

A NEW METHOD FOR DIRECT AND CONTINUOUS RECORDING OF THE BRAIN TENSION AND THE CEREBRAL PLETHYSMOGRAPH

KAZUYUKI WATANABE

*1st Department of Surgery, Nagoya University School of Medicine
(Director: Prof. Yoshio Hashimoto)*

ABSTRACT

The intracranial pressure is one of the important vital signs in the field of clinical neurology and neurosurgery.

The author reports about the newly designed device which measures the intracranial pressure continuously and directly in the closed system with a pressure transducer using a semi-conductor strain gage which was developed by Igarashi of Toyota Central Research and Development Laboratories, Inc.

The pressure transducer is a small disc of 5 mm in diameter and 1.5 mm in thickness. Its output power is 0.24 mV to the pressure of 100 mmH₂O, and it is recorded after amplifying. Using the device, it is possible to measure the absolute value of intracranial pressure and to record the plethysmograph of the brain surface. The satisfactory results have been obtained in the animal as well as in clinical measurements of intracranial pressure. In clinical measurement, two kinds of plethysmograph were observed: one is the wave caused by respiration and the other is the wave caused by pulsation. The changes of these waves have important physiological significances. On the other hand, the absolute intracranial pressure also changed very quickly and largely by coughing, forced respiration, or the other changes of the condition of the measuring subject.

The author has also described that, in the near future, it will become practical to measure the intracranial pressure by telemetering using this device.

1. INTRODUCTION

The intracranial pressure should be considered as one of the important vital signs in the field of clinical neurology and neurosurgery. Up to now, measurement of the subarachnoid cerebrospinal fluid pressure has been made by means of the lumbar puncture, the ventricular puncture, or the puncture of cisterna magna. It is, however, basically doubtful, especially when blockage of the cerebral spinal path exists, whether or not the measurement of the subarachnoid cerebrospinal fluid pressure by these methods can express correctly the intracranial pressure and direct brain tension. Increased intracranial pressure is first diagnosed by clinical means by observation of papilledema in the eye

渡 辺 一 之

Received for publication November 18, 1967.

ground, nausea, vomiting, headache, or other clinical signs and symptoms, because the lumbar puncture is known to be a dangerous procedure because of possible development of the cerebral herniations of various types such as the herniation under the incisura of cerebellar tentorium or of the cerebellar tonsils. However, the quantitative measurement of the increased intracranial pressure has never been made.

From these points of view, precautions should be taken to measure the intracranial pressure directly, continuously, and safely. For this purpose, various types of apparatus to measure the intracranial pressure have been designed by many investigators. The methods, such as that which measures the intraventricular fluid pressure or the pressure that is transmitted from a brain tension to closed fluid bag placed on the surface of the brain and measured as the hydrostatic pressure through the catheter with water-manometer, kymograph, or extracranial pressure transducer, were developed for this purpose. These methods were developed by Blackfan, Clothiers, and Ganz¹⁾, Aboulker²⁾, Carmichael, Doupe, and Williams³⁾, Lagergren⁴⁾, Väinö Seiro⁵⁾, Antoni⁶⁾, Guillaume, and Janny⁷⁾, Ecker^{8) 9)}, Lundberg¹⁰⁾⁻¹⁵⁾, Keegan, and Evans¹⁶⁾, Rothballer¹⁷⁾, Loroche¹⁸⁾, Vedula, White, and Albin¹⁹⁾, Hoppenstein²⁰⁾, Hemmer²¹⁾, Dunber, Guthrie, and Karpell²²⁾, and Yada, and Tsunoda²³⁾²⁴⁾, etc. But, because it is the principle of these methods that the pressure is changed to fluid pressure for the measurement, there are many disadvantages such as the bending of the catheter, the influence of specific gravity of fluid, and the change of the position of the patient's head during the observation, etc. These disadvantages prevent the practical usage of this apparatus and reduce the accuracy of the result of measurement.

Using another method, Cooper, and Hulme²⁵⁾²⁶⁾, and Jacobson, and Rothballer²⁷⁾, adopted the intracranial pressure transducer to measure the intracranial pressure with the small pressure transducer that was inserted directly into the intracranial cavity. It is considered, however, that the sensitivity of their pressure transducer and their fitting methods into the intracranial cavity did not produce satisfactory results.

Therefore, the present author adopted the new device to measure directly and continuously the intracranial pressure with the special pressure transducer using very sensitive, accurate, and small semiconductor strain gage, which was newly developed for the industrial purpose by Toyota Central Research and Development Laboratories, Inc. An accurate and continuous measurement of the alteration of intracranial pressure and plethysmograph of the cerebral surface have been made with the new device. Therefore, the author could confirm that the method and apparatus are very useful and accurate in the direct and continuous recording of intracranial pressure.

The present report describes the principle structure of the apparatus, and

some experimental results obtained by animal experiments and few clinical measurements of intracranial pressure.

2. MEASURING DEVICE

(A) *Pressure transducer*

The newly developed device is a microminiature pressure transducer using semi-conductor strain gage. Fig. 1 and Fig. 2 shows its exterior view. It is 5 mm in diameter, 1.5 mm in thickness, and 0.3 g in weight, and is made of stainless steel. The shape is a very small disc. The inner structure is shown

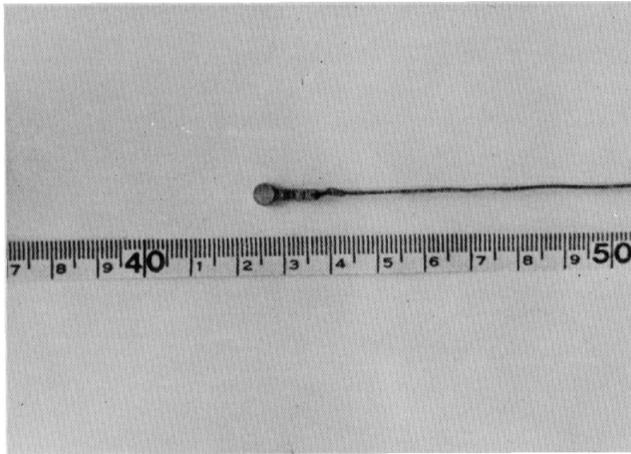


FIG. 1

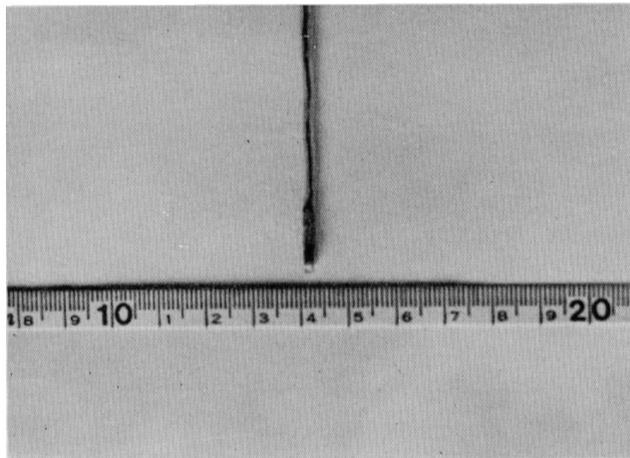


FIG. 2

FIG. 1 and FIG. 2. The exterior view of the strain gage.

in left side of Fig. 3. In the pictures, a_1 and a_2 are the pressure element of Germanium for pressure sensitivity, and b_1 and b_2 are also made of Germanium for temperature compensation. The a_1 and a_2 is the active side, and b_1 and b_2 is the dummy side. Grooves are cut around the supporting parts of the diaphragm so that the stress condition are substantially equalized to the simply supported ones. The right side of Fig. 3 shows the circuit arrangement of the pressure cell. The difference of the thickness of metal between the active and the dummy side is made to compensate the temperature effect and to reduce noise due to acceleration.

As the characteristics of the device, relationship of the pressure and the output power of this strain gage is shown in Fig. 4. The output power of the strain gage is 2.4 mV/1000 mmH₂O pressure. This shows lineal changes in 22.5°C of room temperature. The shift of Zero-level by influence of temperature

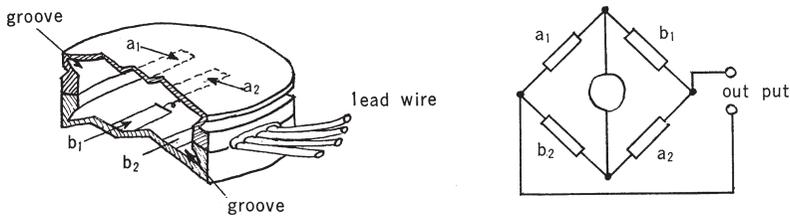


FIG. 3. The arrangement of the semi-conductor element and the electrical circuit of the pressure cell.

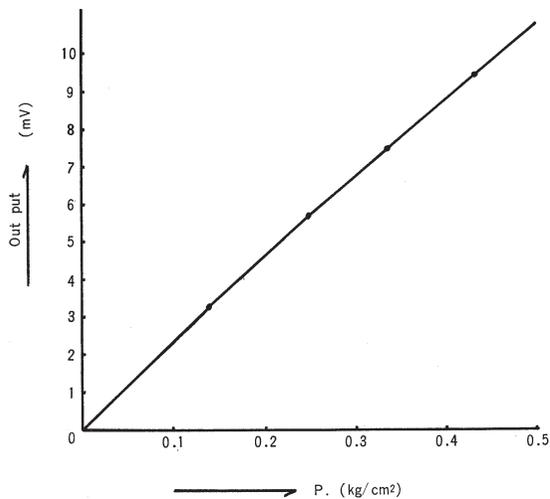


FIG. 4. The relationship of the pressure and the output power of the strain gage.

is under $0.5\%^\circ\text{C}$, and the strain gage is made so as to be able to compensate within 20 seconds from 20°C to 40°C . Applied power was 4 mA in a constant current.

The following device was developed in order to use the strain gage for the observation of intracranial pressure in experimental study and clinical neurosurgery.

(B) *Electrical manometer*

The strain gage is connected to an electrical manometer which has the electrical circuit showed in Fig. 5. A electric bridge circuit is made so that the observation of the change in pressure can be converted into the change of electric current. The electrical manometer has a preamplifier which amplifies 70 times the change of electric current caused by the change of pressure. "NEXUS" of N.E.C. Inc., used as its preamplifier, is very small but very efficient. In Fig. 5, the middle under part of the circuit is made for calibration. In this calibration circuit, when the calibration voltage is 5.2 mV, that is to say, as the electrical manometer used is a amperemeter, and has 0.05 mA in full scals; when this amperemeter shows 0.0371 mA, it is calibrated so that the output voltage of full scale in this strain gage shows 7 mV, that is, 3,116 mmH₂O in pressure.

The specific character as a frequency of this electrical manometer is laid out so as to be DC-30 c/s, and by this character this device can record the sudden change of intracranial pressure and the quick change of the plethys-

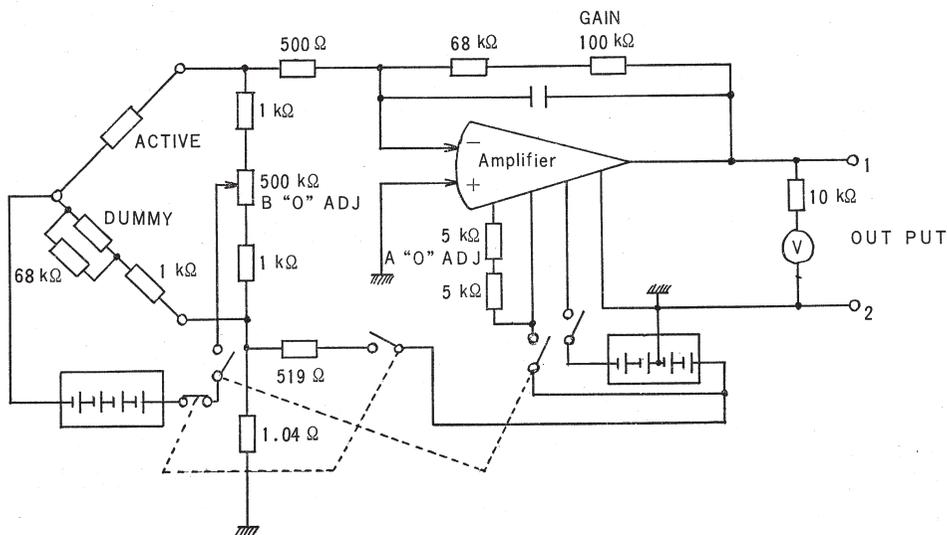


FIG. 5. The circuit of the electrical manometer.

mograph on the surface of brain by the influence of pulsation and respiration without any delay. That is, by a cough or a forced respiration, the intracranial pressure suddenly increase or decrease by 250 mmH₂O or 1,000 mmH₂O of pressure for one second, and the frequency of pulsation wave or respiration wave is 3-4 c/s. Because it changes by the same rhythm with the change of pulsation or respiration rhythm, it is required that the specific character as its frequency must be rapid to a certain degree.

(C) *Recorder*

There are various kinds of such recorder which record continuously, and for this measurement, the model "EPR-2 T" of electronic polyrecorder made by Toa Electronic Ltd., Japan, is used. It has a very high sensitive direct current amplifier in it. The zero range is divided into 14 stages from 100 V to 5 mV, but only 5 zero ranges from 500 mV to 25 mV was used for the purpose of measurement. The chart of this recorder is divided into 10 equal section has 10 smaller sections making 100 small sections in full scale. The calibration of this recorder is set up as follows: when 500 mV scale of zero range is used, 50 chart sections represent 3,000 mmH₂O of pressure and 12.5 mm of the pointer deflection of this recorder represents 500 mmH₂O of pressure. In the case when 250 mV scale of zero range is used, 50 chart sections represent 1,500 mmH₂O of pressure and 12.5 mm of its deflection represents 250 mmH₂O of pressure. In the case when 100 mV scale of zero range is used, 50 chart sections represent 600 mmH₂O of pressure, and 12.5 mm of its deflection represents 100 mmH₂O of pressure. In the case when 50 mV scale of zero range is used, 50 chart sections represent 300 mmH₂O of pressure and 12.5 mm of its deflection represents 50 mmH₂O of pressure. In the case when 25 mV scale of zero range is used, 50 chart sections represent 150 mmH₂O of pressure, and 12.5 mm of its deflection represents 25 mmH₂O of pressure. The chart speed of the recorder can be changed into 6 stages of 180 mm/M, 60 mm/M, 20 mm/M, 180 mm/H, 60 mm/H, and 20 mm/H. Therefore, it can be controlled from relative high speed to slow speed. The balance speed of the recorder is over 300 mm/sec, that is, under 0.3 sec in full scale.

(D) *The arrangement of strain gage, electrical manometer, and recorder*

Fig. 6 shows a photograph of the arrangement from left to right strain gage, electrical manometer, and recorder. Its diagram is shown in Fig. 7. The connecting cords between strain gage, electrical manometer, and recorder are long enough to change freely the positions which is very useful for the clinical measurement.

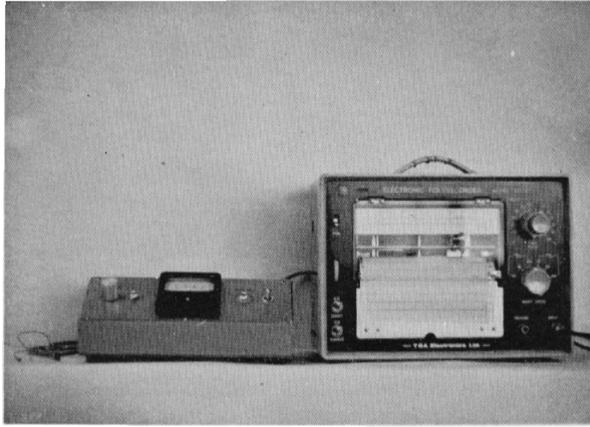


FIG. 6

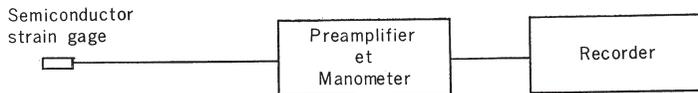


FIG. 7

FIG. 6 and Fig. 7. The arrangement of the semi-conductor strain gage, preamplifier, and recorder.

(E) Special characteristics of the apparatus and method

As its special character, at first, the absolute intracranial pressure can be measured continuously, and next, the plethysmogram of the brain surface and the brain tension can be observed by continuous recording. The frequency of the plethysmogram could record quickly the sudden change of the intracranial pressure. This is the most important characteristic of the device. The results using the device could be expected to be more advanced informations than those obtained by other previously developed apparatus.

As the next special character of the device, it is not needed to be connected to any catheter or any fluid at the time of measurement. Therefore, measurement with the device does not prevent other examinations or operations. The measuring subject can be moved freely to a certain degree. As the heat resistance of this strain gage, it is possible that the strain gage can be disinfected by boiling water just before use without any changes in its reliability. Therefore, its preparation before use is remarkably rapid and simple.

The temperature dependency of the strain gage, which is a disadvantage of the semi-conductor strain gage, is compensated almost completely. In the temperature range between 20°C and 40°C, that is, in the range of the human body temperature, it is so designed that the temperature effect is compensated

within 20 seconds. These facts signify that there is no need to consider the influence of temperature during the measurement. As the sensitive plate of the strain gage is fixed by the supported edge method, and its sensitivity is highly efficient. The other method for fixation of the sensitive plate is the clamped edge method used by Cooper and Hulme. It is proved, however, that physically the clamped edge method is inferior in its sensitivity than the supported edge method.

The fixing method of the strain gage into intracranial cavity is described as follows: it is very simple and after measurement is taken, it can be removed from the intracranial cavity very easily. Therefore, there is very little damage to the measuring subject. There are no leakage of the pressure, and the danger of bacterial infection is also minimal because the measurement system is designed as a complete closed system.

In spite of these advantageous characteristics of this device, there is one slight disadvantage which interferes with operative manipulation even though it is very small. But this disadvantage should be discounted because of possibility of acquiring much informations by the strain gage method. and interference of the operative manipulation is also insignificantly small. Since a part of the operative field is used for its fixation in clinical measurement, it is unnecessary to manipulate the measuring subject.

Since the semi-conductor materials used for the pressure element are extremely sensitive and delicate, the device should be carefully handled.

Because the material of the device is made of stainless steel, there is a danger that the brain tissue measured by the device may be slightly damaged. But this problem can be eliminated by handling the device with extreme care.

(F) *The method of fixation into the intracranial cavity*

Fig. 8 shows the diagram illustrating the device that has been fixed in the intracranial cavity. The strain gage is fixed in the intracranial cavity by the

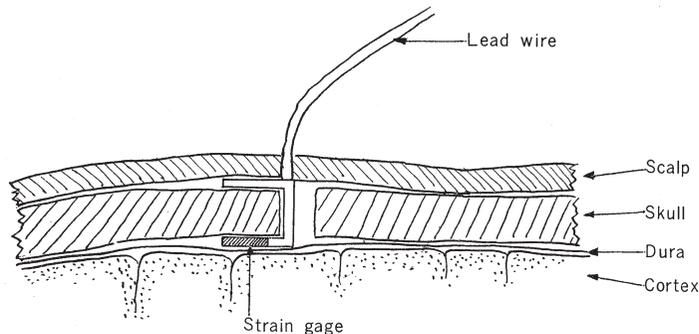


FIG. 8. Method of the fixation into the intracranial cavity.

following method.

At first, the small scalp section is made through a certain region of the head, a burr hole is made (about 1 cm in diameter) by usual method, and the strain gage is inserted into epidural space under the skull so that the dura mater and the sensitive plate of the strain gage come into direct contact with each other. After the completion of its fixation, the section of scalp is sutured.

Cooper and Hulme first developed the special apparatus for fixation of the strain gage in the intracranial cavity. Their methods are as follows. The strain gage is connected in a stainless steel sleeve fastened in a stainless steel strap spanning the burr hole. The open end of the sleeve is closed by Teflon film. Therefore, they measured the brain tension through the Teflon film, as the strain gage has contact with cerebral cortex through the film²⁵). But by this method, the brain tension is transmitted directly to the stainless steel sleeve and not to the strain gage. Furthermore, in the extreme cases, when the intracranial pressure increased abnormally, the strain gage may penetrate into the brain tissue, and there is a great possibility that the Teflon film also prevent the transmission of the pressure.

Fig. 8 illustrates the fixing method of the strain gage to the skull invented by the author. The apex part is fixed with the strain gage; the screws are cut around its vertical parts and the exterior parts of the skull serves as the nut. Insertion of the strain gage into the epidural space and under the skull is shown in Fig. 8. With this procedure the strain gage can be fixed tightly to the skull with this apparatus. Since the developed apparatus is so small, strain gage which is inserted into the epidural space can not be disconnected, nor can the strain gage be pressed from outside of the skull. The advantageous point of the fixation method is that since there are no opening of the dura mater, and it is not inserted into the cerebral nor subdural space, no damage of regional brain tissue is done. There is very slight possibility of infection because the apparatus is a closed system and it can be sterilized easily. The preparation for measurement is very simple and quick because only one burr hole of 1 cm in diameter is made.

In order to decide the fixation region of the strain gage in the intracranial cavity, the author compared the result of measurement from the various points of view such as changing pressure, or the variation of plethysmogram upon the brain surface by inserting the strain gage into epidural space, subdural space, and intracerebral space. Consequently, it could be confirmed that in the epidural space under the skull, the insertion and the fixation of strain gage were easiest, and damage to the brain tissue also minimized. The pressure change and the variation of the plethysmogram upon the brain surface were expressed most correctly, and it was decided that the fixing region of the strain gage was in the epidural space and under the skull.

After measurement, the strain gage can be removed by the following way, under a local anesthesia, cut the suture threads of scalp, reopen a part of operation area, remove the strain gage quickly like removing of a drainage, and suture.

3. THE MATERIALS AND THE METHODS FOR MEASUREMENT

For the experimental measurement, dogs weighing from 15 kg to 20 kg, and rhesus monkeys weighing from 5 kg to 8 kg were used. For clinical measurement, it was measured only on cases that no side-effect would occur. In dogs, they were anesthetized with the administration of 20 mg/kg sodium pentobarbiturate into the abdominal cavity, and in rhesus monkey, they were anesthetized with the intravenous administration of 30 mg/kg Nembutal. After the anesthesia, in both cases, they were laid on the table in prone position, their legs were tied to the table, and their heads were not fixed so that its position could be changed freely in a certain degree. In clinical measurement, in order to measure during the operation, they were given a intubated general anesthesia under semi-closed system by GOF method, and they were placed in position indicated for its operation.

After these preparation, in dogs and monkeys, the calibrated strain gage was inserted with the fixation method mentioned previously into the parieto-temporal region, and the intracranial pressure was changed in various ways. The pressure changes and variations of cerebral plethysmogram were continuously recorded.

In clinical observations, the strain gage was inserted at a part of the burr hole made for the operation, where it did not interfere with the operation, into the epidural space as mentioned previously, and the process was recorded and observed during the operation.

Before usage of the strain gage, it was disinfected with 100°C boiling water for 10 minutes, and after its temperature come down to the room temperature, it was calibrated and inserted into the epidural space and fixed there.

4. RESULTS OF MEASUREMENTS

Fig. 9 shows the continuous recording curve of normal intracranial pressure and the plethysmogram of brain surface of rhesus monkey with 7.5 kg in weight. In this recording, 100 mV scale in the zero range of recorder and 180 mm/M in its chart speed was used. The intracranial pressure in the rhesus monkey showed 120 mmH₂O of pressure, although the recorded time in this recording was 2 minutes. No changes of the intracranial pressure were observed during this recording. As the plethysmogram of the brain surface, two kinds of waves were observed. The one was the wave due to respiration, which was

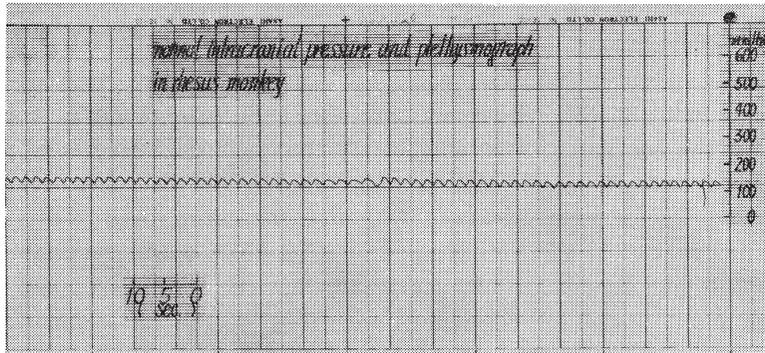


FIG. 9. The continuous recording of the normal intracranial pressure and the plethysmograph of brain surface of rhesus monkey.

the slow wave of 0.6 c/s in frequency and the pressure changes showed the lowest pressure by the inspiration and the highest pressure by the expiration. The differences between inspiration and expiration showed about 30 mmH₂O or 40 mmH₂O of pressure. The other was the small wave above the wave of respiration which was a relatively quick wave of 3 or 4 c/s in frequency, and its pressure change showed about 10 mmH₂O of pressure which was from the pulsation. When the intracranial pressure of the measuring subject is in normal range, it may be confirmed that the slow and big waves are due to respiration. The quick and small waves are observed regularly.

Fig. 10 showed the continuous recording of the intracranial pressure by the prefrontal lobotomy carried out for a psychiatric disorder of a man aged 37 years old. In this case, a burr hole of 1 cm diameter was made in the region near the operation field on the same side, the calibrated strain gage was inserted into the epidural space as described previously and continuous record was made. Under these conditions, the zero range of recorder was changed in 5 stages from 25 mV scale to 500 mV scale and the following facts could be observed. In this recording, it can be observed that the intracranial pressure changes

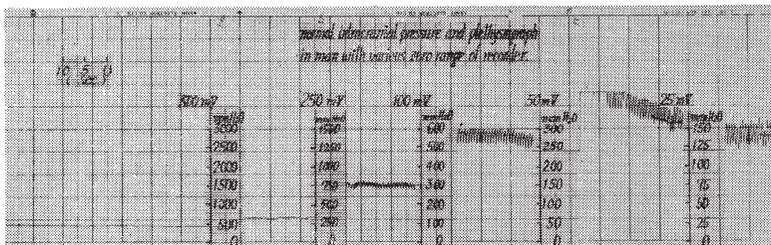


FIG. 10. The continuous recording of the normal intracranial pressure and the plethysmograph in patient of psychiatric disorder under various zero range of the recorder.

between 250 mmH₂O and 300 mmH₂O and showed 1.6 c/s in frequency. These waves are seemed to be due to the pulsation. It will be natural that, if the zero range of recorder is made lower, the wave amplitude can be amplified. From these facts, it can be proved that the high zero range of recorder is suitable for the continuous recording of the change of the intracranial pressure, and the lower zero range of recorder is suitable for the recording of the change of plethysmogram of brain surface. Although the chart speed of this recorder was also 180 mm/M in this recording, this high chart speed is suitable for the observation of the change of plethysmogram, but the slow chart speed is better than high chart speed for continuous recording of the intracranial pressure during a long time. It could be confirmed that for the observation of both change in pressure and plethysmogram with this recorder, it is most suitable for 250 mV scale or 100 mV scale in zero range and for 180 mm/M in chart speed.

Fig. 11 shows the change of intracranial pressure and the variation of plethysmogram of the brain surface which occurred by the compression of neck or abdomen in a rhesus monkey 5 kg that is, at first, it showed 100 mmH₂O of intracranial pressure with a little irregular waves (it may be caused by shallow anesthesia), and the small wave of 2-3 c/s in frequency due to pulsation was observed above over the slow wave of 0.8-0.9 c/s in frequency due to respiration. The amplitude of both waves are 20 mmH₂O of pressure in slow wave and 5-10 mmH₂O of pressure in small wave. By the compression of neck for 6 seconds, the intracranial pressure about 90 mmH₂O for 1.5 seconds and showed no change during compression. After releasing compression, it returned to the previous pressure for 1.5 seconds. In two similar trials of this procedure, almost the same process was observed. In 10 seconds after these trials, by the compression of abdomen, the intracranial pressure for 6 seconds reached the

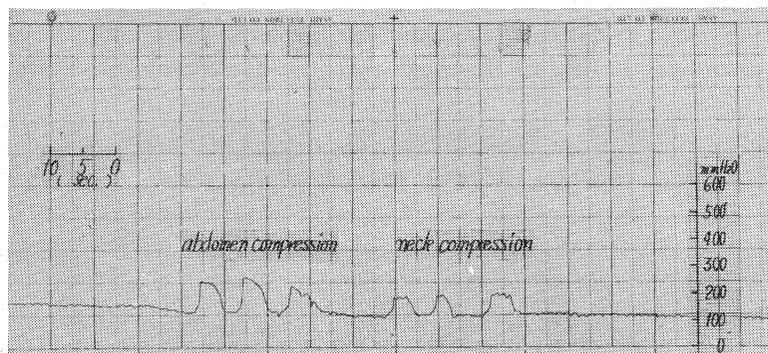


FIG. 11. The change of the intracranial pressure and the plethysmograph of the brain surface which occurred by the compression of neck or abdomen in a rhesus monkey.

upper limit of the pressure; after releasing of compression it returned to the previous pressure in 1 or 1.5 seconds. Consequently, it was observed, that, by the compression of neck, the increasing and decreasing of intracranial pressure showed the same speed by its change, but by the compression of abdomen, the increasing speed of the intracranial pressure was slower than its decreasing speed.

Fig. 12 shows the change of intracranial pressure and plethysmogram caused by apnea. Artificial respiration was practiced during the intravenous administration of 100 mg pentobarbiturate. The intracranial pressure showed 150 mmH₂O of pressure at first. It become apnea by the intravenous administration of 100 mg sodium pentobarbiturate for about 20 seconds which was injected in order to cease the increased body movement. The waves with the amplitude of 30 mmH₂O of pressure and with 0.8 c/s in frequency caused by respiration were observed at first. But these waves disappeared at once by apnea followed after the intravenous administration of sodium pentobarbiturate, and the waves with 3.5 c/s in frequency and the amplitude of 5-10 mmH₂O of pressure caused by pulsation were observed. This apnea continued for about 30 seconds, the artificial respiration has been done. Then, the waves caused by this artificial respiration were observed as almost the same shape as spontaneous respiration. The artificial respiration was continued for 3 minutes. The intracranial pressure increased gradually during apnea. It showed an increase of about 120 mmH₂O of pressure for 20 seconds after the beginning of apnea and ceased to increase temporarily. The decrease of about 30 mmH₂O of intracranial pressure was then observed almost at the same time with the beginning of artificial respiration. But the intracranial pressure began to increase again after continuation of artificial respiration for 1.5 minutes. The increase of the intracranial pressure during artificial respiration was 120 mmH₂O of pressure and, after all, the absolute intracranial pressure increased to 370 mmH₂O of pressure.

Fig. 13 shows the variation of intracranial pressure and plethysmogram caused by the intravenous administration of "Theraptique" for stimulating the

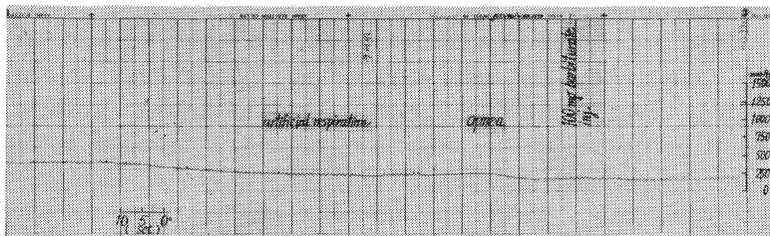


FIG. 12. The change of intracranial pressure and of plethysmograph caused by apnea and artificial respiration through the intravenous administration of 100 mg Pentobarbital.

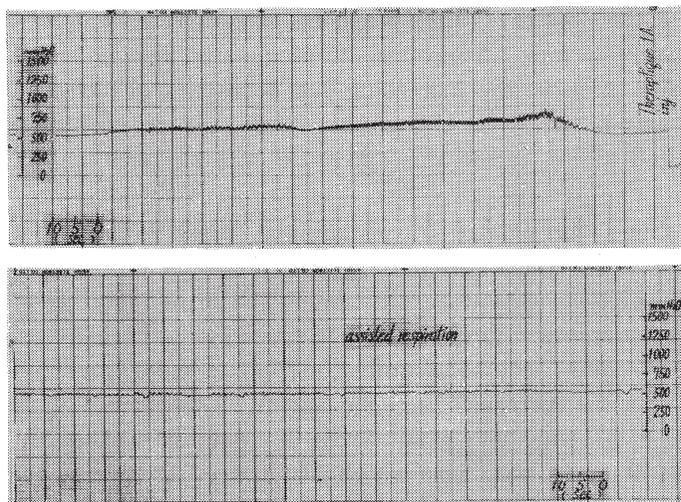


FIG. 13. The change of the intracranial pressure and plethysmograph caused by the intravenous administration of "Theraptique" in rhesus monkey.

respiration in the same rhesus monkey. "Theraptique" was administered intravenous for 13 seconds. After the end of this administration, the quick changed waves which seemed to be caused by pulsation, with 3.0-3.5 c/s in extremely regular and rhythmic frequency and with amplitude of 60 mmH₂O of pressure, appeared at once and they continued for 1 minute. After that, the wave showed 3.6 c/s in frequency and the amplitude of 20 mmH₂O of pressure continued for 6 seconds. After continuance of these waves, it was observed that the slow waves with 1.0 c/s in frequency and with the amplitude of 60 mmH₂O of pressure appeared over the same shaped waves with which described in the above, and they continued for 4 seconds. But since the complete recovery of the apontaneous respiration could not be acquired even by the administration of "Theraptique", and the small shallow respiration continued, the assisted respiration had been given. It was shown in the second part of Fig. 13. Though "Theraptique" was administered 2.5 minutes after the period of Fig. 12, during these period, the intracranial pressure increased to about 500 mmH₂O of pressure. After the end of administration of "Theraptique", it showed the increase of more about 250 mmH₂O of pressure, and the absolute pressure reached 750 mmH₂O of pressure. The increase of 190 mmH₂O of pressure, however, was observed and the absolute intracranial pressure showed to decrease to about 560 mmH₂O of pressure. At the appearance of small spontaneous respiration, the intracranial pressure showed to be decreasing again to 500 mmH₂O of pressure. All the process illustrated in Fig. 13 was seen during 4 minutes 53 seconds,

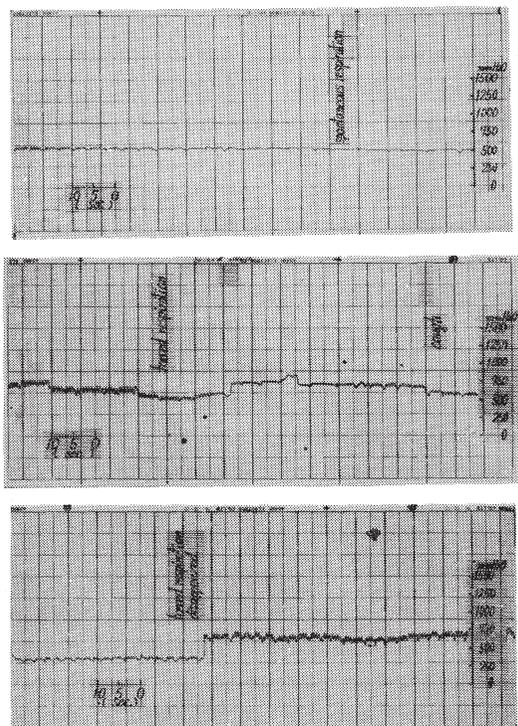


FIG. 14. The change of intracranial pressure and plethysmograph during the forced respiration.

Thirty five seconds after the end of the process, the extremely slow spontaneous respiration of 18 times a minute was noted, but this slow spontaneous respiration changed gradually to forced respiration. These processes are shown in Fig. 14. The intracranial pressure showed a little increase to 650 mmH₂O of pressure again during this process. The moderate quick waves continued with 1.6 c/s in frequency and with the amplitude of 30 mmH₂O of pressure. These waves were seemed to come from pulsation. In this recording, the slow waves with 0.3 c/s in frequency and with the amplitude of 60 mmH₂O of pressure were superimposed over those of the moderate quick waves. During this recording, it was observed that increasing of intracranial pressure of 90 mmH₂O occurred suddenly by cough, and the forced respiration made it also decreased. Furthermore, respiratory influence on the waves was ceased by the condition of the extreme forced respiration at 20 seconds after its beginning for 26 seconds. During these periods, as its plethysmograph, only the quick waves caused by pulsation were observed, and the amplitude itself also increased. And next moment, the spontaneous respiration appeared. The waves observed at the left end of Fig. 14, were caused by pulsation that showed the increasing

amplitude of about 90 mmH₂O of pressure. The waves caused by pulsation were seen as the wave with about 0.25 c/s in frequency in this recording. The increasing amplitude by pulsation continued for 1 minute 50 seconds and at the same time with release of the condition of forced respiration, the increasing amplitude of the wave caused by pulsation began to decrease again, and the waves caused by respiration began to be observed. The change of the absolute intracranial pressure began at 560 mmH₂O of pressure and increase to 640 mmH₂O of pressure with cough, after 20 seconds. It increased to 810 mmH₂O of pressure during 10 seconds together with the increasing of respiration at the condition of forced respiration. After that, it decreased gradually. At the part of the increased amplitude of the waves caused by respiration, the intracranial pressure showed the decrease to 560 mmH₂O of pressure, but it began to increase again, and showed 580 mmH₂O of pressure until the release of forced respiration. The intracranial pressure showed 370 mmH₂O of pressure by suddenly decreasing of 210 mmH₂O of pressure almost at the same time of the release of forced respiration.

Fig. 15 showed the changes of intracranial pressure and plethysmogram by the intravenous administration of 50 ml of 50% Glucose solution which was injected for 20 minutes 47 seconds for the purpose of observing the influence of the hypertonic osmotic therapeutics on brain. In this period, 2 minutes from the period of Fig. 14 passed and the pulsation and respiration returned to normal. Although its administration dose was 6.7 ml/kg, the rhesus monkey ceased respiration. The condition continued from 13 minutes before the end of administration of 50% Glucose solution to 5 minutes 20 seconds after the end of its administration. The respiration was controlled artificially during the period of ceased spontaneous respiration. The observed plethysmogram was only the waves with 1.6 c/s in frequency caused by pulsation and with 7-10 mmH₂O of pressure in amplitude. The waves caused by respiration were observed extremely irregularly with the movement of artificial respiration. The absolute intracranial pressure of 355 mmH₂O in pressure, which showed at the beginning of this recording, was almost unchanged. The decreasing effect for

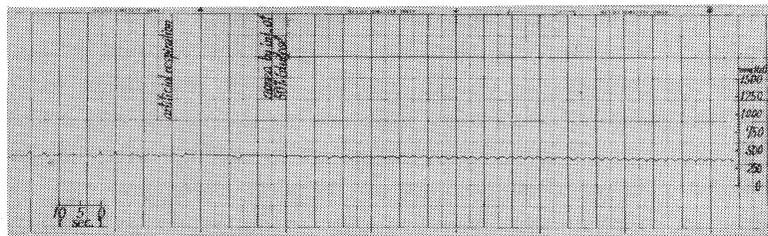


FIG. 15. The change of intracranial pressure and plethysmograph by intravenous administration of 50 ml of 50% Glucose.

intracranial pressure caused by 50% Glucose solution was very little in this case. It was explained that the increased pressure caused by apnea was reduced by the decreasing effect for intracranial pressure by 50% Glucose solution and showed the result in Fig. 15. After the appearance of spontaneous respiration, the intracranial pressure showed the increase of about 250 mmH₂O of pressure which seemed to be a rebounded effect of 50% Glucose solution. It was observed 6 minutes after the end of administration of 50% Glucose solution.

Fig. 16 shows the recording of cough which occurred in the same subject of Fig. 10 just after the intubation of general anesthesia for the prefrontal lobotomy. When anesthesia depth was not so deep and the influence by hyperventilation after intravenous administration of S.C.C., the waves showed at first 250 mmH₂O of intracranial pressure, 1.6 c/s in frequency, 20 mmH₂O of pressure in amplitude. With the occurrence of cough for 1 second, the intracranial pressure increased to 545 mmH₂O of pressure, that is, the pressure increase of 200 mmH₂O and the spindle formed wave of 120 mmH₂O of pressure as amplitude continued for 1 second. These spindle formed waves occurred as the same rhythm with those of cough. Because the measuring subject did

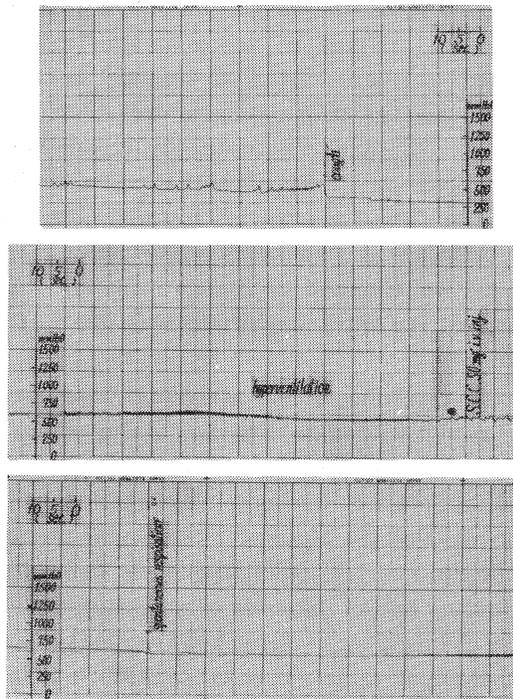


FIG. 16. The recording of the change of intracranial pressure and plethysmograph during cough and hyperventilation.

forced respiration between a spindle formed wave and another, the waves with regular rhythm caused by the amplified pulsation were observed. As the unsuitable condition like these continued for 1 minute 20 seconds, 50 mg of S.C.C. was administered intravenously, then the complete muscle relaxation and apnea was acquired after 10 seconds of administration, artificial hyperventilation was done. The spindle formed waves by cough disappeared naturally together with muscle relaxation by S.C.C. and the intracranial pressure showed the decrease of about 30 mmH₂O of pressure after carrying out artificial hyperventilation. The waves with 1.7 c/s in frequency and 30 mmH₂O of pressure in amplitude were observed regularly. These conditions continued for 40 seconds after the beginning of hyperventilation and after that, the intracranial pressure began to increase again. It showed the increasing of 90 mmH₂O of pressure for 20 seconds, and the absolute value showed 605 mmH₂O of pressure. During this period, the amplitude itself increased and at the time when the absolute intracranial pressure showed 605 mmH₂O of pressure, the amplitude reached about 60 mmH₂O of pressure. But this pressure continued only for 3 seconds, it began to decrease again, and its amplitude also began to decrease. For 1 minute 26 seconds it showed the decrease of 90 mmH₂O of pressure and the absolute value showed 530 mmH₂O of pressure. The amplitude decreased remarkably at 1 minute 26 seconds, slowly disappearing and its rhythm also becoming irregular. After 20 seconds more, the waves almost completely disappeared and its shape became irregular. At this point, the hyperventilation ceased. Then, the spontaneous respiration appeared almost at 6 seconds after the ceasing of hyperventilation, and the wave amplitude also appeared and at the same time, the intracranial pressure began to increase with 75 mmH₂O of pressure at 20 seconds after ceasing of hyperventilation. These variations of the wave caused by hyperventilation were due to pulsation, and the disappearance of the wave amplitude was due to the vasoconstriction of the brain stem arteries.

Fig. 17 showed the recording of the prefrontal lobotomy for a patient of psychiatric disorder as illustrated in Fig. 16. The waves in this recording showed the variation of intracranial pressure and plethysmogram caused by the manipulation of neck near the end of operation (the anesthesia was superficial). 300 mmH₂O of the absolute intracranial pressure, 40 mmH₂O of pressure as its amplitude, and 2.0 c/s in frequency were observed at first with relatively regular rhythm, but at the time of the manipulation of neck, bucking occurred, and at once the increase of about 1,000 mmH₂O of intracranial pressure was observed. The absolute value increased to 1,300 mmH₂O of pressure. At the same time, the waves with regular rhythm, which were observed until that point disappeared, and instead of these waves, the spindle formed waves of extremely irregular rhythm with 250 mmH₂O or 650 mmH₂O of pressure as

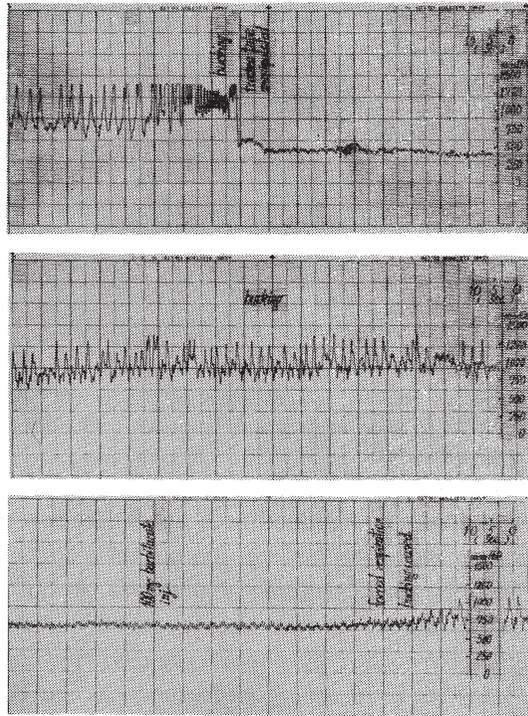


FIG. 17. The change of intracranial pressure and plethysmograph caused by the manipulation of neck.

amplitude continuing for 1.5 seconds were observed to continue for 3 minutes 20 seconds. It was observed that these spindle formed waves appeared at the same rhythm with bucking movement. After these bucking ceased, the pattern of forced respiration continued. As its wave shape showed in the second part of Fig. 17, it was observed that the slow waves with 90 mmH₂O of pressure as amplitude and 0.4 c/s in frequency were superimposed over the moderate quick waves with 60 mmH₂O of pressure as amplitude and 2.0 c/s in frequency. The absolute intracranial pressure decreased again to about 600 mmH₂O of pressure in the pattern of forced respiration. These pattern became quiet again by the administration of 100 mg sodium pentobarbiturate and showed almost same pattern as it before bucking. But, the absolute intracranial pressure did not decrease and showed 600 mmH₂O of pressure.

And in the following, several parts of remarkable change in the variation of intracranial pressure and plethysmogram recorded continuously during the whole process of trepanation for a patient with reoccurrence of brain tumor were shown.

Fig. 18 showed the recording of beginning of the process, just after inserting

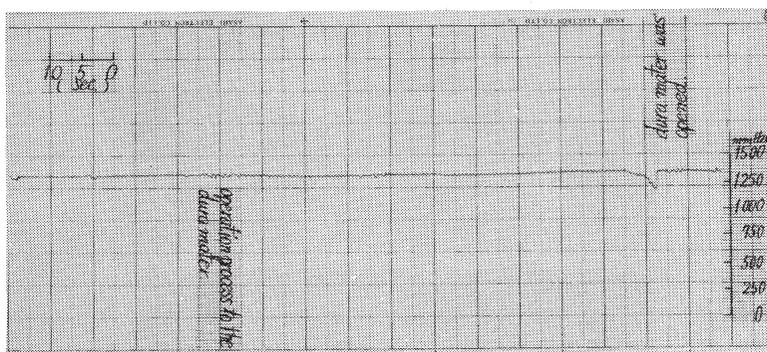


FIG. 18. The recording of intracranial pressure and plethysmograph during the trepanation for the patient with reoccurrence of brain tumor. This shows highly increased intracranial pressure and suddenly decrease by opening of the dura mater.

the strain gage at the edge of trephined hole not to interfere with the operation into the epidural space made for the operation. In this recording, this patient had a relative increased intracranial pressure of 1,300 mmH₂O because of reoccurrence of brain tumor. It was also observed that the small wave with 1.8 c/s in frequency was recorded by almost regular rhythm. By sectioning of dura mater, the intracranial pressure decreased 130 mmH₂O at once, but it increased again very quickly. It began to decrease again gradually, and the decreasing of about 30 mmH₂O of pressure was observed at about 22 seconds after opening of dura mater. The disturbance of waves observed on the way of recording occurred by the manipulation of the dura mater.

Fig. 19 showed the variation by the operative manipulation to the surface of brain. In this recording, the irregular wave and the spindle formed wave by the compression of brain tissue with brain spatula or the other operative manipulation after the corticotomy were observed. This recording shows, by

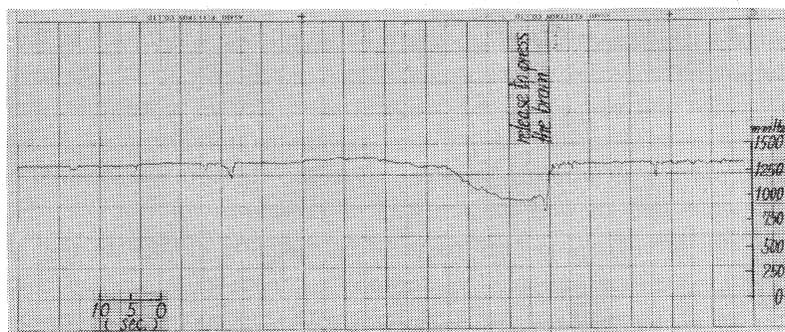


FIG. 19. The change of intracranial pressure and plethysmograph by the operative manipulation to the surface of brain.

the replacement of brain spatula, the intracranial pressure of 180 mmH₂O decreased at once, and it began to increase at the same time with the expansion of operation field by brain spatula, and it returned to the previous intracranial pressure. After this, it showed almost regular rhythm.

Fig. 20 showed the recording in the variation of intracranial pressure and plethysmogram by the complete removal of compression to the brain by spatula for the expansion of operative field. In this recording, the intracranial pressure of 1,300 mmH₂O during compression showed the decreasing of 600 mmH₂O of pressure and 680 mmH₂O of its absolute value during 6 seconds after the removal of the compression. The pressure began to increase again, and the wave with remarkable irregular rhythm was observed.

Fig. 21 showed the artifact in manometer caused by a electrocoagulation to the bleeding points of the brain surface. In this recording, it was observed that, when the electric current is switched off, the previous pattern quickly returns and there was little influence of electrocoagulation to the measurement

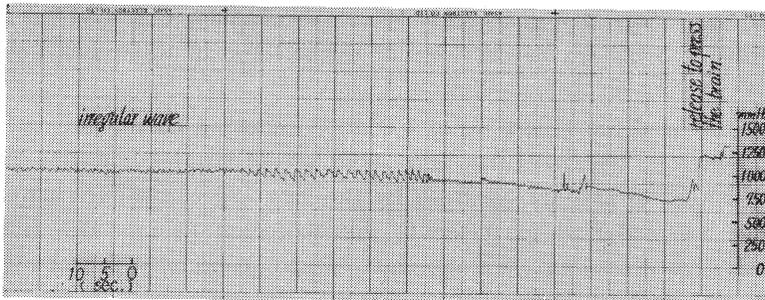


FIG. 20. The recording in the variation of intracranial pressure and plethysmograph by release of compression to the brain by spatula.

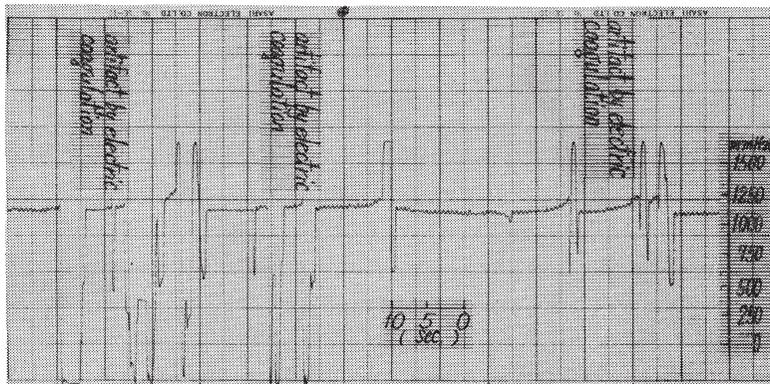


FIG. 21. The recording of the artifact in manometer caused by a electrocoagulation to the bleeding points of the brain.

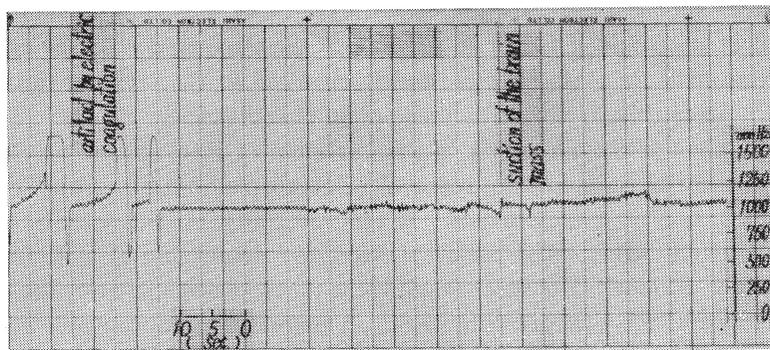


FIG. 22. The recording of the change occurred during the suction of brain tissue.

Fig. 22 showed the recording of the variation occurred during the retraction of brain tissue by a suction apparatus for inner decompression of brain because of the high brain tension. It was observed that the intracranial pressure changed quite irregularly in increasing and decreasing, and also with spindle formed wave. 3 big waves observed at the left end of this recording showed the artifact during electrocoagulation.

Fig. 23 showed the recording of variation occurred during the suturing of the dura mater. The irregular and large waves observed in this recording were also a kind of artifact occurred by the suturing of the dura mater, as the strain gage was inserted too close to the operation field. The remarkable variation in this recording were the wave shape, especially the change of its amplitude, and they showed the decreasing of the wave amplitude. That is, at first, the amplitude of about 30 mmH₂O of pressure decreased to 20 mmH₂O of pressure by the end of this recording. This variation became more remarkable after suturing of the dura mater, and when the scalp was sutured, the decreasing

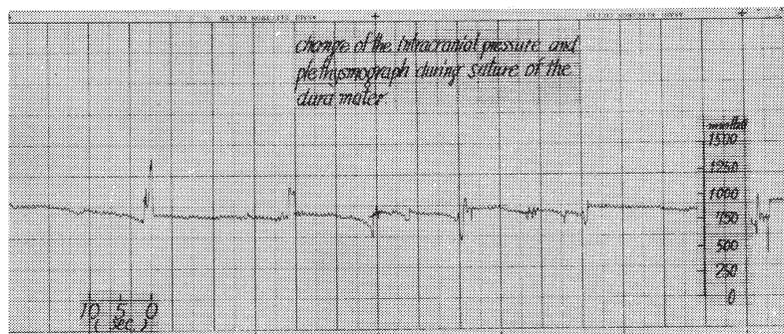


FIG. 23. The recording of the change occurred during the suturing of the dura mater.

of wave amplitude became more remarkable and it decreased to about 10 mmH₂O of pressure. These phenomena were observed at the beginning of the trepanation, with the opposite development after the scalp was sutured, that is, it was observed that, when trephined hole was made in the skull, the wave amplitude increased a little, and when the dura mater was opened, the wave amplitude increased remarkably.

5. HISTORICAL REVIEW

(A) *About the intracranial and the cerebrospinal fluid pressure*

Hippocrates (B.C. 460-377)²⁹⁾ has already observed and described that the brain of the skull defect or the anterior fontanel of infant has a certain tension and a movement with a certain rhythm. Gallen (A.C. 130-200)³⁰⁾ has also observed that this movement of the brain with a certain rhythm is the same with the rhythm of respiration or pulsation. After 200 years of Gallen's description, Oribasius³¹⁾ observed and described about the brain swelling by screaming and the decreasing of brain volume by inspiration. At this time, these observations were not expressed quantitatively, and quantitative measurement began for the first time in the beginning of 19th century. Quinke³²⁾ adopted for the first time in 1891 the lumbar puncture as clinical diagnostic method and measured the lumbar cerebrospinal fluid pressure with water manometer. He described that normal value of the cerebrospinal fluid pressure was 50 mmH₂O by his method.

After Quinke's description, Stadelmann (1795)³³⁾, Lenhartz (1896)³⁴⁾, and Allard (1909)³⁵⁾ used the same method, and described that the normal value of cerebrospinal fluid pressure was 40-130 mmH₂O in lumbar puncture. Claude, Lamarche, and Dunbar (1927)³⁶⁾ described that its normal value was 100-250 mmH₂O with the measuring subject on his side. And Ley (1932)³⁷⁾³⁸⁾ described that these differences between normal value was caused by the measuring method. Besides, Trattner (1932)³⁹⁾, Castex and Ontaneda (1931, 1932)⁴⁰⁾⁴¹⁾, Flexner and Weed (1933)⁴²⁾, Holthaus (1934)⁴³⁾, Lewis and Doune (1934)⁴⁴⁾, Obregia, Dimolescu and Paravanesu (1934)⁴⁵⁾, etc., discussed these normal value of cerebrospinal fluid pressure.

From 19th century, many studies about the lumbar cerebrospinal fluid pressure and the intracranial pressure were published. H. Cushing reported a series of studies about the intracranial pressure from 1901 to 1903 and made a beginning of new and important epoch in this field⁴⁶⁾⁻⁴⁸⁾. That is, he reported "concerning a definite regulatory mechanism of vasomotor center which controls blood pressure during cerebral compression", in 1902 about "some experimental and clinical observations concerning states of intracranial tension", and in 1903 about "the blood-pressure reaction of acute cerebral compression, illustrated by

cases of intracranial haemorrhage". These papers were referred thereafter in many publications about the intracranial pressure. In 1919, the measurement of ventricular pressure by cisternal puncture was also developed, and in 1932 Castex and Ontaneda described about this method.

As mentioned above, all of these methods at that stage were the measurement by water manometer using a glass tube. Lagergren reviewed in detail these methods in 1937, and he described also the optical manometer system which he developed for the continuous recording of the intracranial pressure⁴⁾. The effort to record the intracranial pressure continuously or graphically was made by Blackfan, Crothers, and Ganz in 1929. In their methods, they connected the needle of ventricular puncture or spinal puncture with kymograph, and the cerebrospinal fluid pressure was recorded continuously and graphically¹⁾. Aboulker tried to measure directly the intracranial pressure in 1936²⁾. Carmichael, Doupe, and Williams developed in 1937 a method of the continuous recording of ventricular cerebrospinal fluid pressure, in which the needle of ventricular puncture was connected with a tambour through a catheter in closed system, and the light was reflected by a mirror on the bromide paper³⁾. N. Antoni tried in 1946 to record graphically the cerebrospinal fluid pressure curve with the Lagergren's method which was improved by himself⁶⁾. All of these methods in the early stage were the measurement by connecting the spinal or ventricular lumen and the water manometer, mercury manometer, or the spring manometer. But these methods in the early stages had many undesirable variations and inaccuracy, and many artifacts were observed as Jacobson and Rothballer (1967) described²⁷⁾. Therefore, these methods should be discussed from the following points of view. The removal of cerebrospinal fluid by ventricular tap or lumbar tap influences the pressure itself as a kind of pressure leakage. The measurement is not accurate, because the connecting method is mechanical and incomplete, and there is no effective method to amplify the pressure. The free fluid communication of needle is sometimes blocked by choroid plexus, arachnoid membrane, nerve, or brain itself, and it is difficult to discover this blockage. It would be also difficult to record continuously because there are many artifacts such as by the movement of measuring subject or connecting tube, or by the specific gravity of fluid, and also to prepare the drift free recorder for the long term recordings.

Guillaume and Janny succeeded in 1951 to record continuously the intracranial pressure by using magnetic pressure transducer and connecting it with optical recorder system⁷⁾.

In about 1960, the low-displacement electromanometer was developed together with the development of electronics. Lundberg¹⁰⁾⁻¹⁵⁾, Grote^{49,50)}, Vedula¹⁹⁾, Eckel⁸⁾⁹⁾, Hemmer²¹⁾, Langfitt, *et al.*⁵¹⁻⁵⁵⁾, Hamit⁵⁶⁾, and Keegan and Evans¹⁶⁾, etc.⁵⁷⁾⁻⁶³⁾, reported many studies about intracranial pressure and

cerebrospinal fluid pressure by using electrical pressure transducer connecting with these electrical manometer. The devices which were used by these investigators were, so-called, extracranial pressure transducer. In spite of these methods, Cooper and Hulme (1966)²⁵⁾²⁶⁾, Numoto, Slater, and Donaghy (1966)⁶⁴⁾, and Jacobson and Rothballer (1967)²⁷⁾, used a device to be called as the intracranial pressure transducer to measure the intracranial pressure. But the device of Cooper and Hulme was still not sensitive enough, and there were a few waves which seemed to be an artifact. Device of Numoto, Slater, and Donaghy can measure only discontinuously. Jacobson and Rothballer measured the intracranial pressure still in the stage of animal experiment and did not describe the recorded pressure curve. The intracranial pressure transducer is thought a better method than the extracranial ones, as the transducer was inserted directly into the intracranial cavity in this method. In these points of view, those three methods as described above were not yet complete in the range of recordings. The device developed by the author can be satisfactory enough because its sensitivity is better than the previous device, and there are less artifacts.

(B) About the semi-conductor strain gage

The history of semi-conductor strain gage is very new, and it has been developed as a by-product of the solid physics in the development of transistor industry. C. S. Smith reported in 1954 theoretical ground and the experimental result about a piezoelectric resistance as a physical effect⁶⁵⁾. Its application to the strain gage was a few years after his report. In 1956, I. Igarashi (Toyota Central Research and Development Laboratories, Inc.) reported for the first time about the application of the semi-conductor material to the strain gage using Germanium as the main element²⁸⁾.

After his report, in 1957, W. P. Mason (Bell Research Institute) reported also about its application to the strain gage⁶⁶⁾. Since these reports, the application of strain gage to the transducer was tried by them in various kinds of fields, and is still being activity studied by them. But these applications were all for the industrial usage, and there are only a few medical applications in the world, and there is no record about its medical usage in Japan.

Therefore, its application for medical purpose should be developed further in the future.

6. DISCUSSION

The measurement of the absolute intracranial pressure and the variation of cerebral plethysmogram can be recorded by the strain gage method. It was observed through the simultaneous measurement of ventricular pressure by the previous method and intracranial pressure by the new method that the

measurement using this method is more sensitive and faster responses can be made than measurement by lumbar puncture, ventricular puncture, or the puncture of cisterna magna. That is, all process of the changes of measurement by this method was finished within 1-2 seconds, but those of the change of measurement by water manometer through glass tube began to response more after 3-5 seconds at the beginning of the change. It was also observed that in ventricular pressure, the range of pressure change is smaller than the pressure change in this device. The facts can be explained by the following reasons: it is impossible to observe the pressure change by amplifying its change in the method by watermanometer through glass tube, and since it becomes a leakage for the pressure, to measure by leading the cerebrospinal fluid to glass tube of water manometer, it is questionable whether the actual pressure delivers correctly or not. During the experiment, the author could confirm that the closed system delivers more correctly the pressure change than that of the open system, and same results have been observed by other investigators^{25),26),27)}^{50),51),52),53)}. Since the pressure is observed by water manometer through fine puncture needle with which lead the cerebrospinal fluid, it is natural that the reaction speed become slow because of its passage. Especially, since the cerebrospinal fluid pressure is measured through complicated passage by lumbar puncture, it can be thought easily that the problem become more complicated and the reaction speed became slower. As mentioned previously, this device has many advantages: the device is so small and light that it can be carried easily, and its setting up, handling, and insertion to the intracranial cavity is very simple. Furthermore, the apparatus is sturdy enough to be used for the research and for clinical measurement. But as the sensitive element of strain gage is a semiconductor material and it is very delicate, it must be handled with care. It is advisable to protect the apparatus by a cap and to disinfect it with the cap on.

Although other disadvantages of the device are described in the paragraph of the special character of the apparatus and method, they are of little concern in comparison with the great value of its advantageous points. As the length of the cord between strain gage, manometer, and recorder are long enough to change these arrangement freely, the other operative manipulation are not disturbed and the measuring subject can move to a certain degree. These facts are very convenient in the clinical measurement. Furthermore, the artifact such as movement by lengthening the cord is not recognized.

It is clear that this method is excellent in the comparison with the other methods for the measurement of the intracranial pressure. Though there is additional operative manipulation to the subject when putting this device in place, this method is more excellent than the other methods because the informations acquired by this method and accuracy of the results of measurement

are numerous, and there are minimal artifacts in recording.

This method is also excellent in the comparison with extracranial pressure transducer such as catheter-transducer system or balloon-transducer system in the following points. (a) no leakage of cerebrospinal fluid pressure, (b) no artifact by tubing movement, (c) no artifact by the specific gravity of cerebrospinal fluid, (d) no kinking of the tube or catheter, (e) the measuring subject is freely allowed to move. Most important of all (f) the developed apparatus is very simple and easy in its handling as above described.

So-called, intracranial pressure transducer used by Cooper and Hulme, and Jacobson and Rothballer are also semi-conductor strain gage of the same kind as the author's device. But in sensitivity, the output power of the former's device is 0.05 mV/100 mmH₂O of pressure, those of the latter's device is 0.04 mV/100 mmH₂O of pressure. On the contrary, those of the author's device is 0.24 mV/100 mmH₂O of pressure, accordingly, the author's device has 4.8 or 6 times the output power of their devices.

About the method of fixation to the intracranial cavity of the device, the former inserted the transducer in the skull and fixed it to the skull by using special device which they developed newly and it is a clamped edge method. It is a well-known fact that this clamped edge method is not so sensitive as the supported edge method. That is, in their fixation, the brain tension is transmitted through Teflon film in subdural space to the transducer and there is a possibility which the transmitted pressure is disturbed by the Teflon film, and the metallic sleeve admitting the transducer disturbs the transmission of the pressure. In the extreme cases such as in the increased intracranial pressure as described previously it can be thought that the device may penetrate into the brain tissue. As this transducer is supported not by skull, but by a stainless steel, the pressure is measured actually that which the brain tension delivers to the stainless steel. There seems to be a little difference of the pressure of brain tissue to the skull in the physiological meaning. These differences of the method of fixation seems to come from the difference of the thickness between the author's device and the Cooper and Hulme, that is, the thickness of the strain gage used by author is 1.5 mm in thickness and thinner than those of Cooper and Hulme's device. Jacobson and Rothballer fixed the transducer in the subdural space, but in the author's experience, it was difficult to make contact the sensitive plate of disc with the arachnoid membrane tightly if the special apparatus is not used to fix the transducer. Therefore, the direct intracranial pressure was not measured in subdural space because the disc was liable to be oblique to the arachnoid membrane because of the softness of the brain tissue. Although it was examined by inserting the strain gage into the intracerebral, subdural, and epidural space, the insertion to intracerebral space was unsuitable because of a large damage to the brain tissue and tendency to

push out the strain gage by the increased intracranial pressure, and also there may be some damage to the surface of the brain because of the stainless steel material of the disc. Besides the above described facts, it is especially remarkable in measuring the increased intracranial pressure. In these points of view, it is most suitable to insert the strain gage to the epidural space, because there is a dura mater in the epidural space, and this membrane is strong enough not to allow damage the brain tissue, and the inserting and the removing after the end of measurement also are very simple.

The fixation-apparatus which the author has developed is necessary. Because if the strain gage is inserted to the epidural space without these fixation-apparatus, the strain gage is moved by the body movement of the measuring subject and by the other operative or examination manipulation, and the pressure observed has a kind of artifact.

About the method of disinfection, the author's device can be disinfected by boiling water of 100°C, but Cooper and Hulme's device are disinfected by immersion in 70% alcohol containing 0.5% of hibitane and one hour at least is needed for disinfection. As the temperature of the strain gage was compensated almost completely, as described previously, it is not necessary to be concerned about it. That is, it can be thought that human body temperature can not change over 1°C within 20 seconds, and it can not be also thought that the temperature changes over the range from 20°C to 40°C.

And about the recorder as the zero range of the recorder is divided into 14 stages, and its chart speed can be changed into 6 stages, this recorder has a advantage which the range and speed can be chosen freely for the purpose of measurement. That is, when the continuous recording of the change of the intracranial pressure is observed mainly, high zero range and slow speed must be chosen, and when the change of the plethysmogram is observed mainly, low zero range and high chart speed must be chosen. These facts are clear in the result of recording as described in Fig. 10. It could be confirmed that the base line stability of the author's device is also completely stable by placing it in the room. But as the sensitivity of a transducer is very high, if the atmospheric air in the room moves, or if the wind touches to the transducer, it is observed that this device records the pressure according to the strength of the wind or the air movement.

And next, the data of actual measurement is discussed as follows. There are two kinds of waves observed in the intracranial cavity as a pressure wave. And it was observed already from the early stage that these waves come from respiration and pulsation. Recently, Grote⁴⁹⁾⁵⁰⁾, Hamit⁵⁶⁾, Lundberg¹⁰⁾⁻¹⁵⁾, Schild and Dunber²²⁾, and so on, discussed this fact from various point of view. There are many theories about the origin, that is, one says that they are a capillary effect of arachnoid membrane, the other says also that they are caused by brain

stem arteries, and another says that they come out from the pressure gradient of the chest cavity. The author also could observe these facts as showed in Fig. 9.

The intracranial pressure changes by compression of neck or abdomen shown in Fig. 11 were the same fact described by Queckenstedt⁶⁸⁾ and Stookey⁶⁹⁾⁷⁰⁾, and Gilland described that the pressure increasing by compression of neck was $250 \text{ mmH}_2\text{O} \pm 8.5 \text{ mmH}_2\text{O}$ ⁷¹⁾⁷²⁾, but the author's record showed $90 \text{ mmH}_2\text{O}$, and these difference could be explained as follows. Gilland compressed the neck completely by using the special cuff around the neck developed by him, but as the author compressed it by fingers, it was not compressed so completely as Gilland. Jerema reinvestigated in 1966 about Queckenstedt's test and Stockey's test in the cases of passage obstruction of cerebrospinal fluid, in which he observed that there was no increase of pressure in spinal canal⁷³⁾. These facts are very useful for the diagnosis of the blockage of cerebrospinal fluid. The special character of frequency in this device is determined so that the quick change observed in this recording also can be caught correctly.

The increasing of intracranial pressure by apnea was showed in Fig. 12, and it was caused by the hypoxia occurred in the brain tissue. These facts were reported by Michenfelder (1965)⁷⁴⁾, Alexander (1964)⁷⁵⁾, and Lundberg (1965)¹⁰⁾, etc.

It could be confirmed, as showed in Fig. 16, that the increased intracranial pressure was reduced by hyperventilation, and the other authors also described about these facts, that is, Michenfelder (1965), Lundberg (1960), Gotoh *et al.* (1965)⁷⁶⁾, McHenry *et al.* (1965)⁷⁷⁾, etc. These facts show that the ventilation in anesthesia for the operation in the field of neurosurgery has a important role especially in regard to the control of the intracranial pressure⁷⁴⁾.

The variation by intravenous administration of "Theraptique" showed in Fig. 13 are caused by those of pulsation, as "Theraptique" tend to increase the systemic blood pressure; consequently, it was shown that the amplitude of the wave caused by increased respiration. The increase of the absolute intracranial pressure was explained that, when no respiration appeared, the pressure increase by hypoxia of brain tissue through apnea was added to the increase of systemic blood pressure.

The increase of absolute intracranial pressure by forced respiration as shown in Fig. 14 can be explained that the intrathoracal pressure is increased by forced respiration and the venous pressure also increased, consequently, the pressure of juglar vein increased. From this observation, it can be said that the role of venous pressure as one of the factors which supported the intracranial pressure, is very important. And it was observed that, at the same time when the condition of forced respiration was released, the intrathoracal pressure decreased and then the intracranial pressure also decreased.

And next, by the intravenous administration of 50 ml of 50% Glucose solution, the measuring subject became apnea, but in this case, the increase of the intracranial pressure was not observed. This fact is explained as the result which the 50% Glucose solution decreasing effect of intracranial pressure depresses the increasing pressure caused by hypoxia. The increasing of the intracranial pressure which was observed after the appearance of spontaneous respiration could be thought as a rebound effect of 50% Glucose solution.

The decreasing of intracranial pressure and of amplitude of pressure wave was observed by giving hyperventilation as showed in Fig. 16. As the pressure wave began to disappear, it was thought to be due to vasoconstriction¹⁰⁾⁵⁷⁾⁷⁴⁾. Accordingly, the artificial respiration in the anesthesia during operation in neurosurgery must be done by strictly normal ventilation as long as possible.

The pressure change by coughing as shown in Fig. 16, and Fig. 17, increased from 200 mmH₂O of pressure to 1,000 mmH₂O of pressure (due to a forced respiration) and amplitude of the wave changed in the range of 250-650 mmH₂O of pressure. In this recording, it is observed that the amplitude of the change of intracranial pressure is larger than by the measurement of the other method. This fact expresses that the actual amplitude of the changing intracranial pressure is practically so large as it is observed in this recording, and the intracranial pressure is not so stable.

About the intracranial pressure during the trepanation is shown in Fig. 18, Fig. 20, Fig. 21, Fig. 22, and Fig. 23. The intracranial pressure during the trepanation shows various kind of change not only in the absolute pressure value but also in the plethysmogram as it is shown in Figs.

But in these operative cases, as a big skull fragment is removed and a same sized trephine hole is made, it can be thought as a kind of open system. Because of these open system, the remarkable increasing of intracranial pressure can not be observed, because trephined hole is similar to a leakage of the pressure. But with these recordings, it can be observed that human can resist to a considerable change of the intracranial pressure.

The waves showed irregular and large change during the suturing of the dura mater. In Fig. 23, there are kinds of artifact caused by picking up or letting go the dura mater. In order to make these artifact disappear, the inserting position of the strain gage, under the skull as deep as possible, should be chosen at the edge of the operation field, so that there is no disturbance caused by operative manipulation.

In the case of intracranial pressure in which the brain tissue is compressed to the upper limit of the capacity of the intracranial cavity and the strain of the blood vessel is reduced by vasomotor paralysis, the amplitude of pressure wave was also reduced⁵⁴⁾⁶²⁾⁶³⁾. In the normal cases, the amplitude of pressure wave increased in closed system because of no leakage for pressure. All of

the pressure change and of the change in plethysmogram in clinical measured data were also described by many of the other authors. In these points of view, the results recorded and measured by this device can be reliable.

Now, here the author will describe about the future of this device. The trial for the telemetering of the physiological value become active by using the technique of electronics which has been developed recently. In 1957, Mackay and Jacobson⁷⁸⁾⁷⁹⁾⁸⁰⁾, and in the same year, Zworykin and Farrer⁸¹⁾⁸²⁾, reported about this method for telemetering, and in Japan, Wtanuki, *et al.* (1962)⁸³⁾, also reported about this telemetering.

The author found out the possibility of telemetering in this device, and in the near future, the author wants to deliver the continuous recording of the intracranial pressure to this telemetering. Already, in Toyota Central Research and Development Laboratories, Inc., the development of transmitting capsule for telemetering connecting with strain gage is finished. Fig. 24 shows its exterior view and its electrical circuit arrangement. The under part of Fig.

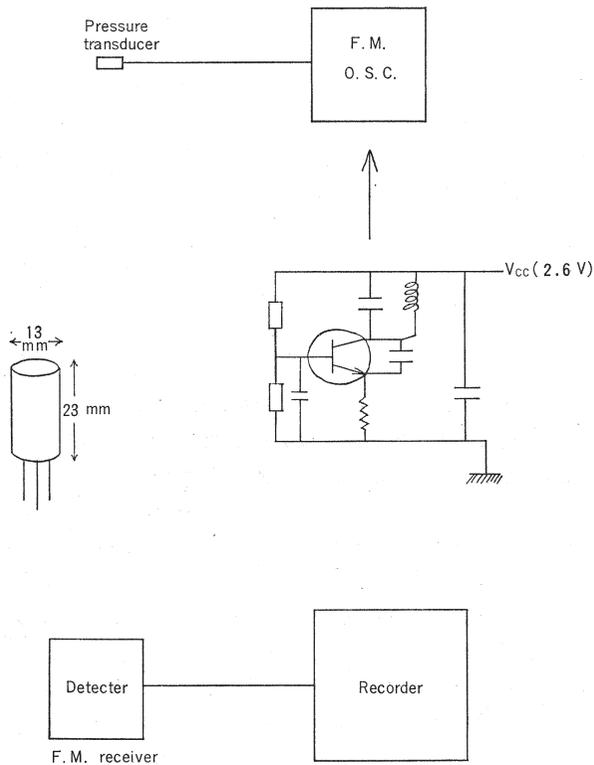


FIG. 24. The exterior view and the electrical circuit arrangement of the transmitting capsule for telemetering.

24 is the arrangement of receiver and recorder. FM receiver is usable for its receiver, and the recorder which is used at present also is usable. As transmitting capsule is a very small as in Fig. 24 shows, the fixation of strain gage, transmitting capsule, and battery for capsule does not cause any disturbance to the measuring subject. The measurement of intracranial pressure by this telemetering is very convenient, that is, as the measuring subject does not need cord or recorder, they can quite freely move, and its management also become easy. And the efficiency range to receive by this transmitting capsule, is within 1,000 m, and the telemetering between the ward and the nurse station become possible, and it can be utilized more effectively.

But in this stage, the output power of transducer is not enough for the telemetering. Since the output power of 10 times of this device is needed, the telemetering is impossible by this device at present. But in the near future, transducer with such a output power will be developed, and the author predicts that there is a great possibility of telemetering of the continuous recording of the intracranial pressure and the cerebral plethysmogram.

7. SUMMARY

The author described the device for the direct and continuous measurement of the intracranial pressure and also reported the result of animal experiments and of a few clinical measurements. The variation of the intracranial pressure and also of the plethysmogram on the surface of brain was recorded and observed continuously.

This device and measuring method have the advantageous points as follows :

1) It is possible to record quickly and correctly the sudden change of pulsation, respiration, or intracranial pressure by its special character about the frequency.

2) It is more sensitive than the previous devices.

3) It is a small and portable, easy to set up and simple to handle.

4) In measurement, it uses no fluid nor catheter.

5) The disinfection of this device can be done in boiling water of 100°C.

6) The fixation method to the intracranial cavity is also simple as described previously.

7) After measuring, it can be removed very easily.

8) The other examination and operation is not disturbed by its insertion, and since the body movement of the measuring subject is free in a certain degree, its insertion gives little damage to the measuring subject.

9) The temperature dependency of semi conductor strain gage is almost compensated.

10) As the sensitive plate is fixed by the supported edge method, its sen-

sitivity is very high.

11) As the measuring system is completely closed, in comparison with extra-cranial pressure transducer, there are no pressure leakage of cerebrospinal fluid pressure, the risk of infection is minimal, and there are also no artifacts like by tubing movement, by cerebrospinal fluid gravity and by catheter blockage.

Therefore, it can be expected to get useful and accurate information about intracranial pressure and cerebral plethysmogram by this device and method.

And on contrary, the disadvantageous points of this device and method are as follows:

1) An additional operative manipulation to the measuring subject is necessary.

2) As the semi-conductor element is very delicate, it should be handled with extreme care.

Furthermore, the author has reported that it is possible to develop the continuous measuring device and method for the variation of the intracranial pressure and the plethysmogram on the surface of brain with telemetering in the near future by further developing the device.

ACKNOWLEDGEMENT

The author wishes gratefully to acknowledge Prof. Dr. Yoshio Hashimoto and Dr. Kinjiro Iwata, 1st Dept. of Surgery, School of Medicine, University of Nagoya, Japan, for their constant advice and encouragement, and also to thank Dr. Isemi Igarashi, Toyota Central Research and Development Laboratories, Inc., for his technical assistance for this device.

REFERENCES

- 1) Blackfan, K. D., Grothers, B. and Ganz, R. N., Transmission of intracranial pressure in hydrocephalus in infancy, *Amer. J. Dis. Child.*, **37**, 893, 1929.
- 2) Aboulker, H., Tonométrie cérébrale directe. Assai de mesure directe de la pression intradurale, *Ann. oto-laryng. (Paris)*, **11**, 1144, 1936.
- 3) Carmicahel, E. A., Doupe, J. and Williams, D. J., The cerebrospinal fluid pressure of man in erect posture, *J. Physiol. (London)*, **91**, 186, 1937.
- 4) Lagergren, S., Studien über den spinalen Block mittels optischer Registrierung und mit besonderer Berücksichtigung der respiratorischen Druckschwankungen, Helsingfors: Mercators Tryckeri., **538**, 1937.
- 5) Väino Seiro, Über die Messung des intraduralen Reserveraums. (Measurement of intradural reserve space by recording changes in CSF pressure following intraspinal injection of saline.), *Acta chir. scand.*, **89**, 139, 1943.
- 6) Antoni, N., Pressure curves from the cerebrospinal fluid, *Acta Med. scand., suppl.*, **170**, 439, 1946.
- 7) Guillaume, J. and Janny, P., manométrie intracranienne continue., *Rev. neurol.*, **84**, 131, 1951.
- 8) Eckel, K., Liquortonograph; A clinical apparatus for determination of the dynamics,

- Acta Neurochirurg.*, **2**, 431, 1952.
- 9) Eckel, K. und Gund, A., Liquordruck-Registrierung bei raumfordern Prozessen, *Wien Z. Nervenheilk.*, **21**, 191, 1963.
 - 10) Lundberg, N., Kjaellquist, A. and Bien, C., Reduction of increased intracranial pressure by hyperventilation, *Acta Psychiat. Neurol. Scand.*, **34**, Suppl., 139, 1, 1959.
 - 11) Lundberg, N., Continuous recording and control of ventricular fluid pressure in neurosurgical practice, *Acta Psychiat. Neurol., Scand.*, **36**, Suppl., 149, 1, 1960.
 - 12) Ingvar, D. H. and Lundberg, N., Paroxysmal symptoms in intracranial hypertension studies with ventricular fluid pressure recording and electroencephalography. *Brain*, **84**, 446, 1961.
 - 13) Lundberg, N. and Ponten, U., Some observations on postoperative ventricular fluid pressure. *Acta Neurol. Scand.*, **39**, 264, 1963.
 - 14) Kjaellquist, A., Lundberg, N. and Forter, U., Respiratory and cardiovascular changes during rapid spontaneous variations of ventricular fluid pressure in patients with intracranial hypertension. *Acta Neurol. Scand.*, **40**, 291, 1964.
 - 15) Lundberg, N., Continuous recording of the ventricular fluid pressure in patients with intracranial hypertension. *J. Neurosurg.*, **22**, 581, 1965.
 - 16) Keegan, H. R. and Evans, J. P., Studies of cerebral swelling, III. Long term recordings of cerebrospinal fluid pressure before and following parenteal urea. *Acta Neurochirurg*, **10**, 466, 1962.
 - 17) Rothballer, A. B., Continuous recordings of intracranial pressure in man and animals, Presented at meeting of Harvey Cushing Society, Philadelphia, *Penn.*, 1963.
 - 18) Loroche, G. S., An aid to be measurement of cerebrospinal fluid pressure. *Lancet*, **1**, 24, 1964.
 - 19) Vedura, J., White, R. J. and Albin, M., Chronic measurement of cerebrospinal-fluid pressure in the dog. A new method and results. *J. Neurosurg.*, **21**, 1047, 1964.
 - 20) Hoppenstein, R., A device for measuring intracranial pressure. *Lancet*, **1**, 90, 1965.
 - 21) Hemmer, R., Cerebrospinal fluid dynamics, *Deutsch. Med. Wschr.*, **91**, 867, 1966.
 - 22) Dunber, H. S., Guthrie, T. C. and Karpell, B., A study of cerebrospinal pulse wave, *Arch. Neurol.*, **14**, 624, 1966.
 - 23) Tsunoda, M., Experimental studies on acute increase of intracranial pressure. Pathological studies using continuous recording of intracranial pressure. *Brain nerve*, **18**, 1087, 1966 (in Japanese).
 - 24) Yada, K., Abe, H. and Tsuru, M., Intracranial pressure following intracranial surgery, *Brain Nerve*, **19**, 565, 1967 (in Japanese).
 - 25) Hulme, A. and Cooper, R., A technique for the investigation of intracranial pressure in man, *J. Neurol. Neurosurg. Psychiat.*, **29**, 154, 1966.
 - 26) Cooper, R. and Hulme, A., Intracranial pressure and related phenomena during sleep, *J. Neurol. Neurosurg. Psychiat.*, **29**, 564, 1966.
 - 27) Jacobson, S. A. and Rothballer, A. B., Prolonged measurement of experimental intracranial pressure using a subminiature absolute pressure transducer, *J. Neurosurg.*, **24**, 603, 1967.
 - 28) Chiku, T. and Igarashi, I., Some applications of semiconductor strain gages. 20th Annual ISA Conference and Exhibit, Preprint No. 17. 11-3-65, 1, 1965.
 - 29) Hippokrates, refered from Grote, W.
 - 30) Gallen, refered from Grote, W.
 - 31) Oribasius, refered from Grote, W.
 - 32) Quinke, refered from Grote, W.
 - 33) Stadelmann, E., Ein Beitrag zur diagnostischen Bedeutung der Lumbalpunktion, *Verl. Klin. Wschr.*, 581, 1895.
 - 34) Lenhartz, H., Über den diagnostischen und therapeutischen Wert der Lumbalpunktion,

- Munch. Med. Wschr.*, **43**, 169, 1896.
- 35) Allard, E., Die Lumbalpunktion, *Erg. Inn. Med.*, **3**, 100, 1909.
 - 36) Claude, H., Lamarche, A. and Dubar, J., Procédé de mesure de la tension du liquide céphalo-rachidien, *Progr. méd.*, **55**, 1384, 1927.
 - 37) Ley, A., La manométrie du Liquide C. R. dans la clinique, *Fol. neuropath. Eston*, **12**, 165, 1932.
 - 38) Ley, A., Die Manometrie des Liquors in der Klinik, *Rev. cir. Barcelona*, **4**, 391, 1932.
 - 39) Trattner, H. R., Graphic recording of spinal fluid pressure with the hydrophorograph, *J.A.M.A.*, **98**, 1081, 1932.
 - 40) Castex, M. R., Ontaneda, L. E. and Mazzei, E. S., Änderung des Liquordrucks bei Stellungwechsel, *Rev. oto.-neuro.-ophthalm. sud.-amer.* **6**, 11, 1931.
 - 41) Castex, M. R. and Ontaneda, L. E., Der Druck des Liquors in großen Zisterne, *Prensa med. argent.*, **18**, 1427, and 1473, 1932.
 - 42) Flexner, L. B. and Weed, L. H., Factors concerned in positional alterations of intracranial pressure, *Amer. J. Physiol.*, **104**, 681, 1933.
 - 43) Holthaus, B., Bemerkung zur suboccipitalen Liquordruckmessung, *Psychiatr. neurol. Wschr.*, **80**, 1943.
 - 44) Lewis, R. N. and Daune, W., Observations on cerebrospinal fluid pressure, *Arch. Neurol. Psychiatr.*, **31**, 204, 1934.
 - 45) Obregia, A., Dimolescu and Parvanescu, Untersuchungen über den Druck der Cerebrospinalflüssigkeit und über das postpunktionelle Syndrom, *Z. Neurol. Psychiatr.*, **150**, 748, 1934.
 - 46) Cushing, H., Concerning the definite regulatory mechanism of the vaso-motor center which controls blood pressure during cerebral compression, *Bull. Johns Hopk. Hosp.*, **12**, 290, 1901.
 - 47) Cushing, H., Some experimental and clinical observation concerning states of increased intracranial tension, *Amer. J. Med. Sci.*, **124**, 375, 1902.
 - 48) Cushing, H., The blood-pressure reaction of acute cerebral compression, illustrated by cases on intracranial hemorrhage, *Amer. J. Med. Sci.*, **125**, 1017, 1903.
 - 49) Grote, W. und Wülenweber, R., Über Liquordruckkriesen. Spontane Druckschwankungen bei intracraniellen Liquorpassagestörungen. *Acta Neurochirurg.*, **9**, 125, 1961.
 - 50) Grote, W., Gehirnpulsationen und Liquordynamik, *Acta Neurochirurg Suppl.*, **12**, 1964.
 - 51) Langfitt, T. W., Shawaluck, P. D., Mahoney, R. P., Stein, S. C. and Hedges, T. R., Experimental intracranial hypertension and papilledema in the monkey, *J. Neurosurg.*, **21**, 469, 1964.
 - 52) Langfitt, T. W., Weinstein, J. D., Neal, B. S., Kassell, N. F. and Simeone, F. A., Transmission of increased intracranial pressure. I within the craniospinal axis, *J. Neurosurg.*, **21**, 989, 1964.
 - 53) Langfitt, T. W., Weinstein, J. D., Kassell, N. F. and Gafiardi, L. J., Transmission of increased intracranial pressure. II Within the supratentorial space, *J. Neurosurg.*, **21**, 998, 1964.
 - 54) Langfitt, T. W., Weinstein, J. D. and Kassell, N. F., Cerebral vasomotor paralysis produced by intracranial hypertension, *Neurology*, **15**, 662, 1965.
 - 55) Langfitt, T. W., Kassell, N. F., and Weinstein, J. D. Cerebral blood flow with intracranial hypertension, *Neurology*, **15**, 761, 1965.
 - 56) Hamit, H. F., Beall, A. C. Jr. and DeBakey, M. E., Hemodynamic influences upon brain and cerebrospinal fluid pulsations and pressures, *J. Trauma*, **5**, 174, 1965.
 - 57) Stern, W. E., Studies in experimental brain swelling and brain compression, *J. Neurosurg.*, **19**, 676, 1962.
 - 58) Beks, J. W. F. and Walter, W. G., Needle with fixation system for permanent pressure measurement, *J. Neurosurg.*, **22**, 515, 1965.

- 59) Troupp, H., Intraventricular pressure in patients with severe brain injuries, *J. Trauma*, **5**, 373, 1965.
- 60) Beks, J. W. F., Ter Weeme, C. A., Ebels, E. J., Walter, W. G. and Wassenaar, E. J., Increase in intraventricular pressure in cold induced cerebral oedema, *Acta Physiol. Pharmacol. Neerl.*, **13**, 317, 1965.
- 61) Thomas, L. M., Roberts, V. L. and Gurdjian, E. S., Experimental intracranial pressure gradients in the human skull, *J. Neurol. Neurosurg. Psychiat.*, **29**, 404, 1966.
- 62) Noell, W. and Schneider, M., Zur Hämodynamik der Gehirndurchblutung bei Liquordrucksteigerung, *Arch. Psychiat. Nervenkr.*, **180**, 713, 1948.
- 63) Hedges, T. R., Weinstein, J. D. and Stein, S., Cerebrovascular responses to increased intracranial pressure, *J. Neurosurg.*, **21**, 292, 1964.
- 64) Numoto, M., Slater, J. P. and Donaghy, R. M., An implantable switch for monitoring intracranial pressure, *Lancet*, **1**, 528, 1966.
- 65) Smith, C. S. Piezoresistance effect in Germanium and Silicon, *Phys. Rev.*, **94**, 42, 1954.
- 66) Mason, W. P. and Thurston, R. N., Use of piezoresistive materials in the measurement of displacement, force, and torque, *J. Acoust. Soc. Amer.*, **29**, 1096, 1957.
- 67) Schild, W., Weise, H. and Siemons, K., Recording of pulse and dynamics of cerebrospinal fluid, *Ärztl. Wschr.*, **11**, 107, 1965.
- 68) Queckeustedt, H., Zur Diagnose der Rückenmarkskompression, *Dtsch. Zschr. Nervenhhk.*, **55**, 325, 1916.
- 69) Stookey, B. and Klenke, D., A study of the spinal fluid pressure in the differential diagnosis of disease of the spinal cord, *Arch. neurol. psychiat.*, **20**, 84, 1928.
- 70) Stookey, B., Merwart, H. R. and Franz, A. M., A study of the spinal fluid pressure in suspected spinal cord tumors, *Surg. Gynec. Obstet.*, **41**, 429, 1925.
- 71) Gilland, O., Cerebrospinal fluid dynamic diagnosis of spinal block II. The spinal CSF pressure-volume curve, *Acta Neurol. Scand.*, **41**, 487, 1965.
- 72) Gilland, O., Cerebrospinal fluid dynamic diagnosis of spinal block V. Uniform lumbar electromanometrics, *Neurology*, **16**, 1110, 1966.
- 73) Jerema, M., The disturbances in the dynamics of the CSF as auxiliary diagnostic sign, *Bull. Pol. Med. Sci. Hist.*, **9**, 67, 1966.
- 74) Michenfelder, J. D. and Terry, H. R., Current practices and trends in neuroanesthesia, *Clin. Neurosurg., Congr. of Neurol. Surgeons*, **13**, 252, 1965.
- 75) Alexander, S. C., Wollman, H., Cohen, P. J., Chase, P. E. and Behar, M., Cerebrovascular response to PaCO₂ during halothane anesthesia in man, *J. Appl. Physiol.*, **19**, 561, 1964.
- 76) Gotoh, F., Meyer, J. S. and Takagi, Y., Cerebral effects of hyperventilation in man, *Arch. Neurol.*, **12**, 410, 1965.
- 77) McHenry, L. C., Slocum, H. C., Bivens, H. E., Mayes, H. A. and Hayes, G. J., Hyperventilation in awake and anesthetized man: effects on cerebral blood flow and cerebral metabolism, *Arch. Neurol.*, **12**, 270, 1965.
- 78) Mackay, R. S. and Jacobson, B., Endoradiosonde, *Nature*, **179**, 1239, 1957.
- 79) Mackay, R. S. and Jacobson, B., Pill telemeters from digestive tract, *Electronics*, **51**, 1958.
- 80) Mackay, R. S., Radio telemetering from within the human body. *IRE Trans. on Med. Electronics ME-8*, 100, 1959.
- 81) Farrer, J. T., Zworykin, V. K. and Baum, J. Pressure sensitive telemetering capsule for study of gastrointestinal motility, *Science*, **126**, 975, 1957.
- 82) Farrer, J. T. and Bernstein, J. S., Recording of intraluminal gastrointestinal pressure by a radiotelemetering capsule, *Gastroenterology*, **35**, 603, 1958.
- 83) Watanuki, T., Hori, M. and Suma, K., The present and the future of clinical application of telemetering by capsule. Especially, in the field of digestive disorder, *Shinryo*, **16**, 28, 1963 (in Japanese).