

1933

History and complications of spinal anesthesia

Stanley Peterson
University of Nebraska Medical Center

This manuscript is historical in nature and may not reflect current medical research and practice. Search [PubMed](#) for current research.

Follow this and additional works at: <https://digitalcommons.unmc.edu/mdtheses>

Recommended Citation

Peterson, Stanley, "History and complications of spinal anesthesia" (1933). *MD Theses*. 282.
<https://digitalcommons.unmc.edu/mdtheses/282>

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

HISTORY AND COMPLICATIONS OF
SPINAL ANESTHESIA

By

Stanley W. Peterson.

UNIVERSITY OF NEBRASKA MEDICAL COLLEGE.

Senior Thesis

1933

FOREWORD

It may be said that anesthesia induced by ether is safe, effective and easy of administration. The margin of safety is wide, and it has rightly come to occupy the premier place in anesthesia. Nitrous oxide anesthesia has a very definite place but its field of usefulness is limited and there are many conditions in which it cannot be satisfactorily employed. The dangers in the administration of ethylene, by reason of its explosive possibilities, are ever present and are sufficiently grave to be a deterrent to its wide application. Furthermore, in ethylene anesthesia the degree of abdominal relaxation that is obtained is many times insufficient and leaves much to be desired. Increasing experience with spinal anesthesia demonstrates its efficacy together with an almost absence of danger. However, there are complications which must be taken into consideration.

In this paper, an attempt is made to discuss some of the complications of spinal anesthesia, with special reference to those observed most frequently as well as those of major importance.

HISTORY

Spinal Anesthesia, sometimes called subarachnoid, medullary, or lumbar anesthesia is insensibility to pain produced by the injecting of an analgesic substance into the arachnoid cavity of the spinal cord.

Like many great discoveries, spinal anesthesia resulted from an accident. J. Leonard Corning of New York City, in 1885 was the first to produce anesthesia by the interspinal method. He did it accidentally, however, while giving a supposed therapeutic extraspinal injection of cocaine (14).

In 1885, Corning (5) published his first report regarding "Spinal Anesthesia and local medication of the cord." Harley had shown that poisons have an effect upon the spinal cord through the instrumentality of the blood vessels. On the strength of this observation, Corning believed that he could cause medicaments to act upon the spinal cord, by injecting them near it. The numerous blood vessels, especially the veins in the neighborhood of the spinal cord, were supposed to absorb the remedies and to carry them to the cord. He did not dare to make an injection upon the spinal cord itself because he believed that this was not possible with-

out removing some vertebral arches. Corning, therefore, injected his remedies between the spinous processes of the lower lumbar vertebrae, because in man the spinous veins have numerous anastomosis with the internal spinal plexes. He believed that the anesthetic reaches the spinal cord by means of these connections and would be capable of inhibiting the sensory and perhaps also the motor conduction, in a similar manner as in the case of a transverse myelitis or a bisection of the cord.

In 1888, Corning published a new report in which he stated his remedy had so far exerted an important influence upon certain diseases of the spinal cord. He treated four cases of spinal irritation by means of cocaine and pyrogallie acid. He describes the technique of his method in detail. This method avoids the spinal cord itself, but brings the medicinal substances into soluble form into its immediate proximity, from where they are carried to the cord through the blood vessels.

So far Corning had anxiously avoided bringing his medicaments into contact with the spinal cord itself. In 1894, however he published a book "Pain", in which the 17th chapter deals with the local medications of the spinal cord. In a sub-section of this chapter, "the irrigation of the cauda equina with medical fluids", Corning clearly expresses

his intention of bringing the medicaments directly into the lumbar sac and there with upon the cauda equina and the spinal cord. He emphasizes, that it did not matter if one injured one of the nerves of the cauda equina with a fine puncture needle.

Corning describes the technique of the procedure as follows: a trochar is pushed through the skin between the first and second lumbar vertebrae. Through the opening of this trochar a long, fine, hollow needle, which is screwed to an ordinary sub-cutaneous syringe, is pushed forward as far as the lumbar sac. Thereupon the contents of the syringe is injected upon the cauda equina. Corning does not permit any cerebro-spinal fluid to escape. He does not state how he knows that the point of the needle is within the lumbar sac.

Corning bases his priority claim upon the above publications.

The methods of Corning, notwithstanding several successful experiments, did not meet with general favor and was accordingly dropped until taken up some years later by Bier, of Kiel (1898). In 1898, Bier made his first experiments with spinal cord anesthesia, which he described in 1899, without having any idea that somebody else had also worked in this field.

Corning's experiments have remained completely unknown

and unconsidered in Europe. His book "Pain" in which for the first time, he proposed to carry cocaine into the lumbar sac, was so difficult to procure in Europe, that Bier required the aid of an American colleague in order to get it.

Evidently, no consideration was given to Corning's experiments even in America. Those American surgeons who state that the spinal cord anesthesia something long known to them, and that Corning merits the priority, as nothing further was needed to translate his complete experiments into surgical experience, were asked the following questions by Bier: "Why have you never, prior to me, carried out an operation under spinal anesthesia, while you have received my first experiments with tremendous and really not very critical enthusiasm and have imitated them?" "You really give a very poor attestation to your intelligence, if you claim, that although you have known the procedure fourteen years prior to my experiments, it was necessary that an European had to make you acquainted with the importance of the work of one of your countrymen."

In accord many physicians have rejected the claim of Corning. Of these, Bier only desires to mention three. Thus in America, Robinson, in the Medical Record of 1901, pointed to the past that Corning had only a vague idea of the consequence of the method, and was not aware of its importance.

In Germany, Hahn came to the conclusion: "It is not proper, therefore, that one designate their method as Corning's, as is done in America. It is connected with the name of Bier for all times."

In France, Paul Reclus in two articles entitled "De la methods de Bier" and "La methods de Bier" has very energetically given credit for the spinal anesthesia to Bier alone.

After all, Bier claims, in its fullest extent, the discovery and the introduction of spinal cord anesthesia. He, however, concedes to Quincke by far the greatest credit. Not only did he come upon the idea of the procedure by his lumbar puncture, but this method of anesthesia has only become possible by means of the lumbar puncture. The blind injection of cocaine with a Pravoz syringe, which is supplied with a long, fine, cannula such as was carried out by Corning in his few experiments, would not lead to successful results, even if one attempted to determine the distance from the skin surface to the spinal cord by means of measurements according to Corning's directions. By virtue of other very great experiences, it is now known that only the dripping of cerebrospinal fluid proves that the needle is in the lumbar sac, and that it is a gross technical error to perform the injection without this manifestation.

Since 1885, the methods of inducing spinal anesthesia

has been carefully elaborated, and has been practiced in thousands of recorded cases by numerous observers both in America and in Europe, where it is much more popular than it is in this country. Credit for the first surgical operation under spinal anesthesia in America, is given to Tart and Cagliariere (25).

A large number of drugs, capable of causing loss of sensation by contact with the unsheathed roots of spinal nerves, have been used for this purpose, principally, cocaine, tropacocaine, eucaïne, stovaine, novacaine, alypin, pantocaine, morphine, antipyrine, magnesium sulphate and so forth. In the early history of spinal anesthesia, cocaine was almost exclusively employed, but, on account of its admitted danger, its use has been practically abandoned and it has been superseded by other substances which are closely allied to it both chemically and therapeutically, but which have proven far less toxic and consequently less dangerous.

HEADACHE:- The most frequent complication that has been noted is postoperative headache. Dana (13) says the symptom is more common in patients whose cerebrospinal fluid is negative, and in whom the fluid comes out under low pressure. In other words, the healthy cord reacts badly to puncture. It is more common and severe in young adults, in women, in persons of high-strung nervous temperament, in the anemic and in the thin blooded.

Calmon (11) believes that the headache is caused by meningeal irritation and not by loss of cerebrospinal fluid while Jaschke (30) attributes the cause of headache after lumbar anesthesia to changes in the anesthetic.

It has been reported as occurring in from one to ten per cent of the cases where spinal anesthesia has been induced with novocaine... Spinal puncture for diagnosis is frequently followed by headaches.

The degree of headache varies from a mild sensation of fullness in the head to a severe type of excruciating pain. The majority are of the milder type and last a period of a few days. They are usually controlled by the common analgesic

drugs and the use of an ice bag. The patient should be kept quiet and prevented from raising his head as this makes the aching more pronounced.

The occurrence of a severe type of headache is trying to both surgeon and patient. The occurrence of the headache is due to a decrease or to an increase in the volume of the cerebrospinal fluid. Headaches caused by the loss of the cerebrospinal fluid are usually noticed when the individual tries to raise his head or sit up. Relief is obtained by lying down but sometimes the foot of the bed must be elevated before relief occurs (2). As is to be expected much theorizing has been done in an effort to explain the cause of this type of headache (14). Among the several ideas advanced are the following which are now widely accepted:

1. Loss of cerebrospinal fluid through the puncture wound in the dura by seepage into the extradural soft tissues.
2. An increase in the volume of cerebrospinal fluid contained within the sub-arachnoid space.

Baruch (1) first attempted to prove experimentally the theory of Mac Robert that fluid escapes through the hole in the dura after lumbar puncture. He performed lumbar puncture and without withdrawing any fluid injected 3 c.c. of a 2 per cent indigo-carmin solution into the sub-arachnoid space. He then plugged the needle with mandrin so that no fluid escaped.

He then inserted a permanent catheter into the patient in order to determine immediately the appearance of the dye-stuff in the urine. In this experiment, with the lumbar puncture in situ, no dye appeared in the urine after 63 minutes. On withdrawal of the needle, however, the dye appeared in the urine in 8 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. He took this as evidence of the fact that fluid escapes through the hole created in the dura by a lumbar puncture needle.

The mechanism of occurrence in which spinal fluid has been lost has been ascribed to the lack of elastic and contractile tissue in the fibrous dural membrane. It is believed that the puncture hole caused in the dura by the needle point does not close until a fibrin clot forms or normal healing takes place.

Upon withdrawal of the needle, the dura and the underlying pia-arachnoid are left in one of two possible relations. Either the openings in the two membranes are superimposed, if the pia is drawn back through the dural opening by the needle, allowing leakage to take place or else the movement of the soft membranes upon each other causes the openings

in the respective membranes to be situated at different levels. The force of the cerebro-spinal fluid is then thought to compress the pia-arachnoid against the dura for an interval long enough to permit the pial opening to close by virtue of its inherent elasticity. If the latter mechanism takes place very little cerebrospinal fluid will escape.

As a result of the considerable seepage of spinal fluid during the first few hours after the operation, there is a disturbance of the intra-cranial portion of the cerebro-spinal fluid. It has been claimed that the latter factor, by removing the watery cushion, allows the brain to sag against the bony framework of the skull. This in turn, supposedly, irritates the dural fibers of the trifacial and the two occipital nerves. Pressure upon the basilar venous plexes diminishes the outflow of venous blood from the cranial cavity and brings about an increase of venous tension. Lowering the patient's head is thought to remove the brain from contact with aforementioned nerves and venous plexes and thus tend to lessen the severity of the headache. Relief is similarly obtained when the opening in the dura has closed, seepage of fluid has ceased, and the normal watery cushion of the brain has been restored.

This type may be referred to as the seepage type of

headache is ascribed ordinarily to the lumbar puncture itself. The following case history illustrates the above condition.

The patient was a woman 23 years of age who was operated upon April 19, 1922. An appendectomy was performed with syn-caine spinal anesthesia after removal of 20 c.c. of cerebrospinal fluid. The postoperative course was normal with the exception of a headache which was progressive and reached its maximum in 5 days. The patient had no vomiting or meningeal signs. Her temperature was not high and her pulse was 90. She had marked asthenia but no somnolence. Her appetite remained good.

The headache reached its maximum of intensity between 10 a. m. and 4 p. m. It was extremely severe when she was sitting. The patient rested at night but went to sleep very late.

A subcutaneous injection of 150 c.c. of isotonic glucose serum was given on May 4 without any effect. On the following day a subcutaneous injection of normal salt solution gave rapid relief.

The headache returned on May 6 at about 5 p. m. and a second injection was given of 150 c.c. of salt solution and she was given 1 gram of theobromine by mouth. The pain decreased during the night and on the next morning the head-

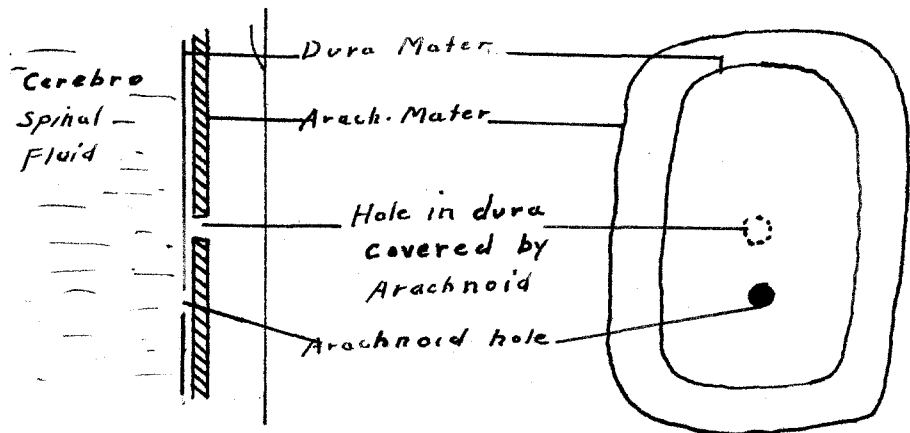
ache had completely disappeared.

Although the pressure of the cerebrospinal fluid was not taken in this case, it is very probable that the symptoms were due to hypotension (3).

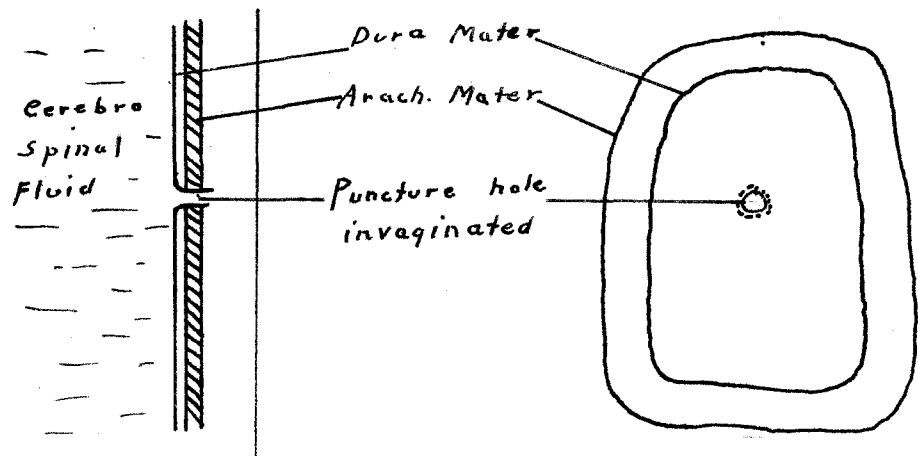
The second variety of headache, however, is thought to be the result of an increase in the quantity of cerebrospinal fluid. Usually, other symptoms and signs are associated as, for instance, stiffness of the neck, photophobia, and more rarely, paralysis of the external rectus muscle of the eye with or without a concomitant diplopia. This syndrome is described as the postanesthetic meningitis or meningismus. Whereas in the former variety, the spinal fluid pressure may be very low, there is usually found an increase in the pressure associated with an increase in cells and globulin constituents of the cerebrospinal fluid in those patients with meningismus. Then either a state of meningeal irritation is present, or else, rather infrequently, a true infectious meningitis. Such headaches do not respond to the lowering of the patient's head.

Unfortunately, the literature is considerably at variance as to the cause of headache after either diagnostic lumbar puncture or after the intra-spinal injection of an anesthetic. Burrus like many others attributes the headaches to leakage and condemns the larger caliber needles because

more fluid can escape through the larger opening and healing is proportionately delayed.



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



Non closure 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 (24).

RESPIRATORY PARALYSIS:- There is very extensive literature concerned with the effect of anesthetics and narcotics on nerve fibers and nerve centers (16). In general, the centers are much more susceptible than the fibers, as every general anesthesia shows, for motor nerves are still capable of stimulation, even when the patient is adequately or even dangerously under the influence of the anesthetic.

Moreover, all nerve fibers are influenced by anesthetics in the same way. This is what is to be expected, of course for we know, are similar in structure. The slight difference in susceptibility between nerve fibers of different functions appear to depend on nothing except their different size. Then the pain fibers yield to the action of cocaine slightly more readily than temperature fibers, and the temperature fibers more readily than the proprioceptive and motor fibers, this being due simply to the small size of the pain fibers and the increasing size of the others.

A consideration of the literature would lead to the conclusion that a spinal anesthesia that involves the cervical region must be dangerous, for the phrenic nerve is made up of fibers that leave the cord in the fourth anterior cervical coat, with varying contributions from the third and fifth. Operations involving arms, neck and face require the application of an anesthetic solution to the posterior roots

in precisely this region. There is, however, some experimental evidence and much more clinical experience that is at variance with this conclusion. Labat and Lericke have contended for some time that respiratory paralysis does not occur in high spinal anesthesia, and that the deaths so interpreted are really due to cerebral anemia (15). Koster and Kasman (21) published an impressive article in which this opinion is supported by an extensive clinical experience as well as by a certain amount of experimental work.

Jones (18) believes the danger in intra-thecal administration is not a vaso-motor effect from a regional block of the thoracic and lumbar roots, which proves to be of much less significance than was formerly thought, but from blood absorption and paralysis of the vaso-motor center, leading in some cases with excessive dosage to severe collapse to a pulseless state. The nausea and vomiting which sometimes occur after injection, apart from surgical manipulation, is similarly due to stimulation of the vomiting center by the poison, as well as low blood pressure. This may be realized by injecting the full contents of a spinal ampoule into a muscle. It is not to be expected, therefore, that the respiratory center will escape entirely, and the degree to which it may be affected will depend on the amount of the dose in relation to the body weight of the patient. The

effect on the cortex shown by the drowsiness and tendency of the patient to sleep also depends on the amount of the drug circulating in the blood. The zenith of blood content is perhaps reached about 15 to 20 minutes after injection by the gravitational method, and a little earlier by the direct method, because of the larger absorptive surface to which the drug is exposed.

Cushny states that the relative non-toxicity of novocaine is due to the fact that novocaine is destroyed by the liver and cocaine is not. The slow absorption from infiltration, especially with adrenalin, gives the liver time to deal with the poisons, but when novocaine is injected intravenously the toxicity more nearly approaches that of cocaine. On account of the rapid absorption of spinal injections their toxicity must be redeemed as only less high than those administered intravenously. If the dosage is high and the body sloped, a variable concentration will reach the anterior roots of the phrenic and intercostals and reduce their conductivity in proportion to the concentration, and the respiration becomes shallow and slow. Anoxemia will further reduce the center and increase a tendency to sleep.

The respiratory center may therefore be paralyzed by an overdose of the drug in the blood, or by a combination of reinforcing agents of which the analgesic and preliminary

narcotic may be the cause. If the center is badly fatigued and poisoned by drugs it may be difficult to keep it active, whatever means may be adopted.

Koster states in relation to the human subjects, "In many patients, particularly in children, when head anesthesia is induced by encouraging diffusion of the drug upward to the medulla, it is found that sleep results." This appears to have been the experience through the history of the method from the earliest cocaine days, and is but due to the fact that the dosage has been relatively high in children compared with body weight. This sleep has also been ascribed to the complete isolation of the consciousness from all body stimuli; a mind deprived of body sensation yet capable of that most potent of all experiences, fear and falling asleep on the operating table out of sheer boredom from want of stimuli. To produce general anesthesia, all that is necessary is to inject a sufficient dose of novocaine in the lumbar region, there after inclining the body in the Trendelenburg position. This spreads the drug over the nerve roots, and at the same time exposing it to an enormous absorptive surface. The general affect on the patient may be profound. Koster admits that in some cases a peripheral pulse is not to be expected, but implies that this is no disadvantage since the surgeon can always refer

to the aorta, a consolation not available to the specialist in anesthesia. This condition almost resembles a state of suspended animation, and it may be said that the patient is "hibernating." And so it seems. Every function of the body is depressed to its lowest level; no pulse can be felt or, when palpable, may be reduced in rate as low as 30 to 40 per minute. The conductivity of the phrenics is reduced and respiration is just sufficient for life. A short period of sleep may be observed and the lips and cheeks may puff out as they do under chloroform.

There are two components at work, a regional nerve root block and a blood dose of novocaine. To what extent the cranial nerves are directly involved is an interesting question. In the dorsal decubitus the novocaine will first be directed towards the cisterna magna, and from here it may spread over the base of the brain, but the arachnoid reticulum is here at its densest and the amount of fluid far greater than it has already encountered in the spine. In such high dilution the novocaine has to attack the largest and most compact sensory coat of the body.

Is it not probable that an amount of novocaine injected in the lumbar region, capable of spreading through the subarachnoid space as far as the fifth cranial roots, may also be capable of producing general analgesia apart from nerve

block, if not actually general anesthesia?

Dickson Wright says that "shallow and slow respirations may be worrying. He records a respiratory rate of six per minute, and says, "The patient was asleep and I did not care to wake her to tell her to breath faster." When admonition is inapplicable, carbon dioxide and oxygen are indicated. The outstanding clinical feature of such cases are a reduced frequency and amplitude of respirations, and a very low blood pressure, sometimes reaching a pulseless rate. The one means imperfect lung ventilation, and the other a reduced rate of blood flow through the lung capillaries. Both tend to anoxemia and a vicious circle which may end in an arrest of respiration, and it is no recommendation to claim that the majority survive.

AFFECT ON BLOOD PRESSURE:- It is stated by authors in lumbar puncture, the puncture of the skin, is accompanied by a slight rise in blood pressure which varies in extent with the degree of consciousness, the pain, and the disturbance produced by pre-operative procedures. This may last from five to fifteen minutes.

Then follows a preliminary fall which is due to the flaccid paralysis of the skeletal and abdominal muscles, the relief from the pre-anesthetic manipulation, and the onset of mental calmness which may amount to sleep.

The main fall in blood pressure which will vary with the height and concentration of the anesthetic. The fall in blood pressure is due to three major factors and it will appear to some degree in each high anesthesia:

1. The main factor is the ascending root paralysis. If the anesthesia reaches only the lumbar nerve root there will be no appreciable drop in the blood pressure.

The fall in blood pressure will be in proportion to the number of nerve roots between the first thoracic and the third lumbar that are involved.

2. The second factor is thoraco-abdominal muscular paralysis, which is compensated for by an increased action of the diaphragm. This paralysis will diminish the aspirating action of the thorax and therefore a smaller volume of blood will be sent to the heart.

3. The third factor is that with higher anesthetics the sympathetic cardio-augmenter (cardiac) nerve is blocked, which allows the vagus nerve, unrestricted, to slow the heart.

Finally, twenty-five to thirty minutes after the injection, the conduction through the white rami will approach normal and there will be a gradual increase in blood pressure.

Variations in pulse rate do not closely follow those of

the blood pressure but a certain degree of resemblance is seen owing to the consciousness of the patient (21) and (14).

PULMONARY COMPLICATIONS:- Brown (8) says it has been his privilege to observe the course of post-operative pulmonary complications following practically all types of anesthetics. The majority of operations were performed under sub-arachnoid block. Like wise the greatest number of cases in which post-operative pulmonary atelectasis were observed were those in which spinal anesthesia had been employed. While the series is numerically too small to give definite statistics for the evidence of this complication following various types of anesthetics, nevertheless, the impression gained thus far is definitely that the evidence of pulmonary atelectasis is greater following spinal anesthesia than following any form of inhalation or regional anesthesia. This impression holds regardless of the type of operation undertaken.

At first thought, one would imagine the opposite to be true, that post-operative pulmonary atelectasis would occur more frequently following inhalation anesthesia. But on closer consideration, several reasons are noted why spinal anesthesia might predispose to this complication.

First, spinal anesthesia definitely inhibits the depth and force of respiratory movements, not only during the

operation itself, but for a considerable period there after. It is these respiratory movements, (both intrinsic and extrinsic) which tend to rid the tracheobronchial tree of foreign matter or secretions.

Second, the normal viscosity of the secretions of the bronchial tree appears to be increased i.e., the material is more tenacious following spinal anesthesia.

Third, following operation under spinal anesthesia the patient tends to remain relatively quiet for a number of hours. One has, then, a more tenacious sputum and decreased or impaired factors that might tend to free the tracheobronchial tree of this material. The increased possibility for this material to obstruct or plug a bronchus and the subsequent development of atelectasis appear reasonable.

Jones (19) believes lung collapse seems to occur quite frequently after spinal anesthesia, practically always after abdominal operation. Whether it is more frequent than after--inhalation anesthetic he does not say, but he is beginning to believe it is. Spinal anesthesia is no protection against lung collapse but lung collapse seems almost never fatal no matter how ill patient seems to be. Apparently the dose of the spinal anesthetic may have some bearing on lung collapse but there are too many other factors present for us to be sure. These factors are of course, the type of operation,

the use of morphine, scopalamine and sodium amytal and so on.

To determine the validity of the impression that the incidence of post-operative pulmonary complications especially atelectasis, appeared to be greater following spinal anesthesia than after inhalation anesthesia, the following statistical studies were reported on an unselected group of 812 consecutive case histories of patients operated on over a period from July 1, 1929, to June 31, 1930 in two San Francisco hospitals. During this period an intensive study was made of all cases regarding the development and prevention of various post-operative pulmonary complications regardless of the type of anesthesia used.

The choice of the anesthesia depended on the individual surgeon. The tables show that about as many "serious major" operations were done under general anesthesia as under spinal anesthesia; but it should be noted that the consensus of the surgical staff is that the old patient, the patient in shock, the patient who is a poor surgical risk generally, should not be given a spinal anesthetic. Hence more of these patients were operated on under an inhalation anesthetic. In spite of this, post-operative pulmonary complications were almost five times more frequent than after inhalation anesthetic. Patients who received both types of anesthesia are not included in these studies (9).

Classification by Type of Post-operative Pulmonary
Complications Diagnosed:

	INHALATION ANESTHESIA				SUBARACHNOID ANESTHESIA			
	<i>Atelectasis</i>	<i>Broncho-pneum.</i>	<i>Hypostatic pneum.</i>	<i>Infarct</i>	<i>Atelectasis</i>	<i>Broncho-pneum.</i>	<i>Hypostatic pneum.</i>	<i>Infarct.</i>
UPPER ABDOMEN	0	0	0	0	5	2	1	0
HERNIOTOMY	0	0	0	0	4	2	0	0
APPENDECTOMY	2	1	0	0	3	3	0	2
LOWER EXTREMITIES	0	0	0	0	1	1	1	0
UPPER EXTREMITIES	2	1	0	0	0	0	0	0
TOTAL	4	2	0	0	13	8	2	2

In the majority of instances, atelectasis was the complication found. Moreover, as has been pointed out frequently, the more closely the operative procedure approaches the diaphragm, the greater is the incidence of post-operative pulmonary complications.

BACKACHE:- Backache occurs in a small percentage of cases. It may be due either to trauma of the soft parts that are traversed by the needle, especially when several attempts

to enter the subarachnoid space have been made, or else to a serous meningitis resulting from the wound in the dura and pia, the liberation of a small quantity of blood by accidental penetration of one of the venous plexuses, or the introduction of rust particles from the needle, or even from the presence of several bubbles of air. No matter what the cause may be, the backache is but short in duration and is frequently relieved by placing a pillow under the small of the back (21).

SHOCK AND SYNCOPE:- The majority of the deaths which have been attributed to spinal anesthesia have been the result of shock. No doubt many of these fatalities were the result of improper technique or disregard of definite contraindications. There are at least five ways in which shock or syncope may be produced during spinal anesthesia (14).

1. Lumbar Puncture or Meningeal Reflex. This may occur in the nervous or overstimulated patient, especially where insufficient preoperative morphine has been given. It may follow an injury to the meninges when a large needle is used or repeated attempts have been made to enter the dural canal, or when the patient has been kept too long in the sitting position. The onset in these cases is usually sudden complete collapse with cessation of the function of the vital centers. The result is variable, from a mild fainting spell

to sudden death.

2. Visceral Reflex Shock. This may occur when the intestines are too roughly handled, when the stomach or omentum is pulled upon, or when the upper abdominal organs are tightly packed off. It is the result of disregarding the principles of atraumatic surgery and should not occur with those who are zealous of their technique.

3. Drug Poisoning (Shock). Very rarely there is an individual who is hypersusceptible to neocaine. Therefore an acute drug poisoning may occur following its intraspinal administration. One should be able to recognize and treat such a condition. The drug poisoning may be due to such hypersusceptibility, or to an unusually strong drug, or to the accidental injection of the drug into one of the meningeal veins. The symptoms are those of anaphylactic shock and the treatment is, as outlined elsewhere, that for vasomotor collapse and respiratory failure.

4. Position Shock. This form of shock occurs when the patient is suddenly changed from one position to another, especially from the Trendelenburg to the horizontal position. It occurs most frequently in weak or very old patients. These patients have had what, for them, is an almost lethal dose of the drug and have had low blood pressure and a decreased respiratory rate throughout the operation. It results from a

sudden added embarrassment to an already overladen heart. It is the most common complication occurring at the end of the operation. Patients who have been quiet and apparently asleep during the operation seem most often affected in this way. It should always be remembered, therefore, that the patient who is too quiet, especially if aged and possessing lowered vitality, should be watched more closely for symptoms of shock and should not be subjected to any sudden change in position or other added embarrassment to his circulatory system.

5. Intracranial Pressure Shock. This may lead to the death which is known as the "lumbar puncture death." It may have been the cause of many deaths which have been cited to prove spinal anesthesia a radical and unsafe anesthesia. It is the commonest cause of death following the simple lumbar puncture, yet diagnostic lumbar puncture continues to be a standard and valuable procedure. Schonbeck thoroughly reviewed the literature and has collected 71 cases. Over 50 per cent had intracranial tumors; 13 per cent had cerebral hemorrhage; 57 or 58 per cent of the cases presented symptoms of some cerebrospinal pathology which was present at the time of the lumbar puncture.

This acute condition results from a loss of the spinal fluid, either at the time of the puncture or by later leakage through the dural opening. Due to the release of spinal

fluid pressure below, the medulla is jammed into the surrounding foramen magnum. Death may be sudden, beginning with a terrific headache, or it may occur within a few hours. It is due to suppression of the medullary centers and the symptoms are those characteristic of severe intracranial pressure.

OCULAR DISTURBANCES:- The ocular palsies, though rare, are considered serious sequelae to this form of anesthetic. They seldom occur in well conducted cases and in all cases clear up spontaneously.

It is limited to the extra ocular muscles and usually the muscle supplied by the abducens nerve. If it occurs it ordinarily appears during the second week after the operation, although cases have been reported wherein they occurred immediately after the lumbar puncture. The first symptom is slight photophobia, followed by diplopia which is very annoying to the patient and surgeon. One or more of the symptoms of meningeal irritation usually accompanies the ocular symptoms. After about ten days to two weeks the paralysis retrogresses and clears up spontaneously by the third or fourth week. It is not known why this particular nerve should be involved. The more important theories are:

- a. This nerve may have a special susceptibility to the drug.
- b. Because of its long passage through the spinal fluid,

it is more exposed than the other nerves or nerve roots to the drug or the spinal contaminations e.g. capillary hemorrhage, air bubbles, foreign bodies, and so forth (14).

A functional optic neuritis occasionally accompanies abducens palsy. There is a disturbance of vision, sometimes insignificant, at other times, rather marked. The degree of disturbance is not always proportionate to the degree of changes seen on ophthalmological examination. There may even be complete blindness, though temporary, as reported by Wells. This alarming complication occurred in one instance out of a total of 557 cases by Wells after the use of anhydrous cocaine. Fortunately the patient recovered completely at the end of five days (21).

Reboy (27) states he has never encountered any case of choked disc as reported occasionally in the literature of spinal anesthesia. According to his views the infrequent paralysis of the tri-facial, oculomotor and external oculomotor nerves, and pressure on the optic nerve (choked disc) can be explained by hypertension of the cerebrospinal fluid consequent to serous, toxic meningitis caused by the injected anesthetic agent.

Drooping of the left upper eyelid (the result of a paralysis of the branch of the oculomotor nerve which supplies the levator palpebrae superioris) was reported by Wells.

This disappeared at the end of two weeks and was encountered in but one case. Martin and Arbuthnot, in a review of over 6000 cases operated on under spinal anesthesia mentioned but one ocular complication, an inability to move the eyeballs without causing intense pain (14).

ANAL AND VESICAL DISTURBANCES:- (20) Often reference is made to anal or vesical incontinence as a possible complication. Most authors, however, remark that they did not encounter such a state of affairs or else an occasional case appeared wherein reestablishment of the normal functions required but several days to a week. Orth for instance, reports a case of fecal incontinence that cleared up spontaneously after five days, and Izquierdo one that persisted for a week.

On the third day after a double inguinal herniotomy under 0.12 gm. of stovaine, a patient of Sourasky developed urinary incontinence, that is, retention with overflow. This was soon followed by occasional fecal incontinence. Fully a year elapsed before complete restoration of functions appeared.

Dazy reported a case of herniotomy in a man of fifty-nine who for eighteen months after the operation experienced no desire to urinate. When the desire had finally returned, spontaneous contraction of the bladder failed to appear. Catheterization was necessary for the entire eighteen months.

After progressive education of the abdominal muscles, catheterization was at last suspended at the end of two years. Still his difficulties were not at an end since, despite his ability to urinate normally at the beginning of his apparent restoration, he was later obliged to assume a crouching posture. Involuntary urination would take place if he neglected to void for from six to eight hours.

The necessity of catheterizing patients post-operatively occurs in about ten per cent of cases. Of this number ninety per cent need only one or two catheterizations and the remainder void spontaneously after the third day.

In no instance did retention last longer than the stay in the hospital necessary for wound healing.

It is our impression that post-operative urinary retention is much more frequently encountered after inhalation narcosis.

Of all the cases presenting this complication, seventy-five per cent occurred after operations involving the perineum or pelvis or both. We rarely encounter vesical disturbances in head, neck or thorax operation.

We have had one experience with vesical incontinence directly referable to the anesthesia. This occurred in a female patient operated on for uterine fibroids. For the first eight days, she voided spontaneously. On the ninth day, it

was reported that she was incontinent. Examination revealed a distended bladder reaching almost to the umbilicus. Fifty-two ounces of urine were obtained on catheterization. Following this, spontaneous urination reappeared. No case of fecal incontinence has come to our notice (21).

MENINGITIS:- Meningitis is a complication which occurs in a frequency which is proportional to the faults in technique. With those using correct technique it is so rare as to be practically non-existent (14).

1. Non-infective Irritative Meningitis: This is an irritative condition of the meninges which is often spoken of as "meningismus." It is essentially a congestion with resulting exudation following the exposure of the meninges to any toxic substance. It may follow a toxic dose of the drug, capillary hemorrhage when a dural blood vessel is punctured, trauma to the dura when several attempts are necessary to enter the dural cavity, the injection of air bubbles into the dural cavity or the introduction of foreign material such as rust or dirt which may come from the syringe, needle or drug.

Beginning twelve to twenty-four hours after the injection the patient shows mild meningeal symptoms such as pain along the spine, stiff neck, photophobia and nervousness. There may be rarely a persistent headache, while the more severe cases

may show some involvement of the eye muscles. There is an increase in the spinal fluid pressure and an examination of this fluid shows an increased cell count but no marked increase in globulin.

The condition is very rare. The symptoms are mild and disappear after six to ten days with no after effects, except possibly a later neurosis which should be prevented.

2. Purulent Meningitis: This is due to a faulty technique, just as is the infective peritonitis that follows a clean laparotomy. It is the same risk of sepsis encountered in every surgical case. The symptoms show themselves, usually, during the first twenty-four to forty-eight hours. Headache and restlessness are followed by the characteristic signs and symptoms of infectious meningitis. Lumbar puncture will usually show a turbid spinal fluid. The pressure will be abnormally high and the cell count and globulin content greatly increased. The literature for the last twenty-years shows only nine such cases with two deaths. As a rule the meningitis tends to clear up spontaneously in ten to fifteen days with no after effects.

AFFECTS ON CENTRAL NERVOUS SYSTEM:- In reviewing the literature on spinal anesthesia, one is surprised to find a large number of articles in which thousands of cases have been reported, but few of them give the clinical observations

of the neurologic examination and the neurologic complications and necropsy observations (23).

Barker, Pitkin, Babcock, Koster, Labat and others have described the technic of spinal anesthesia. In the series of cases composing the present report, the method of administering the anesthesia was the same.

The most common sequel was the complaint of pain in the extremities, particularly the legs, but also in the back, and, in one instance, the head. The pain in the legs persisted in three cases for several months and on the last examination was still in existence. In these cases there resulted a marked muscle tenderness in the extremities, which persisted for several months.

In this particular series, two patients died; the first, twenty days after operation. Autopsy being done on both and neurological findings noted.

His only complaints up to two days before his death were weakness and pains in the extremities. He suddenly went into coma, at which time his blood pressure was 138 systolic and 72 diastolic. The nonprotein nitrogen was 55.6 mg. The heart showed mild cardiac decompensation. The neurologic examination was negative except for diminished tendon reflexes. An autopsy was performed five hours after death. The pathologic diagnosis by Dr. C.V. Weller was as follows: Section of

the spinal cord and meninges showed congestion and numerous small psammoma bodies in the meninges. The brain demonstrated congestion and edema in the meninges and brain substance. Throughout the lower portion of the pons and upper medulla there were numerous small false psammoma bodies of the myelin droplet type.

The second patient, who died twelve days following operation and who had been treated previously for syphilis, showed no evidence of organic disease of the central nervous system at the first examination. Following operation, he complained of pains in his arms and shoulders. The Kahn reaction, as well as other spinal fluid examinations, was negative. He had good anesthesia during the operation, but immediately following it he was confused and cooperated poorly. Apparently he was able to feel pain and the tendon reflexes were hyperactive. The pathologic diagnosis made by Dr. C. V. Weller was as follows: There was edema of the meninges and spinal cord. No myelinosis was present in the upper cervical cord and in the medulla, but in the lower cord there was a marked myelinosis, especially near the meninges. Some small nerve roots showed an extensive degeneration of the fibers with a loss of myelin sheaths.

In 1903, Spielmeyer found involvement of the ganglion cells in that there was chromolysis, dissolution of the

Nissl bodies and rounding of the cell bodies, also dissolution of the nuclear membranes. This was believed to be a circulatory disturbance. He also saw similar lesions in the large anterior cells of the spinal cord, so he believed there was a direct toxic effect on the axis cylinders with secondary degeneration of the ganglion cells. The most intense reaction was present in the nerve fibers in the anterior column of the cord. Brunn (10) in 1922, noted swelling of the nucleus of the ganglion cells and wandering toward the periphery after spinal anesthesia. These changes were visible after the cessation of anesthesia.

One may advance the theory that all spinal anesthetics produce an acute myelitis. This condition usually lasts a few hours and may leave the patient with complaints of pain and paralysis for a period, but there are times when there is a permanent involvement of the nervous system both clinically and pathologically. The fact that there may be certain definite residuals from this method of anesthesia must be recognized (23).

On the basis of observation of thousands of cases of spinal anesthesia, the authors believe that lumbar anesthesia in competent hands is one of the safest and most satisfactory of all forms of anesthesia. Any surgeon who once becomes familiar with its ease of administration and its ad-

vantages both to the patient and to himself will seldom want to return to other methods. The complete relaxation of the muscles (which is found only in deep stages of general anesthesia) and the quiet abdomen, facilitates rapid and thorough surgery. The lack of post-operative vomiting and abdominal distention reduces nursing care to a minimum.

However, spinal anesthesia is a sharp edged tool and in the higher doses and in upper abdominal surgery it should be employed only by those skilled and experienced in its use. It is not fool-proof and the day perhaps will never come when it will entirely supersede all other types of anesthesia.

BIBLIOGRAPHY

1. Alpers, Lumbar puncture headache., Arch. Neurol and Psych. 14: 806. 1925.
2. Anderson, Ernest, R., Complications of spinal anesthesia., Journal Lancet 51: 403-407. July, 1, 1931.
3. Arnaud, M. and Cremieux, A., "Secondary" headaches of spinal anesthesia., Marseille Med. 64: 31-40. January, 5, 1927.
4. Babcock, M. E., Spinal anesthesia deaths (a survey)., Anesthesia and Analgesia . July- August, 1932.
5. Bier, A., History of spinal anesthesia., Munchen Med. Wehnschr 53: 1059-1061. May, 29, 1906.
6. Boros, J., Cystic purulent cerebrospinal meningitis following lumbar anesthesia., International Abst. of Surgery 43: 56. 1926.
7. Brown, A. L., Spinal anesthesia., American Journal of Surgery 39: 2-7. January, 1925.
8. Brown, A. L., Pulmonary atelectasis., Arch. of Surgery 22: 976-982. June, 1931.
9. Brown, A. L. and Debenham, M. W., Postoperative pulmonary complications., J. A. M. A. 99: 209-210. July, 16, 1932.
10. Brunn, Von, M., Die lumbal anasthesie in neue deutsche chinegie., Stuttgart 29. 1922.

11. Calmann, Lumbar puncture headache., J. A. M. A. (abs) 81: 1827. 1923.
12. Dameno, E., Oculomotor paralysis after intraspinal anesthesia., J. A. M. A. 71: 77. 1918.
13. Dana, C., Puncture headache., J.A. M. A. 68: 1017. 1917.
14. Evans, C. H., Spinal anesthesia. 1929.
15. Gray, H. T. and Parsons, Blood pressure variations associated with lumbar puncture and spinal anesthesia., J. A. M. A. 59: 2190. 1912.
16. Harrison, P. W. and Frank Ruth, Respiratory paralysis in spinal anesthesia., Arch. of Surgery 571-573. September, 1932.
17. Ingvar, Leakage after spinal puncture., J. A. M. A. (abst) 81: 175. 1923.
18. Jones, H. W., Respiratory paralysis, fallacies and methods., Anesthesia and Analgesia 11: 228-231. 1932.
19. Jones, H. W., One thousand spinal anesthetics., Annals of Surgery pp. 9, 88. July, 1932.
20. Koster, H. and Weintrob, Complications of spinal anesthesia., American Journal of Surgery 8: 1173. 1930.
21. Koster, H. and Kasman, Surgery, Gyn., and Obst. 49: 617. December, 1929.
22. Levi and Cohn, Meningitis following spinal puncture 85: 1968. 1925.

23. Lindemulder, F. G., Effect on central nervous system.,
J. A. M. A. 99: 210-212. July, 16, 1932.
24. Mac Roberts, R. G., Cause of lumbar puncture headache.,
J. A. M. A. 70: 1350. 1918.
25. Orth, D. A. Spinal anesthesia., American Journal of
Surgery 39: 27. January, 1925.
26. Pechel, R., Complications of spinal anesthesia., J. A.
M. A. 85: 1676. 1923.
27. Rebay, H., Ocular complications of intraspinal anesthe-
sia., J. A. M. A. (abst) 84: 2031. 1925.
28. Seevers, M. H. and Waters, R. M. 99: 961-968. 1932.
29. Symonds, G. P., Meningitis following lumbar puncture.,
Lancet 1: 434-435. 1925.
30. Von Jaschke, Puncture headache., J. A. M. A. 84: 714.
1925.