1939

Headaches following diagnostic lumbar puncture

Dale H. Davies
University of Nebraska Medical Center

HEADACHES FOLLOWING DIAGNOSTIC LUMBAR PUNCTURE

Senior Thesis Presented to
The College of Medicine,
University of Nebraska

Dale H. Davies

1939

OUTLINE

I. Introduction

II. Symptoms of the Different Types of Postpuncture Headache

III. Mechanism of the Lumbar Puncture Headaches

IV. The Causative or Predisposing Factors

V. Methods of Combating the Headaches

VI. Treatment

VII. Conclusions

I. INTRODUCTION

Corning (1885) was the first to puncture the subarachnoid space of a living person. His punctures were for the purpose of injecting novocain and no fluid was removed. Corning reported only using it in one case and that patient he reported suffered from headache, and slight vertigo. It is not possible to tell whether this was a true lumbar puncture headache or not. Punctures for the removal of fluid were first performed by Quincke, Wynter, and Morton, each in 1891. Although all three of these men carried out their work simultaneously Quincke deserves the most credit. He was the first to show that the subarachnoid space could be punctured with a needle without incising the skin, that the fluid could be removed, and that diagnostic aid could be derived from its study.

Since these men first used the lumbar puncture, its sequelae have awakened much interest and occasional comment. The most frequent of the aftereffects is the postlumbar puncture headache, and it is with this subject that this paper is concerned.

II. SYMPTOMS OF THE DIFFERENT TYPES OF POSTPUNCTURE HEADACHE

Evans (1928-29) has published an elaborate and complete description of the various types of postpuncture headache. He divides the headache into type A, that due to decreased cerebrospinal fluid pressure and type B, that due to an

increased cerebrospinal fluid pressure.

Type A is the most common, and differs from others in that being present when the patient is sitting up, it completely disappears when he lies down. This is characterized by an occipital or parietal headache appearing the first twenty-four hours and gradually increasing in severity. The pain comes quickly when the patient sits up, being present fully in about twenty seconds. It also subsides in about the same time when the patient lies down. The headache may be accompanied by nausea and even violent vomiting, perhaps also with some giddiness, mental confusion, and faintness. No drugs give sufficient relief to allow the patient to sit up or do any form of work. A lumbar puncture during the course of the headache will show a hypotension of the cerebrospinal fluid and the fluid will show a decrease in the cell count and globulin content.

Type B headache is characterized by a stiff neck and photophobia and other signs of meningeal irritation, and although it is made worse by raising the head and exercise, it is not relieved to any extent by lowering the head. This type of headache may be relieved by the use of sedatives. A lumbar puncture during the course of this headache shows an increase in cerebrospinal fluid pressure and an increase in cells and globulin content.

III. MECHANISM OF THE LUMBAR PUNCTURE HEADACHES

In considering the mechanism involved in the production of postpuncture headache due to decreased cerebrospinal fluid pressure, the theory advanced by MacRobert (1918) appears quite feasible, and is concerned with the non-closure of the puncture hole in the dura mater.

The cerebrospinal fluid is contained in a closed sac, and forms a pad for the brain and spinal cord. At the base of the brain this pad becomes a veritable cushion or water bed. When the fluid leaks away through a hole in the dura at the lower end of this sac, the base of the brain loses its supporting cushion. This support then would be expected to be absent until the puncture hole heals and the fluid again fills and distends the sac sufficiently to reestablish this water bed. The loss of this supporting basal cushion may be regarded as the secondary causative factor in the production of the headache, leakage being the primary cause. How does this loss of a cushion produce pain?

A headache, which, let us recall comes on when the patient sits up, and disappears when he lies down, must obviously be mechanically produced. Pressure of the meninges by the brain weight, against the irregular bony surface of the base of the skull, which would ensue when the patient sits up, in the absence of a basal fluid cushion, might be considered to be sufficient cause for headache. However, the following explanation is more pleasing to MacRobert.

"A mechanical accident following spinal puncture has caused sudden death in certain brain tumor and other intracranial conditions accompanied by increased intracranial pressure. The withdrawal of fluid deprived the base of the brain of support, and allowed such forcible descent of the pons on the clivus of the occipital bone that prolapse of the medulla through the foramen magnum occurred. In the average normal case, if the supporting fluid cushion is lost by continuous leakage through a patent puncture hole, we may expect the pressure of the brain weight transmitted through the pons to the clivus when the patient sits up to be considerable.

The basilar plexus on the clivus of the occipital bone is formed by an extensive anastomosis of flat venous plexuses. It is connected on either side with the cavernous and inferior petrosal sinuses, and with the neighboring blood channels. The other venous channels that drain the cranial cavity at the base are rigid inelastic tubes and are thus safe from closure by pressure. The basilar veins differ in this matter of severity of closure. They depend on the cushion of cerebrospinal fluid to keep off the pressure of the pons, which is directly above.

When the patient sits up, and the cushion of the fluid is absent, the weight of a good part of the brain is suddenly imparted through the pons to this communicating plexus. The

blood about to leave the skull is impeded and forced to turn back and travel by other crowded pathways. The resulting congestion causes a sudden rise of venous pressure.

The sudden onset of severe headache when the patient sits up can now be understood as due to the sudden heightened intracranial pressure due to rise of pressure in the cerebral veins; its entire relief when the patient lies down, as due to the fall of pressure when the weight is removed from the veins on the clivus. In the course of a week the puncture hole heals, the fluid is rapidly made in sufficient quantities to fill and distend the entire sac, and the integrity of the brain cushion or water-bed is reestablished. The headache, which was purely a mechanical affair dependent on the loss of that cushion, is gone."

Another theory has been advanced as to the mechanism of the production of the headache of type A. This was propounded by Dana (1917) and Zappala (1934) as being due to an inhibition of the secretory power of the choroid plexus. Zappala reports a study of one hundred cases presenting cephalalgia, in which he found a marked hypotension of the cerebrospinal fluid in the majority of them.

The headache due to an increase in cerebrospinal fluid pressure has been conceded by Stillwell (1932), Pitken (1929) and others as being due to meningeal irritation, from poor technique in the performance of the spinal puncture.

Kennedy (1932) offers as a suggestion for the mechanism of production of this headache the following: "The leakage of cerebrospinal fluid from the puncture is greatest during the first 21-24 hours. This leakage stimulates the choroid plexus to pass into the cerebrospinal fluid spaces an increased amount of fluid. This addition of fluid does not cease immediately after the puncture has become sealed, and thus the condition of increased intracranial tension arises."

IV. THE CAUSATIVE OR PREDISPOSING FACTORS

In a consideration of the causative factors of spinal puncture headache it is found, as is usual, where the exact factor or factors is not known, numerous theories to attempt to explain the phenomenon. Following is an enumeration of the various factors which will subsequently be discussed more in detail.

1. Age, sex, and temperament.
2. The disease condition of the cerebrospinal fluid.
3. The condition of the spinal fluid pressure.
4. The rapidity with which the fluid is withdrawn.
5. The amount of fluid withdrawn.
6. The presence or absence of minute amounts of calcium in the cerebrospinal fluid.
7. The effect of blood oozing into the spinal canal.
8. The position of the patient during puncture.
9. The length of time that should be spent flat on the

back after puncture.

10. Leakage of cerebrospinal fluid into the epidural space.

11. The relation of negative pressure in the epidural space to postpuncture headaches.

12. Meningeal irritation; Aseptic Meningitis.

1. Dana (1917) reported that lumbar puncture headache occurred more frequently and severely in young adults, in women, and in people of a nervous temperament. Dana had headaches in 50% of his cases and thought they were more frequent in the above mentioned types. Heldt (1929) and Nelson (1930) made observations on their series with regard to this factor and both concluded that this factor was not important.

2. Dana (1917) also mentioned that the lumbar puncture headaches were more common in patients whose spinal fluid was negative. MacRobert (1918) reported an incidence of 37.5% of headaches in those patients with a positive fluid, and an incidence of 40% in those with a negative fluid. This he interpreted as not being a significant difference. Baar (1920) substantiated Dana's findings and reported an incidence of 26% in patients with a positive fluid, and an incidence of 66% in patients with a negative fluid. Nelson (1930) also reported an incidence of about one-half (10%) in patients with a positive fluid as in those with a negative fluid. The concensus of opinion seems to uphold the fact that post-

lumbar puncture headaches occur more frequently in those patients whose spinal fluid is negative.

3. The condition of the spinal fluid pressure has been suspected by many men as a causative factor in the production of the headache. Dana thought that the headache was more commonly found in patients whose fluid came out under low pressure. Baar (1920) from his observations concluded that the degree of intraspinal pressure had no effect whatsoever on the production of the headache. Frazier (1918) was convinced that rapid pressure change was important and advised manometric control of withdrawal of the fluid. Alpers (1925) in trying to puzzle out the answer to the cause of the headaches also decided that it was concerned with the pressure. He observed the pressure before and after withdrawal of fluid and found that his headaches were more frequent in those patients who exhibited a marked fall in pressure after withdrawal of the fluid. Nelson (1930) made observations with regard to this factor and came to the conclusion that it was of no importance. At the present time it is not considered as being of primary importance.

4. The rapidity with which the fluid is withdrawn, that is, whether it be permitted to drop from the needle according to the spinal fluid pressure or whether it be gently aspirated has also been considered as a possible causative factor. Baar (1920) investigated this by

controlling the flow with a water manometer, and came to the conclusion that it had no bearing on the production of the headache. Greene (1923) used aspiration to obtain fluid when he used a small needle and even though Webster (1913) states that, "No aspiration should be used at any time, as this procedure is extremely dangerous", Greene found no disadvantages and claimed that there was an advantage in that the fluid could be rapidly reinjected in the face of untwoward symptoms.

Heldt (1929) also dismisses the rapidity with which the fluid is withdrawn as an unimportant factor.

5. The amount of fluid withdrawn, and its relationship to the production of postlumbar puncture headache is interesting. The liability for headache to occur when only two cubic centimeters of fluid is withdrawn as readily as when twenty cubic centimeters is withdrawn has often been demonstrated. This lead many to believe that headache did not depend on the loss of cerebrospinal fluid. To support their contention; they have cited the fact that headache is not a frequent complaint following spinal cord operations in which a great deal of fluid is lost. MacRobert (1918), however, explains this by pointing out that after spinal cord operations the patients are left on their backs for at least two weeks; lumbar puncture headache is not in evidence while a patient is on his back, and the situation responsible for the headache, whatever it is, is never present even in the worst

cases for as long as two weeks. Another fact which also tends to disprove this assumption is that the choroid plexus probably secretes or dialyzes sixty cubic centimeters per day, and it is hard to believe that an alteration in the intracranial situation sufficient to produce violent headache of seven or more days can be made by the removal of from two to five cubic centimeters of fluid.

6. Neustaeder, Hala, and Tolstoouchow (1925) read a paper by Depisch and RichterQuittner in which it was stated that the presence or absence of minute amounts of calcium in the cerebrospinal fluid was the cause of headache. They also reported that the administration of calcium relieved the headache. The above authors and Critchley and O'Flynn (1924) investigated this question and report that while the calcium concentration is slightly lower than normal in those patients suffering from postpuncture headaches, it is not significant in any sense as the cause of the headache. They also found that the administration of calcium did not relieve the headache.

7. Baar mentioned that blood oozing into the spinal canal might have an effect on the production of the headache. Out of eighteen cases in which he collected bloody fluid thirteen developed headaches of from two to nineteen days duration. This gave him a percentage of 72 in those cases. Nelson and many others from their observations regard this as unimportant.

8. The position of the patient during puncture has long been considered as a possible causative or predisposing factor. However, in the many observations made it is now considered as unimportant. The position now in use throughout the world is like that described by Keegan (1937). He advocates a comfortable prone position with the patient on the side near the edge of the bed. The patient's head and knees are approximated to bow the back out and separate the lumbar spines. A board under the mattress will bring the hips up when the springs sag, or the foot of the bed may be elevated upon pegs or a chair. The head should be at the same level as the spinal needle in order to obtain a reliable spinal pressure reading.

9. Lying flat on the back for 24 hours after puncture has also been considered as a possible factor since lumbar puncture headaches have been subjected to thought and study.

Baar (1920) performed punctures on ambulatory patients; that is they were ordered to lie flat on their backs when they reached home. His incidence was not higher than that of Dana (1917), who had forty-seven headaches out of ninety-four cases or an incidence of 50% in hospital patients.

MacRobert (1918) had a series of thirty patients lie flat on their backs for 24 hours after puncture and twelve of the thirty or 40% developed headaches which was no less than his ambulant cases.

Traub (1922) urged the abandonment of puncture in ambulatory patients.

Greene (1923-26) and Bleumel (1924) performed punctures in ambulatory patients using small needles. Greene's incidence in two hundred and fifty-two patients was a minus 4% and Bleumel's 10% in one series of fifty and 2% in another series of fifty.

Stokes (1926) set up a dictum "After the patient lies down following puncture, he should not sit up again for at least 24 and preferably 48 hours". However, Torbert (1934) made a very comprehensive study as to the advisability of having the patients lie on their back for 24 hours after puncture. He studied two groups. In his outpatient group he had an incidence of 20.1%, while in his hospital group he had an incidence of 22%. Torbert, however, believes that the postpuncture reactions last longer and are somewhat more severe in the ambulant groups than in the hospital groups.

In drawing a conclusion from the above statements it may be said that the incidence is no higher in ambulatory patients than it is in hospital patients, therefore, whether a patient lies on his back 24 hours after puncture or not is not important as a causative factor.

10. The theory that leakage from puncture hole into the epidural space of cerebrospinal fluid has long been considered as a factor and still is supported strongly.

Sicard (1902) was the first to propose this theory. Marie (1913) also believed that this was the primary factor concerned. MacRobert (1918), whether being ignorant of the above authors work or not, was not satisfied with the theories of his day as to the cause of lumbar puncture headache, analyzed the question and brought to light a factor to him not previously considered. This factor occurred to him in answering the question "After the puncture, is everything within the same as it was before, with the exception of an absence of a few cubic centimeters of spinal fluid?"

To obtain fluid by puncture, the needle must pierce two membranes, the dura and the arachnoid. The dura forms a rigid, tough, fibrous sac, just within the vertebral canal. The arachnoid tissue, which is non-vascular and delicate in texture, is full and loose, and it is in close apposition to the dura. The fluid is contained in a space between the arachnoid and pia mater, the latter closely investing the spinal cord.

MacRobert (1918) performed some punctures on cadavers and examination revealed that a puncture in the rigid dural membrane persisted as a clean edged round hole. Since the spinal fluid is always under some pressure in its sac, MacRobert thought that there could be continuous leakage into the epidural space of the spinal canal following the extraction of the needle.

MacRobert studied the point and became convinced that closure of the puncture hole takes place in the following manner: "The arachnoid tissue, as it drops from the point of the departing needle, is swept snugly against the dura mater, by the pressure of the fluid within. In this way the dural hole is blocked by an intact area of the arachnoid, as the puncture holes, being small are unlikely to approximate. See Fig. I.

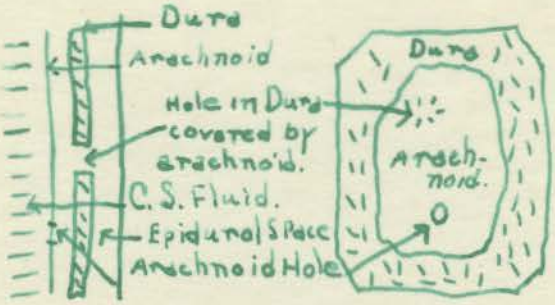

Fig. I: Spinal membranes with normal closure of puncture hole: no epidural leakage; no headache.

If the puncture hole is not blocked, it is because the delicate arachnoid tissue clings around the departing needle, and its hole is pulled into and through the hole in the dura. There it impinges and this invagination forms a spout or wick for the easy drainage of the whole cerebro-spinal fluid sac, and also prevents the rapid healing, which

would otherwise occur, of so small a dural opening." See Fig. II.

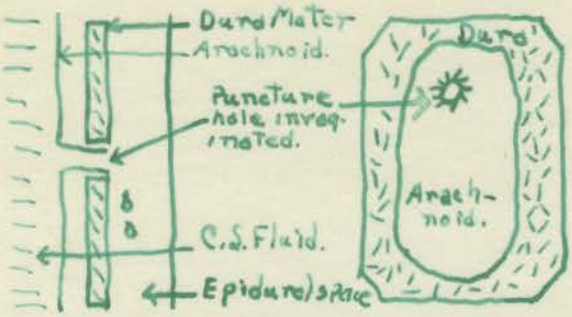

Fig. II: Nonclosure of puncture hole, because of arachnoid tissue being pulled through dural opening as needle was withdrawn, resulting in prolonged epidural leakage and lumbar puncture headache.

From the above it may be seen that all the fluid secreted by the choroidal glands during seven or eight days, the time seemingly necessary for the hole to close by tissue growth, will be lost by leakage into the epidural space where it can be absorbed readily, because the epidural space of the spinal canal is comparatively very large, and contains only loose connective tissue, with rich venous plexuses and lymph channels.

It is evident, therefore, that the amount of fluid collected in the test tube will be no indication of the great loss that occurs, when a puncture hole does not become properly occluded when the needle is withdrawn.

Calmann (1923) reported a case of typical lumbar puncture

headache after five unsuccessful attempts at lumbar puncture. He contended, therefore, that the headache was caused by meningeal irritation and not through leakage of cerebrospinal fluid. MacRobert (1918), however, had an experience which could easily explain the above case. He was interrupted while doing a lumbar puncture by the nervousness and impending syncope of the patient. He was obliged to quickly withdraw the needle, which he believed from his sense of touch to have already pierced the membranes, without collecting or even seeing fluid. Nevertheless the patient developed a typical lumbar puncture headache which lasted with great severity for eight days. It is obvious that an apparently dry tap, if the membranes are pierced, may as truly become oneof epidural leakage as any other puncture.

There has been much work done in an attempt to prove the theory of MacRobert that fluid escapes through a hole in the dura after puncture. Baruch (1920) was the first to work on this problem. He performed puncture and without drawing off any fluid injected three cubic centimeters of a two percent indigo-carmin solution into the subarachnoid space. He then plugged his needle with mandrin so that no fluid escaped. Next he inserted a permanent catheter into the patient, in order to determine immediately the appearance of the dye in the urine. In this experiment, with the lumbar puncture needle in situ, no dye appeared in the urine after

sixty-three minutes. On withdrawal of the needle, however, dye appeared in the urine in eight minutes. Baruch interpreted this as meaning that as long as the hole in the dura was blocked no fluid escaped and no dye appeared in the urine, but as soon as the hole in the dura became patent, fluid escaped and dye appeared in the urine.

Greene (1923) obtained sections of dura with the cord still attached; he suspended the sections and filled the dural space with water and then punctured the dura with different types of needles and found that the amount of leakage was directly proportional to the size of the needle. He also examined puncture holes microscopically and obtained an idea of the amount of trauma done with different types of needles. As a result Greene believed that the headaches, nausea, vertigo, etc. which followed lumbar puncture were due to leakage of the cerebrospinal fluid through the puncture hole.

Perkel (1925) also believed that lumbar puncture headaches were caused by leakage into the epidural space.

Heldt (1929) set about to prove that leakage into the epidural space did occur. He carried out a number of punctures in which the second puncture was carried out from three hours to five days after the first puncture. At the time of the second puncture he inserted the needle only to the depth of the epidural space and from this space he

repeatedly recovered spinal fluid that had leaked into it from the previous puncture. Verification of the fact that the needle was in the epidural space was obtained by manometric changes as influenced by efforts on the part of the patient, or the Queckenstedt maneuvers. Heldt (1929) also inserted the larger cannula of a Hoyt needle into the epidural space and then made a puncture in the same interspace but just above the Hoyt cannula with another 18 or 19 gauge needle. The dura was punctured with this second needle and entrance into the subdural cavity verified by collection of spinal fluid. The second needle was then withdrawn and it was observed that no fluid escaped immediately from the Hoyt cannula in the epidural space. If, however, at this point both jugular veins were compressed, spinal fluid dropped from the previously dry cannula. To do so, the fluid must escape from the puncture hole in the dura into the epidural space and then out the Hoyt cannula.

Nelson (1930), while studying the pressure relations in a large group of spinal puncture patients, decided to take a reading during a typical lumbar puncture headache. This he did on three patients and in each case he found the pressure to be remarkably low during the headache. He explained this as being due to leakage of the cerebrospinal fluid from the puncture hole in the dura.

Nelson also removed a portion of dura mater at autopsy

from a patient who had had a lumbar puncture eleven days previously. He found that the dura varied in thickness and vascularity in different regions, and thought that this variable factor may have a place in the production of leakage.

Kennedy (1932) supported the view that leakage is the essential factor in causing the headache. Also Merritt and Fremont-Smith (1938) believe that this is the primary causative factor.

11. The relation of negative pressure in the epidural space to postpuncture headaches was first expressed by Heldt and Maloney in 1929. These men, while attempting to prevent the loss of the few drops of fluid which occurred before the spinal fluid pressure could be taken, thought of attaching the manometer directly to the puncture needle. They observed that just before the puncture was completed, which would be indicated by a positive pressure that there was a sudden declination of the mercury in the manometer. They then made several punctures with the same technique and confirmed their first observation. They construed this manometric declination as evidence of a negative pressure existing in the cavum epidurale. They then proved this to be the correct location in an experiment upon a cadaver by injecting india ink through the spinal needle after a negative pressure had been encountered. Subsequent dissection revealed the ink in this

cavity.

Heldt and Maloney (1928), and Sheppe (1934) made the following similar observations, but it was up to Heldt and Maloney as stated above to explain them.

1. The appearance of a drop of spinal fluid on the skin following the withdrawal of the needle.
2. In punctures where there was difficulty in entering the dural sac, or if for any reason the stylet was withdrawn from the needle before the dural sac was entered, they often noticed a distinct hissing sound as if there were a sudden inrush of air into the needle.
3. They observed that if the needle is slowly withdrawn following successful puncture that the drop of fluid in the hub of the needle was sometimes aspirated inward. This they thought occurred just after the needle point emerged from the dura.

Heldt and Maloney to study this problem used the following method. "To study this problem, we selected a spinal puncture needle equipped with a stopcock. When such needle is inserted to the depth of the ligamentum flavum, the stylet is withdrawn and the spinal manometer attached. To the stopcock of the needle, we attached, by means of rubber tubing, an ordinary five or ten cubic centimeter Luer syringe loaded with normal saline solution. The needle is then thrust

forward slowly until a negative pressure is recorded by the manometer. The tap of the stopcock is now turned until communication is established between the barrel of the needle and the syringe. Then it may be noted that normal saline is aspirated into the epidural space until the negative pressure disappears. Should the negative pressure be small, -6 mm. of Hg. or less, aspiration does not occur but a little pressure on the plunger of the syringe causes ingress of the saline and consequent equalization of pressure to zero or slightly above." Heldt and Maloney found the negative pressure to vary from -1 to -18 mm. of mercury. Sheppe constructed a set-up similar to the one above and confirmed Heldt and Maloney's findings. As regards the measurement of this negative pressure, two points must be kept in mind (1) if the needle is advanced too far and impinges on the dura without puncturing it, a false negative pressure will be registered as the epidural space is increased by the forward pressure on the dura (2) it is difficult to demonstrate negative pressure in individuals who have had multiple lumbar punctures. It is probable that the degree of negative pressure in the epidural space is affected by (1) the amount of fluid present, i.e., the expansion or contraction of the dural sac; (2) filling and emptying of the epidural veins with change of posture. If the presence of a negative pressure in the epidural may be assumed as seems justified by the observations

of Heldt and Maloney and Sheppe, it is obvious that the opportunity for leakage to occur depends upon the balance of the pressure existing in the subdural and epidural spaces at the needle opening. Negative epidural pressure would tend to aspirate fluid through the dural opening no matter how small this opening may be and such aspiration might be expected to continue until an equalization of pressure was established. So long as such leakage continues, postpuncture headache might be expected from small but steady withdrawal of fluid from the subdural space, also the withdrawal of fluid from the spinal dural sac decreases the space occupied by the spinal membranes, thereby increasing the volume of the epidural space with a resulting increase in negative pressure.

12. Meningeal irritation has been considered by some men as a causative factor of postlumbar puncture headache. This has been explained to be an aseptic meningitis by Stillwell (1932), Pitken (1929) and Heldt (1929). These men believe that this is a definite factor, and is probably due to poor technique in the performance of the puncture.

V. METHODS OF COMBATING THE HEADACHE

Since the exact causative factor of lumbar puncture headache is not known we find here a great variety of methods of combating the headache. The methods reflect their exponents belief as to the causative factor they consider of primary importance.

The following discussion will reveal the various attempts made by different men to successfully reduce the incidence of headache as well as treatment of the headache.

(A) Raising Cerebrospinal Fluid Pressure

Baar (1920) in the course of treating his neurosyphilitic patients made routine spinal taps for prognosis. He noticed that the cases he tapped more frequently made better clinical and laboratory showing, so he concluded that systematic tapping of the canal before injecting the salvarsan might be beneficial to these patients, by quasi drawing the salvarsan from the blood stream to the spinal canal. He also noticed that the patients treated in this way rarely developed post-puncture headaches. This suggested to him to follow every spinal puncture immediately with intravenous salt solution if they were not given salvarsan.

Number of Headaches Without Salt Solution

93 cases; 51 headaches 55%

Non-syphilitics, 66 cases; 44 headaches.66%

Syphilitics , 27 cases; 7 headaches.....26%

Number of Headaches With Salt Solution

50 spinal punctures (17 cases); 3 headaches..... 6%

Nonsyphilitics 7 cases; 1 headache 14%

Syphilitics, 43 punctured(10 cases); 2 headaches.. 5%

Baar gave no explanation as to how the hypotonic salt solution aided in reducing the incidence of the headache.

Solomon (1924) made some experiments and found that one cubic centimeter of pituitary extract intramuscularly or 100-200 cubic centimeters of distilled water intravenously raised the cerebrospinal fluid pressure. The effects of hypotonic solutions in increasing the cerebrospinal fluid pressure has been demonstrated by Weed and McKibben (1919), Weed and Hughson (1921) and others. The effect of pituitary extract on raising the cerebrospinal fluid pressure was studied by Weed (1922) by injecting the drug and noting the outflow from a catheter which had been placed in the aqueduct of Sylvius. Cushing and Weed (1915) demonstrated an increased flow of fluid following the injection of pituitary extract and Halliburton and Dixon (1913) have demonstrated an increased flow after the injection of choroid plexus extract. However, Becht and Gunnar (1921) do not agree with these findings. These investigators, by means of an apparatus arranged so that volume changes could be detected in the spinal fluid, concluded that no increase in the production of fluid occurs with administration of epinephrin, pituitary extract or atropine, but what occurs is a displacement of preformed fluid due to increased venous pressure. Solomon (1924), however, found that in the majority of cases of lumbar puncture headache relief was obtained by the use of either pituitary extrace or distilled water or a combination of the two. In some cases he reported the action of pituitary

extract was very striking and to the patient wonderful. In a few cases the effect was nil. He also observed that in most cases if relief was obtained it was permanent; while in a few cases where the effect was marked and immediate, it lasted from five to ten hours and then wore off, the headache returning. In several cases a second injection or even a third had an effect similar to the first. Similarly with the distilled water injections, the effect was at times quite striking, in other cases less so; and in a couple of cases in which the effect was very satisfactory, it lasted a number of hours and then the headache returned. Alpers (1925) reported using pituitary extract in twelve of sixteen patients who developed the headache. Two obtained no relief and ten permanent relief. He feels that this drug is very good in less severe reactions; while the hypotonic solutions are better in the more severe type with the effect more lasting. Perkel (1925) also thought that intravenous distilled water or intramuscular pituitary extract gave the best results in treatment in headaches with hypotension.

(B) Insertion of Catgut Through Needle

Heldt (1929) working on the principle of preventing leakage of the spinal fluid from the puncture hole hit upon the idea of inserting into the puncture hole a small piece of anhydrated catgut. The catgut is of a diameter slightly less than the bore of the needle and is placed in position by

special modifications and additions to the ordinary lumbar puncture set. The rate of absorption in the anhydrated catgut is such that by its rapidly increased size, it apparently very promptly seals the hole in the dura.

Fifteen punctures with the use of catgut were not followed in a single instance by headache, while two control cases developed severe reactions.

Heldt and Whitehead (1936) studied this procedure further and below is presented a summary of their results.

	Series Without Catgut	Series With Catgut
Total number of patients	110	110
Sex	M 63; F 47	M 67; F 43
Average age	38	37
Number having postlumbar puncture symptoms for more than 12 hours	39 (35%)	59 (54%)
Average duration of symptoms	89 hrs.	45 hrs.

The reaction in the second group was quite different in character from the usual postlumbar puncture difficulty. The most frequent symptom complex was as follows: a dull headache, aching in the lower back and thighs, and slight stiffness of the neck, all relieved to a certain extent by being in the upright position and by activity. This symptomatology presents an interesting contrast to the usual postlumbar puncture syndrome. These patients are not incapacitated, and they are able to be up and about continuously following

puncture. Also fifty-five of the catgut series showed an elevation of temperature of over one degree, as contrasted to only five of the first group. Heldt and Whitehead feel that the whole syndrome, when the catgut was used, was due to a mild meningeal irritation, caused possibly by the influence of the foreign body introduced. They think that these reactions should be termed catgut reactions rather than postlumbar puncture reactions. These men felt that the use of this special technique is worthwhile since it allows the patient to be safely up and about following puncture.

Nelson (1930) working with the same idea in mind, but with a different technique inserted pieces of catgut 3 cm. long into the hole left by the needle in the spinal meninges of one hundred and two patients. At the same time, lumbar puncture was done, for control purposes, one ninety-two patients, in the routine manner, without plugs of catgut. The patients on whom the catgut method had been used were kept flat in bed for twenty hours after puncture. Those on whom puncture had been done in the customary way also were kept in bed, but with the head lowered, for the same length of time. Of the ninety-two patients used as controls, sixteen (17.4%) had the characteristic postpuncture headaches. Of the one hundred and two patients in whom catgut was inserted at lumbar puncture five (4.8%) had reactions that would be interpreted as characteristic postlumbar puncture

headaches. However, approximately half of the one hundred and two complained of aching pains in the back, in the popliteal region and in the posterior muscles of the thighs. These reactions were not nearly so severe and did not last as long as the typical lumbar puncture reaction. Hence they fall into the group of "catgut reactions" as postulated by Heldt and Whitehead. Nelson believes that the use of this technique is therefore advisable and justified.

Merritt and Fremont-Smith (1938), however, do not believe that this procedure should be used because of the possible complications attendant to introduction of a foreign body into the spinal canal.

(C) Use of a Small Needle

Since the theory of leakage of cerebrospinal fluid into the epidural space through the patent puncture hole was brought forth as a causative factor much work has been done to counteract this factor.

Greene (1923-26), Bleumel (1924), Kennedy (1932), Allen (1934), Erskine and Johnson (1938), Cresswell (1936) and others have all worked with the small needle in an attempt to reduce the incidence of headache.

Greene (1923) used a number 23 needle, and he advocates that the point be smooth and round. He performed two hundred and fifty-two punctures with this type of needle, and had an incidence of a minus 4%.

Kennedy (1932) used a variation of the small needle, using a small needle inside a larger one. The large needle was inserted to the depth of the ligamentum flavum from which point the smaller needle was used to pierce the dura and arachnoid.

It is now accepted and advocated that a small needle be used especially in ambulatory patients.

Sheppe (1934) also advocates the use of a small needle and in addition thinks that the incidence of postpuncture headaches may be reduced by slow withdrawal of the needle without the stylet. He does this to allow an inrush of air to neutralize the negative pressure in the epidural space, and thereby aid in preventing leakage.

VI. TREATMENT OF THE DIFFERENT TYPES OF POSTPUNCTURE HEADACHE

The treatment of lumbar puncture headache is not satisfactory at the present time. In some patients a given form of treatment works beautifully, while in others it fails completely.

The treatment of the headache due to an increased intracranial pressure includes many measures. Evans (1928-29) advocates (a) keeping the patient flat in bed with an ice bag to the head; (b) elimination of all excietment and stimulating drinks; (c) repeated lumbar puncture to relieve the spinal fluid pressure; and (d) diuretics, cathartics and hypertonic salt solutions by mouth and intravenously. Perkel (1925) and

Kennedy (1932) also advise hypertonic salt solution intravenously in this type of headache. Koster (1928), and others include retention enemas of six ounces of a 50% solution of magnesium sulfate, repeated every four hours if necessary.

Treatment of the headache due to a low cerebrospinal fluid pressure, is managed by Evans (1928-29) in the mild cases by (a) placing the patient in the Trendelenberg position for twenty-four hours; (b) removing all forms of stimulation and excitement; (c) injecting intramuscularly one ampule of surgical pituitrin or ephedrine hydrochloride. In the more severe cases he advocates (a) hypotonic solution of saline intravenously, about 100 cc. of a 0.5% sodium chloride; (b) forcing fluids, one glass of water every hour by mouth, and if this is impossible to give 1000 cubic centimeters every six hours by the Murphy drip.

Various other authors have attempted to treat the headache due to decreased cerebrospinal fluid tension by the direct restoration of the spinal fluid pressure to normal. Frazier (1918) suggests an isotonic solution directly into the spinal canal. Heldt (1929) interrupted the headache temporily for three to forty-eight hours by injecting 20-30 cc. of distilled water or normal saline into the epidural space.

Zappala (1934) found that intradural injection of a 10% solution of dextrose caused the headache to disappear during the injection and this occurs when the mercurial

manometer shows a pressure of from twenty-one to twenty-three.

VII. CONCLUSIONS

1. The incidence of postlumbar puncture headache lies between 10 and 15%.
2. The exact causative factor of headache following spinal puncture is not known.
3. Leakage of the cerebrospinal fluid into the epidural space through the puncture hole in the dura is probably the primary factor concerned.
4. A small needle not greater than 22 gauge should be used in diagnostic punctures.
5. Diagnostic spinal puncture on ambulatory patients is not contraindicated, because the incidence of discomfort and seriousness of this complication is far outweighed by the diagnostic value of this procedure.

BIBLIOGRAPHY

1. Allen, H. W.: 1934. Headache Following Lumbar Puncture. Brit. M. J. 2:349

2. Alpers, B. J.: 1925. Lumbar Puncture Headache. Archiv. of Neurol. & Psychiat. 14: 806-812.

3. Baar, Gustav: 1920. New Procedure For Prevention Of Spinal Puncture Headache. Med. Rec. 98: 598-599.

4. Baruch, H.: 1920. Zur Ursache unangenehmer Nebenerscheinungen der Lumbar anesthenic ein Beitrag zur Funktionsprufung dis Liquorseptems. Berl. klin. Wehnschr. 38: 298. (From Alpers. 1925)

5. Becht, F. C. & Gunnar, H.: 1921. A Study Of The Volume Changes Of The Cerebrospinal Fluid After Adrenalin, Pituitrin, Pilocarpine And Atropine. Am. J. Physiol. 56: 231-240.

6. Bleumel, C. S.: 1924. Small Needle For Avoiding Lumbar Puncture Headache. J. A. M. A. 82: 209.

7. Calmann, A.: 1923. Headaches After Lumbar Puncture. Deutche Med. Wehnsch. 49: 1058. Ab. J. A. M. A. 81: 1827.

8. Corning, J. L. 1885. Spinal Anesthesia And Local Medication Of The Cord. New York Med. J. 42: 483-485.

9. Cresswell, G. W.: 1936. Cerebrospinal Fluid In Treated Syphilis - 532 Cases - Discussion Of Post-Spinal Headaches. Virg. Med. Month. 63: 503-505.

10. Critchely, M. & O'Flynn: 1924. The Calcium Content Of The Cerebrospinal Fluid. Brain 47: 337-344.

11. Cushing, H. & Weed, L. H.: 1915. The Effect Of Pituitary Extract Upon The Secretion Of Cerebrospinal Fluid. Am. J. Physiol. 36: 77-103.

12. Dana, Charles L.: 1917. Puncture Headache. J. A. M. A. 68: 1017.

13. Erskine, D. & Johnson, A. G.: 1938. Lumbar Puncture In Out-Patients. Lancet. 2: 371-373.

14. Evans, C. H.: 1928. Possible Complications With Spinal Anesthesia. Their Recognition And The Measures Employed

To Combat Them. Am J. Surg. 5: 581-593.

15. Evans, C. H.: Spinal Anesthesia, Principles And Technique. New York. Paul B. Hoeber, Inc. 1929.

16. Frazier, C. H.: Surgery Of The Spine And Spinal Cord. 834-835. New York and London. D. Appleton Co. 1918.

17. Greene, H. M.: 1923. A Technic To Reduce The Incidence Of Headache Following Lumbar Puncture In Ambulatory Patients With A Plea For More Frequent Examination Of The Spinal Fluid. Northwest Med. 22: 240-245.

18. Greene, H. M.: 1926. Lumbar Puncture And The Prevention Of Postpuncture Headache. J. A. M. A. 86 pt. 1: 391-392.

19. Halliburton, W. D. & Dixon, W. E.: 1913. Secretion Of The Cerebrospinal Fluid. J. Physiol. 47: 215-239.

20. Heldt, T. J.: 1929. Lumbar Puncture Headache. Med. J. & Rec. 129: 136-139.

21. Heldt, T. J. & Maloney, J. C.: 1928. Negative Pressure In The Epidural Space. Am. J. Med. Sc. 175: 371-376.

22. Heldt, T. J. & Whitehead, L. S.: 1936. Clinical Studies In Post-Lumbar Puncture Headaches. Am. J. Psychiat. 93: 639-648.

23. Keegan, J. J.: 1937. The Dangers And Technique Of Spinal Puncture. Nebr. M. J. 22: 285-288.

24. Kennedy, A. J.: 1932. Spinal Anesthesia. Clinical Experiences In 430 Cases, And A Technique To Eliminate Post-Operative Headache. M. J. Australia. 1: 40-45.

25. Koster, Harry: 1928. Spinal Anesthesia. Am. J. Surg. 5: 554-570.

26. MacRobert, Russell G.: 1918. The Cause Of Lumbar Puncture Headache. J. A. M. A. 70: 1350-1353.

27. Marie. 1913. Revue Neurol. 26: 712. Cited by Greenfield and Carmicheal, E. A. The Cerebrospinal Fluid In Clinical Diagnosis. pp. 14. London. MacMillan & Co. 1925.

28. Merritt, H. H. & Fremont-Smith, F. The Cerebrospinal Fluid. pp 224-226. Philadelphia & London. W. B. Saunders Co. 1938.

29. Morton, C. A.: 1891. The Pathology Of Tuberculous Meningitis, With Reference To Its Treatment By Tapping The Subarachnoid Space Of The Spinal Cord. Brit. M. J. 2: 840-843.

30. Nelson, M. O.: 1930. Postpuncture Headaches. Archiv. Derm. & Syph. 21: 615-627.

31. Neustaedter, M.; Hala, W. W.; Tolstoouchow, A.: 1925. Relation Of Calcium Content Of The Spinal Fluid To Postlumbar Puncture Headache. J. A. M. A. 85: 347-349.

32. Peluse, Samuel: 1935. Post-Spinal-Anesthetic Headache. A Review Of The Literature And Suggestions For Treatment. Ill. M. J. 67: 372-377.

33. Perkel, J. D.: 1925. Complications Of Lumbar Puncture. Presse Med. Ab. J. A. M. A. 85: 1676.

34. Pitkin, G. P.: 1928. Controllable Spinal Anesthesia. Am. J. Surg. 5: 537-553.

35. Pitkin, G. P.: 1929. Spinocain: The Controllable Spinal Anesthetic. Brit. M. J. 2: 183-187.

36. Quincke. 1891. Verhandlungen der Cong. f. innere Medizin. 10: 331. (From Greenfield and Carmicheal. 1925).

37. Sheppe, W. M.: 1934. The Relation Of Negative Pressure In The Epidural Space To Postpuncture Headache. Am. J. M. Sc. 188: 247-252.

38. Sicard. 1902. Le Liquide Cephalorachidien. (From Allen 1934).

39. Solomon, H. C.: 1924. Raising Cerebrospinal Fluid Pressure With Especial Regard To Effect On Lumbar Puncture Headaches. J. A. M. A. 82: 1512-1515.

40. Stillwell, W. C.: 1932. Lumbar Anesthesia. Resume Of 751 Cases. Minn. Med. 15: 73-79.

41. Stokes, J. H.: Modern Clinical Syphilology. Philadelphia. W. B. Saunders Co. 1928.

42. Torbert, H. C.: 1934. The Safety Of Lumbar Puncture For Ambulatory Patients. Archiv. Derm. & Syph. 30: 692-696.

43. Traub, H. W.: 1922. The Status Of Lumbar Puncture And

The Prevention Of Its Complications. New York M. J. 116: 91-93.

44. Webster, R. W.: Diagnostic Methods. Ed. 3. pp. 646. Philadelphia. P. Blakiston & Sons. 1913.

45. Weed, L. H.: 1922. The Cerebrospinal Fluid. Physiol. Reviews. 2: 171-203.

46. Weed, L. H. & Hughson, W.: 1921. Intracranial Venous Pressure And Cerebrospinal Fluid Pressure As Affected By The Intravenous Injection Of Solutions Of Various Concentrations. Am. J. Physiol. 58: 101-129.

47. Weed, L. H. & McKibben, P. S.: 1919. Pressure Changes In The Cerebrospinal Fluid Following Intravenous Injection Of Solutions Of Various Concentrations. Am. J. Physiol. 48: 512-530.

48. Wynter, Essex W.: 1891. Four Cases Of Tubercular Meningitis In Which Paracentesis Of The Theca Vertebralis Was Performed For Relief Of The Fluid Pressure. Lancet 1: 981-982.

49. Zappala, G.: 1934. Pathogenesis And Treatment Of Cephalalgia Following Spinal Anesthesia. Policlinic Rome. 41: 932-936. (From Peluse 1935).