CPQ Medicine (2019) 7:1
Research Article

Open Cerebral Decompression -A New Method for the Treatment of Severe Traumatic Brain Injury

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Received: 22 May 2019

Published: 12 June 2019

Keywords: *Severe Traumatic Brain Injury (S-TBI); Open Cerebral Decompression (OCD)*

Abstract

Severe traumatic brain injury (S-TBI) carries high rates of mortality and disability. We developed a technique of Open Cerebral Decompression (OCD) in the treatment of patients with S-TBI based on our experience in clinical practice in neurosurgery and animal experimental studies. The use of this technique may result in a fundamental change in the treatment of S-TBI, and increase the survival rate and reduce the occurrence of complications and sequelae in these patients.

Animal experiments showed that OCD and closed cerebral decompression (CCD) were used to observe brain edema and pyramidal cell damage in rats after S-TBI. Brain edema in OCD was confined to cerebral cortex, most pyramidal cells were intact and vacuolation was rare. Brain slices using CCD showed that brain edema was very obvious, the range of edema was about 5 times that of OCD, and most pyramidal cells were vacuolated, only a few pyramidal cells survived.

Five patients with S-TBI, GSC coma index 3-5, were treated with OCD. One patient with GSC coma index 3 prolonged the survival period. The other four patients survived and took care of themselves.

In this article, the authors introduce the local mild hypothermia technique of the head, the procedure of OCD operation, the head protection device after operation, and the reduction technique of cerebral hernia, etc.

Introduction

The mortality and disability rates in cases of traumatic brain injury (TBI), especially in severe TBI (S-TBI), remain high. TBI is considered severe when the patient's Glasgow Coma Scale score is three to five. Of the 150,000 patients who died of traumatic injury each year in the United States, one third (approximately 50,000) died of brain injury [1]. In China the death rate in cases of S-TBI was as high as 60% to 90%. Therefore, the current methods used for the treatment of S-TBI are unsatisfactory [2] (DOI). To promote a remarkable increase in the TBI survival rate, we conducted a series of systematic studies of open cerebral decompression (OCD) to compare the rate of survival and adverse outcomes and sequelae against rat model data using closed cerebral decompression (CCD), including histopathological examinations of surgical tissue samples. Experiments were made in 80 rats, and the correlation between pathological sections of brain and clinical brain trauma was observed at serial time points after trauma. An improved free-fall apparatus was used to give impacts on the vault of skull of anesthetized rats with a 50-g to 200-g weight from a 20-cm to 50-cm height. The rats were observed for four hours to 12 days after impact, and the brains were collected after anesthesia to produce 652 pathological sections. We also conducted a study of the manifestation of closed decompression and open decompression in animal models with a comparison of the pathological changes. From these, we gained useful comparator data [3]. [https://www.cientperiodique.com/article/CPQME-5-2-120.pdf]

Materials and Methods

We included patients aged from 28 years to 68 years who were sent to the Shanghai Navy hospital with S-TBI (i.e., a Glasgow Coma Scale score of three to five) and intracranial pressure (ICP) greater than 5.3kPa in our study. Our inclusion criteria also included patients with severe brain contusion combined with intracranial hematoma and obvious brain swelling or encephalocele occurring during the operation. We also included patients with a cerebral hernia and diffuse cerebral axonal injury accompanied by intracranial hypertension. Our study included a total of five male patients with an average age of 40 years. One case suffered from severe coma, bilateral dilated pupils, bilateral subdural hematoma and encephalocele during operation. This patient received open decompression and a result, had a prolonged survival time. Before the operation, his GCS score was 3. Two cases had intracranial hypertension and antihypertensive drugs were used before and during operation for these cases. They each recovered and lived free of support. Their GCS scores before surgery were 4 and 5. In two cases of TBI respectively the skull was buried subcutaneously in the lower abdomen. They recovered well after the operation (Their GCS before surgery were all 5).

Preopeative Preparation

We maintained free air passages for the patients via tracheal intubation, tracheotomy and/or ventilator support. We used at least two venous passages, one of which was a deep venous passage. Patients received lumbar drainage tube placement after general anesthesia.

We monitored the patients' consciousness, pupils, blood pressure, pulse, pulmonary arterial pressure, and the partial pressure of oxygen and carbon dioxide. We also monitored ICP, cerebral perfusion pressure (CPP), cerebral blood flow and arteriovenous difference of oxygen. To prevent brain swelling and acute encephalocele during the operation, patients were hyperventilated at the beginning of the operation [4]. The systolic pressure was controlled at the lower limit of the normal CPP to ensure the normal supply of brain blood flow as the blood pressure dropped simultaneously [5]. Patients received a venous infusion of 20% mannitol, furosemide and human albumin to maintain a normal blood volume.

Surgical Operation

A T-shaped or H-shaped scalp incision was made for each patient. We used a new type of scalp incision nail to prevent bleeding on incision (Figure 1, Figure 2).

Figure 1: *Hemodynamic and transfusion*

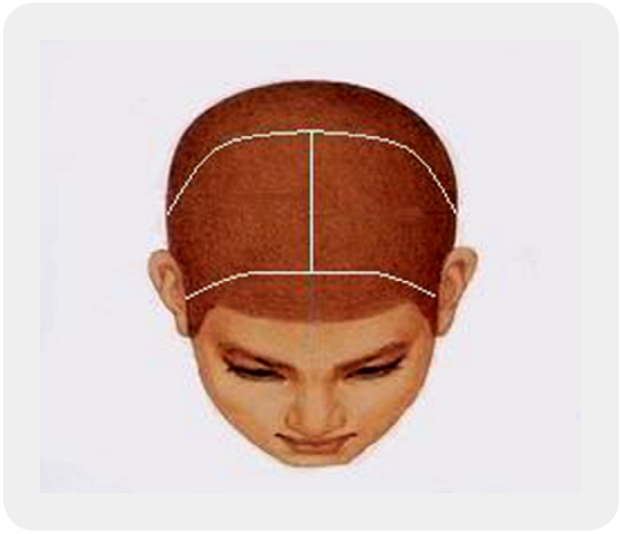

Figure 2: *H-type scalp incision*

We created a large bilateral bone flap extending to the lower edge of temporal bone and near the middle cranial fossa. In the event of a cerebral hernia during the procedure, we used a brain spatula equipped with light-emitting diodes to reposition the cerebral hernia followed by water infusion. If necessary, reposition was achieved using our fingers. We also created a tentorial margin incision with a knife specially made for cerebellum tentorial incisions and opening the bilateral cistern ambiens [6]. We performed a lateral ventricle puncture drainage and monitored ICP throughout [7].

Intracranial hematomas were removed as quickly as possible, and bleeding was stopped completely. After the cerebral hernia was relieved by incising the lateral dura mater of bilateral cistern ambiens, the lumbar drainage tube was opened, and a closed drainage bag connected [8]. The dura mater was incised but not sutured. The exposed surface of the brain was lightly covered with the dura mater. We used the artificial dura mater to cover the part lacking coverage.

Throughout the operation after dura mater suspension, cotton soaked with antibiotics was used for extensive coverage.

We used local epidural hypothermia rather than whole body cooling to prevent fluctuations in vital signs and protect the damaged brain tissues (Figure 3) [9]. Patients received a continuous drip of 35°C mild hypothermic solution containing antibiotics [gentamicin, 16u; add 500ml physiological saline (0.032mg/mL)] This could not only protect the brain tissue but also prevent intracranial infection. Excess fluid was removed via the drainage tube placed under the scalp. After head surgery, the removed skull was placed on the left and right sides of the skin under the straight incision of the lower abdomen of the umbilical part of the patient. After stabilization of the injury, the skull was restored to its original position.

The scalp was not sutured at this stage. The patient's whole head was protected by an aseptic helmet invented by the authors to protect the exposed brain tissue and prevent intracranial infection. The subcutaneous

drainage tube, ventricular drainage tube, ICP monitoring, low temperature control tube, ICP monitoring wire and brain temperature monitoring tube were all led out through corresponding channels in the helmet.

Postoperative Management

In the neurology intensive care unit (N-ICU) of an adjacent operation room, the patient was placed in a special aseptic cabin where all the monitoring and treatment were conducted until the patient's vital signs were stabilized, the ICP was normal, and the patient's pupils were bilaterally symmetrical. Patients usually spent seven to 10 days (mean, eight days) in the N-ICU to overcome the risk of cerebral edema, swelling, and hemorrhage. Patient blood pressure was regulated according to the ICP and CPP, and the use of hypotensors was gradually stopped.

Antibiotics were used to prevent and control infections [10]. The cabin was sterilized at regular intervals. The medical personnel were required to enter the cabin and dress themselves according to operating room protocols. The temperature in the cabin was kept at approximately 25°C. Rewarming of the head was carried out after the ICP gradually returned to a stable condition. After the injury condition was stabilized, the patient was sent to the operation room for dura mater suture, cranial bone repositioning and fixation, and scalp suture. The patient was transferred to the general ICU ward for further treatment and monitoring.

Results

By comparing the outcomes of our study in patients with the experimental models of rats with S-TBI, we found that OCD was more effective than CCD, and OCD produced a lower degree of brain edema and swelling than CCD (Figure 3, Figure 4). Moreover, pyramidal cells in the cerebral cortex were well preserved (Figure 5, Figure 6) [11].

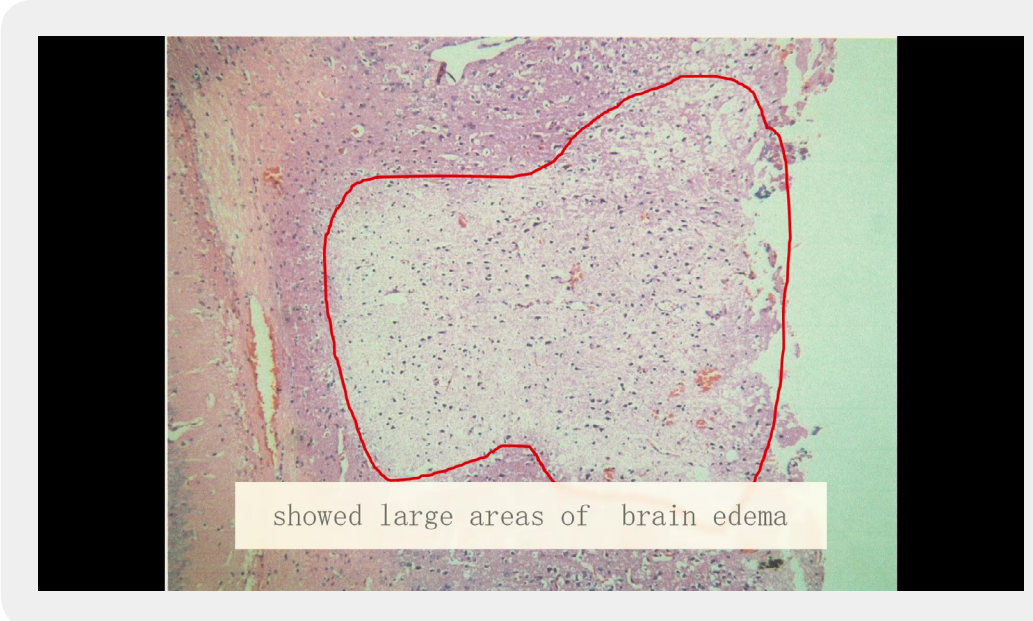

Figure 3: *Rats receiving closed decompression showed large areas of brain edema in the cerebral cortex zone*

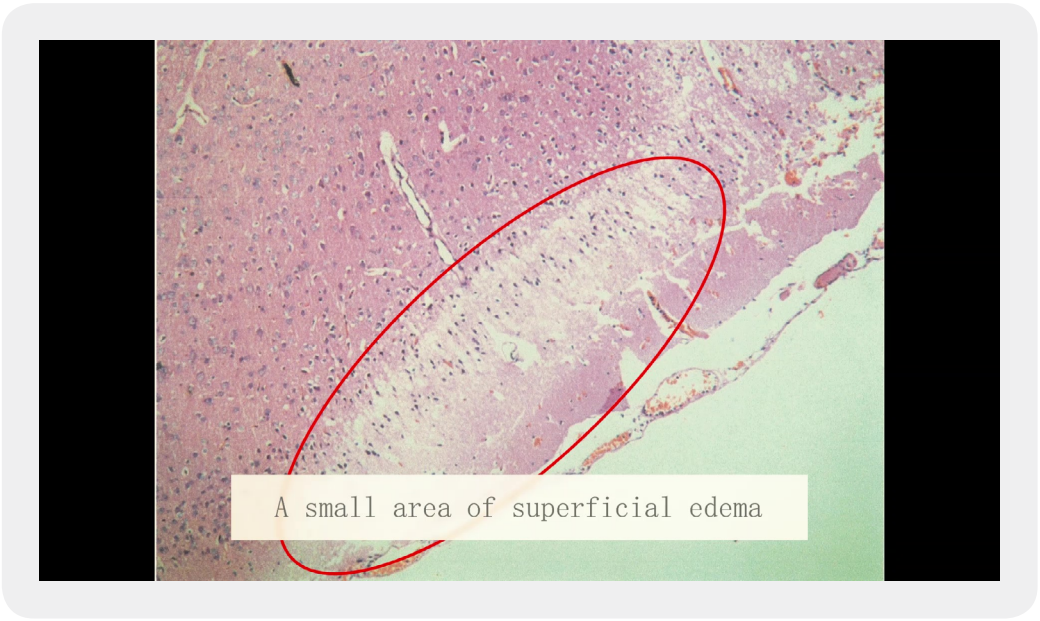

Figure 4: *A small area of superficial edema was found in the cerebral cortex zonein rats receiving open decompression*

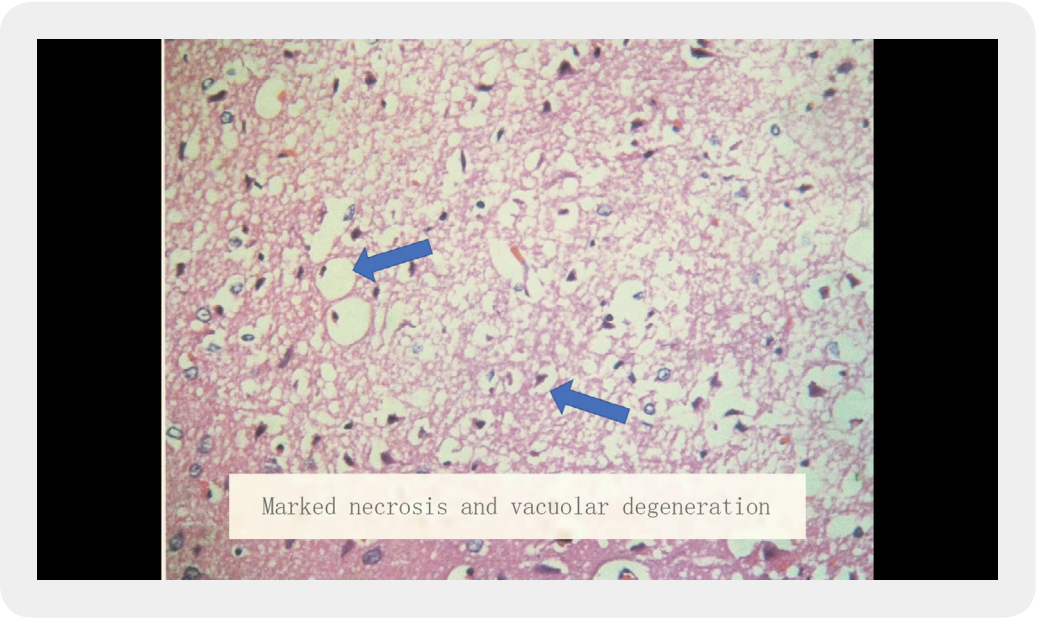

Figure 5: *Marked necrosis and vacuolar degeneration in most pyramidal cells after closed decompression*

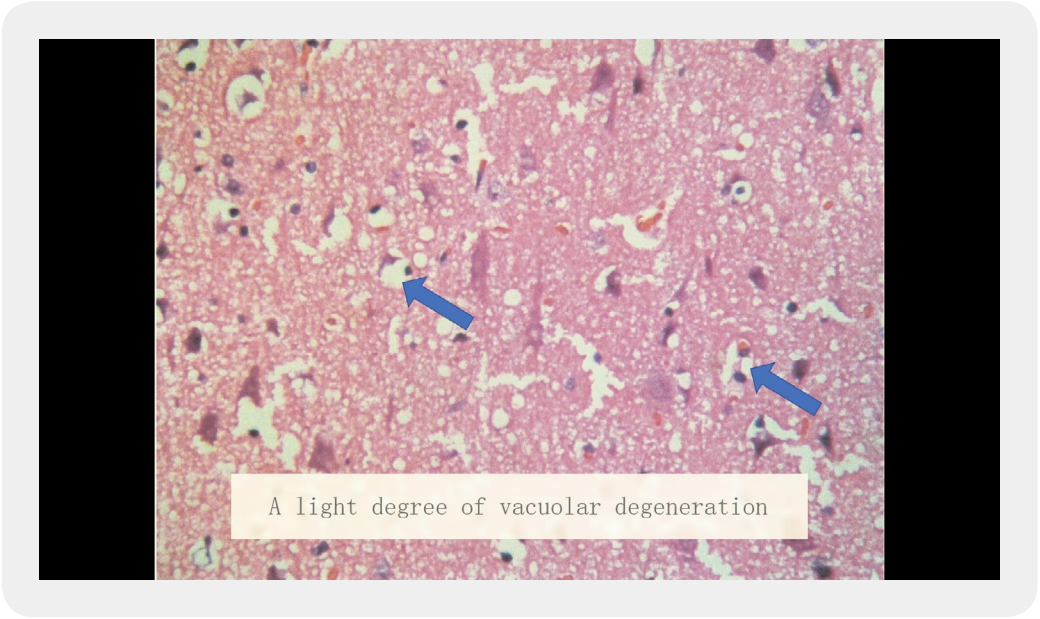

Figure 6: *A light degree of vacuolar degeneration after open decompression; most pyramidal cells*

From histopathologic samples in rat model experiments [3], we noted OCD had a lower degree of brain edema after injury than tissues collected from CCD. After OCD, edema was limited to the cortex and involved only a small area (Figure 4), while CCD showed edema five times thicker in a large area, including the deep glial region (Figure 5). Under 100x magnification, approximately half of the pyramidal cells of the cerebral cortex were well preserved in OCD cases (Figure 6), while in CCD samples, most pyramidal cells were necrotic (Figure 5).

Five patients with S-TBI were treated with OCD technology. Four patients recovered and discharged from hospital, and their normal life was restored. One patient's survival time was prolonged for 4 days.

Discussion

The main problem associated with OCD treatment was infections. Following measures were taken to prevent intracranial infections. After the operation described above had been completed, the brain was covered with the dura mater. A layer of brain cotton soaked with antibiotics was applied to the dura mater on its outside. Then the scalp enveloped in an aseptic membrane was laid upon the brain cotton. The head of the patient was protected by a special aseptic helmet (Figure 7). The patient was sent into an aseptic cabin with all kinds of instruments, first-aid devices, respirators and monitors. The cabin was sterilized at regular intervals. The medical personnel were asked to enter the cabin and dress themselves up as required in the operation room. The temperature in the cabin was kept at about 25°C. Since a series of preventive measures were taken the probability of intracranial infections was low and controllable in open cerebral decompression.

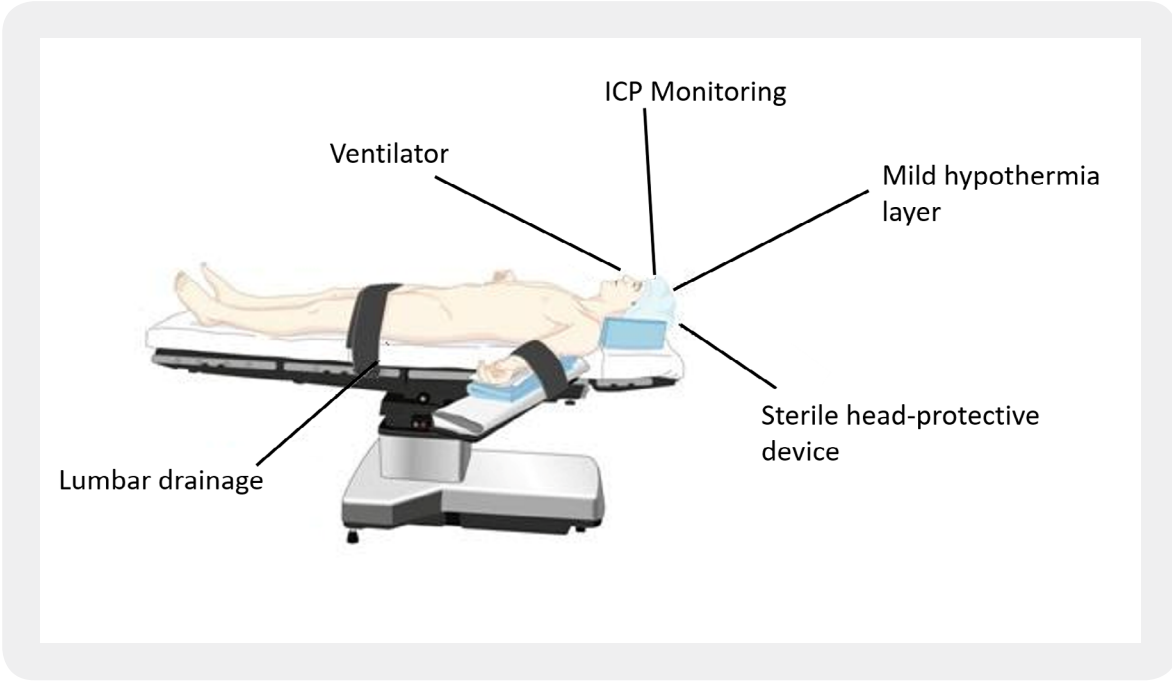

Figure 7: *The head of the patient was protected by a special aseptic head-protective helmet. Abbreviation: ICP, intracranial pressure.*

Measures for prevention and treatment of malignant intracranial hypertension were taken to prevent a series of primary and secondary brain injuries caused by intracranial hypertension, thus the occurrence of late complications and sequelae was reduced in S-TBI.

The S-TBI patient was able to overcome the risk of cerebral edema, swelling, hemorrhage and others in 7 to 10 days after injury. To protect the brain tissue a thin layer of brain cotton was placed under the scalp and a continuous drip of 35°C mild hypothermic solution containing antibiotics of a certain concentration was given. This could not only protect the brain tissue but also prevent intracranial infection. Of course it was necessary to drain the excess liquid with the drainage tube.

From pathologic sections in animal experiments it was seen that open cerebral decompression had a lesser degree of brain edema after injury than CCD. In the former (OCD) edema was limited to the cortex and involved only a small area (Figure 4), while in the later (CCD) edema was found in a large area and was 5 times thicker than the former, and involved the deep glial region (Figure 3).

Under the extra powerful microscope (with the visual field magnified 100 times) almost more than half of the pyramidal cells of cerebral cortex were well preserved in open decompression (Figure 6), while in closed decompression nearly all of them were necrotic Figure 5.

These experimental findings suggested that the technique of open cerebral decompression had a greater practical value. It could reduce cerebral edema and swelling after severe brain injury and help the patient to

pass through the period of cerebral edema safely. It was beneficial in maintaining the function of brain cells and reducing sequelae resulting from brain injury.

It was believed that the technique of open cerebral decompression (OCD) invented by the authors would provide a new method for the treatment of severe traumatic brain injury (S-TBI). It would play an important role in raising the survival rate and lowering the death rate, and decreasing and alleviating the sequelae in the S-TBI patients, and therefore bring more hope of survival to the patients. All these could be achieved by the use of this technique.

As for the treatment of S-TBI, neurosurgeons around the world generally use the guidelines for the treatment of severe craniocerebral trauma compiled by the American Association of Neurosurgers and the Brain Trauma Foundation for clinical operation. In recent years, mild hypothermia after brain injury has been used by neurosurgeons in some countries. After years of clinical practice, mild hypothermia does play a better role in brain protection. However, its application also brings some serious complications, such as pulmonary infection, Arrhythmia, decreased blood pressure, immune dysfunction and other consequences. In our OCD application, mild hypothermia is applied only to the head of the patient and normal temperature is applied to other parts of the body, which not only protects the damaged brain but also avoids serious complications of mild hypothermia in the whole body.

In view of this, the treatment of severe craniocerebral injury is still an important topic in neurosurgery research. Our ODC series technology provides an effective method and approach.

Conclusion

Our findings suggested that OCD has a greater practical and clinical value and may reduce cerebral edema and swelling after S-TBI compared to traditional treatments. OCD may maintain brain cell function and reduce brain injury sequelae.

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