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Complications of spinal anesthesia

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THE COMPLICATIONS
OF
SPINAL ANESTHESIA

SENIOR THESIS

UNIVERSITY OF NEBRASKA COLLEGE OF MEDICINE

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THE COMPLICATIONS OF SPINAL ANESTHESIA.

INTRODUCTION

Spinal anesthesia is that special form of anesthesia induced by the injection into the subarachnoid space of an anesthetizing solution. This results in spinal block due to local absorption of the solution with effects on the spinal cord and spinal nerve roots within the dura. This form of anesthesia is essentially a root block of the spinal nerves, both sensory and motor. There is a further effect upon the vasomotor nerves which has claimed the attention of surgeons using this form of anesthesia. (Poe, 1932) Maintenance of consciousness is one of its characteristic features. (Labat, 1928)

To begin with we find some dissension over the names applied to this form of anesthesia. Some terms used are spinal anesthesia, spinal analgesia, lumbar anesthesia and subarachnoid block. Case (1928) states that the word lumbar is strictly correct and prefers its use. The first name is the one most commonly used although some of the others maybe more suitable and scientifically correct.

Most authors are fairly well agreed upon the possible complications and their treatment, but somewhat at variance as to their etiology. However, through experimentation the profession has been considerably enlightened, more so in the past eight to ten years than in the three decades preceding.

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With regard to the incidence of complications the various authors show a difference of opinion on this matter. In 1100 cases, Case (1928) found three with complications. Fawcett (1931) and Levine (1930) stated the complications are rare, a statement which probably represents the facts and findings of most of the surgeons today.

HISTORY

Spinal anesthesia is not a new procedure but for the past three decades has had a limited vogue in this country and Europe. It failed to attain the popularity its advocates had hoped it might, and so very little was done with it. Just prior to the World War there was a reawakening of interest but it again waned and fell to a low ebb. About ten years ago it was employed by surgeons in various sections of the country and it became a topic for scientific discussion. Articles by the score appeared in the medical and surgical journals. Soon the subject occupied a conspicuous position in the minds of hundreds of surgeons in this and other countries. (Welton, 1928)

Spinal anesthesia has been gradually improved through the years by the efforts of various men. J. Leonard Corning (1885) of New York City is credited with being the first to launch the subject. He performed the first lumbar puncture on a dog. In 1885 he produced the first anesthesia by the intraspinal method. Bier (1899) reported six cases. He carried

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on an experiment on himself and his assistant and described the sensations. The method became popular for a short time in America and France where Tuffier was responsible for bringing the procedure into prominence. (Garside, 1931) From 1900 on spinal anesthesia was employed sporadically and its use was confined to elderly people when Pitkin (1928) first used it in 1912. These were the so-called bad risk cases.

After the World War there appeared occasional articles on the clinical aspects of the subject. Babcock (1925) and Bower, Wagoner and Clark (1926) were some of the contributors. The stimulus for various articles on the subject was given by Pitkin (1928) when he published his article on "controllable spinal anesthesia" in which he discussed the use of solutions lighter or heavier than spinal fluid. This also gave a stimulus for a wide spread use clinically. Its use in head, neck and thoracic surgery was discussed by Koster (1928); Evans (1928) delved into the technique and treatment of complications; Labat (1928) considered the importance of the Trendelenburg position. Other articles were published by Sise (1932); Severs and Waters (1932); Cotui and Standard (1932); Babcock (1928); Bower, Wagoner, Clark and Burns (1932), and many others.

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FALL IN BLOOD PRESSURE

Fall in blood pressure in spinal anesthesia has been the topic of much discussion with authors on this subject, especially during the past ten years. It has always been in the mind of the surgeon and has given him great concern. "The most dreaded of all complications of spinal anesthesia" is Evans' (1928) opinion of it. It is fairly well agreed that there is a slight fall in blood pressure, between 10 and 20 mm. Hg., following injection in spinal anesthesia. This should cause little concern, be compatible with safe anesthesia and is usually well born. (Aigner, 1926; Domenech, 1932) The latter author states that this hypotension is not associated with respiratory disorders.

The blood pressure may fall so low, however, as to produce nausea, vomiting, pallor of the skin, thirst and air hunger, cold sweats, and collapse. The pressure may fall to zero. The functions of the liver, kidneys and other organs may be greatly diminished. The patient may become unconscious with cessation of the heart beat. If the respirations are not allowed to cease and the heart beat is maintained, with proper treatment the blood pressure may be restored and the patient none the worse because of it. This type of fall has given the greatest concern and has been the object of experimental work through the years, more especially in the past eight or ten years.

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Several explanations have been offered for this fall in blood pressure. Dönitz (1903) and Klapp (1904) thought it due to absorption of the drug into the circulation with a toxic action. That the drop in blood pressure was due to direct action upon the nerve centers was the contention of Heineke and Laewen (1906). Tuffier and Hallion thought it was due to action on the cord or nerve roots. (Grodinsky and Baker, P.C.) Schilf and Ziegner (1924) stated it was due to action upon the preganglionic fibers of the thoracic region. Smith and Porter (1915) advanced the theory of action on the splanchnic nerves by the anesthetic solution to cause paralysis and a consequent fall in blood pressure. Stout (1929) agreed with this theory. Wislanski (1929) thought it due to congestion of blood vessels in the skin.

Experimental work in the year 1932 produced enlightening data. Bower, Wagoner, Clark and Burns stated the fall in blood pressure was not due largely to splanchnic dilatation. They thought that paralysis of the intercostal and phrenic nerves interfered with respiration causing venous stasis in the right heart and its tributaries due to loss of the sucking action of the chest; therefore, fall in blood pressure was the result of myocardial weakness. Severs and Waters found only a moderate alteration in blood pressure with anesthesia to the tenth dorsal segment. In high spinal block they found a progressive loss of vascular tone over the whole body and acute cardiac

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incompetence, both the result of oxygen deprivation as the sucking action of the chest is interfered with through intercostal and phrenic nerve paralysis. This produced a profound fall in the pressure. Domenech found that high spinal block caused a marked hypotension.

Grodinsky and Baker (P.C.) performed an experiment in which they carried on artificial respiration to prevent any possible myocardial weakness through interference with respiration and found the fall in blood pressure despite the precautions. "Out of this maze of evidence one fact stands forth, the effect on the blood pressure is due to local action upon the nerve roots, cord or medulla, and not to absorption into the circulation.....the usual fall in blood pressure must have been due to reduction in peripheral resistance, which means the anaesthesia must have affected the vasomotor system.....the medullary center or the pre-ganglionic sympathetic fibers."

If the idea that the fall in blood pressure is due to the effect on the vasomotor system is accepted, it is evident that the anesthetic solution should be confined below the midthoracic region of the cord for anaesthesia in that part of the body below the diaphragm. This is due to the fact that the vasoconstrictor fibers emerge from the first thoracic to the third lumbar segments of the cord (Starling, 1930), and the higher the anesthetic reaches in the thoracic

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region the more extensive the vasomotor paralysis and the greater the fall in blood pressure.

This fall is a gradual one when the effect is produced on the fibers below the medulla, but abrupt when the solution reaches the medullary centers. Ferguson and North (1932), Bower, Wagoner, Clark and Burns (1932), and Grodinsky and Baker (P.C.) agree in their conclusions that about one-third of the blood pressure change is due to dilatation in the splanchnic area, abdominal wall and lower extremities; and two-thirds to vasodilatation in the head, neck, upper extremities, thorax and its contents. "Whether or not there is agreement upon the percentage influence of regions mentioned upon the blood pressure, it seems quite clear that there is a cumulative action with upward progression and one must expect a greater fall in blood pressure with higher anesthesia." (Grodinsky and Baker, P.C.)

RESPIRATORY PARALYSIS

Another possible complication of spinal anesthesia is that of respiratory paralysis. It may occur immediately or follow very shortly upon the completion of the operation when the patient is raised to the horizontal position after being in the Trendelenburg position during the operation. (Evans, 1928; Poe, 1932) Severs and Waters (1932) state that respiratory paralysis is the usual cause of immediate death from spinal anesthesia.

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Various reasons have been assigned for the cause of respiratory paralysis. Jones (1931) attributes it to an overdosage of anesthetic solution in the blood and action then on the respiratory center. He agrees with Poe (1932) that preoperative medication with narcotics or other adjunct drugs may be a contributing factor. Bulbar anemia is advanced as a cause by Evans (1928) and Roeder (1932). The latter author includes a central or peripheral origin or a block of the motor nerves of thoracic respiration as possible causes. Ferguson and North (1932) speak of the dual control of respiration, the intercostal nerves and the phrenic nerves. If the former are paralyzed the latter carry on with an adequate diaphragmatic type of breathing. When the latter of the center itself are paralyzed, complete cessation of respiration occurs. Grodinsky and Baker (P.C.) agree that definite changes in respiration may result as outlined above by the injection of volumes of the anesthetic solution sufficient to reach the various levels. The majority of authors seem to favor the paralytic action of anesthetic solution upon the thoracic and cervical nerve roots and the respiratory center as the etiological factor in the production of respiratory paralysis.

In the production of this complication there are three mechanisms or combinations of them which may be involved. In the first instance there may be direct action on the

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respiratory center in the medulla by diffusion of the drug to the fourth ventricle. This type may be excluded from serious consideration as excessively high concentrations of the anesthetic solution are required to bring about this effect. Another mechanism is that of insufficient nutrient flow of blood through the respiratory centers in the medulla secondary to cardio-vascular depression which is, in some degree, attendant on intradural block involving the upper part of the abdomen and the thorax. Seever and Waters (1932) attribute the majority of cases of respiratory failure to this mechanism. A third mechanism concerns the ascending block of the intercostal and cervical nerves. The above authors think this type is unquestionable preventable, and secondary to inexperience in the technical aspects of the procedure; or to unsuccessful attempts to produce a selective sensory nerve block of the upper thoracic and neck regions. Grodinsky and Baker (P.C.) feel that the greatest safety in this type of anesthesia consists in confining the solution below the midthoracic region of the cord because of the effect upon the respiratory nerves and medullary centers. Evans (1928) states "true respiratory failure is due to high anesthesia and it will vary with the height and concentration of the anesthetic."

If the anesthetic solution be confined to the lower or lumbar regions of the spinal canal by the Trendelenburg

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position or through other means, the danger of this complication is practically nil. When upward diffusion takes place the thoracic nerve roots are affected and consequently the action of the intercostal muscles is lost. This throws the burden of respiration upon the diaphragm. If diffusion proceeds to the cervical region, because of involvement of the fifth, fourth and third cervical nerve roots, this unaided diaphragmatic breathing may cease such that tidal air cannot be moved. True respiratory paralysis has begun and the condition may be overlooked until far advanced. The patient may still be able to move the lips and tongue but be unable to talk. Cyanosis and unconsciousness may develop and the heart stop beating unless artificial respiration is begun. "This is the important complication of high anesthesia and may prove fatal unless the operator understands what is happening and is prepared to meet it." (Evans, 1928)

PULMONARY COMPLICATIONS

Pulmonary complications have not received attention in the same degree as other complications. Sise (1932) gives some idea of the incidence. He says that Elwyn found an incidence as low as 0.75% in operations on the extremities, while Pasteur found as high as 13.4% in operations on the stomach.

In the pulmonary complications the dominant factor is evidently in the operation itself and may well have a

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relationship to the changes produced in the lungs by operation. They are most conspicuous after upper abdominal operations. The changes, briefly, are as follows: The diaphragm is elevated; the circumference of the chest is increased; the tidal excursions are reduced to one-half their preoperative amounts and the maximum excursions to one-third. Before operation the tidal excursions are in the lower one-third of maximum and after operation in the upper one-third. X-rays in most cases show increased trunk shadows, haziness and mottling. Pictures taken during full inspiration after operation have much the same appearance as those taken during expiration before operation. Often there are temporary clinical signs as tubular breathing and rales. (Sise, 1932)

Brown (1931) found the greatest number of cases of post-operative pulmonary atelectasis in cases in which spinal anesthesia had been employed. He found few statistics available but his impression is definitely that the incidence 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.

Reasons that spinal anesthesia might predispose to this complication are: (1) There is decrease in the depth and force of the respiratory movements during the operation and

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for a considerable period afterward which retards the rid-
dance of foreign matter or secretion from the tracheobron-
chial tree. (2) The viscosity of the secretions of the
tracheobronchial tree appears to be increased; the material
is more tenacious following spinal anesthesia. (3) The
patient tends to remain relatively quiet for a number of
hours following operations with spinal anesthesia. The
subsequent development of atelectasis appears reasonable.

NAUSEA AND VOMITING

Evans (1928) states "nausea and vomiting occur only
where there have been errors or omissions in the proced-
ure.....there is no severe post-operative nausea and vomit-
ing after spinal anesthesia alone." When these symptoms
occur during a spinal anesthesia there may be several diffe-
rent causes and the majority of them may be eliminated.

During the first fifteen minutes there may be nausea
and vomiting from cerebral anemia or sudden moving of the
patient. It may be slight nausea or may go on to severe
vomiting. In another type the patient may develop certain
mental impressions which may be termed a psychic nausea.
These upsets commonly follow upon a lack of preoperative
sedation and poor psychology on the surgeon's part. The
patient's mind may be overactive with terrifying thoughts
racing through it and, in conjunction, the surgeon may ask

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thoughtless questions, all of which results in nausea and, possibly, vomiting. All may be proceeding well when the patient suddenly becomes extremely nauseated, vomits and extrudes the viscera through the abdominal incision. With sufficient preoperative sedation and an entertaining and cooperative anesthetist this complication may be eliminated.

In a third type of vomiting the term reflex may be applied. In such a situation, to an already existing cerebral anemia is added a disfunction which may be sufficient to start reflex vomiting. With thoraco-abdominal paralysis causing excessive action of the diaphragm in breathing, a sudden change in position of the patient, packing against the stomach and manipulation of the stomach and duodenum, all interfere with the normal function of the stomach and may lead to this type of vomiting.

As an accompaniment of the spinal puncture headache, post-operative nausea and vomiting may develop. It may be one of the symptoms, too, of meningeal irritation or infection. When the other conditions improve this symptom disappears. (Evans, 1928)

HEADACHE

The complication of headache is one that has produced a great deal of comment by the authors in their various

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articles. It has been termed the most frequent, troublesome and annoying complication of spinal anesthesia. (Koster, 1928; Evans, 1928; Koster and Weintrob, 1930) The incidence has been stated as low as ten per cent and as high as forty per cent. (Korgonova-Mueller, 1930; Koster and Weintrob, 1930)

The headache is usually either parietal or occipital in location; diffuse or localized and associated with some degree of stiffness of the neck muscles. In severe forms it may be lancinating or pulsatile pain accompanied by pains over the spine, vomiting and insomnia. The majority of patients afflicted begin to complain, as a rule, within twenty-four hours after the operation. The intensity of the headaches varies and in most instances they are of such mild degree that they disappear within twenty-four to seventy-two hours. The severer forms may persist as long as several weeks.

There have been various theories advanced as to the etiology of the headaches which follow spinal anesthesia. These theories are the toxic, circulatory, mixed, meningeal irritation and hydraulic. Chief among these, however, are the circulatory, meningeal irritation and hydraulic. The supporters of the circulatory theory include such authors as Angelesco, Buzianu and Caramzulesco (1932). In this type the vascular mechanism is, as yet, imperfectly determined. Changes in the normal tonus of the blood

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vessels may produce disturbances of blood circulation in the brain. Vasomotor paresis may cause dilatation of the capillaries of the dura mater and compress the sensory nerve filaments to produce headache. In addition to the first theory the above-mentioned authors with Kennedy (1932); Case (1932); De Courcy (1928) and Evans (1928) support the second theory. Meningeal irritation is produced by trauma because of repeated punctures or through the use of too large needles. The supporters of the third theory, Anderson (1931); Case (1928); De Courcy (1928); Pitkin (1928) and Koester and Weintrob (1930), postulate an increase or decrease in the amount of spinal fluid following the spinal puncture. In the event of an increase there are the signs and symptoms of a meningitis or meningismus; stiffness of neck, photophobia, headache and a possible paralysis of the external rectus muscles of the eye. With a decrease in the amount of spinal fluid the brain may sag against the bony framework of the skull resulting in irritation and a consequent headache. Pressure upon the basilar venous plexus may result in increased venous tension and result in headache.

If the headache is of the type that results from an increase in the amount of the spinal fluid, no relief is obtained by lowering the head and shoulders. If of the type in which there is decrease, no drugs give relief but lowering the head and shoulders give relief. (Evans, 1928)

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MENINGITIS

Meningitis, as a complication of spinal anesthesia, is one dependant upon the skill and command of technique of the operator, making its appearance in that order. The cases reported are usually of a mild type. (Evans, 1928; Hertz, 1922) Meningismus, a non-infective meningitis, is essentially an irritation of the meninges. Trauma to the dura, puncture of a dural vessel, injection of toxic doses of the drug or of foreign material may result in a congestion of the meninges with a resultant exudation. Within twelve to twenty-four hours the meningismus is usually ushered in with meningeal symptoms such as headache, dizziness, stiffness of neck and photophobia. These usually persist for two to three days and then subside spontaneously. No relief is obtained by the administration of coal tar derivatives or codein. (Evans, 1928; Koster and Weintrob, 1930)

The true purulent variety of meningitis is a rarity. The appearance of this type of meningitis depends again on the technique and skill of the operator, but it is the same risk that accompanies any surgical procedure. Within the first twenty-four to forty-eight hours the symptoms usually appear. Headache and restlessness are usually attended by stiff neck, increased temperature and ocular aberrations. Then the characteristic signs of purulent meningitis

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become evident. The course is usually ten to fifteen days, after which it tends to clear up spontaneously with no residual. (Anderson, 1931; Koster and Weintrob, 1930; Evans, 1928)

NEURITIS AND PALSIES

The after-effects of spinal anesthesia in regard to the possibility of a complicating neuritis and palsy are evidently quite rare and fortunately, if they occur, only transient in nature and of short duration. (Koster and Weintrob, 1930; De Courcy, 1928; Evans, 1928; Anderson, 1931) There are two types generally recognized, those occurring because of irritation or trauma at the site of puncture and those further removed, the ocular palsies.

In the first type the motor or sensory disturbances may be due to trauma to the nerve roots, spinal or cauda equina; through repeated attempts to puncture or through the use of too large needles. (De Courcy, 1928; Babcock, 1928) The symptoms may be sharp, lancinating pains along the course of the nerve, muscle spasm, anaesthesia or paresthesias of the lower extremities, or irritation to the anal or vesical sphincters. If the damage is severe the resulting disability may be quite permanent. Interruption in the continuity of the nerve roots results in permanent damage. Puncture of the cord usually causes neither pain or after-effects. (Evans, 1928)

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The later palsies are confined to the extra-ocular muscles and, of these, the external rectus, which is supplied by the abducens or sixth cranial nerve, is the one commonly affected. The condition may be unilateral, bilateral and as frequently in either eye. (Koster and Weintrob, 1930, Fawcett, 1931; Evans, 1928; Anderson, 1931) The more important theories concerning the particular involvement of abducens nerve are two in number. The first theory assigns it to a special susceptibility to the drug. The second refers to the longer passage of the abducens nerve through the spinal fluid, in which it suffers more from exposure to the drug or spinal fluid contaminations than do the other cranial nerves.

The symptoms are usually a preliminary photophobia, possibly headache and dizziness, and a supervening diplopia which is usually homonymous. Outward motion is limited and there may be a convergent squint. After about ten days the condition usually shows signs of beginning cure, and generally clears up spontaneously in a month or two. (Koster and Weintrob, 1930; Evans, 1928; Fawcett, 1931; Bufill, 1930)

Backache occurs in a small percentage of cases. Koster and Weintrob (1930) found a two per cent incidence. Trauma to the soft parts or strains or bruises, at the time when the patient is unable to react to a cramped or uncomfortable

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position may be the cause. The duration is short, sometimes a week. Relief may be obtained by placing a pillow under the back.

PSYCHIC DISTURBANCES

Very little consideration is given to psychic disturbances in the literature. This leads to the conclusion that such a complication is rare. Koster and Weintrob (1930) report a case by Izquierdo which developed into a hypochondriac. From the same source there is a report of a case of insanity. Dassen (1930) had a case of grave neuro-psychosis after eleven days. It was characterized by mental confusion. The patient was inactive for four months. Dassen considered a toxic influence was the most probable cause of the disturbance.

CONCLUSION

Although spinal anesthesia has been employed for over three decades, it has been only within the past few years that much has been accomplished with it. The complications which followed in its wake made many surgeons cautious and fearful of its use. Extreme fall in blood pressure and respiratory paralysis are rarely encountered, but when they do occur may readily result fatally. They are to be feared and avoided if possible. The other complications are troublesome and annoying, but seldom result in death, and may be avoided

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with proper use of this type of anesthetic, and by using proper technique.

With the knowledge at hand from experimental work and experience of the years to guide, the surgeon is now better informed and prepared to meet the complications should the occasion arise. It may be seen that spinal anesthesia has its limitations, and should be used only in that part of the body below the mid-thoracic region of the spinal cord. If such procedure be followed there need be little concern about complications following the use of spinal anesthesia. It should then follow that much of the fear of the patient will be dispelled. Knowing that it is not more dangerous than any other form of anesthesia used in its proper place, he will have more confidence in its use when such is indicated.

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CASE REPORTS

Case I. Respiratory failure and fall in blood pressure.

The following case illustrates the dangers of high anesthesia. Mrs. S., age 70. Interposition operation for cystocele and uterine prolapse. Spinal anesthesia by means of 300 mg. neocaine dissolved in 1 c.c. of spinal fluid, and injected into the first lumbar interspace. The patient was immediately placed in the dorsal lithotomy position with the head lowered. The blood pressure before injection was 160 systolic and 90 diastolic. Ten minutes after injection it had fallen to 80 systolic and 50 diastolic, but the patient's general condition was good. The respirations were quite normal except for the fact that they were more diaphragmatic in character. Pin tests showed that cutaneous anesthesia had reached the first thoracic segment. Ten minutes later the blood pressure was about the same, 75/45, but the cutaneous anesthesia had reached to the first cervical segment, and the respirations were slower and more shallow. Five minutes after this the respirations suddenly stopped. The patient became cyanotic and the blood pressure could not be recorded. The pulse was feeble and slow, and soon could not be gotten at all. The heart sounds could not be heard. Artificial respiration was immediately instituted. Strychnine sulphate gr.1/30 and alpha-lobelin gr.1/20 were given hypodermically; adrenalin, 1c.c., was injected directly into the heart. Several minutes later the heart sounds could be made out, and a feeble pulse could be felt. After about fifteen minutes of artificial respiration the patient took a few spontaneous breaths which gradually increased in rate and depth until her condition was normal again.

A careful study of this case shows the typical progressive effects upon the respiratory mechanism seen in the laboratory experiments. (Grodinsky and Baker, P.C.) First there is a change to abdominal breathing to compensate for loss of the intercostals; then interference with the abdominal breathing by paralysis of the cervical segments (phrenics), and even possible effect upon the medullary centers (though this would be relatively unimportant since all the peripheral respiratory nerves were already affected). It is to be noted that there was an early drop in blood pressure (50%) but that this was compatible with good respiration (though modified), and good general condition, showing the independence of changes in blood pressure and respiration. Furthermore, the blood pressure remained about the same until the respirations ceased entirely when it became imperceptible due to the anoxemia of the medulla, or possibly to direct

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action of novocaine upon the vasoconstrictor as well as the respiratory centers. Until that time, however, the blood pressure had reached its maximum fall due to peripheral vasodilatation, and this correlated well with the level of cutaneous anesthesia, to and above the first thoracic segment. That the head was already down when the respiratory difficulties began, gives further evidence that the latter was not due to anemia of the medulla, resulting from fallen blood pressure. It is interesting to note that, although there was some fixation of novocaine early, this was only partial, and the effect continued to spread upward for twenty to twenty-five minutes after injection. The recovery of this patient must be ascribed to the artificial respiration instituted, since the essential cause of the reaction was respiratory failure, and restoration of breathing was all important for recovery. However, the use of stimulants may have had some additional favorable effect. Adrenalin may have stimulated the waning circulation particularly. (Grodinsky and Baker, P.C.)

Case II. Ocular Complications.

Bilateral paralysis of the external recti (sixth pair). This patient came under observation in July of 1926, with a history of having been operated upon under spinal anesthesia a month and a half previously. The diplopia supervened a fortnight after the operation, following a series of general disturbances and severe headache. During the past month, he had undergone various treatments without any result and was now referred to the author by his family physician. Examination showed a well marked, although not absolute, paralysis of both external recti muscles; vision was normal and the ocular fundus was normal. As the only treatment he was given potassium iodide and galvanic and faradic currents, with the result that after three months of treatment, he was completely cured. (Eufill, 1930)

Case III. Headache.

The patient was a woman 28 years of age, who was curetted on July 1st for placental retention. She was given spinal anesthesia with syncaïne without removal of the cerebrospinal fluid. Her temperature immediately

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descended but on the next evening she began to complain of headache in the neck and frontal regions. This headache was increased in severity when she sat up, and was practically continuous without any sharp paroxysms during the day or night. It was accompanied by vomiting. She could not sleep.

On the evening of July 3rd she still complained of headache and in addition had marked stiffness of the neck with photophobia but without Kernig's sign. A lumbar puncture was made and a few drops of clear fluid were obtained. The fluid followed only when she coughed or talked. There was evident hypotension but she seemed relieved and passed a good night.

The headache and vomiting reappeared on July 4th. She was much better on July 6th and was able to sleep. However, on the 7th the headache returned and was almost continuous, although it did not prevent her from sleeping.

The headache returned about 10 A.M. on the morning of the 8th with the same characteristics. She had a sensation of general fatigue and the neurologic examination showed slight stiffness of the neck, active tendinous reflexes, and photophobia but no ocular paralysis. Another lumbar puncture was made but only 1 or 2 drops of a clear fluid were obtained. She was given a subcutaneous injection of 250 c.c. of salt solution. She passed a good night and the headache, stiffness of the neck and photophobia completely disappeared on the next day. (Wideroe and Dahlstrom, 1922)

Case IV. Headache.

The patient was a man 28 years of age, who was operated upon for a bilateral hallux valgus. The operation was performed on September 3rd. The cerebrospinal fluid escaped in a jet.

About 11 A.M. on the second day the patient had slight headache. The headache increased in severity but he was able to rest. The headache returned on the morning of the 5th and 10 c.c. of distilled water was injected intravenously. The pain seemed to be increased when he sat up. On the morning of the 6th there was contracture of the neck but no Kernig's sign. His pulse was 70 and he had no fever. A lumbar puncture was made and only a few drops of normal fluid could be obtained. He was

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given theobromine but the headache became much worse in the evening.

The occipital headache was continuous and there were paroxysms without any apparent cause. There was no vomiting. Percussion in the occipital region was painful. He had rather marked stiffness of the neck, but no paralysis or nystagmus. There were no disorders of sensation. His pupils were slightly dilated but were equal and reacted to light. Another injection of 30 c.c. of distilled water was given intravenously without any immediate improvement. The theobromine was continued and was so severe that morphine was necessary. He was much better on the 9th and was able to eat. Chloral hydrate and bromides were given in small doses for 48 hours. He became completely well on the 12th. (Wideroe and Dahlstrom, 1922)

Case V. Paralysis of lower extremities.

The case concerns a woman who was operated for cystoma. Lumbar anesthesia with 0.15 novocain and 5 drops of 1:1000 suprarenin solution. Further course was uneventful, and the patient was discharged on Sept. 10. End of December, pains manifested themselves in the lumbar portion of the spine. January, paresthesia in both legs. Up to June, a gradual paralysis of the legs developed. June 20th, normal childbirth. Findings a month later reveal a severe paresis of the flexor muscles of the legs with loss of position-muscle-and-touch-sense. A combination cord disease of the pyramidal tract and of the posterior tracts was assumed. Whether it was genuine or whether it was a case of a diffuse inflammatory process cannot be determined, as the autopsy findings were not complete.

The author considered pregnancy myelitis as becoming steadily less frequent. Some such cases have revealed themselves as multiple sclerosis. In those cases in the literature in which exact autopsy findings are available, a circumscribed or diffuse inflammatory disease was always present, never a systemic disease. (Muller, 1921)

COMPLICATIONS OF SPINAL ANESTHESIA.

Case VI. Vesical and anal sphincter involvement.

A man 59 years old consulted the author for a rectal disorder which he believed was due to hypertrophy of the prostate. He had an inguinal hernia which was operated upon by means of spinal anesthesia. The operation was painless at first but later became painful and he had a violent reaction in 12 hours. His nervous condition was such that on the third day he had a syncope lasting three hours. He was not able to urinate after the operation and catheterization was necessary. He had no appetite for 8 days and when he began to eat he had diarrhea. His surgeon believed that he had a relaxation of the sphincter and a contraction of the vesical sphincter.

The patient could not sleep and he had pains and cramps in his legs. There was absolute loss of sensation in the entire lumbar and genital regions and the muscles of the cortex were hard on palpation. An attempt was made to re-establish the functions by means of electricity at the end of the third week. A specialist found inertia of the anal and vesical sphincters. There was no improvement after treatment for one month. Since that time the patient has had constipation and has been obliged to use laxatives constantly. He has had urinary disorders constantly and his urine has been cloudy and alkaline.

A rectal examination showed a small prostate and the bladder appeared to be empty. There was loss of sensation of the scrotum. The patella reflexes were sluggish. Since the operation he has always had a sensation of cold feet. He has no spontaneous desire to urinate and his rectum always has the sensation of being full.

Hence, there may be complications due to spinal anesthesia which may persist for years. (Bazy, 1927)

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