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Sinography: A Method of Radiography in the Diagnosis of Sinus Thrombosis

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SINOGRAPHY is a method of investigation by means of which, with the aid of X-rays and of X-ray-opaque substance injected into the dural sinuses of the skull, an obstruction in the circulation of the blood in the said sinuses or the jugular vein can be demonstrated.

Three years have passed since I made my first experiments with sinography, published in *Acto-Oto-Laryngologica*, xx, 1934. Since then, owing to the force of circumstance and certain local conditions, I have not had the opportunity of continuing these investigations until the last half year.

I have already stated that this method is not intended for use in all cases of sinus thrombosis, but should be reserved for those in whom the picture of the symptoms in general is not clear, necessitating, therefore, further support for the diagnosis and subsequent treatment. It should be used, for instance, in cases of bilateral otitis, in which one is not sure in which of the sigmoid sinuses a fairly positive sinus thrombosis is situated. It may also be used in cases of pyæmic and septicæmic conditions with high fever, in which one is unable to determine as to whether a sinus thrombosis or some other disease is the cause of the fever, and in cases in which a thrombosis is highly probable clinically, but is not encountered at operation, because it was localized in the bulb of the jugular vein or in some other part of the sinus, difficult to reach.

Many may think that the diagnosis of an otogenous sinus thrombosis is so simple that we are not in need of any methods of investigation other than those we already have. And this is indeed the case when one is confronted with the classical picture of an otitis, complicated with acute septicæmic conditions, manifesting remittent temperature, chills and metastases, but there

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are many cases in which these typical symptoms are absent or at least are so indefinitely expressed as to be insufficient for a positive diagnosis. Especially as regards children, one not seldom meets with a noteworthy digression from the usual typical course, with perhaps a temperature which rises by degrees and stays at a moderate height.

An otitis described as silent mastoiditis, or another form of mastoiditis unaccompanied by chills or other positive symptoms, may occur. In such cases, it is specially difficult to determine whether the fever and effect of the general condition are caused by a sinus thrombosis or by affected glands. Amongst adults even the course can vary considerably and be particularly divergent from the typical, and the difficulty in obtaining a correct diagnosis is augmented owing to the simultaneous occurrence of some other fever-promoting disease. The second case affords an excellent example of such a case.

It is possible in bilateral otitis to diagnose sinus thrombosis and yet not be able to determine on which side it is situated. I should like to say a few words concerning the possibilities of making a decision in such a case. There are times when, in a case of bilateral otitis, one has arrived at certain conclusions, supported by the course of the disease. The side on which the otitis first developed, or on which it is seen to be making greater progress, may be considered as the more suspicious. This is also the case in mastoiditis, when on one side tenderness is found over the glands in the neighbourhood, or when the X-ray picture shows greater changes. But these factors are very unreliable in reality. Griesinger's test obviously renders excellent aid in cases in which the outcome is positive. Crowe's test, on the other hand, is unreliable and difficult to estimate. The most discussed test of later years is undoubtedly the Tobey-Ayer, which, in its way, is considered to be fairly reliable. As I have said previously (1934), we in Sweden were unable to share this opinion. As a matter of fact, in numerous cases of lumbar punctures the rise in pressure of the spinal fluid frequently occurred to the same height on the healthy side as on the other, where we had a positive sinus thrombosis, and we also obtained a negative result in several cases in which a true thrombosis was found, or in which the jugular vein was previously tied. This occurred in cases in which only one examination was made, as well as when repeated punctures were performed.

We must confess that we still lack a fully reliable method of diagnosing and localizing a sinus thrombosis, and that, owing to this, we are obliged now and again to perform big operations, with no definite diagnosis, on patients in a poor general condition. It is only after a mastoidectomy has been carried out, perhaps first on one side and then on the other, that a correct diagnosis can be

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obtained. For this reason I would emphasize the justification for continuing to seek new methods of investigation, which would be a mainstay in diagnosing these conditions, when correct treatment at the psychological moment is of great importance.

With the aid of sinography I had hoped to be able to obtain a method of investigation which would, if possible, unfailingly demonstrate the presence or otherwise of sinus thrombosis. We know that the longitudinal sinus empties itself through the lateral sinus and the jugular vein on either side and, should there be no anatomical anomalies, one might assume that the emptying process runs equally on both sides. Should an absolute obstruction exist, or a less tangible obstacle prevent the passage on one of the sides, the flow would turn either wholly or mostly in the opposite direction. It should be possible to demonstrate this with the aid of Roentgenology by injecting a Roentgen-opaque substance into the longitudinal sinus and photographing the area during the injection, at the moment when the sinuses are adequately filled.

My first sinography test was carried out as follows: With the aid of a coarse drill (Marell's automatic trepan) I trephined an opening more than 1 cm. in diameter towards the longitudinal sinus at a suitable point, inserted a cannula and made exposures in various planes, whilst the injection was in progress. It was a successful experiment, but with the disadvantage that trephining the skull with such an instrument proved to be an unnecessarily big operation. The method has therefore been simplified and one now proceeds in the following manner: The patient lies with his forehead resting on the table (Lysholm skull table, Fig. 1). Under local anæsthesia, an incision about 2 cm. long is made in the midline between the parietal protuberances and about 5 cm. above the external occipital protuberance. After the tendinous aponeurosis has been removed with a raspatory and the interparietal suture is made visible, the soft tissues are kept apart with a small instrument, leaving a small passage to the bone. A small and specially made drill which may be attached to any handle (Fig. 2), conical in shape with a sharp point, is now inserted in the suture. One knows exactly when the bone is trephined from the movements of the drill and from slight bleeding from the pierced longitudinal sinus. The trephining accomplished, a cannula (Fig. 2) is inserted into the opening. The distal conical end of this cannula corresponds exactly in size to the drill and fits precisely into the aperture. In the cannula an injection needle, which can be moved through a few millimetres in and out, is pushed in 1 or 2 mm., whereupon the point lies in the middle lumen of the sinus and a fair amount of bleeding takes place. The needle is held in place by means of rubber packing, and the injection may begin.

During the exposure the X-ray tube was adjusted so that the

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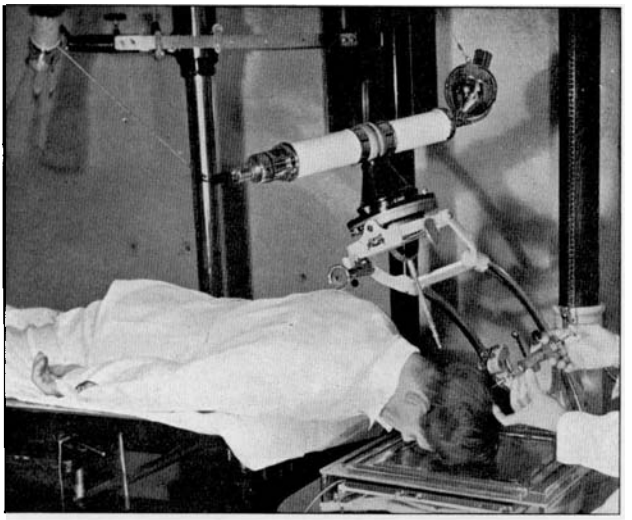


Fig. 1.

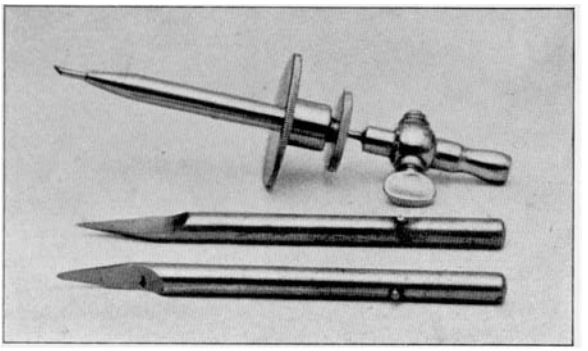


Fig. 2.

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posterior cranial fossa was projected above the middle and anterior fossa, that is, an angle adjustment about 30° caudally from the eye-ear plane. The X-ray-opaque substance used was the ordinary 35 per cent. solution of perabrodil in the same concentration as is used in intravenous pyelography. In 2 to 3 seconds 10 c.cm. of this solution was injected, and during the last second of injection the exposure was made. To be more exact, the time of exposure was 0·7 second. In order to obtain a satisfactory result of an investigation it is necessary that the exposure should be made just at the right moment, and this demands excellent team work and a preparatory collaborative training in Roentgenology. If the operation is carried out in the above manner, and in a well-arranged operating theatre, the whole procedure need not occupy more than about ten minutes, with no more difficulty for either patient or operator than in an ordinary lumbar puncture.

My earlier tests, already mentioned, mainly comprised a number of experiments on monkeys. Here, after the mastoidectomy, an artificial thrombus was placed in the sigmoid sinus. The sinography showed in these cases (Figs. 3 and 4) that the contrast material on the thrombosed side only moved forward somewhat in the transverse sinus, whilst the outflow went on through the lateral sinus on the other side. As it was of interest to know whether the blood on the thrombosed side circulated up to the thrombosis, or whether the stream in the blood ceased to move somewhere on the way, I made the following experiments: A sinography was carried out without touching either sinus or vein engaged in the outflow. This done, the jugular vein on one side was tied and a new sinography was made. After this the sinus was tamponed as near to the jugular bulb as possible, and another sinography followed. Further tamponings were made at the upper knee of the sinus and at various parts of the transverse sinus, and after each there was a sinography. The results of these tests are reproduced in Figs. 5 to 9. In Fig. 5 both jugular veins are seen to be filled. In Fig. 6 one sees that the opaque material has reached the ligature of the jugular vein. In Fig. 7 where the thrombosis is seen against the jugular bulb, the opaque material has not filled the sigmoid sinus but stops at the upper knee of the sinus and continues towards the zygomatic region through a blood-vessel running in this direction. An anatomical investigation made here, shows in monkeys a relatively wide vessel, the capsulo-parietal vein, which from the upper sinus knee runs forward in the dura and empties itself through superficial vessels. This blood-vessel may be found in certain other mammals, but not in man. In the other figures (8, 9), where the thrombosis is seen to be placed near the upper sinus knee or somewhere behind, one finds that the contrast material goes right up to the obstruction.

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With the exception of the blood-vessel at the upper knee of the sinus, the vessels and sinuses of this species of monkey (*Macacus rhesus*) as regards both size and topographical situation are very like those of the human being. In consideration of this, should one venture to assume that the circulation of the blood within these areas is the same in man and monkey, one is in a position to assert that if an obstruction arose somewhere between the torcular Herophili and the jugular vein, the blood would continue its circulation to the place of the obstacle. Thus it is seen that no stationary unit of blood exists between the torcular Herophili and the posterior end of the thrombus, but on the contrary one finds a continuous circulation of blood which is of enormous practical importance. This movement prevents additional formation of thrombosis behind the anterior end, but considerably favours the ejection of thrombotic material from the posterior end into the systemic circuit.

Obviously I do not mean to assert an exact analogy between man and monkey, seeing that the blood-vessel in question in man is at least ten times greater in width than that in the monkey, but I am putting forward a likely hypothesis which can be proved or set aside by forthcoming practical experience.

It is too early to define the value of sinography as a method of investigation in man, when up to the present I have had the opportunity of using it only in a few cases, but amongst these were two so instructive that I would like to give a detailed account of them.

CASE I.—A woman, aged 28. No previous trouble with ear or throat. On 19.10.36 had sore throat, high remittent fever and chills. Glands swollen on the outside. Remained in bed a few days. Admitted to hospital twelve days later; was there ten days with a temperature beginning at 39° C., followed by remittent temperature 38·5° C. to 41·5° C. On arrival at the hospital no ear trouble was found. The disease was believed to be a septicæmia, originating probably from the throat, with some suspicion of jugular thrombosis. A consulting otologist was called in some days after patient's arrival. The right drum membrane had not reddened but manifested a certain change of colour, which might possibly give suspicion of a transudate. During the first few days the patient had a certain dull pain in the whole right side of the neck and head, which was thought to originate from the lymph-nodes on the neck. Four days later (7.11.36) the otologist was unable to note any changes on the drum membrane but performed a paracentesis, as he had previously suspected a transudate. The drum membrane was found to be normal, the mucous membrane in the middle ear was pale, and there was no discharge. The patient still lay in her septicæmic condition, the cause unsolved, and three days later,

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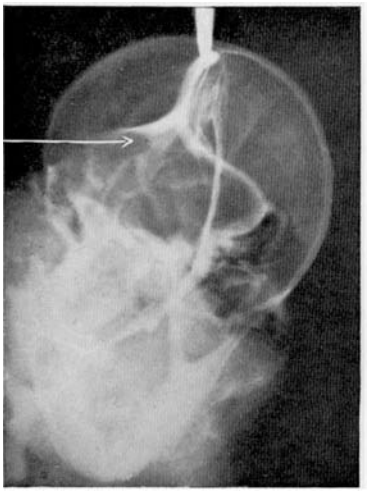


Fig. 3.



Fig. 4.

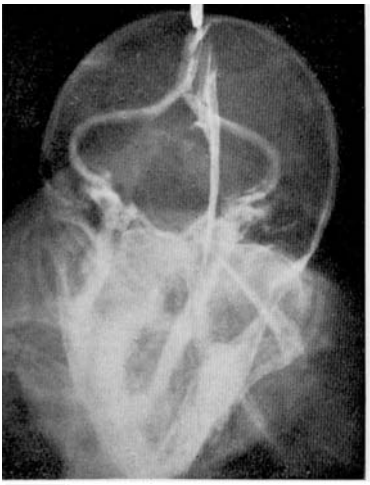


Fig. 5.

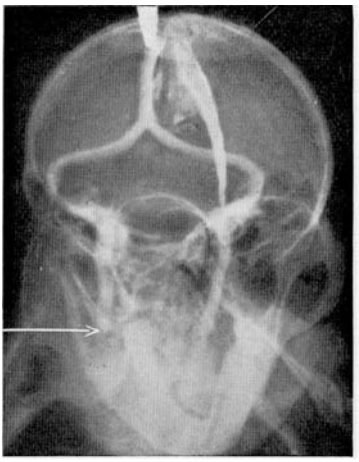


Fig. 6.

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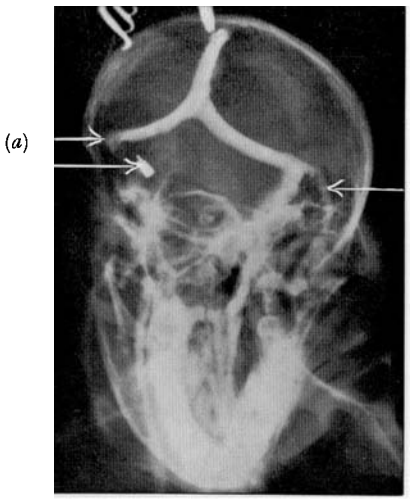


FIG. 7.
(a) Vena capsulo-parietalis.

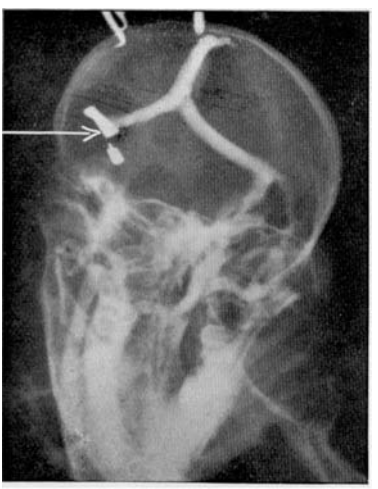


FIG. 8.

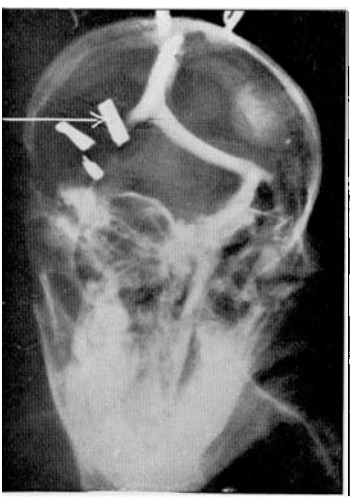


FIG. 9.

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10.11.36, was taken to the ear department of the Sabbatsberg Hospital with suspected thrombosis of the right jugular vein.

An open perforation with moist edges was seen on the drum membrane. A very slight radial injection was also visible. Whispers could be heard at a distance of one metre. No sign of otitis, no tenderness over the mastoid process. The pharynx was pale. By the right angle of the mandible and along the sterno-mastoid muscle there was a group of swollen, tender glands. The pain reached as far as the mastoid process. No tenderness over the jugular vein below the glands. X-rays showed a slight shadow over the whole cell system on the right mastoid process.

It seemed impossible to get any exact diagnosis or explanation of the septic condition, but one was unable to rid oneself of the suspicion of a thrombosis in the sinus or jugular vein on the right side. This led to a sinography and the surprising picture obtained is reproduced in Fig. 10.

The longitudinal sinus, the left lateral sinus and the left jugular vein were completely filled. On the right side there was only a plug approximately 1 cm. long, pushing its way into the posterior part of the transverse sinus from the torcular Herophili. The picture disclosed an extensive thrombosis of the right lateral sinus. On immediate operation the mastoid process was found to be full of serous and seropurulent matter, a swollen mucous membrane but no evident changes in the bone. The lamina interna covering the sigmoid sinus appeared intact. Towards the jugodigastric region there was found, within a very small area, a considerable decalcification of the cell walls and an osteitic portion lying close to the wall of the sinus. The sinus was thrombosed and filled with a suppurating grey-green substance. It was opened, and the thrombosis was traced backwards until fluid blood was encountered about 1·5 cm. from the confluence of the sinuses. A tamponing was done instantly. The thrombosis was then followed forwards in the usual manner as far as one could get towards the jugular bulb. The jugular vein, which was found in a state of collapse, was tied. The recovery was ideal, the temperature falling by degrees without chills or high temperature remissions.

This case deserves to be considered as having diagnostically succeeded as far as sinography goes, and one is almost persuaded to conclude that this result must be absolutely reliable. The fact that mistakes may occur must be carefully reckoned with, as may be seen from the following parallel case.

Case II.—A man, aged 41. Previously operated on for chronic otitis on both sides. Was taken ill 30.9.36 with dizziness, vomiting, indisposition, and a temperature approximately 40° C. On arrival at the hospital a subperiosteal abscess was found under the operation

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scar on the right side. No sign of labyrinthitis. Slight meningeal irritation, otherwise nothing definite intracranially. On operating immediately there was found an extensive osteitic nest in the jugo-digastric region and towards the jugular bulb. A perisinus abscess lay ensconced here and had broken down the inner side of the mastoid process towards the muscles. No sign of sinus thrombosis. Some days later the patient grew worse, had high fever and at a lumbar puncture a meningitis presented itself with 500 leucocytes per cubic millimetre. Sinus thrombosis was suspected and a sinography was carried out. The picture shown in Fig. 11 was obtained. According to this the longitudinal sinus, the lateral sinus, as well as the jugular vein on the one side, was completely filled, whilst there was no sign of the transverse sinus and its continuation on the other. The diagnosis was considered quite clear and an immediate sinus operation was performed. To one's surprise, however, the sole change found was that the sigmoid sinus had a somewhat thickened wall. There was no sign of thrombosis or obstacle to the circulation of the blood. On comparing the Roentgen plate it was seen that the sinus of the diseased side was filled, whilst the healthy side was quite obturated. The explanation was made clear some days later at the *post-mortem* examination. The torcular Herophili presented an anatomical anomaly; the longitudinal sinus went direct into the right transverse sinus, which, like the sigmoid sinus, was singularly wide. In the sigmoid sinus was a small non-obturating thrombosis; otherwise there were no pathological changes. The left transverse sinus, on the other hand, was connected with the longitudinal sinus solely by a thread-like passage, into which only with difficulty could the finest probe be passed. The lumina of the left transverse and sigmoid sinuses were considerably smaller than were those on the right.

In comparing these two cases, the first impression might be that anatomical anomalies of this or a similar kind could derange the entire result of the investigation, and sinography give rise to diagnostic conclusions which might be extremely dangerous instead of helpful. Such is indeed the case, so long as one is ignorant as to the manner in which such mistakes should be detected and faulty conclusions thereby forestalled. The anatomical variations in the construction of the sinus system chiefly concern the torcular Herophili. One can therefore presume that changes in the contrast shadow, caused by the appearance of the sinus lumen, should be apparent right from the outflow from the torcular Herophili, and a mistake of the above nature ought therefore never to occur, save only when a sinus thrombosis is located exceptionally far back. But even in such relatively rare cases

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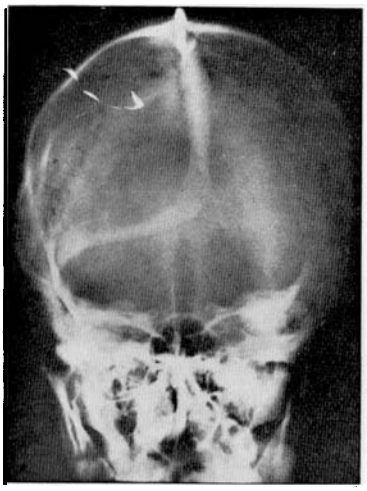


Fig. 10.

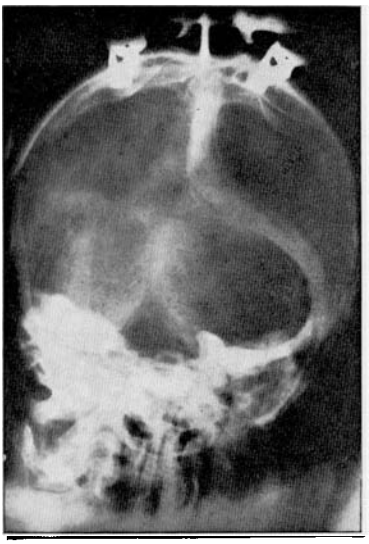


Fig. 11.

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one should, with a good measure of certainty, be able to distinguish between a thrombosis and an anatomical anomaly.

In X-ray investigations of the skulls of both these cases, special attention being given to the state of the grooves of the sinuses and of the jugular foramen, we find that in Case I, with the sinus thrombosis, the sinus groove from the longitudinal sinus divides itself Y-shaped and equably to the grooves of the transverse sinuses, which are equally large on both sides. The jugular foramina are also similar in size on both sides. On the other hand, in Case II, with a congenital defect of the transverse sinus of the one side, one finds the sinus groove of the healthy side considerably widened, whilst on the diseased side it appears only as a narrow streak, and the jugular foramen is on this side considerably less than on the healthy one. Had this been quite clear in Case II, when issuing judgment, one would naturally have been more careful in one's diagnostic conclusions. In order to judge the practical importance of the sinus anomaly in this connection, further investigation of *post-mortem* material is being made. The X-ray picture of the sinus grooves and jugular foramen will be compared partly with the X-ray appearance of the sinus filled *post-mortem* with an X-ray-opaque substance and partly with the gross anatomical appearance of the sinus.

In a direct skiagram of the skull the dural sinuses can be studied only very incompletely, and in some parts very inadequately, by means of corresponding grooves. Recently this was discussed by Lindblom in a Roentgenographic study of the vascular channels of the skull. As pointed out by Lindblom, the breadth of the sinus grooves cannot give any true idea of the size of the sinuses themselves. Only the exit, the lateral portion of the jugular foramen, may have this attribute in relation to the jugular vein.

Even if one is able in most cases to judge as to whether a thrombosis or an anatomical anomaly is the cause of an obstruction in the sinus, there remain in all probability a number of cases in which such a determination is impossible. One can assume, for instance, a thrombosis in a lateral sinus which is defective as it was in Case II.

Misleading anomalies signify a shortcoming in sinography, but this flaw is also incident to other methods, for instance in the Tobey-Ayer test, in which, obviously, a congenital defect has the same effect as a thrombosis. The danger of being misled is considerably greater in the Tobey-Ayer test than in this procedure, as one is not able to determine the type of the obstacle as one can by sinography.

It should be of great significance in some cases to be able to show the defect of the transverse sinus on one side and the subsequent flow of all the blood which comes from the longitudinal

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sinus. If such a condition is verified, one should not, as a matter of course, either tie the jugular vein or tampon off the sinus on that side which has a normally wide sinus, if the diagnosis of an obturating thrombosis is not absolutely indisputable.

Should the X-ray examination and sinography of the sinus show that only on one side is the sinus filled, and that an anomaly is suspected, some still further certainty can in all probability be obtained by compressing the jugular vein on the free side and making a new sinography. If the other side still remains unfilled, the surmise of a complete obstruction is pretty sure to be correct. On the other hand if there is a certain stricture with impaired circulation, a more or less contrast-filled sinus should manifest itself on this side also.

I will here mention a third case, which is of no little interest. The sinography, though not completed, gave a certain very plausible diagnosis.

CASE III.—A girl, aged 17. Radical mastoid operation for chronic otitis in the beginning of *May*, 1936. The recovery was normal, but suppuration from the ear remained. A month and a half after the operation the patient was taken to a hospital, owing to headache and diplopia. Two weeks later double-sided choked discs were observed. A week later when removed to the ear department she still had some headache, but the diplopia had practically disappeared. No paresis of the eye-muscles ; no clinical or radiological signs of apicitis. Apart from the still slightly suppurating ear only high-graded choked discs and a lumbar pressure increased to 350 mm. could be testified. There were no clinical symptoms of abscess of the brain. Ventriculography showed normal conditions. Sinography gave the following result. When the trepanning was over, and the drill had penetrated the skull, there was no bleeding. The point of the cannula when withdrawn was tipped with blood. The cannula was again inserted, and an attempt was made to aspirate blood by means of syringe, but to no purpose. The X-ray plate showed the point of the cannula in the middle of the lumen of the longitudinal sinus, and when the instrument was removed the point again showed blood. Thrombosis was now suspected and in order to determine if it was present an arterio-phlebography was made with injection of X-ray-opaque substance into the internal carotid artery. The phlebogram presented a normal picture as far as the fore-part of the brain was concerned, with a number of small vessels running towards the longitudinal sinus within the brain. Within the further part, however, from approximately the parietal tuberosities and backwards, there was no such normal circulation to be seen. As the subjective symptoms vanished there was no fever and nothing

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significant occurred; the patient was just allowed to lie still. Four months later she was both subjectively and objectively free from symptoms, but the high-grade choked discs remain. The case is diagnosed as creeping thrombosis in the longitudinal sinus where, due to the protraction, collateral veins developed, sufficient in number to prevent a fatal issue.

In cases in which sinography has hitherto been practised, no trouble has arisen either as regards the operation itself or in consequence of the fluid used for injection. Nowadays perabrodil is used as contrast material. This substance has been well tested in arteriography and phlebography in other fields. However, until one has had sufficient practical experience, sinography must be used only in cases in which, owing to lack of other clinical symptoms, additional contributions are needed to complete the clinical picture. Circumstances of that kind have been mentioned above. If one finds that harmful effects are practically excluded and that the method shows the possibility of gaining important clinical enlightenment, it should be applicable, without reservation, in any case of suspected thrombosis whatever.

For the solution of the Roentgenological questions in this problem I am greatly indebted to Dr. E. Lysholm and Dr. E. Lindblom for their valuable advice and suggestions, and for their never-failing interest and help in the practical work.

Discussion.

Dr. Graham Hodgson said he thought that nowadays there was a tendency, which was not very good for radiology, to stretch radiology rather too far. There were definite parts of the body where the radiologist could afford good and very valuable help, but radiology never was, and he hoped never would be, intended to take the place of ordinary clinical methods.

With regard to the danger of the particular method of examination which had just been described, it was scarcely for him, as a radiologist, to speak, but in assessing the value of the method one had to take into account the possible dangers, with which members of the Section were better acquainted than he was. The patient was a sick person, and was having his superior longitudinal sinus punctured and a foreign substance introduced into his blood.

The paper had pointed out one possible source of error, namely a developmental abnormality in the dural sinuses. If there was complete blockage of a sinus, no doubt in a normally-developed sinus it could be clearly seen in the skiagram; but what was expected to be shown in the case of a mural clot which was not completely blocking the sinus? Filling defects in the intestine were sometimes difficult to see unless one looked at the wall from all angles. If there was a mural clot on the anterior or the posterior wall of the sinus and one was taking a lateral view, one would not see it when it was

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filled with opaque material, for those surfaces would be hidden. There might be a mural clot so situated that nothing would be shown on the skiagram. Yet one might make such an examination and rely on it, but it would be misleading, allowing one to think there was no lateral sinus thrombosis. In carrying out these methods of X-ray diagnosis there was always the danger of being misled by a negative finding.

The method shown was, however, very interesting from the academic point of view, and in some cases it might be of great use, but he thought it would put too much strain—an unwarranted strain—on the radiologist, to ask him for a negative—or sometimes even for a positive—diagnosis by this method. In the case of mural clots it would be impossible by this means to say whether or not there was lateral sinus thrombosis.

Mr. H. V. FORSTER said that he had read, some time ago, a preliminary paper by Dr. Frenckner on this subject, and he clearly remembered that the writer had called attention to the unreliability of the Tobey-Ayer test in suspected lateral sinus thrombosis.

When faced with the difficult and anxious problem of generalized otogenic sepsis, he (Mr. Forster) had found comfort in the conservative views of the late Holger Mygind, and also of S. H. Mygind, of Copenhagen, who, in his work at the Commune Hospital on these cases, though dealing very thoroughly with the primary focus of disease about the middle-ear cleft, had hesitated to open the lateral sinus, unless it was thought to contain pus. The results had compared very favourably with those of other Continental clinics. He (Mr. Forster) appreciated, however, that there would be difficult cases in which otologists might endeavour to emulate the courage and skill of Dr. Frenckner. The physician-neurologist, after all, had learnt to explore the cranium and to co-operate with the radiologist in such a delicate subject as ventriculography.

The PRESIDENT said that the subject which Dr. Frenckner had brought forward was an important one, and his contribution was of real value. The evidence was not only experimental but also clinical. As Dr. Graham Hodgson had well said, the difficulty arose in determining the degree of obstruction, as the value of the method depended on that. If complete obstruction was not present, the method was naturally of less value.

He wondered in what proportion of cases of "closed" thrombosis, spontaneous cures arose. A number of cases had been reported in which the thrombus had become organized and the sinus was thus sealed.

He was interested in Mr. Forster's remarks on the conservative attitude; he (the President) had left the sinus alone in several cases of doubt, and had been agreeably surprised at the favourable results.

Dr. FRENCKNER (in reply) said he would emphasize the fact that at present the method was mostly experimental. He had had so few cases that he could say little about the dangers.

It was difficult to say whether one would eventually be able to see a mural thrombosis; exposures had been tried in different directions

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to see whether there was any indication of sinus wall. Further experience might show this to be possible. If the X-ray picture was negative, it was impossible to say whether or not there was mural thrombosis.

CASES

Three Cases of Mastoid Asymmetry.

—E. WATSON-WILLIAMS.

I.—R.M.S., female, aged 49, has been subject all her life to attacks of pain and discharge from the right ear. X-rays show complete absence of cell development on right side, well-developed cells on left. Radical mastoid operation 12.9.36: Mastoid cortex thick, concave, process acellular. Ear quite healed 26.10.36. No cholesteatoma found at operation. Does the failure of cell development indicate infection in early infancy?

II.—V.L.R., male, aged 26, has suffered from deafness and discharge in the right ear during two years (possibly longer). X-rays show complete absence of cell development on right side, normal development on left.

Operation: Radical mastoid (right). Thick cortex; acellular process; granulations in aditus; cholesteatoma in attic, almost destroying incus. Careful inquiry has failed to obtain any suggestion of aural disease before 1934. Does the cellular asymmetry indicate infantile infection?

Ménière's Syndrome with Mastoid Cellular Asymmetry.

III.—W.T.A., male, aged 54, a sanitary inspector, had sudden giddiness in *July*, 1935, in the evening, with violent sickness and deafness in the right ear; he was giddy for a week. Since then the ear has been deaf (not completely) and there has been slight puffing tinnitus. He has since had several such attacks, and now gets them about once a week, chiefly at night in bed; the sickness has not been prominent in recent attacks, but he never has an appetite for breakfast ("breakfast sign"). He is always a little unsteady in walking, and becomes giddy if he turns the head to the right; he tends to fall to the left. Both tympanic membranes are indrawn and glossy. He is certain that he has never had aural discharge. The right ear shows a severe degree of nerve deafness (Rinne positive). He has always been quite healthy except for this disorder. Wassermann reaction negative. X-ray examination of mastoids was undertaken for evidence of fibroma; there was none, but the right mastoid was seen to be completely acellular, in contrast with the good cell development of the left side. The deafness worries him, as he is an amateur musician. He is having a small dose of quinine to minimize giddiness.

Suggestions for treatment (? ossiculectomy) welcomed. How far is the asymmetry significant?

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Comment on the above three Cases

These are three examples of patients in whom one mastoid is normal in regard to cellular development, and the other shows no cells. A point I raise is as to what is the best treatment to adopt for the patient in the first case, in order to get rid of his vertiginous attacks? If operation is advised, what is the best procedure—ossiculectomy, injection of the labyrinth with alcohol, or division of the VIIIth nerve? I think ossiculectomy is worth trying; there is great loss of hearing by bone conduction on the right side.

With regard to asymmetrical mastoid processes, what is the present feeling as to the origin of the acellular mastoid? In chronic mastoiditis it is almost the rule, if one has to operate, to find that the mastoid is acellular. Yet when one examines ordinary skiagrams of the skulls of patients chosen without reference to ear disease, nearly all of them show normal cell development. It is very unusual to find a cellular mastoid on one side and an acellular one on the other. Is the mastoid acellular because the cells never develop? That is an attractive theory. But if that is so, why do so many of the acellular cases require operative treatment? Did the cells not develop because the mastoid was infected in early infancy? If so, that must have been in the first year of life, because at the end of that time, cell development is well on its way.

Finally, is it possible that cells, having developed, can fill up again? I do not think it is. If one examines a non-cellular mastoid it is not merely a question of the interior being different, but the bone is different in external shape, the wall is deeply grooved for muscular attachment, the floor of the middle fossa dips so much that one sometimes has to burrow upwards in order to get at the antrum, and it is impossible that that change should take place in a bone which has been of the normal convex shape.

But if it is a case of infantile infection, why is it not very common to find acellular mastoids in ordinary people? Infection of the ears is a very common event in infancy. A large proportion of the population—perhaps one in six—have running ears at some time in their early life, and it is difficult to understand the exact degree of infection which is necessary to stop cell development.

Why are so many cases of cellular mastoid processes infected later in life? I find the infection theory very difficult to support; I tend towards the developmental view.

Chronic Labyrinthitis; Fracture of Mastoid Process, involving Facial Canal.—E. WATSON-WILLIAMS.

W.T.W., male, aged 35, has been subject to attacks of giddiness for three years, with sickness and headache. At first they were frequent and severe; during the last two years he has had five attacks, each lasting two or three days. In 1918 he was suffering

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from chronic otitis media, and had a polypus removed from the left ear.

November 2nd, 1936: Admitted to Bristol Royal Infirmary suffering from a worse attack than ever before, which had begun the previous day without warning. Headache was especially severe, with pain in and behind the left ear. He could not even sit up in bed, and thinks he fell down when the attack began (he falls to the right side). He lay on the left side (i.e. bad ear down); any movement brought on vomiting. He had been bleeding severely from the left ear, and was pallid. Temperature 98·6°, pulse 92, respiration 22.

On examination.—Left cheek weak, not completely paralysed; severe frontal headache; mastoid process very tender, without swelling. Mixed nystagmus to right, worse on looking to right; falls over to right on sitting up. Pupils equal, react to light; fundi normal; knee-jerks increased, equal; plantar response normal; no inco-ordination or muscular weakness (except of face), no rigidity of neck. Kernig's sign negative. No dysdiadokokinesia; no past-pointing (but tendency to inward deviation with both hands). Ear full of clot. Apparently complete nerve deafness on left side; Weber to right. Caloric test of vestibule: No response to five minutes' syringing with cold water.

November 4th.—Temperature 98°, pulse 56, respiration 20. Radical mastoid operation. On exposure of the bone a transverse fracture was seen running across the mastoid process from the external meatus. The cortex was dense, the mastoid medulla acellular; cholesteatoma filled the antrum. The line of fracture ran up the facial ridge, apparently along the canal; every tap on the mastoid (in any place) produced a marked twitch of the left side of the face. The facial ridge was therefore not pared down as closely as usual. Malleus and incus completely ankylosed. In view of the clinical findings vestibulotomy was attempted, but the vestibule was found to be completely solid, several attempts failing to find any trace of stapes or to open the vestibule. The symptoms were completely relieved after the operation. No headache, no vertigo; facial weakness rapidly disappeared. Complete nerve deafness in left ear. Suggested pathology; old (healed) labyrinthitis left side, Eustachian catarrhal process on right, sudden vertigo, falling; fracture of left mastoid, hence tenderness, and bleeding from lateral sinus (latter was left alone at operation, as it was desired not to disturb local clot). Subsequent progress satisfactory.

Comment

I brought this case which shows a different condition from that in the previous three, because it was a "catch". I think the

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labyrinthitis, which was diagnosed as "acute" when the patient came in, was an old condition. I think he fell on the mastoid during an attack of giddiness, and that the resulting fracture caused tenderness in that situation. When he was brought in to us he was practically bled white and the bleeding had stopped. The ear was "dead" to all tests. He had nystagmus and weakness of the face, and the deduction seemed straightforward: chronic otitis media, acute suppurative labyrinthitis. But when I found he had a fractured mastoid and an old bone-filled labyrinth, I reflected that this was the sort of thing which caught one out occasionally. This is an example of bony ankylosis of the malleus and incus, a condition to which I have drawn attention on previous occasions; I have not found it of any clinical significance.

Discussion.

Mr. E. A. Peters said that, as the proportion of cellular to acellular mastoids varied as much as showing a 70 per cent. occurrence in some races, it was to be expected that there should be variations with a cellular mastoid on one side and an acellular on the other side. His experience had been that in acellular mastoids the symptoms of pain and giddiness had been more acute than in the cellular ones; probably this was a question of tension. On opening up such a mastoid, relatively little disease was to be seen, while in cellular mastoiditis the symptoms of pain and giddiness were slight, and possibly extensive deep-seated disease was found.

Mr. J. F. O'Malley said he had formed the opinion that when a mastoid condition was related to chronic discharge of the ear, the acellular condition was consequent upon sclerosis, following long-standing low-grade inflammation. That was the conclusion he arrived at after performing many radical mastoid operations. There was a chronic suppurating ear, and one anticipated finding something in the mastoid to account for it, but nothing was found except sclerosed bone. He thought it was a result of chronic infection of the mastoid which showed asymmetry of cellular distribution.

Mr. H. V. Forster said that of a group of children of early or middle school age suffering from chronic middle-ear suppuration, a certain number were found to resist carefully supervised cleansing or ionization treatment. When these resistant cases were submitted to the appropriate type of mastoid operation, he had quite often found a mastoid area showing marked cellular development, the cells being filled with a dark fluid which could be recognized even before the cortex was uncapped. What condition would be found in these cases if the patients survived without operation until later life? Was it possible, in such a temporal bone, for these cells to be obliterated by Nature's healing process?

Mr. F. Brayshaw Gilhespy: A paper by Bunch and Groves from the Otological Research Laboratory of Johns Hopkins University is of interest as it answers Mr. Watson-Williams' question as to whether the acellular condition of the mastoid bone is due to suppuration early

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in life. The authors studied a group of children whose ages ranged from seven to sixteen years, and the onset of otorrhœa from two days to four years—at the age when suppuration might be expected to modify the mastoid development. It is interesting to note the fact that only those children who had myringotomy performed at least three times, on one side or the other, were included in the survey. In looking at the skiagrams, in many cases it would be impossible to say that there had been an attack of otorrhœa at any time.

Essinger in 1928 described seven patients who were operated upon at the age of under one year, with good pneumatization at the age of seven years. However, he offers an ingenious explanation, as this in some ways tends to be against Wittmaack's theory. It was stated that the mucosa was brought back to normal by the mastoid operation, and was able to carry on its normal function of pneumatization. Of course certain bone conditions such as rickets must be taken into account.

I am interested in this subject from a medico-legal point of view. Could the fact of an acellular mastoid be produced in a court of law as evidence of previous ear-trouble in a case of injury causing deafness? If pneumatization was not present the deafness might have been present in some degree before the accident. It would depend on whether the judge was a disciple of Wittmaack or belonged to the English school of thought.

Mr. W. Stirk Adams said that he had not examined the patient in Mr. Watson-Williams' first case, but, having heard the history and seen the skiagram, he thought it highly probable that in spite of a normal drum-head being found on otoscopy, a chronic infection was present in the mastoid, and he suggested that the first treatment should not be a destructive operation on the ear but a cortical mastoid operation, in order to ascertain whether infection was present. If this proved to be the case no further treatment should be necessary, but he suggested that if the mastoid proved not to be infected Mr. Watson-Williams should proceed to a Portmann operation. He had no personal experience of the operation, but two such patients had been operated on by colleagues in Birmingham. In the first case a complete relief of vertigo was obtained for four months after the operation and then the attacks recurred. In the second case, that of an elderly man whose attacks of abdominal disturbance and vertigo had persisted for three years before the operation, complete relief of symptoms had been obtained since the operation up to the present time, a period of over six months. He found, when testing this patient's hearing, that the ear which had previously been his better ear heard a whisper only at 15 inches, while the operated ear, which had previously been the worse ear, now heard a whisper at 15 feet.

He regarded mastoid sclerosis, in the presence of chronic suppuration, as a result of the infection, either primary or secondary. The primary sclerotic mastoid, as met with in surgery, had a well-developed mastoid antrum but no cells were present. When the secondary sclerotic mastoid was opened, the antrum at first appeared to be single, but a careful search in this type revealed minute tracts leading in the

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direction of the mastoid tip, the retro-facial groove, and the petrous angle. No explanation other than a secondary development of sclerosis in a pneumatic mastoid could explain the presence of these tracts leading, as they often did, to well-developed cells in the periphery of the mastoid.

Mr. F. W. WATKYN-THOMAS said he wished to mention one point only in connection with this alleged secondary sclerosis of the mastoid due to infection. Had anyone ever demonstrated the presence of osteoblasts in such mastoids? If there were a proceeding sclerosis, there should be osteoblasts in the mastoid laying down bone. So far as he knew, they had never yet been found.

Dr. J. P. STEWART said that the generally accepted idea of sclerosis of the mastoid bone was that, in infancy, the growing membrane, which hollowed out the marrow-spaces in the bone, in order to produce the air-cells, was damaged by infection—not necessarily a purulent infection—so that it lost its function and the mastoid bone remained as it was—diploic. In addition, this infective process might also transform the marrow-spaces into dense bone, thus giving rise to the sclerotic or dense mastoid bone. In acute cases of mastoiditis in a cellular bone, it was quite possible for the mastoid process to become sclerosed, for under the stimulus of an infective process, new bone-formation occurred in the mastoid air cells, and if this process was continued long enough, what was originally a cellular mastoid bone could be transformed into a dense or sclerosed one.

The PRESIDENT said that Mr. Watson-Williams had brought forward an important perennial problem, which was not yet settled. The interference with cellular development which might take place as a result of mastoiditis must necessarily be produced during the very early years of life. One could not imagine a fully-developed, pneumatized mastoid process becoming sclerosed by an otitis.

Mr. ERIC WATSON-WILLIAMS (in reply) thanked members for the views they had expressed, but could not say that his mind had yet been made clear on the matter he raised. It was difficult for him to accept the view that once a mastoid had become cellular it could become acellular. But if infection was the true story, one would expect, on the law of averages, that unilateral acellular development of the mastoid would be relatively common. For a child almost always to have both ears affected to the same extent, or to have both ears escape infection to the same extent, seemed unlikely. He therefore thought infection was not a convincing explanation of failure of mastoid cell development.

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