleration or deceleration of the head is essentially similar.

Holbourn also believed “that the shearing strains set up during rotation are less the smaller the [animal’s] brain,”51,66 and accounted for the difficulties that experimental workers had in producing brain damage (or concussion) in small animals, which often develop permanent neurological signs or death after blows to the head.47,84

Holbourn was perhaps the first to provide factual knowledge of physical properties of the brain, surface membranes, and skull, which conformed to the known facts of brain injury and afforded a plain explanation by the laws of physics.51 He believed that if the precise details of how the head was struck were known, brain damage could be predicted with reasonable accuracy.51,53 Finally, as the hemispheres are not exact replicas but mirror images of each other, under certain conditions such as rotation in multiple planes, damage would be asymmetrical in the 2 hemispheres.51,84

### Hugh Cairns After WWII

Following the war Cairns returned to surgical practice, teaching, and research, and published on a wide range of neurological topics. Cairns and Symonds were knighted in 1946 for their military contributions. In that year Cairns was also called to attend to the American general George S. Patton, who subsequently died of a fracture/dislocation of the cervical spine. Much of the neurosurgical load at Oxford was taken by his associate Joe Pennybacker (1907–1983), as Cairns traveled widely to lecture and assumed more administrative activities. Projects included disturbances of consciousness with diencephalic injury, the use of streptomycin in tuberculosis, treatment of meningitis, and studies of cingulate gyrus excision in the monkey. Cairns subsequently performed cingulate gyrus excision in select psychiatric patients with mixed results. He also became interested in hemispherectomy.

Cairns smoked about 15 cigarettes per day and had been in robust health until late December of 1951, when he had an attack of abdominal pain and vomiting. He saw his friend, the Oxford internist Leslie Witts, who found an abdominal mass on examination. He was operated on the next day, and a large lymphosarcoma was resected from his right colon. Despite radiation therapy and reoperation he stoically died of disease progression about 6 months later at age 56. He had a very happy marriage, leaving a widow and 4 children.

W. R. Russell, who collaborated with Cairns during and after the war years, stated:

> He was amazingly easy to work with...Always appreciative of the work of his colleagues and provided every facility to further their researches. He formed the most important training-ground for neurosurgeons in this country, and there must be over a score of his trainees...who have formed similar neurological clinics in many parts of the world. He played the game of life with enthusiasm and an insistence on highest possible standards which were an inspiration. His work was his life, and holidays were short intervals during which vigorous physical activity strengthened him for the next session. He was always convinced that things could be improved, and his insistent pressure for changes naturally provoked opposition at times; but his motives were so transparently honest and sincere that he usually gained much support. We had looked forward to many more years of happy collaboration, but as this cannot now be, we must maintain what he has built.

Lord W. R. Brain, a personal friend, wrote:

> In Cairns’ success there were many ingredients...athletic physique, untiring capacity for work, manipulative skill and...
high intelligence... (but) Cairns added an imaginative vision, which served him alike in his everyday activities and in larger affairs. He had an unusual insight... which contributed to his minute attention to detail. His approach to larger questions was empirical, and guided by an intuition which led him successfully to explore possibilities in research and treatment... He lived with a humorous zest, which sprang from some perennial youthfulness. One could not picture Hugh Cairns as an old man... 

Three decades later, Leslie Witts stated, “There was always an aura and magnetism about Cairns which was very difficult to define, but which attracted people to him and made them work with him along the lines that he wanted... setting them to work quite happily, and they would stay with him. Very few... had this manifest aura of greatness that surrounded Cairns.” Cairns was unique in his “personal confidence, integrity and a capacity to brush aside obstacles. His plans were far-sighted, yet simple and direct, and people found themselves co-operating because of his unique resolution, charm and utter dependability.”

Similarly, in the manner of his mentor Harvey Cushing, Cairns could not neglect detail in his projects, publications, or the treatment of patients. Consequently, his legacy has been lasting.

Conclusions

Cairns, like his mentor Harvey Cushing, believed it important to definitively treat and debride head injuries as soon as possible after injury. Early in WWII he formed well-equipped MNSUs near the front lines for this purpose. Cairns also mandated crash helmets for motorcycle riders and enlisted the help of Oxford physicist A. H. S. Holbourn to improve upon helmet design and to study the biomechanics of brain injury. Cairns saw to it that careful records of the injured were kept for later analysis and publication. His Oxford team established the first hospital and rehabilitation centers for brain-injured patients, as well as the first spinal cord injury center. During this period Cairns, in conjunction with colleague Howard Florey, facilitated the earliest clinical trials of penicillin usage in hospitalized civilians and WWII military injuries, and demonstrated dramatic results despite the small amounts of the drug then available.


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Correspondence
James L. Stone, Department of Neurosurgery, NorthShore Neurological Institute, NorthShore University HealthSystem, 2650 Ridge Ave., 3rd Fl., Kellogg Bldg., Evanston, IL 60201. email: jlstone4@gmail.com.