diminished. This fact may in part account for our lack of successful results in some cases. In the permanent apparatus now under consideration measures are being taken to overcome this difficulty. The results obtained in those cases (for example, intra-abdominal or mammary or cutaneous neoplasms) in which the lesion is accessible to the full intensity have been so much more favourable than in those in which, either for anatomical reasons or on account of the patient's comfort, the distance has had to be increased, that it would appear that a smaller apparatus is necessary for dealing with some lesions.

The "bomb" is in use continuously for twenty-four hours daily for six days each week. During the day out-patients are treated, and during the night in-patients, the average time given to each case being two hours. It has been found that most patients are very nervous during their first period of treatment, but that on the second and subsequent occasions they are quite reassured and comfortable. The majority of our patients sleep quite peacefully, and say that their sense of comfort and well-being is increased while they are on the "bomb" and for some time afterwards. Thus it is no great hardship for the patients to leave their beds in the wards and go to the "bomb" at night. Two patients have even said—and it has been confirmed—that they slept better while having their treatment than when in bed in the ward.

When the patient is put on the stretcher the "bomb" is adjusted as near as possible to the correct focal distance at the port of entry decided upon, the patient is made comfortable, and in the majority of cases falls asleep in a short time. The ports of entry are changed on alternate days, and in this way not only is the danger—and this is apparently very slight—of damage to the skin minimized, but the intensity at the growth is increased.

Periodical examinations of the blood are carried out, as are tests of liver function and other biological tests, and in this way the effects of irradiation on the organism as a whole are controlled. A register is kept and discussed at a weekly meeting of the staff, at which the past week's results are examined and the programme for the succeeding week is decided upon.

SPINOCAIN: THE CONTROLLABLE SPINAL ANAESTHETIC.*

BY

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To discuss the clinical features of spinocain would seem somewhat far-fetched. Dr. Frank A. Kelly of the Grace Hospital, Detroit, Michigan, has worked with the solution from its early experimental days, and he, I believe, will gladly enlighten you as to its clinical advantages. With your permission I will endeavour to enumerate some of the theories we had concerning spinal anaesthesia and explain the reasons for our conclusions.

Since the introduction of painless surgery our dreams have been an ideal anaesthetic—one that would abolish pain completely, be reliable and certain in its action, constant in duration and intensity in direct ratio to the amount employed; one that would not alter the normal secretions of the body; one that would not affect respiration and circulation; one that would be free from all complications brought about by the action of inhalation anaesthetics, such as pulmonary oedema, bronchitis, pneumonia, lung abscess, acidosis, distension, ileus, acute circulatory failures, etc.; one that would not materially disturb the central nervous system. Such an anaesthetic should essentially be non-toxic, be simple of administration, lessen morbidity, and abolish anaesthetic mortality altogether. However, such an anaesthetic is still a dream. The reality is our present-day anaesthesia, the deleterious effects of which are an ever-growing concern of thoughtful surgeons.

The short time allotted to this paper does not permit going into the problem of the causes of anaesthetic morbidity and mortality. It should be pointed out, however, that the problem is a part of the great problem complex of cardio-respiratory physiology and pathology, that has remained largely unsolved to our very days.

However, we know that it is through their interference with the normal cardio-respiratory correlation that anaesthetics cause their ill effects. We see the phenomena of cardio-respiratory decompensation ensue as soon as the anaesthetic action reaches and depresses higher central nervous elements regulating respiration and circulation, regardless of whether the anaesthetic was inhaled through the lungs or injected into the subarachnoid space in a manner enabling it to diffuse in a cephalic direction. The respiratory embarrassment as manifested then in the degree of anoxaemia, or the circulatory collapse as manifested in the degree of hypotension, is in both cases directly proportionate to the action of the anaesthetic. The functions involve central nervous mechanisms upon which the effect of the anaesthetic varies greatly even from one individual to another; it is therefore beyond our power to estimate or control. In this lack of control lies the source of all complications of anaesthesia, caused by the anaesthetic, regardless of whether inhaled or injected. The action of the anaesthetic depends on its diffusion. To control diffusion of the anaesthetic thus becomes the paramount goal of all anaesthetists.

The fatalities of respiratory embarrassment or circulatory collapse at the time of operation, are not to be compared with the secondary mortality, which is 99 per cent. of the whole as compared to a scant 1 per cent. of a primary mortality. How many anaesthetists or surgeons can truthfully say that they have had 100 consecutive cases without some post-anaesthetic complication, such as shock, bronchitis, pneumonia, lung abscess, distension, ileus, acute cardiac or gastric dilatation, albuminuria, suppression, or acidosis; many or all of which may be directly attributable to a partial or impaired respiratory or circulatory dysfunction?

The surest method of eliminating anaesthetic complications is by the use of local or conduction anaesthesia. Unfortunately its art is not familiar to all of us, nor is the method adaptable for all operative procedures. But, with a combination of these and controllable spinal anaesthesia, any operative procedure may be carried out with greater protection to the patient and better facilities to the surgeon. To eliminate the possibility of respiratory and circulatory disturbance, permit me to suggest a spinal anaesthetic that will not diffuse with the spinal fluid, one that can, with the proper technique, be placed under the direct control of the operator.

As we have known spinal anaesthesia in the past it produces hypotension. In fact, so great is the circulatory depression that one well-known author in America states that "the radial pulse cannot be felt by the finger, nor can it be detected by the ear when the arm is subjected to the pressure of the cuff of the sphygmomanometer." This extreme drop in the blood pressure is due to diffusion of the anaesthetic fluid cephalically. If the anaesthetic fluid is confined to the lumbar spine there is no change in the blood pressure; if it reaches the first thoracic the pressure becomes *nil*. If the nerves controlling the diaphragm and intercostal muscles are anaesthetized, respiratory embarrassment arises. If it reaches still higher levels the sympathetic cardiac accelerators are anaesthetized, and unrestricted vagus action slows down the heart. Extreme pallor, nausea, vomiting, a cold clammy sweat, a feeling of pressure over the precordium, thirst, air-hunger, and the fear of impending danger are the alarming symptoms such diffusion of the anaesthetic causes.

With an endeavour to overcome any or all of these objectionable symptoms of an otherwise ideal anaesthetic a series of experiments were conducted to ascertain the physiology and the physiological action of the various drugs advocated for spinal anaesthesia. We attempted to bring the anaesthetic under the control of the operator, to prevent uncertainty as to the parts involved, to stabilize the intensity and regulate the time of anaesthesia, to diminish the tremendous drop in blood pressure, to minimize nausea and vomiting, to abolish cold sweats and

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anxiety of the patient, to eliminate air-hunger and cardiac pains, to solve the mystery of post-anaesthetic headaches, to prevent localized peristalsis and systemic disturbances.

We set out to produce a spinal anaesthetic that would minimize the toxic reactions, be certain in effect, constant in duration, act in direct ratio to the amount of anaesthetic agent injected, would not in any way affect the heart action, vascular tension, or respiratory function; one that would lessen nausea and vomiting, abolish pallor and cold sweats, that would eliminate the anxiety of the patients as well as systemic disturbances and after-effects; one that would restore, if possible, the confidence of the surgeon to this form of anaesthesia. A new anaesthetic compound had to be devised and the technique so modified that the procedure could be carried out almost with the exactness of the micrometer, if the desired results were to be obtained. Some of the experiments and the reasons for our deductions in devising spinocain may be of interest.

To obviate anaesthesia—temporary paralysis of the thoracic voluntary nerves and sympathetic chain—diffusion of the anaesthetic within the subarachnoid space must necessarily be prevented. Our experiments were carried out with tubes shaped to conform to the human spine. In the larger tubes we employed an isotonic solution, in the smaller tubes with a capacity of 40 min. human spinal fluid was used. In these tubes we studied the action of various viscid substances in an endeavour to secure a solution that would mix but not diffuse with spinal fluid, that would remain *en masse* until the anaesthetic drug had become fixed or absorbed. A substance that would not inhibit anaesthesia but regulate its durability and intensify its action had to be found, always bearing in mind the oscillatory action of spinal fluid within the dural sac due to vascular pulsations, and the external physical disturbances, such as are caused by respiration, coughing, motion of the body, straining, vomiting, etc.

To simulate the first, a clock-like arrangement was devised that would pulsate against a rubber cap; to produce the action of the latter disturbances, a bulb arrangement was attached to the tubes as well as an oscillating device. The first would increase or decrease the inter-spinal pressure in the tubes from 1 to 8 mm. of mercury; the slight oscillation compensated for the motions of the body. To prevent diffusion we employed many viscous agents, some of which had been advocated before. But, by the use of a solution of starch paste, we were enabled to prevent dissemination for forty minutes. This solution, even in sealed ampoules, could only be used when fresh. It would ferment, precipitate, and become unstable within six to eight weeks. Later we discovered that gliadin (the mucilaginous content of wheat starch) was the substance that really produced the viscid properties desired. This is relatively insoluble, being soluble only in certain alcoholic solutions. If the alcoholic content is increased or diminished it will precipitate out of the solution in crystalline masses. From gliadin we were able to produce a jelly-like gelatinous substance that we chose to call amyloprolamin, which was stable, soluble, prevented dissemination for over two hours, or until the anaesthetic drug was spent. This was necessary because we observed that by dissemination any anaesthetic solution became weakened by dilution. This accounted in a great part for the uncertainty of duration and variation of intensity of various methods formerly employed in spinal anaesthesia.

To further check the action of the solution within the subarachnoid space and to determine whether a definite and fixed volume could be maintained throughout the duration of the anaesthesia, to prove that dissemination would not occur in a cephalic direction, we coloured the solution with methylene blue and tapped human spines from twenty minutes to one hour after the original injection. We obtained no coloured solution from the sixth or seventh thoracic interspace. Its action was further checked by adding stains to the solution, injecting it into the dural sac of dogs, and noting the stained regions of the cord and arachnoid in necropsy specimens.

To enable us to control the anaesthetic so as to bathe only the spinal nerves desired, it became necessary to determine the capacity of the dural sac at various heights in adults and children of various ages. This was accom-

plished by colouring the spinocain with methylene blue and making taps along the spine until we obtained clear fluid. A further check was made by adding eosin to the light and heavy solutions and injecting these into cadavers, the body being maintained in a position similar to that of the patient on the operating table. The solution was found to expand in a like manner. The results were observed by performing a laminectomy. We added sodium iodide to a solution of the same specific gravity and viscosity as spinocain and injected this into the spinal canal of cadavers and radiographed them. The results verified our other findings. With the dural sac capacity determined it was easy to anaesthetize various pairs of spinal nerves at will, by expanding the solutions as has been described in the technique.

The toxicity of novocain injected intradurally is practically the same as when introduced intravenously. When 300 mg. of normal saline were injected into dogs weighing 9 kg. (20 lb.) convulsions occurred, followed by death. If liquid alboline or olive oil was employed as a solvent for the novocain before it was dissolved and injected into the subarachnoid space, satisfactory anaesthesia was obtained. Although it was delayed, the duration was extended, and by elevating the hips of the dog we were enabled to limit anaesthesia to the legs only. Anaesthesia appeared to be more intense, and with the oil preparation convulsions did not occur in the six dogs injected. There were no toxic reactions when as much as 750 mg. of novocain were employed. The viscosity of the amyloprolamin solution acts similarly to the oils, and materially reduces the toxicity of novocain within the subarachnoid space. This is well exemplified by the introduction of a sufficient amount of spinocain to be equivalent to 1,200 mg. of novocain into a patient weighing 135 lb., when no toxic symptoms or vascular relaxation resulted. It prolongs the time of absorption and extends the duration of anaesthesia. When novocain is dissolved in normal saline or spinal fluid, 120 mg. is considered a maximum dose, but with the use of spinocain we do not hesitate to use amounts equivalent to 400 or even 600 mg.

The selection of novocain as the most desirable anaesthetic agent resulted from a study of the physiological action of the drugs ordinarily used in spinal anaesthesia. Stovaine produces the most intense anaesthesia—a complete and absolute transitory paralysis and anaesthesia of not only the sensory nerves, but also the motor nerves, the tactile and thermo sensation, the sphincters and the vasomotors of the anaesthetized field. It may, if not acidified, produce incomplete anaesthesia. The alkalinity of the spinal fluid often causes the stovaine to crystallize out of the solution injected into almost insoluble particles. Traces of stovaine have been found in the spinal fluid twenty-six hours after injection. As the anaesthetic is subsiding it often produces severe pains in the lower limbs; headaches of a hypertonic nature occasionally follow its use. Tropicocaine produces anaesthesia not unlike stovaine, with perhaps a slightly lessened action of the vasomotors. Apthesin gives a perfect anaesthesia and complete relaxation. Tactile sensation is not constantly impaired. Primarily there is very little vasomotor disturbance, its absorbability is slow or retarded. Secondly, and in one or two hours after injection, it produces a severe vasomotor disturbance. Cases have been observed without more than a ten to fifteen points drop in blood pressure within forty minutes. Two hours after the injection the pressure had dropped to zero in many cases. This secondary action does not occur with any other drug but butyn. At times it is impossible to raise the patient's head for twenty-four hours after administration. Apthesin, stovaine, and butyn have been considered too dangerous to use, and we have abandoned them for this reason. Novocain gives a complete anaesthesia of the sensory nerves, satisfactory relaxation, and affects the tactile and muscular sensation only slightly. It affects the vasomotors less than any other drug used. Thermo-sensation disappears with all drugs. Occasionally the motor nerves are not involved with novocain, but this is not constant.

It is interesting to note that a 100 per cent. solution of novocain-amyloprolamin solution, used to produce intra-

dural sacral anaesthesia, caused no appreciable increase in the cell count of the spinal fluid. But the acid anaesthetics, as apothosin and stovaine, produce a marked increase in the number of cells, and not infrequently we have observed 15, 20, and as many as 25 cells to the cubic millimetre after a 5 per cent. solution of the latter had been injected. The anaesthetic agents with an acid reaction increase also the intradural pressure.

A transitory or prolonged paralysis of the hind legs was produced in 30 per cent. of our dogs after a 5 per cent. stovaine solution had been introduced into the subarachnoid space. This paralysis lasted from six hours to nine days. All cases in which stovaine was employed showed a perivascular round-cell infiltration, and by the Cajal method of nerve staining we found the axis cylinders swollen, marked degeneration of the fibres of the anterior roots, and in a dog that was paralysed for five days the sciatic nerve showed marked degeneration of the nerve fibres. On the other hand, novocain in 5, 10, and 20 per cent. solutions failed to produce any macroscopic or microscopic changes.

The absorption time of various anaesthetic agents from the spinal fluid shows great variations. Traces of stovaine have been found in the spinal fluid twenty-six hours after introduction; whereas all traces of novocain had disappeared in innumerable cases in four hours, and in all cases in six hours. In mentioning novocain, I include neocaine and procaine, as they are all nearly identical, the only variation being in the manufacture of the product and not in the primary formula.

Deductions led us to believe that novocain is absorbed by the nerve trunks, and enters the system in this way, rather than through the veins or lymphatics by osmosis. As has been shown, the osmotic rate between spinal fluid and the venous blood is very slow, due to the similarity of intravenous and intraspinal pressure. With the patient in a reclining position the former is 3 to 4 mm. Hg. and the latter is 6 to 8 mm. Hg. This may also account for the fact that if a vein is accidentally punctured during a spinal tap, bleeding does not occur into the spinal canal, but there is a possibility of seepage of spinal fluid into the vein. Possibly this accounts for the frequent hypotonic headaches, as these headaches are observed more frequently after blood enters the spinal needle or after bungling attempts at a spinal tap.

Intraspinal pressure with the patient in a sitting posture is greater than when the patient is recumbent, but there is no difference between the pressure of a recumbent patient and one in a 45 degree Trendelenburg position. This may be accounted for by the cerebral congestion produced when the patient is placed in a position of so marked a degree. This fact is observed if the spinal manometer is permitted to remain attached to the needle intact, when the patient is recumbent, and then placed immediately in a 45 degree Trendelenburg position. A slight remission in the pressure is seen, which almost at once assumes its original reading. We have observed that headaches are more likely to occur when the spinal tap is made with the patient sitting, as with this method a greater amount of spinal fluid is permitted to escape. This is also true when large needles of 15 to 17 gauge are employed. They permit seepage through the dura into the surrounding tissues, and not infrequently cause post-operative backaches, either as a result of the oedema by the spinal fluid or trauma of the tissues. A long tapering point is likely to produce more bleeding than the short bevelled fine-gauge needle.

We conducted a series of tests and animal experiments with all the drugs that have been advocated as vaso-constrictors. Strychnine, intradurally, was the only one that showed satisfactory results in the presence of novocain. These tests and animal experiments were not only in relation to intradural circulation, but the drugs were introduced intravenously, intramuscularly, and subcutaneously. Of the so-called stimulants, strychnine is the only one with any definite value, with the exception of ephedrine and suprarenin. Ephedrine and suprarenin combined, and introduced intravenously, proved the most effective as well as lasting. Suprarenin has little or no value when injected into the muscles, where it produces a localized ischaemia which delays absorption. These experiments enabled us to

establish a ratio of the approximate amount of ephedrine required for the stabilization of definite amounts of novocain, with anaesthesia at various heights on the body surface. We also learned how much ephedrine-suprarenin mixture should be used to cause a hypotension case to approach normal and maintain a constant pressure for from four to six hours, as well as how to gauge the dosage of ephedrine in hypertension cases. Given a case of hypotension of 67/42, 50 mg. of ephedrine and 0.7 mg. of suprarenin introduced intravenously will increase the pressure to approximately 120/70, which will be maintained for at least four hours from the time of injection, during which time one can proceed as with a normal case. A hypertension case of 200/110 pressure, however, should not have over one-half the amount of ephedrine that would ordinarily be used in a case with a pressure of 120/70, or 25 mg. of ephedrine.

Bearing in mind that the vaso-constrictors leave the cord by way of the anterior nerve routes from the second dorsal to the third lumbar vertebra, one must estimate the amount of ephedrine necessary to maintain a constant pressure. With intradural sacral anaesthesia none is required. When the lumbar nerves are bathed in spinocain, with anaesthesia of the legs, 25 mg. should be used. If the anaesthetic is to be carried to the ninth dorsal for anaesthesia up to the umbilicus, 50 mg. should be employed. If the anaesthesia is to expand to the sixth dorsal and anaesthesia to the unciform process, 60 to 100 mg. of ephedrine will be necessary. Strychnine intradurally stimulates the vaso-motor constrictors, but will have little or no effect if injected intravenously or intramuscularly. Ephedrine will fortify the anaesthetized nerves, stimulate the unaffected vaso-constrictors, and if administered before the spinocain will stabilize the arterial pressure. Ephedrine should fortify the vasomotors against the attack of the anaesthetic. To obtain the best results it must be injected before the spinal tap is made. The result will be disappointing if used after the pressure has fallen, and the vaso-constrictors have become anaesthetized, as it will not re-establish a normal pressure if employed at this time. We should then resort to an intravenous injection of 50 to 60 mg. of ephedrine and 0.7 mg. of suprarenin combined.

There has been considerable discussion as to the toxicity of ephedrine. Two of my colleagues have reported deaths which they attributed to the administration of 50 mg. doses. There must have been some error in their conclusions. With an effort to estimate the maximum safe dose we used a 1 in 1,000 solution in rats weighing 100 grams, and by transposing our findings in ratio to a human being weighing 72 kilograms (160 lb.) the lethal dose was estimated at 13,200 c.c.m., or 13 grams. The maximum safe dose, according to Solis-Cohen and Gilhens, is 5 grams.

As a result of these studies and reports on 1,100 primary inductions (the total is now 160,000) with controllable spinal anaesthesia—performed by surgeons in every State in the Union and several foreign countries—we feel justified in asserting that spinocain is controllable within the dural sac. The physiological action of spinocain is quite different from that of any other spinal anaesthetic, inasmuch as nearly all of the objectionable features of the others are eliminated. When local conduction or spinal anaesthesia is used, the paramount issue at all times is never to hurt the patient in any way. The patient once hurt may lose confidence in the method and in the operator. A lost confidence may be very hard to regain, and frequently constitutes the direct cause of unsuccessful anaesthesia. A patient who is immediately subjected to one painful manipulation becomes hypersensitive to every following procedure. In spinal anaesthesia with novocain it must be remembered that tactile sensation is not abolished. All the more reason why every precaution should be taken, and the technique employed with such finesse that spinal anaesthesia may be administered without producing any pain. With an entirely satisfactory anaesthesia the patient should be cheerful and comfortable throughout the operative procedures.

To enable us to carry out this technique with as few manipulations as possible, and to place at our disposal solutions that could be controlled within the subarachnoid space, we devised the ephedrine-novocain solution, and

the light and heavy spinocain. The ephedrine-novocain ampoule consists of ephedrine 50 mg. and novocain 10 mg. The light solution of spinocain has a specific gravity of 1.0005. It contains: strychnine 2 mg., novocain 200 mg., amyloprolamin 130 mg., ethyl hydrate 300 mg., normal saline q.s. 2 c.cm. The heavy solution has a specific gravity of 1.109, and contains: strychnine sulphate 2 mg., novocain 200 mg., amyloprolamin 130 mg., propantriol 100 mg., and normal saline q.s. 0.5 c.cm.

The heavy solution should always be given with the patient sitting or in a marked Fowler's position. After administration patients should never be placed in a Trendelenburg position; they should, if possible, be maintained in Fowler's position for at least two hours. If for any reason it becomes necessary they may be placed flat for from fifteen to twenty minutes after the anaesthetic has become fixed.

It would hardly seem necessary to repeat the details of technique for the use of spinocain anaesthesia, as that was described in the October issue of the *British Journal of Anaesthesia*. The method of using the heavy solution is again explained by the author in the current July issue; also in an article by Dr. Frank A. Kelly. There are, however, a few points that should be emphasized as essential to the perfection of this form of anaesthesia. The needles used should be of small calibre, 22 gauge, with a short tapered point, and made of a special alloy of chrome steel that will neither rust, corrode, nor break on bending. To reassure oneself that they are safe for use they should be bent into a semicircle, with the stylet removed, before sterilization. The point of the needle should at all times be very sharp; in fact, it is a good practice never to use a needle twice without resharpening. There is a certain sense of "touch" that one acquires which materially facilitates a lumbar puncture. The development of this is essential to obtain the best results. A large needle will traumatize the tissues, permit seepage of the spinal fluid, produce an increased cell count, and cause post-anaesthetic headaches, to say nothing of the injury to the strands of the cauda. A long tapered pointed needle causes bleeding more frequently at the time of puncture, and it is often the direct cause of unsatisfactory anaesthesias. Owing to the fact that a part of the taper is within the dura and the remainder outside when the solution is injected, a portion escapes extradurally. A part of the long tapered needle may be within a vein, and when concentrated solution is introduced intravenously novocain convulsions may result. An all-steel needle is very apt to break within the tissues if the patient moves or jumps, or if side pressure is applied. The extraction of a needle from the interspinous ligament is not a pleasant experience.

The syringes should be light, and preferably of the all-glass type. It is advisable to fit the needles to the syringe before they are sterilized, to reassure oneself that there is no leakage between needle and syringe, or against the possibility of aspirating air into the syringe. They should be so constructed that the piston works freely in the barrel; there should be no leakage about, or sticking of the plunger. It is advisable, but not essential, that the syringe should have a suitable locking device that will not permit the needle to become detached when in use.

When the anaesthetic is to be injected with the patient in a prone position the posture must be essentially correct—that is, lying on the side with the knees flexed upon the abdomen and the head bent forward, the chin resting on the chest, the back bowed outward. The hips and shoulders should be in the same plane. If the shoulders are tilted and the hips vertical, or the hips tilted and the shoulders vertical, "a corkscrew spine" results. With this condition dry taps may be produced, bleeding may be caused, or the needle may deviate to the side of the dura, and unsatisfactory results will be obtained. The size of the subarachnoid space varies in different individuals, but with a little experience one will quickly learn the extent of expansion of spinocain necessary to produce anaesthesia on the body surface at various heights. Better results will be obtained if the patient is allowed to remain flat on the table for three to five minutes after injection, or until such time as anaesthesia has reached the desired height.

Then the degree of Trendelenburg position is assumed as measured by the tiltometer.

Children tolerate spinocain anaesthesia very well; in fact, they are able to withstand larger doses in proportion to the body weight than the adult. It must be remembered that the cord extends lower in infants and children than in the adult. Therefore it is necessary to make the spinal puncture in the fourth interspace so as to avoid accidentally injuring the cord. To determine the amount of spinocain one should consider the body weight of the infant rather than the age. The spinous processes are not fully developed, and this lessens the distance from the skin to the dura. The accompanying table shows the age, weight, distance from skin to dura, amount of spinocain advocated, the mechanical expansion required, and the approximate duration of anaesthesia. I am offering this as an aid to those who may be unfamiliar with the technique in children.

Age.	Weight.		Height.		Distance from Skin to Dura.		Amount of Spinocain.		Mechanically expand to c.cm.	Duration of Anaesthesia (minutes).
	lb.	kg.	in.	cm.	in.	cm.	Minims.	c.cm.		
1 mth.	8	3.6	23.8	52.7	0.65	1.8	6	0.40	1.0	15-25
2 mths.	10	4.5	22.3	57.0	0.65	1.8	6-8	0.4-0.55	1.0	15-30
4 "	14	6.6	23.7	60.1	0.75	2.0	6-8	0.4-0.55	1.0	15-30
6 "	16	7.2	25	63.7	0.75	2.0	6-8	0.4-0.55	1.0	15-30
8 "	18	8.1	26.5	67.4	0.85	2.2	8-10	0.55-0.65	1.3	20-40
10 "	21	9.9	28.3	72.3	0.85	2.2	8-10	0.55-0.65	1.3	20-40
1 yr.	23	10.4	29	73.8	0.88	2.3	8-10	0.5-0.65	1.3	20-50
2 yrs.	28	12.6	32.5	82.8	1.0	2.5	10-12	0.65-0.8	1.5	25-60
3 "	32	14.7	35	89.1	1.0	2.5	10-12	0.65-0.8	1.5	25-60
4 "	35	15.8	38	96.7	1.2	2.8	12-14	0.8-1.0	1.8	40-80
5 "	41	18.9	41	104.3	1.2	2.8	12-14	0.8-1.0	1.8	40-70
6 "	45	20.3	44	111.7	1.2	3.0	14-16	0.8-1.0	2.0	60-90
7 "	49	22.5	46	116.8	1.2	3.0	16-18	1.0-1.2	2.3	80-110
8 "	54	24.8	48	122.0	1.2	3.1	18-20	1.2-1.3	2.5	80-100
9 "	60	27.0	50	127.0	1.3	3.4	20-22	1.3-1.5	2.7	90-120
10 "	65	29.4	52	132.0	1.3	3.5	20-24	1.3-1.6	3.0	80-120
11 "	72	32.1	54	137.2	1.4	3.7	20-26	1.3-1.65	3.3	70-120
12 "	79	35.1	56	142.2	1.4	3.7	22-26	1.5-1.65	3.5	70-100
13 "	88	39.8	58	147.3	1.5	4.0	22-26	1.5-1.66	4.0	60-90
14 "	93	45.0	61	155.0	1.5	4.1	24-30	1.6-2.0	4.5	70-100
15 "	110	50.3	63	160.0	1.7	4.4	25-40	1.6-2.6	5.0	80-120

For intradural sacral anaesthesia, with the light solution, a 15 degree Trendelenburg position should be assumed at once. With the heavy solution the patient should remain sitting after injection until the anaesthesia has become fixed; he should then be placed in a reverse Trendelenburg position. *One should not confuse the light and heavy solutions; if this happens, a case of collapse may occur, due to the ascent of the solution in a cephalic direction.*

Morphine and scopolamine are necessary, not to intensify the anaesthesia, but to allay the fear and apprehension of the patient. For upper abdominal work the first lumbar interspace should be selected. With the patient in the prone position the second interspace is usually the wider and more accessible; it is the interspace of choice. In children the level of the conus medullaris should always be borne in mind. In infants it terminates at the fourth lumbar; in children from 4 to 5 years of age it is found between the second and third; from 10 to 11 years at the second lumbar. It does not ascend to the dorso-lumbar interspace until early in adult life. The site of puncture should be selected accordingly.

For prolonged operations 2, 3, or 4 c.cm. of spinocain may be used with safety, provided an equalizing amount of ephedrine is employed. For nervous individuals it is always advisable to use 0.5 to 1 c.cm. more than in a normal case,

as this produces a deeper and more intense anaesthesia, that will greatly diminish tactile sensations, which often disturb the hypersensitive patient. One should never attempt to introduce spinocain except into the lumbar spine, never above the conus medullaris. The introduction of a local anaesthetic directly into the cord substance may, and probably will, result in an instantaneous catastrophe.

Spinal anaesthesia is adaptable in all classes of cases, be the blood pressure high or low, the patient young or old, fat or thin, cardiac, diabetic, alcoholic, or addict. Should the blood pressure be extremely low it may be elevated to normal by the intravenous injection of suprarenin and ephedrine combined. Our youngest patient was 2 months and the oldest 91 years of age.

Patients suffering from tuberculosis, bronchitis, asthma, renal toxæmias, pyelitis, diabetes, or arterio-sclerosis should not be subjected to the harmful influences of inhalation anaesthesia.

In the acute abdomen this form of anaesthesia almost becomes a matter of life or death; morbidity is lessened, convalescence shortened, and the mortality rate lowered.

Spinocain offers more protection to the patient and better working facilities to the surgeon. It has been taken up as a routine anaesthetic by innumerable American operators, because they realize the above conditions. The revived enthusiasm of spinal anaesthesia in America can hardly be estimated. An idea of the recent increase in spinocain anaesthesia may be obtained by attending any of the National Surgical Association meetings. It is the safest form of anaesthesia we possess for operative procedures below the costal margin.

Concluding, I do not wish to advocate the use only of spinal, conduction, or local anaesthesia, but I wish to emphasize that past experiences have taught me that with their use we can prevent the aftermath of operations—the nausea, vomiting, distension, ileus, acidosis, pneumonia, and innumerable other complications—which are directly or indirectly attributable to inhalation anaesthesia. Patients no longer fear an operation or dread the effects of the anaesthetic. Permit me to foretell that within ten years, possibly less, local, conduction, or spinal anaesthesia will be the anaesthetic procedure of choice.

THE CLINICAL ASPECTS OF CONTROLLABLE SPINAL ANAESTHESIA.*

BY

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It has been said, and we thoroughly agree with the statement, that "all textbooks on spinal anaesthesia will have to be rewritten as a result of the epochal work of Dr. George Pitkin." My object in writing this article is twofold: first, to try and shed some light on the subject; and, secondly, to correct some of the erroneous impressions that are abroad concerning it.

This subject is a very old one, dating back to the work of Corning in 1888, and during the forty years there have been many successes and failures, many advocates and detractors. In some localities and with some operators it has gradually gained and held favour, but in most cases it has been doomed to early failure and disuse. In the last few months, however, it has received a tremendous impetus, as note the great number of articles published in the last year; particularly I would call attention to the December number of the *American Journal of Surgery*, where no fewer than fourteen different articles appear on the subject. I believe this great impetus is largely due to the admirable work of Dr. Pitkin and his associates of Teaneck, New Jersey, and his development of a new solution. Dr. Pitkin has used every effort to have this method thoroughly tried out, and has worked

out a technique which to my mind does away with the objections to the older methods of spinal anaesthesia.

The main objections in the past have been, first, the transitory nature of the anaesthesia; secondly, lack of control of the height or extent of the anaesthesia; thirdly, the serious depression of the blood pressure. The technique devised and first advocated by Dr. Pitkin, and now by a host of his followers, completely negatives all of the above objections, and with them other objections that one so often hears.

First, the transitory nature of the anaesthesia is corrected by making the solution viscid by the addition of gliadin (the active principle of wheat starch). This very greatly prolongs the period of the anaesthesia—our usual anaesthesia now lasting from one and a half hours to two hours.

Secondly, the height or extent of the anaesthesia is controlled by the tilting of the table and expansion of the solution. Owing to the fact that the anaesthetizing agent is lighter than the spinal fluid and floats upon it, the upper extent of the anaesthesia is very definitely controlled by placing the patient in the Trendelenburg position as soon as the anaesthesia has reached the desired height.

Thirdly, the severe drop in the blood pressure is controlled by ephedrine, used before the spinal injection in an amount to be determined by the existing blood pressure, and is repeated or added to as the case requires, always keeping the blood pressure within the range of normal. One must bear in mind, however, that the ephedrine will not act as efficiently after the spinal anaesthesia has been administered as before.

Formerly the systolic blood pressure of less than 100 mm. of mercury was considered a contraindication to spinal anaesthesia. Now, having a patient with a low systolic pressure, ephedrine is used in sufficient quantities to raise the blood pressure to the desired amount and the operation is started. Frequent blood pressure readings are taken and the blood pressure is maintained by the use of further ephedrine, if needed; 1/30 grain of strychnine is also added to spinocain, or spinal anaesthetizing agent, as it has been shown that this has a decided effect on the vasomotor nerves if given intraspinally.

To act intelligently one must thoroughly understand the cause of the drop in the blood pressure. It must be remembered that *all* the vasomotor nerves controlling the blood vessels of the body are given off the spinal cord from the second dorsal to the third lumbar segment. Therefore, if the anaesthetizing agent is allowed to extend as high as the second dorsal all the vasomotors will be paralysed and a very considerable blood pressure drop will take place. This illustrates the very great value of the controllable feature of this anaesthetizing agent, because it can be limited to lower levels so that a large portion of the vasomotor nerves are not affected.

Another safety factor is of the greatest importance in the use of this lighter specific gravity solution: if a blood pressure drop of serious proportions should occur, and even if the ephedrine should not control it (a circumstance we have never encountered), the head of the table, which is already lowered, should be lowered still more, and this in itself would cause blood to flow to the brain by gravity and prevent a cerebral anaemia, even if all other methods fail. It is a well-understood fact that the blood pressure drop is due to the involvement of the vasomotor nerves, with the consequent "pooling" of most of the blood in the splanchnics. The method of putting the patient into a decided Trendelenburg position always causes a flow of pooled blood to the brain, even if there is no recordable blood pressure. I think we will all agree that gravity will not fail us even though all other methods may, and we will continue to have a sufficient amount of blood in the brain to carry on the necessary functions of life. The importance of this is at once recognized by the fact that our great interest in blood pressure depression is really one of fear of serious cerebral anaemia.

The use of ephedrine before the spinal injection is made is essential for two reasons: first, that ephedrine will act much better before anaesthesia; and secondly, that we believe in the *prevention* of a serious blood pressure

* Read in a discussion in the Section of Anaesthetics at the Annual Meeting of the British Medical Association, Manchester, 1929.