one foot to the other, lest the jar of putting his heel heavily on the ground should hurt him.

By the beginning of June the nasal process and a large adjacent portion of the left maxilla was isolated and necrotic. The tissues of the face were attacked above the fold of the nostril and necrosed, leaving a large perforation with tender granular edges between the nose and the cheek, through which the dead bone could be seen and felt over a large area.

The treatment adopted during this time consisted in cauterization of the ulcerated surfaces by chemical caustics, such as acid nitrate of mercury; but though these appeared to arrest the disease now and then in places, it was quite evident that the general trend of the malady was from bad to worse.

We decided to try the effect of radium emanations, and accordingly, in June, four tubes obtained from the Laboratory of the Royal Dublin Society, of about 12 millicuries activity, in steel needles wrapped with tea lead, were placed through the perforation in the palate and left in for eighteen hours. In about two weeks it was quite evident that great improvement had resulted. The posterior part of the palatal perforation had epithelialized, and was no longer tender to the probe. A second dose of radium was given on July 17th, consisting of six tubes of about 20 millicuries activity in steel needles covered with eight wrappings of pure tinfoil. The needles were embedded in dental wax and left in for eighteen hours. In ten days the change was striking. The swelling had subsided, the tenderness reduced to a trifle, and the pain had eased off to such an extent that the patient had no difficulty in sleeping without drugs. Improvement was sustained, and in October the front of the left maxilla, which had become quite loose, was easily removed through the palatal perforation.

He put on flesh rapidly in the latter half of 1920, and remained quite well until Easter of this year, when a swelling appeared on the inner wall of the left nose near the body of the sphenoid. It was certainly a recurrence, but subsided within ten days after an exposure of 360 millicurie hours of emanation, screened through two layers of tea lead.

At my invitation the patient was seen in June by my laryngological colleagues Drs. Dempsey, Gogarty, Graham, Keogh, and Law, all of whom confessed that the case was unique in their experience.

One must hesitate before concluding that a case belongs to a new class, even though its clinical features differ from everyday experience. The vagaries of syphilis in particular are so notorious that the greatest caution must be exercised. I felt that in these cases syphilis was the one disease that needed the most rigorous exclusion. Not being a believer in the constancy of the Wassermann reaction in syphilis I placed no reliance on a negative report, though I am of opinion that, at least in the second case, the acuteness of the disease was such that the negative Wassermann amounted to a positive exclusion. But when, in addition to this and other evidence, all the best tried and most valued remedies are applied, as they were in both these cases, without the least effect on their progress, the case for its exclusion is overwhelming.

There remains to be considered the nature of the process in these two cases, for, although differing from one another in some important respects, their similarities make it likely that they resulted from the same cause.

Granulation tissue is usually the result of that attempt at repair that follows tissue destruction, and when the cause of destruction is bacterial there is little difficulty nowadays in recognizing the organism. In these cases no such organism could be found, and the presumption is raised that the granulation tissue, so far from being an evidence of attempted healing, is itself the primary cause of destruction.

Until we understand what is meant by a cell taking on malignant action we cannot doubt that what happens to one kind of cell may happen to another, and if we can have a malignant epithelioma, why not malignant granuloma?

The pathological side of the question, while of the highest interest and importance, must from the clinician's point of view give place to the interest attaching to the fact that the first case failed to be cured by x ray, and the second was certainly cured by radium.

INFECTIVE EXTERNAL HYDROCEPHALUS

("SHIFTING EPILEPSY").

BY

CECIL E. REYNOLDS, M.D., D.P.H., M.R.C.S.,

LOS ANGELES.

[With Special Plate.]

THE following case, at one time or another, presented all the symptoms of the various other cases of epilepsy due to localized hydrocephalus which I have reported elsewhere.

History.

L. H., female, born October 29th, 1911, of good stock, was healthy until she had whooping-cough in June, 1916, the cough of which dragged on until December, 1916, during which month she had sore throat and fever. Her tonsils were removed in January, 1917. From this time until March she would cry out in her sleep. During March she contracted a habit of constantly rubbing the palm of the right hand upon the head of her "Teddy Bear," and when questioned, said her hand itched. Later in March, 1917, she ran to her mother during the day with her right hand in a state of involuntary flexion. This contraction was repeated with increasing frequency and severity, and in a few weeks it became so strong as to resist her father's efforts to open the hand. Consciousness was unaffected in these attacks. In April, 1917, it was discovered that she was having, about four times a night, attacks in which the whole body stiffened and the right arm was convulsed. The attacks from June onward became solely nocturnal, and were more severe and frequent, especially after a febrile attack in July, in which the temperature was 103°. Her memory appeared to be failing. During this time she complained often of headache "all over," and of nausea, but seldom if ever vomited; she was obstinately constipated, and her nose bled almost every week. The appetite was poor, but the diet had been greatly restricted. A Wassermann test of the blood was negative. The condition became steadily worse, until about October 5th she was having almost continuous fits every night, and on October 18th remained unconscious, cyanosed and rigid for so long that death was expected.

On October 19th, 1918, I met Dr. J. H. Utley and Dr. William A. Edwards in consultation, and, operation being agreed upon, she was admitted to the California Hospital under my care.

Condition on Admission.

The patient is a fairly well nourished child of 7 years, mentally bright to the point of precocity, over-restless, and inclined to laugh hysterically, good tempered, walks "pigeon-toed," the right foot being more inverted. The optic discs have clear, sharp margins; the temporal sides are rather pale, and the veins a trifle large. Alternating internal strabismus definitely present though slight. Slight lateral nystagmus to the left seen once. Pupils normal in size and contour, equal, and react well to light and accommodation. Slight weakness of the right angle of the mouth when at rest and on voluntary movement. No other muscular weakness discernible, but, although right-handed, she is at least as strong in her left hand as her right. Sensation everywhere very keen and well localized. Slight Rombergism present. Position sense of right arm rather less certain. Arm co-ordination excellent and pointing normal. Knee-jerks equal and exaggerated. No ankle clonus, plantar reflexes flexor. Temperature 98.6°, pulse 94; weight 50 lb.

Before she was soundly asleep at night the fits began with tonic contraction of the flexors of the body, extension and adduction of the arms, and extension of the knees with flexion of the hips. The emprostotonos was so great that the head nearly touched the thighs. Shortly afterwards there occurred blinking movements of the eyelids, the right angle of the mouth was drawn clonically to the right, the formerly extended right wrist was clonically flexed and the convulsion spread to the right arm. The left arm and both legs remained rigid throughout. During some fits she would awaken whilst the right arm and face were in clonus, and ask to have them stopped. Often she awoke aphasic or anarthric, and then would be greatly terrified. Usually she wept bitterly on coming out of a fit; at night, if awake without having had a fit, she seemed peevish and made even "fighting and striking-out" movements. During the fit the eyes were obscured by the clonus of the lids, but it is probable that they were jerked to the right, for after the fit they moved slowly to the left, as if the opposite group of muscles were weak. The right arm was paretic after the fits, and the right side of the mouth drooped more. In less than 1 per cent. of the fits there was opisthotonos instead of emprostotonos. These fits, solely nocturnal, occurred every ten minutes or more all through the night.

FIG. 1.—Second, third, and fourth operations (subtemporal decompression; right osteoplastic flap; and insertion of gold tube).

FIG. 2.—Operations 1 to 5. A. Left osteoplastic flap. B. Right osteoplastic flap and subtemporal decompression. C. Gold tube. D. Trephine hole for gold tube. E. Suboccipital decompression.

FIG. 3.—Fifth operation (suboccipital incision): exposing both cerebellar hemispheres and dura covering fourth ventricle and medulla.

FIG. 4.—The patient nine months after the last operation.

BROOK: TRANSFIXION AND IMPALEMENT INJURIES.

FIG. 1.

FIG. 2.

There were thus three types of fit seen, namely—

Cerebello-medullary or anterior vermis defect (tonic emprostotonus and tonic rigidity of limbs).

Inter-peduncular or posterior vermis defect (tonic opisthotonus and tonic rigidity of limbs and alarming depression).

Left Rolandic (clonic contractions of right angle of mouth, blinking of eyelids, deviation of eyes to right, and, later, clonic flexion of the recently hyper-extended wrist and arm of the right side).

TREATMENT.

First Operation: First Stage.

October 24th, 1918, at 10 a.m. The blood pressure at time of incision was 117, and pulse 100. A bevel-edged osteoplastic flap reflected, and palpation over the left arm and hand centres revealed fluid under the dura, which was incised at the upper margin of the window; much subarachnoid oedema was evident. The blood pressure then fell suddenly from 115 to 80, and the pulse rate increased to 153. Accordingly I tied off the larger branch of the middle meningeal artery by under-running, and replaced the bone-scalp flap, leaving a thin flat rubber drain at the postero-superior angle, and returned the patient to bed at 10.55 a.m.

The shock became more profound as fluid drained from the wound, probably forced through the arachnoid by pressure. In spite of this, it seemed best to combat the shock by keeping the head low, bandaging the legs, hypodermic injections of camphorated oil, and a rectal drip of hot tea and brandy. An anxious time for about five hours. She improved until October 29th, when she had twenty fits during the night, each about fifteen seconds' duration.

Second Stage of First Operation.

October 30th. Blood pressure at commencement 98, pulse 140. Osteoplastic flap non-adherent to the dura prised up without difficulty. The dura was covered with a little fibrin. The former incision was easily recognized, and reflection of the dura was continued from the point where it had been formerly interrupted; it was very thick. A flap, comprising the whole exposed area except a thin margin around the edge of the bony defect, was reflected and retracted sufficiently to leave an area of denuded cortex a good inch wide at the sides and above when the dura was laid back upon the bulging brain. Ventricular puncture was not performed. Marked subarachnoid oedema was visible over this area and the channels that lodge the middle cerebral vessels were filled with a clear yellowish fluid. The superficial wall of arachnoid covering this marsh was incised in several places, especially just above the Sylvian point. The meningeal vessels did not bleed, but fine sutures were passed around both sides of the flap base as a precautionary measure. The cortex was washed with lotio hydrarg. perchlor. 1 in 4,000, the dura laid smoothly on the cortex, and the bone-scalp flap replaced leaving an unstuffed thin rubber cigarette drain in the postero-superior angle of the flap. Blood pressure 94, pulse 136.

This completed the second stage of the Rolandic de-duralizing operation, but it was now clear that a permanent subtemporal decompression to relieve the pressure would have to be done at the earliest favourable moment on the opposite (right) side. Largely owing to the admirable even, light ether anaesthesia by Dr. T. C. Low, the patient's condition was excellent after this operation. She was returned to bed with the head kept high, and improved until, at 2 a.m. on October 31st, there was much trembling of the right hand and arm for about a minute; had seven spasms of the old emprostotonus type, some with the legs flexed and some with legs extended, and followed by right arm clonus and accompanied by blinking of the eyes. At 7.45 a.m. she developed anarthria after a fit. At 11.30 a.m. I found her in very good condition but complaining of pain in the right great toe. As the drainage tube was lying against the cortex in the neighbourhood of the sensory toe area I removed it, and this symptom abated very shortly. She had a good day, but that night, following a tonic (cerebellar) spasm, she had a Jacksonian attack, with definite coarse clonic contractions of the right arm, of which she was conscious throughout, asking to have them stopped. At 1 p.m. next day she had a tonic spasm, in which both eyes turned upwards. At 1.40 a.m. I found that she had a marked right-sided paralysis of the arm and angle of the mouth and was unable to speak, but understood what was said. Power of speech had returned at 4.30 a.m. At 5.30 a.m. involuntary micturition during a fit was noticed for the first time. She had four severe and twelve light fits during the night; temperature 100.4°, pulse 136, respirations 20. At 10 a.m. (November 1st), when the wound was dressed, clear fluid escaped. At 2 p.m. the arm was

somewhat recovered as she had been sitting very vertically, but she looked very ill and the jaw had a tendency to drop. Slight anarthria. 4 p.m.: Deep sighing and yawning (cerebello-medullary oedema).

Second Operation.

Blood pressure 110 and pulse 120. A generous and low-situated subtemporal decompression was performed on November 1st and the dura opened to the fullest extent. A great excess of fluid, about a demi-tasse, escaped from the subdural space. The blood pressure at once rose from 110 to 130 and the pulse frequency dropped from 145 to 133. The dura was left wide open, the vertical separation in the temporal muscle brought together with catgut, the temporal fascia replaced, and a thin unstuffed cigarette drain left under the fascia at the postero-inferior angle. Blood pressure 130, pulse 130.

The patient was now turned around and, while still in the semi-recumbent posture, the left Rolandic area was further inspected. The bone flap was readily lifted and had not appreciably adhered either to the pia arachnoid or to the dural flap. The latter was quite flat and smooth but even more shrunken than before, greyer, and less congested. The arachnoid was glazed and closely applied to the pia and cortex, and there were only slight traces of blood around the margin of the skull defect. On raising the dural flap the under side was slightly sticky. Everything was replaced and the patient returned to bed at 10.3 p.m. with a blood pressure of 94 and pulse 148. Head raised.

She slept well from 10.15 until 12.30, when she had a customary emprostotonus with both arms extended in front of the body. Slept till 1.30 a.m., November 2nd. At 2.25 a.m. a very severe Jacksonian fit began, affecting the right arm and angle of the mouth. Morphine gr. 1/20 hypodermically and bromide and chloral were given per rectum, but, until she was changed from the sitting posture and laid on the left side, the violent contractions were only checked by full chloroform narcosis and recurred when it was withdrawn. The drain was removed. After this the fits were easily controlled by chloroform. They were probably due to too rapid drainage.

These fits, in lesser degree and combined at times with those of the basal type, recurred all day on November 2nd, and in the intervals the right hand kept up a coarse tremor with the thumb flexed into the palm. The head and eyes were forcibly deviated to the right during the seizures, and she was conscious throughout many that I saw, her attempts to cry out being stifled by the spasms of the mouth and throat. Amyl nitrite and oxygen seemed of little value, but chloroform relieved at once; only in the first attack did it have to be pushed. I was ably relieved during this trying time by Dr. A. Zuber of the California Hospital, to whom I am indebted for the blood-pressure records taken during all the operations. I made the following notes at the times specified at the bedside, and they seem to be highly significant observations in regard to the fluid-pressure cause of these fits:

November 3rd, 8.45 a.m. Patient recumbent. Pure Jacksonian fit right arm and face (clonic). 4.20 p.m. Still recumbent. Jacksonian clonic fit of the right arm and eyes to the right. Speech impossible, hebeticude marked. Temperature 98°, pulse 122, respirations 20. 4.45 p.m. Up on back-rest. 5 p.m. Clonic cortical fit of mouth to the right. The arm did not participate in this attack. 5.15 p.m. Still up on back-rest. Tonic basal spasm, both arms extended. No clonic. Mentally clear after it. Pulse 120, good volume. 5.50 p.m. Still on back-rest. Tonic basal spasm, followed by clonic of angle of mouth and tongue. Fully conscious of clonus and asks for CHCl₃, which at once arrests the twitching. Speech clearing markedly. Whole attack much milder. 6.23 p.m. Still on back-rest. Another fit, still milder.

This was the last clonic fit until November 30th, when they temporarily reappeared as described later.

It seems obvious why the cortical fits disappeared when she was raised in bed on the afternoon of November 3rd, since they vanished first from the arm, which is highest of the affected centres, and later from the mouth. The subsequent fits were tonic in character, being mild forms of what I had previously regarded as cerebellar or basal fits. The "shifting epilepsy" could only have been caused by fluid as it shifted according to posture—if one lowered the head the fits became cortical-clonic; if one raised it they became basal-tonic; and as one moved the patient they disappeared first from the higher placed centres.

On November 17th she had seven very slight and brief tonic spasms during the night; this was the best night she had had for more than a year. She was not herself aware of having had any "spells" since November 3rd.

On November 23rd the temperature at 8 a.m. was 99°. At 9 a.m. I performed a lumbar puncture under light chloroform narcosis, and with the patient recumbent upon her left side. Fluid came out under very great pressure. In all less than

20 c.cm. were allowed to escape; it was quite clear and colourless.

There was no ill effect afterwards on this day, although the temperature rose to 99.4° and the pulse to 104 in the evening. It was considered safe to remove this quantity of fluid because the lack of bulging in the subtemporal region argued against hydrocephalus ventriculi—a condition which, when present, would render lumbar puncture dangerous—and also because the subdural location of the fluid found at the second (the subtemporal) operation gave the assurance that the intrathecal pressure was due to a pond under the cerebellum.

On November 24th, 1918, she had only three very slight spasms. There were no fits of any kind on the night of November 27th, and she slept well.

Report on Spinal Fluid.—Cell count: 2 mononuclears per 1 c.mm. Butyric acid test: Negative with 0.2 c.cm. spinal fluid. Wassermann test: Negative; 1 c.cm. spinal fluid produces no fixation with 1 unit of complement.

The trouble began again on November 30th with seven fits; five light, two severe. December 1st: Six fits last night (basal type). Discs normal; no congestion of the retinal vessels. Urine highly alkaline, otherwise normal. December 2nd: Ten fits last night (basal type). December 3rd: Eleven fits. On December 4th the fits had again assumed a cortical-clonic character, and at 7 a.m. next day I found her comatose with eyes deviated to the right, but the limbs at rest, and was informed that these urgent symptoms had been present for half an hour; also that she had had severe opisthotonos. She soon developed myoclonic shocks of the left arm, hand, and face, and the head and eyes were severely jerked to the left. Throughout this attack, which lasted about twenty minutes, and until she was under chloroform, the right side remained passive; the right side had been clonically convulsed before the first operation.

While she was under chloroform I performed a lumbar puncture between the fourth and fifth lumbar spines, and the fluid oozed out slowly drop by drop, so that in ten minutes I had only collected 3 c.cm. of clear fluid. It was therefore under greatly reduced pressure. I now put the needle well into the theca between the third and fourth spines, but no fluid came. I had felt the needle puncture the dura as usual, and the stylette found the needle clear, so that it was evidently a genuine "dry tap." At the same time it was noticeable that the subtemporal region on the right side was very tense.

The opinion was formed that an acute accession of internal hydrocephalus had followed the valve-like closure of the ventricular outlets, especially the foramen of Magendie. In other words, the insufficient relief of the external hydrocephalus had secondarily brought about a condition of internal hydrocephalus, which in turn displaced the fluid of the cisterna basalis upward and corked up the foramen magnum. It will be remembered that at the previous lumbar puncture the fluid was under high pressure, and remained so in the spinal theca after the needle had been withdrawn. It was now discovered, during the semi-lucid period, that she was hemiplegic on the left side, the leg being only slightly affected.

Third Operation.

She was removed to the California Hospital, where I enlarged the subtemporal decompression. The adhesions between the cortex and the temporal muscle were so firm that they could not be separated without haemorrhage. It was possible, however, to continue the enlarging with rongeurs from the edge of the old opening. Above this decompression an osteoplastic flap was cut with its base upward, the two sides being made with a Gigli wire saw, which was easily passed up to the drill holes from the large opening below, and on breaking the flap upwards an excellent hinge was obtained in the upper parietal region. The dura bulged, and pulsation was not observed. Reflection of the dura revealed considerable general pressure and much subarachnoid fluid, which flowed away freely after the arachnoid had been opened at the lower part of the flap and just above the old subtemporal adhesions. The dura was left open, being laid back upon the brain under the osteoplastic flap, but in the temporal region a large strip of dura was brought through the temporal muscle and stitched to the deep aspect of the skin flap in the hope of increasing the drainage. Two rubber cigarette drains (unstuffed) were left in contact with the temporal cortex and brought out externally. Blood pressure was 105 mm. at the commencement and at the end of the operation, and the pulse 120 to 140.

There was no vomiting after the operation and consciousness was soon regained, her mental condition becoming excellent. Immediate and profuse flow of cerebrospinal fluid necessitated frequent change of outer dressings during the night. There were no fits of any kind this

night and she slept well. On the morning of December 6th, while sitting up, she had a severe *grand mal* attack, lasting a minute and a half. This ceased, and was not repeated when she was placed recumbent, and was evidently due to too rapid drainage. The drains were removed at noon. She remained in excellent condition (lying down) and slept from 6 p.m. till 8.40 p.m., when she had a slight spasm. Next day, at noon, there was a spasm lasting two minutes, and another at 4 o'clock. At 5.30 a.m., while still lying low, she had another fit, cortical-clonic in character, the convulsions of the mouth continuing after she awakened. I gave instructions to raise her to a sitting posture; the twitching promptly ceased, and the speech, which had been much affected for more than an hour, returned and she lost the dysphagia. It was now more obvious than ever that we had to maintain a balance between the fluid accumulating at the base and that collecting at the vertex, according to symptoms. I left standing instructions that if, after a fit, the colour was bad and there were signs of collapse, she was to be placed in the recumbent posture; if the colour and pulse were good but the speech thick and the mind dulled, she was to be propped up. This had a good effect, and at 10 a.m. she was as bright as usual.

Fourth Operation.

The fourth operation was performed on December 7th. Somnoform and ether was administered by Dr. T. C. Low. Blood pressure 108, pulse 145. A quarter-inch trephine hole was made over the posterior end of the right superior temporal sphenoidal convolution, and another small hole made with the perforator of a Hudson drill about half an inch lower. By passing a protector between the bone and the dura this second small hole was reamed out so as to fit exactly the central part of the female half of the wide-bore flanged gold tube (14 carat). The dura was now cut in a circle as large as the drill-hole, and the female half of the tube passed from the subarachnoid space through the dura and the drill-hole in the bone; the male half of the tube was screwed home, so that the bone and dura were clamped, not too tightly, between the two flanges. In view of future growth enough space was allowed to sew the periosteum and some loose tissue between the male (external) flange and the outer table of the skull. During the insertion of the female or inner half of the tube a fine thread was fitted through a hole in the flange in case the tube slipped during insertion; this thread was removed as soon as the tube was well home. The whole procedure was made more difficult by the increased intracerebral pressure existing at this time, but as this was not very excessive I did not tap the ventricle, preferring puncture of the corpus callosum when driven to it. Moreover, I was working on the principle that if the external hydrocephalus were permanently relieved the internal hydrocephalus could take care of itself, as there was reason to believe that the iter and foramen of Magendie were patent as long as the fluid was being absorbed with sufficient rapidity external to them. The patient was returned to bed at 12 noon, with a blood pressure of 116, and pulse 156; good volume. Diastolic pressure, 78.

Since this operation the patient has never had another cortical-clonic fit, with one doubtful exception on December 17th. The scalp healed promptly over the tube, which did its work well in combination with the dural valve in the subtemporal region, for fluid was evident in the loose cellular tissue of the scalp at the time of the fifth operation (suboccipital) described below, and marked oedema of the right side of the face was periodically visible as late as January 10th, 1919.

There were no more fits of any kind, either day or night, until December 12th, and during these five days of freedom and absorption the temperature was normal in the mornings and averaged 99.6° F. in the evenings; pulse 110. The decompression did not bulge. The nose bled once.

On December 12th, at 3.40 a.m., while sleeping and fairly low on the pillows, she had a slight tonic basal spasm, and at 10 a.m. her articulation was temporarily affected but her mentality bright as usual. December 13th: A good night and very bright and cheerful; slight epistaxis. December 14th: A good night and no more spasms. December 15th: A good night, though slightly more restless; bright all day. On December 16th slight basal spasms reappeared, and on December 17th, at 5.35 p.m., she had in my presence, while awake and lying low in the bed, a purely basal tonic spasm lasting 35 seconds. In this the head approximated the thighs, as in the initial basal element of her pre-operative fits. Lumbar puncture revealed fluid under strong pressure, but not so strong as on November 23rd. (It will be remembered that on December 5th the spinal theca was practically dry.) During this night she had eight purely basal spasms. December 18th: The lumbar puncture seemed to have brought about an exacerbation of basal pressure, as she had increasingly frequent basal fits, until December 20th, when I again operated.

Fifth Operation.

9.19 a.m. Operation under somnoform and ether. Blood pressure at the time of incision 104, pulse 126. The skin flaps

of Cushing's crossbow method to expose both cerebellar hemispheres were turned down with minimal bleeding; but when the patient, who was lying in the semi-prone position, was turned a little further on to the right side to permit easier reflection of the muscles, the pulse disappeared at the wrist. Accordingly, the skin flaps were replaced and covered with a dressing, the head was lowered, and venesection of the median basilic vein and saline infusion of the median cephalic vein were performed. The collapse, probably due to a cerebellar fit under the anaesthetic, occurred at 9.36 a.m., and by 10.20 a.m. the operation could be resumed with the patient's blood pressure 98 systolic, 50 diastolic, pulse 110. The cellular tissue of the scalp over the occiput was found on incision to contain considerable fluid from drainage out of the openings above the tentorium previously described.

The patient being placed now in an almost upright sitting posture, a trephine hole was made to the left of the occipital sinuses and just above the margin of the foramen magnum. The bone was further removed with rongeurs and the de Vilbiss forceps until the posterior half of the foramen magnum had been taken away, the dura over the medulla, fourth ventricle, and occipital sinuses fully exposed, and both cerebellar hemispheres denuded of bone within the limits of the superior curved line. The dura was more adherent to the bone than usual and in places appeared thickened. Pulsation was not readily felt or seen, and upon incising the dura the arachnoid was found to be adherent to its deep aspect. Much fluid between the dura-arachnoid and the cerebellar substance obscured the latter from view even in the highest part of the wound, a bluish thin-walled sac of fluid coming into view, upon opening which much fluid gushed out.

The blood-pressure records (mm. Hg) and pulse rates are as follows:

A.M.		Systolic.	Diastolic.	Pulse.
10.35.	Before opening skull ...	92	50	130
10.39.	After opening skull ...	100	50	102
10.49.	Bone removal complete ...	92	53	140
10.51.	Dura reflected ...	96	50	120
11.15.	Subsequent manipulations...	84 (lowest)	50	124
11.18.	Wound closed ...	96	52	140

The dura was fully opened over both cerebellar hemispheres, leaving intact that over the occipital sinuses. The dura covering the fourth ventricle was not incised, as it was felt that the posterior medullary velum would, in this case, allow the discharge of fluid from the ventricle when its lateral supports had been removed. Accordingly, a large flap of dura-arachnoid was turned upwards on the left side and brought through a slit in the upper fringe of complexus muscle and stitched to the fascia covering the occiput just above. Upon attempting to do the same thing on the right side by turning a dural flap outwards, the dura here was found to be so friable that it would not hold a stitch, and so, time pressing, the rest of the dura was everywhere left wide open as it was, and the muscles were brought together by iodized catgut suture. The wound was closed without drainage.

The condition that had been found was therefore a posterior basic meningitis, evidenced by thickened dura, adherent arachnoid, excessive subarachnoid fluid, and fine strands and adventitious vessels bridging across between the pia and arachnoid and dura. No tubercles were seen.

From this time on the progress has been uneventful, and the temperature has been normal since March, 1919. In June, 1919, the weight was 69 lb. She is the brightest child mentally I have known. In January, 1920, the report was received that she has remained in perfect health and with increased mental development. There have been no fits of any kind since December 22nd, 1918.

OBSERVATIONS.

I have reported this case in great detail because of its rare nature and because of the physiological points brought out.

In regard to its apparent rarity, Professor Thomas M. Rotch, in his *Pediatrics* (1901), says: "Both the congenital and acquired forms of external hydrocephalus are so rare that they need not be more than mentioned..." In *Diseases of the Nervous System*, by Jelliffe and White (Lea and Febiger, 1917, p. 559), there appears the statement that "External hydrocephalus, synonymous with serous meningitis, is, as an entity, infrequent," and the better known fact is also mentioned that "in chronic hydrocephalus, contractions and partial and general spasms are of ordinary occurrence."

The title of this paper requires a few words of explanation in regard to its various elements.

I believe that there was a loculated serous meningitis of the left Rolandic area where the first craniectomy revealed a subarachnoid accumulation of fluid, because the child's first symptom was a paraesthesia of the right hand, followed in a few days by Jacksonian epilepsy of the right hand and, later, the right arm. In this respect the case was identical with Case 4 and Case 6 which I reported in *International Clinics*, vol. 3, series 28, 1918, under the title "The operative treatment of chronic polio-encephalitis superior"; these were cured by craniectomy over the

affected area alone, leaving the dura open. This same procedure had a similar beneficial effect in the present case as regards the Jacksonian fits of the right arm, some time before the other types of epilepsy had been abolished by the combined subtemporal and suboccipital deduralizations.

F. Krause (*Surgical Operations*, 1917, vol. ii, pp. 496-499, reports a case similar to Case 6 of the paper referred to above) except that Krause's patient suffered three fits a month, whereas mine had twenty fits per night. Fluid was also found by Krause, who performed Kocher's valve-flap operation, with a resulting immunity from fits for at least six months, after which the patient was lost sight of.

The first element of the title, "infective external hydrocephalus," needs but little elucidation, for it is self-evident from the clinical notes, but emphasis may be placed on the large amount of fluid found in the middle fossa during the second (the subtemporal) operation, and in the posterior fossa during the fifth (the suboccipital) operation, as well as in the spinal theca on November 23rd; also upon the shifting character of the epilepsy, especially on November 3rd between 8.45 a.m. and 6.23 p.m., when it was well demonstrated that raising the patient to a sitting posture caused the fits to change their character and disappear in such order that the centres that were posturally highest were the first to be relieved from the spasms. Moreover, after 6.23 p.m. on November 3rd, when the fits ceased in response to the change of posture, there was no recurrence of cortical fits for twenty-seven days. It is difficult to imagine any other cause than shifting of fluid to account for such a disappearance of these symptoms and particularly on such a sliding scale. Moreover, confirmation was also present in that hebétude and aphasia rapidly disappeared when the patient sat up.

This does not tell us whether the fluid produces convulsions by mechanical pressure upon the blood vessels, causing anaemia of the cortex (which is my belief), or whether it acts by virtue of its abnormal chemical constitution. It appears certain that children more readily than adults react to varied intracranial pressure by convulsions, and convulsions are commonly observed a considerable time before the other signs of pressure appear in children.

Sir Charles Ballance (*Some Points in the Surgery of the Brain*, 1907) describes diffuse external hydrocephalus as follows (p. 46):

"In the subdural space, which is not divided into compartments, a pond of fluid will form, while in the subarachnoid space of the cortex the tissue under normal circumstances being traversed by countless rivulets of fluid (like marshy ground) will become oedematous and swollen."

This work does not mention polio-encephalitis as a cause for some of these conditions, but Krause mentions the likelihood in his works, and a good description of some of the symptoms attributed to this cause can be found in the article on epilepsy by the late Sir William Gowers in *Quain's Dictionary of Medicine*.

I believe that the internal hydrocephalus, which she must have had during the basal and cortical fits on December 5th, when the spinal theca was found almost dry, the subtemporal hernia bulged, and inadequate supratentorial drainage had been established for a month, was secondary, thus:

Fluid accumulated beneath the tentorium until, for example, the foramen of Magendie was occluded, the back pressure being assumed capable of causing a temporary valve-like closure when the posterior medullary velum was sticky with inflammatory products. Then, more fluid being formed within the ventricles by the choroid plexuses, the cerebellum was forced downwards and the fluid in the cisternae magna and basalis displaced upwards to the cortex, which at first could accommodate it, the former operations also giving egress to the subdural space and to the subcutaneous tissues from the subarachnoid level, but later the cortical region became overburdened and the clonic fits reappeared. By this time the medulla had corked up the foramen magnum, and the fluid in the spinal theca was absorbed by the spinal veins and lymphatics at the time when I failed to get more than 3 c.cm. by puncture; hence the urgent condition of the patient, the severe basal spasms and the return of the cortical fits on this date, all of which were relieved by enlarging the subtemporal decompression, turning back a flap of dura through the muscle and inserting a gold tube.

It is clear why absorption of the fluid was arrested in the first place, since at the subsequent (fifth) operation it was found that the arachnoid was adherent to the dura all over the posterior fossa. Without doubt removal of one

half of the foramen magnum improved the venous circulation, and gave "elbow room" to the fourth ventricle.

In seeming contradiction to the above theory is the fact that on November 23rd I removed 20 c.cm. of fluid from the spinal theca under great pressure, and thereafter she had no fits of any kind for four days; but this contradiction is only apparent, because the fluid was still coming out in a continuous stream when I withdrew the needle, so that just enough fluid was removed, and not too much—that is, not so much that the medulla should sink into the foramen magnum; whereas, on December 17th, when the pressure in the spinal theca was less, removal of 15 c.cm. of fluid did increase the basal fits.

A similar mechanical condition to the above seems to have occurred eventually in a case of primary internal hydrocephalus reported by Dr. Sutherland and Sir Watson Cheyne in the *Transactions of the Clinical Society of London*, based on experiments by Dr. Leonard Hill (*Physiology and Pathology of the Cerebral Circulation*), who states:

"If we could only establish a permanent communication between the ventricles and the subarachnoid space, then no matter how much fluid is poured out into the ventricles it would be at once absorbed and carried off by the veins of the subarachnoid and subdural space, and thus all injurious pressure intra- or extra-cerebral would be removed and the brain would be free to develop if it could."

Accordingly, Sutherland and Cheyne put the ventricle of a six months old hydrocephalic child into communication with the subdural space by means of a bundle of catgut which was left within the skull and the scalp closed.

The hydrocephalus greatly diminished, but the child showed no improvement in intelligence, and nine weeks later developed symptoms of basal meningitis, dying twelve weeks after operation. Necropsy showed the ventriculo-subdural drain patent and free from inflammation, the ventricles not distended, but a pool of fluid in the subdural space. The authors add that "expansion of the brain, the factor necessary for the removal of the fluid, had not taken place." The veins had not taken up the subdural fluid as Dr. Hill asserts is normally to be expected, owing, I suspect, to the basal meningitis spoken of above, and possibly for this reason the brain did not expand.

The patient whose case I have recorded had an extra-cerebral pool of fluid at every operation, and certainly the brain was then and is now well "expanded," but in spite of the palliative effect of the various supratentorial measures her symptoms did not disappear until the inflamed dura and arachnoid had been completely and permanently reflected from both cerebellar hemispheres. In regard to the original cause of the posterior basic meningitis and the localized serous meningitis of the left cortex that was discovered in this case it will be remembered that within a few days after the removal of inflamed and pus-laden tonsils she cried out in her sleep for several nights—an unusual thing for her—and within a month she began to complain of itching in the right palm, which was followed by the Jacksonian fits. It seems highly probable that a septic thrombus in the tonsillar branch of the ascending pharyngeal artery was carried off into the more distal posterior meningeal branches of the same vessel; and more septic matter may have been conveyed via the middle meningeal or middle cerebral to the left Rolandic area. I have no doubt that the tonsils are responsible for many such infections, although it is curious that the left Rolandic area should be so often the first to show symptoms of the infection. One moral is to render all tonsils as clean as possible before removal.

It may be asked why I did not perform a suboccipital decompression in the first instance in this case. The first and most weighty reason is that I have usually had greater shock with the suboccipital operation than when operating above the tentorium. In the four supratentorial operations upon this child not more than 1 oz. of blood in all was lost. The second reason is that I am by no means convinced that in cases of epilepsy having Jacksonian characters any mere decompression ever suffices to effect a cure, and believe that the most favourable outlook is produced by leaving a large dural flap wide open over the area indicated by focal signs. The dura eventually regenerates, as I have demonstrated elsewhere. The third reason is that as regards the type of fit that has been designated "basal" in this case, I have known these to clear up completely after the safe and simple subtemporal decompression of Cushing.

I reported such a case in the *California State Journal of Medicine*, November, 1914, under the title, "Decompression in acquired hydrocephalus." The fits were identical with the

opisthotonic ones of the present case (L. H.), and the trouble had progressed to almost total blindness before operation cured both conditions.

For these reasons, in the case of L. H. I did not approach the base until compelled to do so by a process of exclusion. Again, the osteoplastic flap causes the least deformity, leaves no permanent weakness of the skull, and will cure some at least of the most severe cases, even when the Jacksonian fits do not comprise the whole clinical picture, as was the result in Case 6 of my paper already referred to (*International Clinics*, 1918, series 28, vol. 3), in which the whole operation was completed in one stage without shock—a procedure generally inadvisable, as the two-stage operation allows more gradual adaptation of the vital centres to the alterations of pressure.

Ballance, in his work already referred to, speaking of the attitude of the head in cerebellar lesions, says: "Also the head may be retracted or the chin depressed on the chest; the anterior or posterior part of the vermis is then probably involved." This is in accord with Pagano's experiments on animals, in which he found that stimulation of the anterior part of the vermis caused the head to look upwards and the subject to fall backwards, whereas stimulation of the posterior vermis brought the chin down on the chest and caused a falling forwards.

Several additional points of moment have been emphasized in the course of this case, three of which I have noticed in previous cases.

First, that the greatest shock of most brain operations is apt to occur after the skull is opened for the first time. When the intracranial contents have had a few days to become accustomed to the change of pressure, the brain can be judiciously attended to without much fear. In this connexion one is reminded of the views of Leonard Hill on the influence of atmospheric pressure, and, in spite of the statement of so experienced a cerebral surgeon as Sir Charles Ballance that no profound changes are observed upon first opening the skull, my experience from careful blood-pressure records has been that stage 1 is the only time in many operations that I had any cause to fear a sudden depression. I have never lost a patient from operation except from one cause—to wit, doing too much at stage 1, so that it became necessary to enforce recumbency to combat shock at a time when the mechanical conditions within the cranium called loudly for a sitting posture. I think that more stress should be laid on the question of posture both during and after cranial operations.

In none of the subsequent operations upon L. H. was there any disturbing shock due to the surgical procedure, and even after the suboccipital operation, which is usually considered a severe measure in a debilitated patient, her condition was excellent, because I believe there was a decompression above the tentorium that allowed the atmosphere to follow the changes in the fluid pressure and intracranial bulk. Thus, after I released the large amount of fluid from the cisterna magna, any fluid that followed it from the fourth ventricle could be replaced without tension by that from the third and lateral ventricles, for the atmosphere was able to press into the subtemporal region. The chief danger, therefore, of brain surgery lies in alterations of pressure, positive and negative. I have met with severe but not fatal shock under similar circumstances in at least three other cases of extracerebral fluid upon which I operated.

The next point is that cortical-clonic fits appear to be produced not only by too much fluid but by too rapid drainage of fluid from the cortex into the basal cisterns, as may be inferred from a reference to the events that occurred on November 2nd at 2.25 a.m., and on December 6th. In this connexion also Harvey Cushing speaks of convulsions caused by loss of cerebro-spinal fluid after his ventriculo-abdominal drainage (*Keen's Surgery*).

Fourthly, it is not always possible to obtain adequate drainage and decompression of the entire cranial cavity by any operations above it. Fifthly, the arachnoid of the great cisterns is most important for the absorption of excess fluid, but can be replaced by muscles.

It is evident that an accurate knowledge of the pathology of many of these conditions can only be obtained by exploratory operation, since fluid that would be obvious upon the brain during life is usually absent in the post-mortem room.