SPINAL FLUID FINDINGS IN SPINAL ANESTHESIA

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Anesthetists are familiar with the neurologic complications that have been reported following spinal anesthesia. That these complications are relatively rare is attested by the thousands of spinal anesthesias given without untoward results. Minor side effects, such as spinal headaches, are encountered by all anesthetists at one time or another. No definite cause or cure is yet known, but we do know that with relatively simple treatment the headache can be cured and no permanent ill effect remain. Every anesthetist hopes never to have a postanesthetic sequel which permanently cripples or is the causative factor in the loss of the patient.

Complications of a serious nature reported in the literature (1-11) include hemorrhage and paraplegia, loss of bowel and bladder sphincter control, cranial nerve paralysis, radiculitis, epidural abscess, meningitis, chemical meningismus, arachnoiditis and others.

Recently, Haynes and Smith (1) reported one case and Foster Kennedy (6) and associates reported 3 cases of arachnoiditis following spinal anesthesia. These cases illustrate severe complications following so closely the giving of spinal anesthesia that it seems likely the authors are justified in their correlation of cause and effect. Little is known of the changes occurring in the central nervous system between the giving of spinal anesthesia and the development of pathologic lesions.

Barker (12), a British surgeon, writing on spinal anesthesia in 1907, speaks of "by-effects." 'In spite of all the attention bestowed upon their causation, these by effects remain very obscure, the more so as they are not uniformly present, the great majority of cases showing no sign of them. This emphasizes the urgent need of scrutinizing the composition of the injected fluid in all its aspects.'

Cerebrospinal fluid has been studied in conditions of health and disease by many workers, probably the greatest single study being that of Merritt and Fremont-Smith (13, 14) in which 21,000 fluids were examined.

Of cerebrospinal fluid after intrathecal injection they state: "The injection of foreign substances in the subarachnoid space causes an

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aseptic meningeal reaction. The principal changes consist of a pleocytosis of varying degree and an increased protein content. The sugar content of the fluid remains unchanged or is increased.”

Following spinal anesthesia they believe there is almost regularly an inflammatory reaction in the spinal fluid. Their personal experience, they stated, is confined to 10 cases; in 5 the puncture was made within four days of the anesthesia. One case showed increased protein, the highest 162 mg., with pleocytosis of 2028 cells and a sugar content of 85 mg.

In addition, three reports on spinal fluids following spinal anesthesia were found in the literature between 1928 and 1934 and one in 1943. It was not until Konwalter’s (11) report in 1943 that pontocaine was mentioned and then only in 3 cases. He examined spinal fluids, one, two, and three weeks after induction of spinal anesthesia with procaine, metacaine and pontocaine, and found no significant changes in sugar, protein, cell count, chlorides or Kahn test. He did not state whether or not the Sise pontocaine-glucose technic was used.

These findings are in contrast to those of earlier workers, Iason, Lederer and Steiner (2), who, in 1930, reported an increased cell count and sugar content. Bacher-Grondahl (7), in 1934, reported an increased cell count in 65 per cent of cases, albumin and globulin increase in 33.3 per cent and sugar in 24 per cent.

In the time interval between 1934 and 1946 considerable change has taken place in the field of spinal anesthesia (15, 16), the first and most important being the widespread development and acceptance of trained anesthetists, the second, changes in technic—among them being the Sise (17) pontocaine-glucose method of spinal anesthesia and the Lémmon (18) method of continuous or fractional spinal anesthesia.

It seemed to me that a further study of cerebrospinal fluids might be interesting and worth while. Accordingly, a collection of spinal fluids was begun about eighteen months ago. Spinal fluids from 200 patients were studied. They were divided into the following groups:

Controls, 75 patients. Fluid was taken from individuals preoperatively who had no previous record of spinal anesthesia.

Spinal fluid was taken from 60 patients preoperatively who had had spinal anesthesia previously—the time interval being longer than one month and extending for the most part not over one year, although in one case ten years had intervened. This group will be referred to hereafter as the year group.

Spinal fluid was obtained from 40 patients postoperatively who had had spinal anesthesia within the preceding thirty days. This group was made up of patients with bilateral hernias with a week’s interval between operations, an occasional dehiscence, intestinal obstructions having colostomies—followed by resection, and other cases in which a similar two-stage operation was carried out. In this group patients had
received spinal anesthesia for the first operation and before giving the
anesthetic for the second operation, spinal fluid was collected, the time
interval varying from twenty-four hours to thirty days. This section
will be termed the thirty-day group.

And, finally, spinal fluids were obtained from 25 patients before and
after continuous spinal anesthesia.

The method of spinal anesthesia employed is fundamentally the Sise
pontocaine-glucose technic.

Before anesthesia is induced the anesthetist employs to produce
meningeal infection the surgeon's preventative—namely a surgical ten-
minute scrub and sterile gloves. Needles and syringes are dry steril-
ized, then rinsed with saline solution by the anesthetist to eliminate
any possibility of foreign body being injected through flaking of the
needles or inadequate cleansing. The entire back is prepared with ether,
alcohol and iodine and draped with sterile towels.

The ampules of solution are soaked in deeply colored methylene blue
alcohol so that the most minute crack in an ampule is quickly indicated
in the anesthetic fluid.

Procaine, 1 cc. of a 1 per cent solution, with 50 mg. of ephedrine, is in-
jected into the skin and intramuscular tissue at the site of tap. No tap
is done above the second lumbar interspace, the majority being in the
fourth lumbar interspace.

Poncaine and glucose were the agents used in the study and for the
most part, in those cases representing the longer time interval. In a
few instances the agent was unknown or procaine had been used in place
of poncaine.

Fluids were collected in sterile test tubes after a few drops had
demonstrated that the fluid was clear and a free flow was present. All
specimens were examined in the spinal fluid laboratory of the Massa-
chusetts Memorial Hospitals.

Since the principal changes in cerebrospinal fluid following intra-
theecal injection of a spinal anesthetic were thought to be an increased
cell count and an increase in the protein content, the early spinal fluids
were examined for this alone. Later, sugar studies were added.

Protein content was determined by the Ayer-Daley-Fremont-Smith
technic in which sulfa salicylic acid is used to precipitate the protein
and a colorimeter reading is made against distilled water. Protein was
considered normal when present in amounts of 15 to 45 mg., with an
average of 28 mg.

Sugar content was studied by the Folin-Wu method, using protein-
free filtrate to which copper tartrate and phosphomolybdic acid are
added and the results read on the Klett-Somerson colorimeter. A nor-
mal range of cerebrospinal fluid sugar in fasting patients is between 50
and 80 mg. per 100 cc.

Cell counts were made with a blood cell counting chamber, using
Unna's polychrome methylene blue for staining. Cell counts not ex-
ceeding 5 per cubic millimeter were regarded as normal.
Norms established by Merritt and Fremont-Smith were taken as standards of comparison, together with the levels obtained in the control group.

The results of this study are shown in table 1. On the basis of the results shown in table 1, it would appear that the controls are entirely within the normal range previously mentioned.

<table>
<thead>
<tr>
<th></th>
<th>Protein, mg.</th>
<th>Cell Count</th>
<th>Sugar, mg.</th>
</tr>
</thead>
<tbody>
<tr>
<td>75 controls</td>
<td>31.8</td>
<td>Normal</td>
<td>71</td>
</tr>
<tr>
<td>60 1-year</td>
<td>38</td>
<td>Normal</td>
<td>not done</td>
</tr>
<tr>
<td>40 30-days</td>
<td>49-61.3</td>
<td>Normal</td>
<td>81</td>
</tr>
<tr>
<td>25 cont. spinal</td>
<td>Before 28</td>
<td>Normal</td>
<td>79.8</td>
</tr>
<tr>
<td></td>
<td>After 26.5</td>
<td>Normal</td>
<td>404</td>
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</tbody>
</table>

The one-year findings show a mild rise in protein when compared with the controls. This reflects the presence of fluid of some 20 patients in whom the specimens were taken within three months of spinal anesthesia, whose values were higher than those of a longer interval.

The group in which the patients had been given a spinal anesthetic thirty days previously shows a definite protein increase. Three patients are included whose protein values were 133, 137 and 139 mg., respectively. That the increase in protein values is not owing to an increased cell count is shown by the fact that all counts were within normal limits, two cells being the maximum reported in the fluid of any patient. Possibly, some error in cell count may occur on the basis of elapsed time. If fluids are kept refrigerated and sterile, however, they are said to show little change in twenty-four hours. All counts were made within four to six hours of the tap.

Levels of spinal fluid sugars were not determined on the year group since most of these specimens were examined in the early part of the study. The results in the thirty-day unit are within high upper limits of normal.

It would appear on the basis of comparison of the first three groups that a mild rise in protein level occurs in cerebrospinal fluid within the first thirty days after spinal anesthesia and may continue for a time but returns to normal shortly thereafter.

Davis, Haven and Gibbons (10) gave procaine spinal anesthesia to dogs and demonstrated meningeal irritation, demyelination of nerve tissue and hemolysis of cells up to twenty or thirty days. Dogs permitted to live ninety days showed that repair had taken place to a marked extent. They concluded that such changes were not permanent.

That this is true for the majority of patients is proved clinically and in the research laboratories (19, 20, 21). Allergy or individual susceptibility to anesthetic drugs does exist, as evidenced by the recent report of sensitivity to ether. Clark (9) reported that when an allergic
reaction occurs within the cranial cavity, localized edema may increase intracranial pressure or cause local anemia. Neurologic symptoms which may arise as a result are headache, vomiting, convulsions, hyperesthesia, anesthesia, paralysis and psychosis. Patients showing sensitivity to the local use of anesthetic drugs exhibit symptoms not unlike these. It is possible that in the rare patient with a high degree of sensitivity to a specific drug, application of the drug in direct contact with tissue of the nervous system produces a lasting pathologic result rather than a transient one (22).

I have chosen to treat the fluid from patients having continuous spinal anesthesia as a separate unit. The protein differences as noted in table 1 are so insignificant as to be within the realm of error. The majority of continuous spinal anesthesias lasted from two to three hours, with one lasting five hours. Within this period of time there does not appear to be evidence of meningeal irritation or damage to nerve tissue. Presumably, if such change does occur, this is too short a time for it to develop.

<table>
<thead>
<tr>
<th>Sugar Before Anesthesia</th>
<th>Glucose Added</th>
<th>Sugar After Anesthesia</th>
<th>Position</th>
</tr>
</thead>
<tbody>
<tr>
<td>mg.</td>
<td>mg.</td>
<td>mg.</td>
<td></td>
</tr>
<tr>
<td>73</td>
<td>775</td>
<td>700</td>
<td>H</td>
</tr>
<tr>
<td>88</td>
<td>240</td>
<td>400</td>
<td>H</td>
</tr>
<tr>
<td>80</td>
<td>360</td>
<td>400</td>
<td>H</td>
</tr>
<tr>
<td>71</td>
<td>528</td>
<td>445</td>
<td>H</td>
</tr>
<tr>
<td>68</td>
<td>528</td>
<td>475</td>
<td>H</td>
</tr>
<tr>
<td>83</td>
<td>384</td>
<td>250</td>
<td>H</td>
</tr>
<tr>
<td>74</td>
<td>180</td>
<td>265</td>
<td>H</td>
</tr>
<tr>
<td>86</td>
<td>240</td>
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<td>H</td>
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<tr>
<td>83</td>
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<tr>
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<td>550</td>
<td>H</td>
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<tr>
<td>?</td>
<td>528</td>
<td>475</td>
<td>V.S.T.*</td>
</tr>
<tr>
<td>80</td>
<td>456</td>
<td>150</td>
<td>T†</td>
</tr>
<tr>
<td>86</td>
<td>240</td>
<td>87</td>
<td>T†</td>
</tr>
</tbody>
</table>

Average 79.8

* Trendelenburg
† Very slight Trendelenburg

The increase in the sugar content is startling. A more detailed study of this aspect is presented in table 2. Certainly, a part of this increase is the result of the glucose used with the pontocaine for weighting the solution.

The only cases I can find recorded with comparable values for spinal fluid sugar occur in (13): (1) rare cases of poliomyelitis and meningitis; (2) a case of tetanus with intrathecal injection of serum in which the sugar rose to 314 mg.; (3) a thirty-six hour postspinal anesthetic meningeal reaction with 106 mg., and (4) in diabetics in whom the highest I found recorded was 500 mg. The spinal fluid sugar of diabetics is
obviously a result of a general body state with increased blood sugar. Accordingly, blood sugar values were checked in 3 patients with high levels and were found to be within normal limits. Fear, and the use of ephedrine and intravenous glucose solutions (23) may also increase sugar levels, but normal blood sugar values would seem to rule them out. The practically routine use of oxygen with continuous spinal anesthesia combined with no other clinical symptom of anoxia would seem to preclude an anoxic hyperglycemia (24).

It was suggested that pontocaine might be acting as a reducing agent, but this was checked by adding it to blood serum and glucose of known content, and found not to be true.

It is entirely possible that the high glucose levels might well be the result of pooling of the glucose around the needle, and I wondered if removal of spinal fluid postoperatively might be wise. Spinal fluid removed in two portions from one patient showed values of 550 and 500 mg.

It was noted as time went on that such drastic elevations did not occur in a few cases. On checking all factors that might influence the results, it became evident that those patients who, after the original stabilization of height, were placed in the Trendelenburg position did not develop as marked a hyperglycorrhachia.

The flow of spinal fluid is slow and tends to lag or stagnate in the lumbar area. "Flow is influenced by respiration, pulse beat and change in posture." Flow tends to promote diffusion.

According to Long (25), the nervous tissue is unique in that its sole source of energy appears to be glucose or such related substances. Reduction of oxygen is ultimately fatal to all tissues, but reduction of the glucose supply is without effect on any tissue except the nervous system. Himwich and Nahum, studying the metabolism of the brain in situ, found that nerve cells require only glucose and oxygen for their needs. Elliott, Scott and Libet (26, 27) found that the maximum rate of respiration in brain suspensions occurred in the presence of 10 per cent glucose.

Five or ten per cent glucose, given intravenously, has no untoward effect upon the vein wall. When glucose is given in 50 per cent concentrations it tends to cause irritation and sclerosis. Such an increase in glucose in cerebrospinal fluid may conceivably act as an irritant to the nerves and meninges.

If diffusion of the glucose is aided by the Trendelenburg position, it is possible that the metabolism of brain and cord tissue may be carried on under conditions that at least from the laboratory point of view appear to approach ideal. Given an adequate supply of oxygen at the same time, it would seem that glucose, if properly diffused, is helpful rather than harmful to the tissue of the nervous system.

All patients who have spinal anesthesia in our hospital are placed in the Trendelenburg position postoperatively for four hours. Origi-
ually, this was done to prevent spinal headaches; it would seem to have the additional advantage of promoting diffusion of glucose and metabolism of the central nervous system.

Two side effects of this study are included as being of general interest. The practice of withdrawing spinal fluid at the end of operation, to remove excess anesthetic agent, appears to be of little value. Chemical analysis of spinal fluid for pontocaine shows a barely perceptible amount present fifteen minutes after injection. Fear of infecting the subarachnoid space during spinal anesthesia with resultant meningitis, appears to be unfounded if a good technic is followed. Intermittent cultures of fluids were uniformly sterile except in one case of continuous spinal anesthesia in which the first fluid showed bacteria and the final fluid none; it was concluded this was a contaminant.

**Summary**

In conclusion, a study of spinal fluids of 200 patients was made to determine the effect of spinal anesthesia.

In 75 control cases the spinal fluids were within normal limits.

In 60 cases in which the fluid was taken thirty days to ten years after spinal anesthesia there was, in comparison, a mild increase in protein, chiefly because the interval was less than three months in 20 cases.

In 40 cases in which the spinal fluid was taken one to thirty days after spinal anesthesia there was a significant rise in protein, tending to indicate that a mild but transient irritation occurs after spinal anesthesia.

In 25 cases, specimens of spinal fluid obtained before and after continuous spinal anesthesia were examined. Protein variations were negligible, which suggests that the time interval was adequate for meningeal or tissue irritation to be demonstrated.

Spinal fluid sugars in this group present a marked rise at the end of operation when patients were kept in the horizontal position.

It is thought probable that this rise is caused by pooling of the glucose at the site of injection. The Trendelenburg position promotes diffusion and it is believed this diffused glucose promotes central nervous system metabolism.

It is suggested that pontocaine or any similar anesthetic agent used intraspinally may produce transient irritation, the possible exception being the rare individual who may be allergic to the drug. Glucose, when used in conjunction with spinal anesthetic agents and if permitted to diffuse by use of the Trendelenburg position, may be helpful rather than harmful to the tissue of the central nervous system.

**References**


