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TREATMENT OF EMBOLISM AND THROMBOSIS OF THE CEREBRAL VESSELS*

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I should like to call your attention to the possibility of influencing cerebral circulation by very simple methods in cases of cerebral vascular accident caused either by embolism or by thrombosis. I shall not concern myself here with head injuries justifying surgical intervention at the site of the haemorrhage, but will refer only to medical cases in which coma can be alleviated in a few minutes and the extent of the hemiplegia considerably reduced.

I should like, too, to tell you about the hypothesis that has guided me in my clinical researches on this matter for 25 years. I started from the same general pathological principle which I tried to verify in other parts of the body: autonomic dysfunction—that is, a vasomotor disturbance—usually precedes, often causes, and practically always accompanies such a vascular accident.

In the case of the cerebral circulation, I presupposed that isolated spasm could exist, comparable to that which may be observed in the limbs. And particularly I thought that such a spasm would always accompany the occurrence of an embolism or thrombosis, causing around the ischaemic area perifocal zones of diminished circulation.

I knew that the then prevailing ideas did not take into consideration the possibility of vasomotor reactions in the cerebral arteries. It was claimed, with a certain degree of finality, that the brain circulation was protected from vasomotor fluctuations by some extracranial regulating system. About 1920 I undertook to investigate this question. In various clinical conditions, working mostly on war-wounded who had undergone brain surgery, I performed what I called an "internal carotid sympathectomy"; in other words, I removed as much as I could of the tunica adventitia of the internal carotid, starting from its bifurcation. Immediately the artery would contract; then the retinal vessels diminished in calibre and the retinal pressure dropped. After a few hours a vasodilatory reaction would be observed, evidenced by dilatation of the same retinal vessels with rising of the retinal pressures. Simultaneously the flattened trephine scars would be seen to swell. When dealing with patients with Jacksonian epilepsy due to war

injuries, I would first perform this internal carotid sympathectomy under local anaesthesia, then immediately after, still under local anaesthesia, widely open the skull to excise the arachnoid cortical scar tissue. The cortical vessels of those patients were so constricted that it appeared to me that this operation should provide a means of ensuring preliminary haemostasis in brain surgery, and at the time I reported a number of operations performed in this fashion. Denervation of the carotid body gave the same results.

This substantiated the idea that the small brain arteries had their own vasomotor control. Moreover, it was histologically confirmed that the pia mater and intracerebral arterioles had a double innervation of myelinated and amyelinated fibres. This was not consistent with a total functional inactivity. At any rate, from that time on it seemed to me that it was possible to modify the activity of the cerebral blood vessels by surgical intervention, and I should like to give you two examples of what can be accomplished.

Effects of Resection of a Thrombosed Common Carotid Artery and of Pericarotid Sympathectomy in Certain Intracranial Conditions

Case 1

A 35-year-old man was sent to me in 1941 on account of a painful paralysis of the brachial plexus. Because of considerable dysarthria he had great difficulty in telling me how he was wounded by a bullet which entered through the left side of his neck and came out below the scapular region. His arm was paralysed immediately. Picked up by the Germans, he discovered that he could no longer talk. He had complete aphasia, no hemiplegia, but very unpleasant tingling sensations in the right leg. He was repatriated on account of his wound, and when I saw him I noted the following three points:

1. Complete paralysis of the deltoid, supraspinatus, infraspinatus, biceps, triceps, pronator, and supinator muscles. The movements of the hand were normal. I supposed that there must have been an injury of the upper branches of the brachial plexus. The patient complained of acute pains in the arm.

2. A considerable degree of dysarthria; his voice was smothered, non-bitonal, and there was a slight hemiplegia on the right side.

*Being a lecture delivered at the Institute of Neurology, London, on July 3, 1951.

3. In the neck region a long scar extended over the site of the carotid junction, suggesting a carotid lesion. The most careful palpation could detect no carotid beat. It was my impression that the aphasia must have been caused by sudden ischaemia of the brain. I therefore examined the other side and found a slight but unmistakable hemiplegia with hyperaesthesia of the abdominal wall and a definite Babinski sign.

The hospital neurologist concurred in the diagnosis of definite signs of a pyramidal lesion. Examination by the laryngologist showed a paralysis of the hemilarynx with hemianaesthesia, but the difficulty of speech was ascribed to a central lesion. I therefore decided to perform a double exploratory operation: one to verify the condition of the left carotid, and another to explore the left brachial plexus.

Nineteen months after the initial injury I found the common carotid in a mass of scar tissue. In its lower part, and up to the bifurcation, the artery was like a cord filled throughout with scar tissue. Lower down, the artery had the shape of a spindle ending at the site of the obliteration, which began about 2 in. (5 cm.) from the clavicle, and a blood stream seemed to circulate from the external to the internal carotid. The vagus nerve was oedematous, and in the middle of it was a cicatricial neuroma. I injected procaine hydrochloride and resected the whole of the obliterated carotid segment. Histological examination showed an organized thrombosis.

In the evening some improvement of speech could be observed as well as a retrocession of the pain in the neck and shoulder. From this time the patient's condition continued to improve; speech became easier, and the signs of hemiplegia decreased. I shall make no mention of the laryngeal condition, of the second operation on the brachial plexus, or of the arthrodesis of the shoulder which was rendered necessary by paralysis of the deltoid.

When the patient was seen several years later his speech was almost normal and he had regained an important part of his movements. He could carry out his agricultural activities almost normally, and was married.

This case shows clearly the influence of resection of the thrombosed carotid on an old focus of cerebral ischaemia. Nothing is changed in the circulatory mechanism after the arteriectomy, and yet a great proportion of the symptoms attributable to a central lesion have decreased in a short time. This can be explained only by modifications of the vasomotor conditions comparable to those observed after performing an arteriectomy on a limb. It was my impression that the carotid had not been cut by the bullet, but had only been bruised. The endothelium had probably been torn, as I have observed on several occasions in arteries of the limbs. A spasm had certainly occurred at once, since the aphasia was immediately present. Then thrombosis set in. The circulation was re-established by a reversal of the blood stream into the external carotid, so that the ischaemia was of short duration. A zone of ischaemic necrosis resulted—at the margin of which perifocal circulatory disturbances manifested themselves—and disappeared after the arteriectomy.

Case 2

A priest was wounded on May 25, 1940. While crawling to the front lines he suddenly felt a shock, tried to stand up, and fell unconscious. He remained several days without being able to talk or move, and his hospital notes contain the following observations: "Right hemiplegia with aphasia, amnesia, visual disturbances, and meningeal reaction." When he became conscious again he could only utter some inarticulate sounds. During the next few weeks there was some improvement, and after two months he began to read and was able to make some movements. But he could not keep his attention on anything, and most of the time he could not understand what he was reading or what he was being told.

I saw him six months later, in January, 1941. About 2 in. (5 cm.) from the sternum, inside the carotid line, I found the entrance scar of the bullet, which had gone out at the back, near C6. Carotid pulsation was present. There was evidence of a regressing right hemiplegia. The patient walked with a circular swing. He could only move his hand slowly and with difficulty. The reflexes were exaggerated, and a facial weakness was present. There was also a left Claude Bernard syndrome and a lesion of the lower branches of the brachial plexus with atrophy of the thenar and hypothenar muscles. There was anaesthesia of the C8-T1 territory. He had stuttering dysarthria. Ideation was slow, with lapses. The patient could not make conversation and could not follow a lecture. He forgot things, and was unable to say mass through lack of memory.

In view of the presence of carotid pulsation I felt that the ischaemia must be due to a spasm. I decided to try an infiltration of the left stellate ganglion. The result was extraordinary; in a few minutes speech improved, and ideation became easier. The rhythm of the conversation was entirely changed, and this improvement persisted until the next day.

During the next few days 11 injections were made in the stellate ganglion, all with favourable results, so that the patient was soon able to say mass again. In view of this, on January 17 I decided to operate. Under local analgesia the carotid was exposed; this artery was not obliterated, and pulsations were present above and below the scar area. At this point, however, it was immobilized by sclerosis of the tunica adventitia, which was removed entirely up to the bifurcation. The same evening the patient could speak much more easily and stated that he could think almost normally. After a few more days I noted that the improvement of both speech and ideation was maintained. Moreover, there was a definite improvement of hand movements, especially of the index and middle fingers, which could be moved independently; this had been impossible. Walking was unchanged. After 10 days the patient went home delighted with the results obtained.

Eight years later he wrote me: "I take part in discussions and in all conversations. My speech is still improving. Reading remains 50% slower than it was. My right leg is almost normal. I can ride a bicycle 30 to 40 kilometres without fatigue. My memory is quite fair, though not completely restored."

This case is very instructive. The results of the first injection in the stellate ganglion were really dramatic. In less than one minute speech was transformed, and so was ideation. All this can be accounted for only by vasomotor modification of the brain arteries, and indicates that functional disturbances were added to the original ischaemic lesion. The pericarotid sympathectomy had the same permanent results. I cannot find any other explanation than that of an arrested activity of some of the brain vasoconstrictors.

It appears, therefore, that perverted circulation in the neighbourhood of a zone of ischaemic necrosis of the brain can be modified by working on some organ outside the brain.

Local Vasomotor Autonomy of the Cerebral Arteries

To the above I wish to add some clinical observations which seem to indicate that there exists a sort of autonomy of the vasomotor vessels of the brain, although this is not a recognized fact in physiology. Pathological physiology will sometimes reveal possibilities of reactions which experimentation cannot demonstrate. At a time when the methods of haemostasis had not yet been perfected, it was possible to observe in the course of some surgical procedure, or when clipping a vessel, a sudden local vasodilatation evidenced by congestion and an immediate oedema which would hide everything from view. Similarly, in cases of trauma of the brain, small haemorrhagic zones may be observed at a distance from the site of the traumatic lesion, and

sometimes even in the brain stem or in the bulb, which can only be due to diapedetic haemorrhage of vasomotor origin.

This shows that vasomotor reactions in the brain are not always of extracerebral origin and that they may be the consequence of a local injury.

Localized or Clinical Spasms

Observation on patients suffering from disturbance of the cerebral circulation, especially those who probably have had a stroke, reveals some facts which can be interpreted only as resulting from a local spasm of the brain arteries.

First, one will occasionally find in patients suffering from high blood pressure a disappearance of the transient manifestations following a cerebral vascular accident, which cannot be accounted for by anything but excessive vasoconstriction. All of a sudden aphasia will be present, sometimes with paraesthesia of an arm or leg; then everything disappears without leaving any trace, and this for a long period. At times such manifestations will precede a stroke by several months, or even several years. In one case in which the symptoms had lasted several hours a stellate-ganglion block cleared the aphasia in a few seconds.

There are cases, too, in which coma occurs only after hours of slight paralysis. Recently I saw a 42-year-old physician who woke up one morning with a left facial paresis. He attended to his business the whole morning, had lunch as usual, carried out his customary activities in the afternoon, and went out in his car; but towards the end of the day he experienced some difficulty in driving. After he got home, paralysis progressively invaded his arm, then his leg, and finally he became comatose. The coma lasted until the next morning. He recovered from it, but the hemiplegia persisted.

These facts led me to think that in cases of accidents to the brain vessels there always is added to the thrombosis, or to the embolism, a vasomotor reaction which enlarges the field of ischaemic disturbances producing coma.

Attempts at Section of the Cervical Ramifications in Hemiplegia

On the basis of this hypothesis, in 1925 I performed a section of the communicating cervical ramifications on a patient suffering from hemiplegia. There was considerable improvement. Royle had already performed an identical operation, but his object had been to relieve the contraction, whereas my own objective was to modify the circulation. I have always expounded the principle that surgery of the sympathetic system is above all concerned with vasoconstriction.

Subsequently I made three other such operations with appreciable results. In 1936, in a case of Wallenberg (Babinski-Nageotte) syndrome with hemiparesis and hemianaesthesia, the presence of grave angina pectoris attacks induced me to perform a stellectomy. The heart attacks disappeared and there was at the same time considerable improvement of the hemiparesis, ataxia, and lateropulsion. The improvement still persisted after a year.

Attempts at Treatment of "Strokes" by Stellate-ganglion Block

In 1932 I had obtained remarkable results by performing a stellate-ganglion block in a case of hemiplegia due to birth traumatism in a young boy. This fact, added to previous observations, gave Fontaine and myself the idea of trying to treat strokes due to embolism by infiltration of the stellate ganglion. We had an occasion to do this on May 5, 1936.

On that date I was called at 8 a.m. to see an obese woman who, a week before, had undergone hysterectomy for a voluminous fibroma and who had suddenly become comatose. She did not respond to any form of stimulation. Her

breathing was very irregular. Death appeared imminent. An injection was immediately made in the stellate ganglion (approximately an hour after the onset of the symptoms) on the left side, as the right limb seemed more inert than the left one. After five minutes breathing became more even and the pulse slower. Half an hour later the coma subsided and the patient responded to stimulation by groaning. In the afternoon her condition was completely changed: her breathing was no longer stertorous, and the pulse was regular. Hemiplegia still persisted, but the aphasia seemed to be regressing. The next day another injection was given, as a result of which the patient was able to speak slowly but very distinctly, without stuttering. She was also able to move her fingers very slightly. Two more injections were made in the stellate ganglion during the following days, and after each one the motility was improved. After a week speech was clear. The limbs were gradually regaining their movements, although these were still slow and spastic, and there was a persistent Babinski sign. In spite of this the hemiplegia was obviously regressing.

At the beginning of June the patient got out of bed; she dragged her leg in walking. The movements of her right arm were clumsy and slow, and she could not raise her arms above the level of the shoulders. Improvement continued during the next few weeks, and the movements of the fingers became easier.

On August 3 she was sent home with only faint traces of her hemiplegia and a perfectly normal cerebral function.

Undoubtedly the stellate-ganglion block saved this patient and promoted favourable progress. We made the mistake, both Fontaine and I, of not continuing the infiltrations, and perhaps, too, of not having given her two a day. This would probably have shortened her convalescence.

A few months later we were presented with a new case of cerebral embolism in a 56-year-old woman following removal of a uterine polyp. On the third day after the operation there appeared a slight facial paralysis with aphasia and right hemiplegia. Respiration and pulse were normal. An injection was immediately made in the stellate ganglion; half an hour later the patient could, with difficulty, pronounce a few words—she could slightly move her fingers. In the afternoon the improvement became more pronounced: speech, though still difficult, was intelligible. The movements were slow and feeble, but nevertheless present. The next day another infiltration was made, bringing about further improvement. The movements were easier and speech was improving. The next day, except for a slight difficulty in talking and some weakness of the movements on the right side, no trace of the accident to the brain was left. It would have been impossible to know that three days previously the patient had complete hemiplegia. Her reflexes had become normal again—and the recovery lasted.

These two observations were published in the *Revue de Chirurgie* in December, 1936. They did not make much impression in spite of the fact that we had suggested applying this method in cerebral vascular accidents and pointed out that it could occasionally be of great help in cases of hemiplegia of old standing, could improve speech in cases of aphasia, and could reduce the spasticity.

In 1938 Mackey and Lawrence Scott reported in the *British Medical Journal* (vol. 2, p. 1) a group of 19 cases of apoplexy treated by stellate-ganglion block. Nine of the patients showed improvement. Most of the failures were in cases of severe cerebral haemorrhage in old people; it should be noted, however, that Claude Bernard's syndrome was not always present, as it should be if the anaesthetic really reaches the stellate ganglion.

In 1948 Geza de Takats and Gibert tried this method on 25 patients who had had a stroke a few hours before. In 19 cases the results were favourable.

In August, 1950, Naffziger and Adams published in the *Archives of Surgery* (vol. 61, p. 286) a report covering 225 cases, for which a total of 700 stellate-ganglion blocks were

made; 155 of these patients had acute cerebral vascular accidents—thrombosis, embolism, or vasospasm—and 70 suffered from acute neurological deficiency due to trauma or brain surgery. The results were classified as good when recovery was complete, fair when there was only partial recovery, and poor when there was no immediate or subsequent improvement.

In the first group, comprising 155 cases, the result was good in 91 (59%), fair in 37 (24%), and poor in 27 (17%). In the second group, comprising 70 cases, the result was good in 30 (43%), fair in 30 (43%), and poor in 10 (14%). The authors' conclusion is that the stellate-ganglion block should have a definite part in the treatment of cerebral vascular disturbances.

My own experience is less extensive than Naffziger's. The following, however, is the procedure I should advise in such cases:

Make the infiltration as soon as possible on the opposite side to that of the paralysis. Begin with 10 ml. of 1% procaine. Make another injection of 20 ml. six hours later; then continue with one infiltration a day for fifteen days. There seems to be some advantage in occasionally giving the injection on the side of the paralysis and even in associating a lumbar infiltration with the stellate-ganglion block.

I might add that some physicians have observed a modification of the electrocardiographic curve following stellate-ganglion block; moreover, as I reported long ago, it also modifies the spinal fluid pressure.

Recently, A. de Souza Pereira, in view of the favourable results obtained by stellate-ganglion block 50 hours after the occurrence of an embolism, resected the middle-ganglion and the intermediate and upper part of the stellate ganglion, thereby obtaining a marked improvement in the patient's condition. Encouraged by this result, de Souza Pereira treated a number of hemiplegias of more or less long standing by sympathetic block followed by sympathectomy. He sometimes obtained progressive improvement—dramatic in some cases, slight in others—some patients regaining useful movements, while others were scarcely benefited. In order to clarify these facts further, de Souza Pereira experimented on dogs.

After tying the two internal carotid branches, he performed an arteriography, then a sympathectomy, and finally followed this by another arteriography. It was easy to observe on the arteriogram how the circulatory track was enlarged by the operation.

He then used the same method of arteriography to study the effects of ganglionectomy on man. The interruption in the sympathetic chain caused an increase in the blood circulation. Lubsen had already, in 1941, come to the same conclusion, but it was his impression that unilateral ganglionectomy, while increasing the blood flow on the operated side, diminishes it on the opposite side. It therefore appears from this that the intervention should be bilateral.

At the present stage of research, however, I do not believe that a sympathectomy should be performed too soon—I mean immediately after the stroke—on the human being. The stellate-ganglion block seems sufficient to give the desired results.

For the time being I would perform a sympathectomy only on patients who, after one or several cerebral episodes, live in terror of the future—perhaps with good reason. I have operated on this indication eight times during the past fifteen years. Because of the war I lost track of six of my patients. Here is one case report, however, which will make it possible to judge the efficacy of the method.

A man aged 49 had had mild diabetes for a long time. On November 6, 1948, while talking, he suddenly stuttered and could not finish his sentence. After a few seconds his malaise disappeared and he was able to talk again, but he had the impression that his left side was all cold, numb, and senseless. His movements were awkward, slow, and difficult. His blood pressure was 230/90 mm. Hg. From

that time onwards the patient could walk only with difficulty, dragging his leg; the movements of his arm and leg were slow and incomplete, and the whole left side felt numb. He was forced to reduce his business activities considerably. He had difficulty in expressing himself and often had fits of dizziness.

He came to see me at the beginning of June, 1949. I noticed some difficulty of speech and the remnants of a left hemiplegia. Both arm and leg had lost considerable strength; there was hypoaesthesia of the left side, but not total anaesthesia. A feeling of constriction above the left knee annoyed him very much.

His blood urea was 28 mg. per 100 ml.; blood sugar 108 mg. per 100 ml., blood pressure 170/90 mm. Hg. An arteriography showed normal arteries.

On June 30, under local analgesia, I performed a section of the roots of the vertebral nerve on both sides. As usual the operation was followed by intense cephalalgia, which disappeared after a few days.

Twenty months later the patient came to see me for an arteritis of the leg. He stated that a few weeks after the operation he was able to resume his professional and family life, having regained his faculty of speech. He now spoke at a normal speed, without hesitation, and finished all his sentences. It would have been impossible to guess that he had a past history of disturbances of speech. Both arm and leg had regained normal strength; all movements were easy, though there persisted a feeling of numbness and the feeling of constriction above the knee was still present. Everything else was normal. His blood pressure was 170/90 mm. Hg.

Another patient, operated on in similar clinical circumstances a few months ago, is very much satisfied with the results obtained so far.

In all these cases I did not do a ganglionectomy but merely a section of the vertebral nerve roots at the upper pole of the stellate ganglion, as this suppresses most of the constrictors of the basilar artery.* The future will tell what the true value of this operation is. It must be remembered, however, that this operation, which should always be performed bilaterally, in either one or two stages, is followed for a few days by intense cephalalgia, probably due to meningeal congestion. This operation should perhaps be resorted to as a complement to the classical surgical treatment of patients having high blood pressure with cerebral manifestations, and also, perhaps, of those who have severe visual disturbances.

Stellate-ganglion Block in Old Hemiplegias

In cases of long-standing hemiplegia we must for the present be content with the results obtained from repeated stellate-ganglion blocks.

Since 1935 I have tried the stellate-ganglion block in a great variety of cases of hemiplegia. I began with those in which the patients complained of pain, and have generally succeeded in suppressing or at least attenuating the pain. Then I extended this form of treatment to patients whose condition was comparatively recent. Since 1945 I have treated over 100 patients by repeated infiltrations (from 12 to 36). It is impossible for me to give statistics and percentages, owing to the fact that the cases treated varied considerably in severity, extension, and duration. The general impression, however, is that the more recent the case the greater the chance of success; yet it is possible to obtain favourable results after several years. At all stages of the disease a number of failures must be expected: the result can be either insignificant or negative.

In some patients changes will indicate a functional share in the establishment of symptoms. For example, a 45-year-old woman stricken with aphasia was able to say a few

*A report on this operation was published in *Arch. ital. Chir.* 1938, 52, 659.

words within the hour following the stellate-ganglion block ; then she gradually sank back into mutism.

Approximately one-third of the patients have a feeling of relief, are less contracted, and are more supple ; and many walk very much better after a week. This improvement may be temporary, and sometimes the former condition will re-establish itself when the infiltrations are stopped. Or the improvement may be permanent, especially if the patient is determined to collaborate.

An example of this is the case of a 42-year-old physician who had had hemiplegia for 18 months, with a blood pressure of 200/100 mm. Hg, and had walked with difficulty, using a stick ; the arm movements were incoördinate. After 39 stellate-ganglion blocks on the opposite side to that of the paralysis he can walk without his stick, and is now planning to resume some of his activities.

In another case, a man who could not hold a pencil in his contracted hand could, after 16 infiltrations, carry for fifteen minutes a basket filled with fruit.

Possibly these patients who can still be improved by delayed stellate-ganglion blocks could be benefited by sympathectomy. In my opinion they represent 25% of the cases I have treated, but I must say that for reasons of humanity I have made an attempt in some very old cases—of four to five years' duration. Let us hope that in the future such patients will be treated at an earlier stage. We must think of the despair of these unfortunate people abandoned without hope to their terrible fate.

Pierre Stricker, a neurosurgeon from Mulhouse, after performing a stellate-ganglion block for a two-year hemiplegia and seeing the patient resume his occupation, has since 1942 treated several cases of hemiplegia by repeated infiltrations of the upper cervical ganglion, and has just recently published some case reports which fully confirm those recorded above.

A recent attempt has been made to treat hemiplegia by carotico-jugular anastomosis. This operation does not seem to increase the number of arteries visible on the angiogram : the blood is mostly diverted into the veins, which become dilated, especially in the bulbar region.

According to the results of our experiments, the simplest and most efficient method seems to consist in giving emergency stellate-ganglion block treatment in cases of embolism and thrombosis, and, for patients who have missed this early chance, to resort to treatment which may vary from infiltration to resection of the thrombosed carotid and the various methods of sympathectomy—section of the vertebral nerve roots or gangliectomies.

Conclusion

These are the facts and comments I wish to submit. I do not think that I have unduly emphasized the extent of the results obtained.

If it is true, as I have read, that there are each year in the United States 500,000 cases of apoplectic strokes and hemiplegia, I feel that a method which makes it possible to diminish the importance of these accidents deserves some consideration. There is, moreover, another side to the question—the tragedy brought into the life of those who survive the stroke by the persistence of hemiplegia and particularly aphasia. A famous American magazine some months ago very clearly described the facts which I have just stated, and gave the principles and results of the stellate-ganglion block. Since then I have received 200 letters, from different countries of the world, expressing the despair of those whom we call, without conscious irony, "cured apoplectics."

Anyone reading such distressing messages cannot fail to wish for the development of a method which would considerably minimize the gravity of the accident.

FURTHER OBSERVATIONS UPON THE DIAGNOSIS OF DEAFNESS IN YOUNG CHILDREN

WITH PARTICULAR REFERENCE TO THE MAKING OF RECOMMENDATIONS FOR SPECIAL EDUCATIONAL TREATMENT

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Since our first description of the peep-show technique for pure-tone audiometry in children under the age of 6 years (Dix and Hallpike, 1947), the equipment has been in regular use at weekly clinics at the National Hospital, Queen Square. In the course of this work certain developments have taken place.

In the first place, the mechanism of the equipment has been greatly improved and reconstructed in a simple and readily reproducible form. Secondly, we have had the opportunity during the examination of the considerable number of subjects involved—350 in all—of becoming familiar with certain technical and psychological difficulties which may arise in the course of the testing procedure, and have been able to formulate effective means of recognizing and overcoming these difficulties.

Furthermore, in the course of these investigations we have been asked to make recommendations, upon the basis of our findings, for the special educational treatment (S.E.T.) of the children in accordance with the well-known classification of deaf children proposed by the Board of Education (1938) in its special report. Here certain interesting difficulties have been encountered. Finally, we have been able in a number of cases to carry out a follow-up with a view to assessing the validity of our educational recommendations.

The publication of the present communication has, therefore, the following aims : (1) to give a detailed account of the mechanical improvements in the apparatus ; (2) to describe the psychological and other difficulties which may arise in the course of the test procedure and the methods of overcoming them ; (3) to describe and discuss our clinical material and test results ; (4) to describe and discuss the problems arising in connexion with the making of recommendations for S.E.T. ; and (5) to give the results of our follow-up.

Apparatus—1. The Child's Equipment

The general lines of the tests and of the peep-show equipment are best illustrated by two diagrams reproduced from our original paper (Figs. 1 and 2). The test procedure depends upon the conditioned response of the subject to a series of short pure-tone stimuli delivered from a loud-speaker. These pure-tone stimuli are synchronized with the