



BRAIN HEMORRAGE: INTRODUCTION, PATHOPHYSIOLOGY, DIAGNOSTIC STUDIES, CLINIC PROBLEMS AND GUIDELINES FOR TREATMENT

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ABSTRACT :

Brain Hemorrhage is a form of stroke that occurs when an artery in the brain bursts, producing bleeding in the surrounding tissues. CT (Computed Tomography) pictures are used to diagnose internal bleeding and fractures. CT pictures are recommended over MRI (Magnetic Resonance Imaging) images because they are more widely available, less expensive, and sensitive to early stroke. These photos are first pre-processed, then morphologically operated on, and finally segmented using the watershed technique. The retrieved image is fed into an artificial neural network for categorization. It also provides information on the bleeding area and percentage. Brain hemorrhage, which accounts for 10% of all strokes, has a high morbidity and mortality rate. While the incidence of stroke due to occlusive cerebrovascular disease has decreased, the incidence of intracerebral hemorrhage has remained relatively stable. The syndromes caused by brain hemorrhage can be clinically recognized in many patients. Computed tomography (CT) has revolutionized the management of brain hemorrhage.

KEYWORDS: Computer Tomography; Brain hemorrhage; Magnetic Resonance Imaging; Convolutional neural networks

INTRODUCTION :

Machine learning techniques have been widely employed to evaluate medical datasets in recent decade. ^[1-3] Today, most modern hospitals are well-equipped with data monitoring and collection technologies. Computer-aided detection (CAD) systems rely on combining different data from current devices with powerful machine learning algorithms. CAD is now used routinely in clinical settings to detect crucial diseases including lung cancer and breast cancer due to its effective support capabilities. Additionally, CAD has been a significant research topic in medical imaging and diagnostic radiology ^[4]. Probabilistic models, such as support vector machines (SVMs) and artificial neural networks (ANNs), are widely used in machine learning methods. There has been extensive study on using artificial neural networks (ANNs) for medical decision assistance. ^[5] Deep learning, which takes advantage of high-performance computing (HPC) systems, has emerged as a promising solution for image processing. Deep neural networks (DNNs) have won various challenges for pattern recognition and machine learning ^[6]. DNNs require several processing layers with complicated topologies to model complex and partial real-world data, resulting in significant computational complexity. To be effective, DNNs require HPC systems to function properly. DNNs are infrequently employed in medicine, especially in CAD systems, due to their high hardware requirements and pricey datasets. Since 2015, DNNs have been used to detect diabetic retinopathy in retinal images, among other applications. Since 2015, DNNs have been used to detect diabetic retinopathy in retinal fundus pictures ^[7], chest pathology ^[8], thoraco-abdominal lymph node, and interstitial lung disease. ^[9] According to Adam R. D. ^[10], stroke is the third biggest cause of death, behind cardiology, vascular disease, and cancer. The World Health Organization defines stroke as "rapidly developing clinical signs of focal (or global) disruption of cerebral function, lasting 24 hours or longer or leading to death, with no apparent cause other than vascular origin". ^[11] Brain stroke is a significant medical illness with a high death rate, severe consequences, and economic and social costs. The death rate ranks second in underdeveloped countries and third in the US and other advanced countries, following cancer and heart disease. ^[12] There are two main types of stroke. Cerebral infarction and intracerebral hemorrhage. Cerebral infarction accounts for 75-80% of cases, while intracerebral hemorrhage affects 10-15% ^[13]. Over 50% of patients who experience an intracerebral hemorrhage die within the first year. ^[14] This is a medical emergency that requires immediate care to avoid serious, long-term, and potentially fatal consequences. In Vietnam, the annual stroke rate is 416/100,000, with 40.42% of cases resulting in cerebral hemorrhage. Over months, reparative mechanisms reduce the size of the hematoma. The process of reabsorbing the hematoma is delayed due to the need for macrophage activity along the rim. After a year, the hematoma site transforms into a slit-like chamber with orange-stained walls, including hemosiderin-laden macrophages and surrounding tissue that seems normal. ^[15]

PATHOPHYSIOLOGY :

Hypertensive brain hemorrhage typically affects specific areas, including the putamen, thalamus, cerebellum, and pons. Lobar hemorrhage can be caused by hypertension, but it can also occur when blood pressure is normal and no specific cause is identified.^[16] Spontaneous non-hypertensive cerebral bleeding could be caused by aneurysm, arteriovenous malformation (AVM), or primary or metastatic brain tumor. Examples of clotting disorders include leukemia, thrombotic thrombocytopenic purpura, sickle cell disease, cerebral arteritis from collagen vascular disease, amyloid angiopathy, and methamphetamine abuse. Hypertensive hemorrhage is typically caused by bleeding from the lenticulostriate and thalamoperforating arteries at the base of the brain, as well as the paramedian branches of the basilar artery.^[17] Pathological alterations can weaken these arteries, despite their usual ability to sustain high pressure without rupture. Although microaneurysms have been mentioned as a possible source of hemorrhage, histological studies show that fibrinoid necrosis is typically responsible for arterial wall weakening.^[18] The exact cause of the rupture is unknown, however abrupt increases in blood pressure due to effort or emotional stress may exceed the vascular wall's tolerance.^[19] Pathological tests show blood tracing along tissue planes and displacement of tissue. This effect, which is often more visible than tissue damage, offers hope for a better future than the acute deficit suggests. There is no association identified between hematoma size and blood pressure. The cause of eventual clinical worsening is unclear. Rebleeding is uncommon. In a study of chromium-labelled red cells injected at admission for hypertensive hemorrhage, patients who died showed little evidence of labelling in the original hemorrhage. However, Duret hemorrhages, which reflected the post-admission fatal cerebral herniation, were easily labeled. CT scans may show hematoma expansion and dye extravasation in arteriograms taken shortly after admission. Edema and ischemia necrosis around the lesion are the primary causes of further deterioration.²⁰²⁴ Over months, reparative mechanisms reduce the size of the hematoma. The process of reabsorbing the hematoma is delayed due to the need for macrophage activity along the rim.^[20]

DIAGNOSTIC STUDIES :

Patients suspected of suffering a brain hemorrhage should undergo an urgent CT scan. The scan identifies the location, size, and structure of the bleeding, as well as the extent of edema in surrounding brain tissue. The scan can be repeated to assess clinical outcomes. CT scans show a progressive transition from high density to isodense and low density over time. CT-autopsy correlation indicates that the change in scan appearance is caused by altered photon absorption, not hematoma resorption. After a hemorrhage, a CT scan with contrast may show a ring-like enhancement around the location, indicating edema or local ischemia infarction. This can occur from a few days to months. When the clinical condition and CT scan show a typical hypertensive hemorrhage, especially in the putamen, thalamus, cerebellum, or pons, angiography is rarely recommended. If hypertension is not the primary cause of lobar hemorrhages, angiography may be necessary to determine the presence of a vascular malformation or tumor.^[21] However, the method may not detect these lesions, especially during the acute period. We urge that if the initial angiography is negative and no other cause for the hematoma is found, the examination be repeated in 2-3 months when the hematoma's pressure has decreased. If no abnormalities are detected, a CT scan should be performed every 4–6 months to rule out the possibility of an underlying malignancy causing the hemorrhage. Every patient who has had a brain bleed should have their coagulation parameters evaluated. These should include the prothrombin time (PT), partial prothromboplastin time (PPT), and platelet count. Patients who are taking aspirin should have their bleeding time assessed.^[22]

MEDICAL TREATMENT :

After a CT scan confirms a brain hemorrhage, steps are done to stabilize blood pressure, avoid repeat hemorrhage, diminish mass effect, regulate edema, and prevent seizures. Efforts to stop hemorrhage in cases of ongoing bleeding have been challenging to evaluate due to the majority of hemorrhages stopping before the patient gets at the hospital. Recurrence of bleeding is uncommon, except in cases with aneurysms. Aminocaproic acid is exclusively advised for cerebral hemorrhages caused by ruptured aneurysms. Drug therapy helps to regulate hypertension. We do not propose lowering blood pressure below normotensive values due to decreased autoregulation in brain tissue surrounding the hemorrhage. A considerable fall in cerebral perfusion pressure may result in subsequent ischemic injury. When elevated intracranial pressure (ICP) is suspected, it should be treated aggressively. Steroids may be useful. We observed transitory worsening after premature steroid decrease, followed by improvement upon resuming high dosage. However, the efficacy of this therapy in a large number of instances has not been proven. One controlled research found no effect; however the vast majority of patients were in coma or severe stupor.^[23] Intravenous mannitol is an efficient and safe treatment for elevated ICP. Furosemide can be taken alone or with mannitol to increase its effectiveness. The stuporous or comatose patient must be intubated to ensure adequate breathing and the maintenance of a normal or, preferable, somewhat lowered pCO₂. Proper hydration and electrolyte control is crucial for all patients. One must watch for incorrect antidiuretic hormone release, which is not uncommon. Consciousness has proven to be the most accurate predictor of outcome. Most individuals with moderate-sized hematomas, as well as those with minor hematomas, will respond to medical treatment. These individuals are awake and may be seen by a clinician, and ICP monitoring is usually unnecessary. The use of continuous ICP monitoring in the management of patients with massive hematomas has been documented. A subarachnoid bolt or ventricular catheter can be utilized. The effects of hyperventilation, mannitol, and furosemide can then be carefully monitored. When these therapies fail to manage elevated ICP, surgical excision of the hematoma or substantial doses of barbituates are considered.^[24]

SURGICAL TREATMENT :

The indications for surgical treatment of brain hemorrhage continue to evolve. While not all patients have clear indications, clinical and CT guidelines for therapy are evolving. Several studies have looked into the scheduling of operations in patients with hypertensive hemorrhage. Some individuals have analysed the benefits of delayed versus early procedure. Surgery can save a patient's life while they are failing. It is unclear if removing a hematoma immediately or later can reduce morbidity in patients with stable moderate or severe neurological impairment. Single cases and limited series

indicate that surgery can reduce late morbidity. When a hematoma in any site is linked with a history of significant intracranial pressure and brain stem compression, emergency surgery is considered based on CT scan and physical examination results, as well as the initial response to medical therapy. Surgery is usually avoided if there is significant hemorrhage, loss of pupillary responsiveness and brain stem function, and no response to medicinal treatment. Direct surgery offers minimal benefit in situations of thalamic and pontine bleeding. There are clear reasons for surgery in individuals with cerebellar hemorrhage, and surgery may be considered in patients with putamina and lobar hemorrhages. This is explained in the following section. With the introduction of microsurgical techniques, there has been increased interest. With the development of microsurgical methods, there has been a greater interest in performing urgent surgery to remove the hematoma and obliterate the bleeding vessel. It is claimed that this not only stops additional bleeding but also minimizes neurological morbidity by preventing secondary alterations such as edema formation and ischemia damage caused by the hematoma's residual mass effect. Additional expertise is required before urgent surgery may be advised in neurologically stable patients. Typically, a craniotomy is performed to remove the hematoma while minimizing injury to neighbouring walls. When a tumor is detected, the walls of the hematoma should be carefully biopsied. 10 CT-guided stereotactic procedures have been shown to effectively remove hematomas in both acute and chronic stages. Specialized apparatus now allows for the subtotal removal of deep hematomas in individuals who were previously unable to undergo conventional surgery without causing damage to normal brain tissue. Whether this new strategy is safe and effective in a longer series needs to be shown.^[25] Postoperative care must be thorough to prevent recurrence. Blood pressure must be monitored while the anaesthetic wears off. Continuous blood pressure monitoring is required, especially throughout the patient's movement from the operating table to the bed and into the recovery room area. To keep blood pressure within normal limits, rapid-acting intravenous antihypertensive medications are delivered as needed. Steroid treatment is ongoing. In supratentorial lesions, an anticonvulsant is advised. Electrolytes are periodically tested. If the patient's level of consciousness decreases, intubation must be continued. The role of continuous intracranial pressure monitoring in postoperative care has not been determined. Any signs of deterioration should prompt a CT scan for examination. Postoperative consequences include recurrent bleeding, hydrocephalus, infection, and convulsions.^[26]

SPECIAL CLINIC PROBLEMS AND GUIDELINES FOR TREATMENT

Putaminal Hemorrhage

The putamen is the most common site of hypertensive hemorrhages. Hemorrhage may occur locally or spread to the white matter, frontal or temporal lobes, internal capsule, or ventricle. Larger lesions result in greater deficits and a worse prognosis. The clinical syndrome is well described. When patients detect a problem, they tend to become more alert and active. Then hemiparesis develops gradually and steadily. This can lead to hemiplegia, hemisensory loss, hemianopia, dysphasia in the dominant hemisphere, and unawareness of the impairment in the non-dominant hemisphere. The eyes frequently deviate to the side of the bleeding. The sickness can either end abruptly or lead to a coma and death within hours. In 27 cases, 62% had a smooth onset, while 30% had symptoms so quickly that observers felt the impairment was practically maximal at onset. Neither of the patients' deficits fluctuated. Only 14% experienced headaches at onset and 28% at any time, leaving approximately 72% headache-free despite significant neurological damage. Most small and moderate-sized hematomas in the putamen heal well on their own or with medical treatment. For hematomas greater than 3 cm, medical treatment is typically recommended. However, if the patient continues to experience neurological deficits or loss of consciousness despite aggressive medical treatment, surgical excision may be considered. A CT scan analysis of 24 patients with putaminal hemorrhage revealed three distinct groups. In the first group, individuals who were comatose upon admission had severe hemorrhages and had a dismal prognosis. The second group was alert but had significant neurological deficits and moderately large hematomas. While some made respectable recoveries, the bulk still faced considerable deficits. The third group had mild impairments, little hemorrhages on CT scan, and made a good recovery. It's unclear if surgery would have improved the outcomes in the first two groups.^[27]

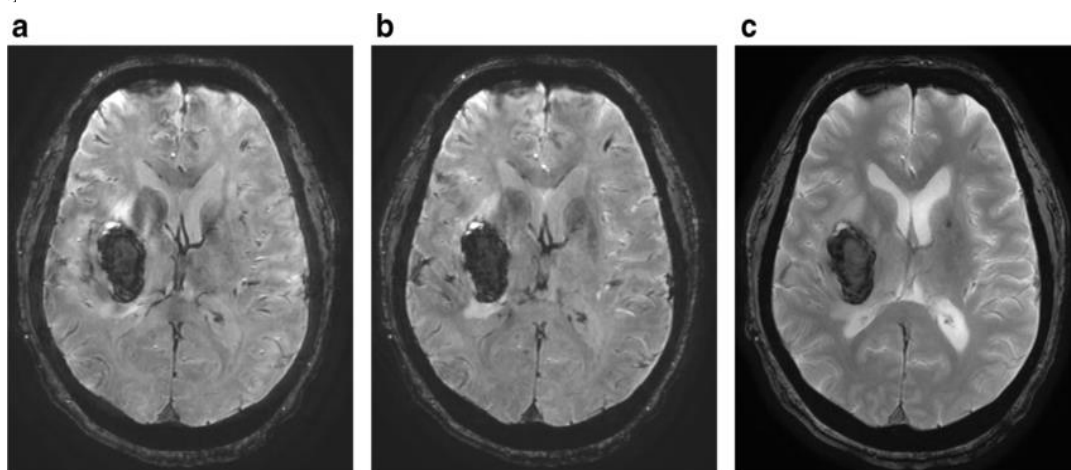


Fig.1 Putaminal Hemorrhage

Thalamic Hemorrhage

Classic symptoms include initial hemisensory loss and, if the internal capsule is implicated, hemiparesis. Extension into the upper brain stem can result in vertical gaze palsy, retraction nystagmus, skew deviation, loss of convergence, ptosis and miosis, anisocoria, or unresponsive pupils. Dysphasia can arise in people with left-sided hemorrhages. If the hematoma is big, profound coma may occur from the start. Headache is uncommon. Compression

of the cerebrospinal fluid channels can result in hydrocephalus. All deaths occurred when the hematoma measured more than 3.3 cm on a CT scan. Patients with minor hematomas generally recovered with impairment. Direct surgery may not benefit patients with bigger hematomas, according to current research. We have not operated on individuals with thalamic hemorrhage in the past, except to relieve hydrocephalus. This may occur suddenly and necessitate immediate ventricular draining. For certain patients, a permanent internal shunt may be necessary.^[28]

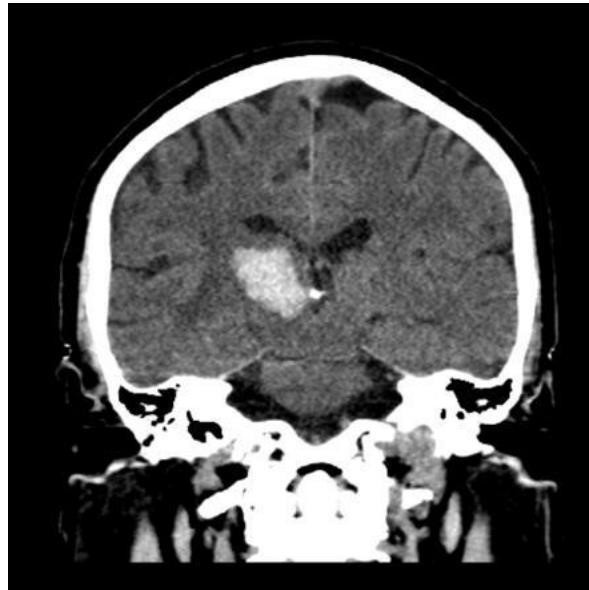


Fig.2 Thalamic Haemorrhage

Cerebella Hemorrhage

Hemorrhage in the cerebellum causes a potentially fatal illness that can be cured with urgent surgical intervention. This hemorrhage typically causes abrupt symptoms such as nausea, vomiting, and difficulty walking. A study of 56 patients with cerebellar hemorrhage found 75% had headaches, 55% had dizziness, and 14% had loss of consciousness at beginning. The examination revealed appendicular ataxia in 78%, facial palsy in 60%, and ipsilateral gaze palsy in 54%. In non-comatose patients, there is no distinguishable clinical sign that can predict survival with minor disability versus progression to brain stem compression and coma. In terms of treatment, the cerebellar hematoma represents a unique situation. Once brain stem compression has started, the damage it causes is unpredictable and frequently irreversible. Treating the patient before compression results in a change in consciousness and an unstable clinical situation is vitally crucial. Based on our observations, just four out of sixteen patients who were unconscious or comatose before to surgery survived, compared to ten of the twelve patients who were awake or sleepy. Other reviews have emphasized the significance of not postponing surgery for patients with acute cerebellar hematomas and the association between awareness level and outcome. However, it should be noted that emergency hematoma evacuation can still lead to a successful recovery for a patient who is in a severe coma. A full recovery may follow hematoma evacuation, particularly if there is little time between the onset of the comatose condition and surgery. Therefore, until there is clinical or CT indication that the brain stem has been obliterated, a patient who is in a coma due to cerebellar hemorrhage should not be denied surgery. Six of the ten patients in a report with cerebellar hematoma had a progressive course with early brain stem compression, and every patient had hematomas of three centimetres or more on CT scan. In general, if a patient is evaluated within the first week of symptom onset, we advise evacuation of hematomas larger than 3 cm in diameter. Individuals who present later and exhibit a stable neurological history may get medical therapy under watchful supervision.^[29]

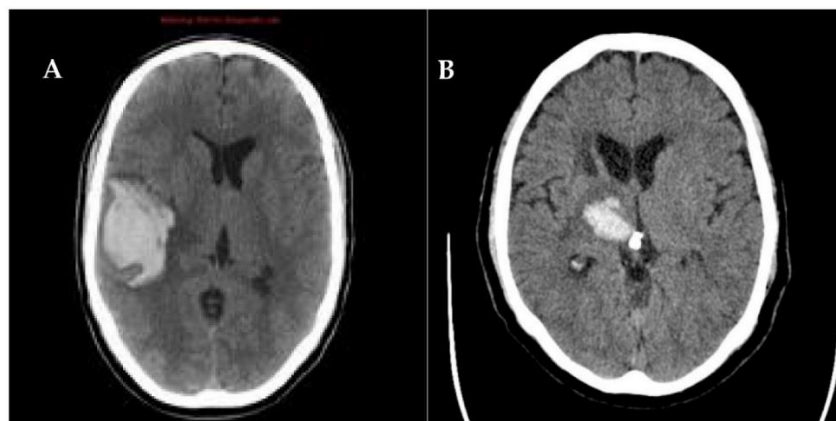


Fig.3 Cerebella Hemorrhage

Pontine Hemorrhage

This brain hemorrhage is among the most severe and difficult to treat. A little hematoma frequently causes a range of ocular motility abnormalities, fast quadriplegia, decerebrate rigidity, pin-point pupils that may hardly react to light, and an instantaneous coma. Ocular movements may allow the patient to communicate despite being paralyzed due to the unusually small bleeding, a condition known as a "lock-in" state. Most patients don't make it through the acute stage. There have been a few documented instances of pontine hematoma removals going well. Sano and Ochiai reviewed 24 patients who had pontine hematomas brought on by hypertension. Six patients with CT-detected hematomas smaller than 1.0 cm in diameter made it out alive, although just one of them is currently employed. Except for one, every patient with a hematoma larger than 1.0 cm passed away. To remove the hematoma, four underwent suboccipital craniectomy, and several underwent ventricular drainage. It was determined that there was little utility in direct operation.^[30]

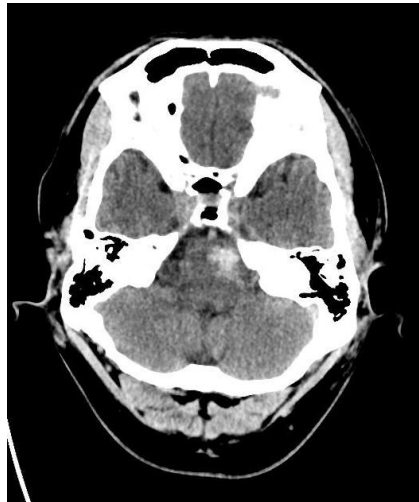


Fig.4 Pontine Hemorrhage

HEMORRHAGES DUE TO COAGULATION DISORDERS

Anticoagulant Therapy

Anticoagulants are widely used to treat a wide range of illnesses, which has led to a rise in the amount of individuals experiencing brain bleeding from this reason. Most individuals with these complications have a prothrombin time greater than the therapeutic range or a localized lesion, such as myocardial infarction, which explains the bleeding. When a patient using anticoagulants is not bleeding in other parts of their body, they may experience an isolated intracerebral hemorrhage. The initial assessment and course of treatment are identical to those outlined for cerebral hemorrhage with hypertension. Fresh frozen plasma transfusions immediately reverse anticoagulation. When vitamin K1 (phytonadione) preparation is administered, hemostasis is often maintained by restoring normal coagulation within 6 hours, with the exception of those who have considerable liver disease. Most of the time, operation can be carried out safely with these precautions. After surgery, prothrombin time should be rechecked and adjusted as needed.^[31]

Haemophilia

The majority of individuals with haemophilia are deficient in factor VIII, a smaller percentage in factor IX, and occasionally in factor XI. There is an extended partial thromboplastin time in every case. Several reports have provided an overview of the issue. While brain bleeding can happen on its own, it is typically linked to minor trauma. Any haemophiliac patient who reports having a constant headache ought to get a CT scan. As soon as bleeding is confirmed, the proper replacement therapy needs to begin. In order to stop additional spontaneous bleeding before or after surgery, transfusions of the right concentration must be used to keep the deficient factor at least 20% of the total. If a procedure is performed, replacement must be administered until the incision heals.⁸⁴ The indications for operation are the same as previously stated.^[32]

CONCLUSION :

Brain hemorrhages are an extra complication that trauma patients—especially those over the age of 55—experience when using antiplatelets and anticoagulants. The current study shows that these drugs come with an additional cost in the form of extra hospital and ICU days, return visits after release, intracranial bleeding requiring neurosurgical intervention, and hospital and mortality within three months. Because of this, trauma patients who experience head injuries should be seen as having a high risk factor for antiplatelet and anticoagulant use in the past. About half of these hemorrhages are caused by hypertension; the remaining ones are brought on by vascular malformations, tumours, aneurysms, inflammatory and degenerative vasculopathies, and hematologic and iatrogenic coagulation abnormalities. In certain cases, the cause is never identified. Depending on where it occurs, hypertensive brain hemorrhage in the deep gray nuclei of the hemispheres, the cerebellum, and the pons causes distinct clinical symptoms. In cases of lobar and putaminal hemorrhages, surgical hematoma removal is recommended if the patient's condition worsens despite receiving intensive medical

treatment. Furthermore, the majority of severe cerebellar hemorrhages (>3 cm) and smaller hemorrhages that cause a notable compression of the brain stem should be assessed. More research is required to determine how immediate surgery, elective late surgery, and intense medical care affect final functional outcomes

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