

Postdural Puncture Headache and the Development of the Epidural Blood Patch

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During a routine attempt at placement of a lumbar epidural catheter for labor analgesia using a loss-of-resistance technique, a distinct “pop” is appreciated. Upon removal of the syringe, cerebrospinal fluid flows freely through an 18-gauge Tuohy needle. A repeated attempt at another interspace is uneventful, and analgesia is satisfactory for the remainder of labor. The following day, this patient reports an incapacitating headache having a strong positional nature. She anxiously desires to hear of her options

Today, the options presented to the patient described would certainly include the epidural blood patch. The blood patch concept now seems intuitive and can be quickly described to patients and expeditiously performed. Yet this common procedure has only been a credible option for a relatively short time.

Whether secondary to accidental dural puncture or purposeful diagnostic or therapeutic procedures, postdural puncture headaches continue to be a prominent concern.^{1,2} Such “spinal headaches” are all too familiar and are well described.³⁻¹¹ Although varying greatly in severity among individual patients, headache symptoms characteristically begin within 48 hours after dural puncture, are either circumferential or radiate bilaterally from the occipital to the frontal region, and tend to be dull or throbbing in nature. Headache symptoms may be accompanied by visual or auditory disturbances, nausea and vomiting, and neck pain. Critical to the diagnosis of postdural puncture headache (PDPH) is

the influence of posture on the severity of the headache, which is aggravated by the upright position and generally relieved with recumbency.

PDPHs have proven to be a problem over the ages and are among the most refractory challenges in anesthesia.¹² It seems likely that the early enthusiasts of regional anesthesia would be quite surprised to find that these headaches have cast such a long and enduring shadow over spinal anesthesia and continue to do so despite the passing of the centennial of this anesthetic technique.¹³ The epidural blood patch has proven to be a major consolation for this problem. The development of this unique procedure is very much a tale of the times and the people who lived in them.

Part I: PDPH

Beginnings

Although others had previously shown the capacity for cocaine to produce local and regional anesthesia effects, Dr. J. Leonard Corning, a New York City neurologist, was clearly the first to conceive the idea of a subarachnoid injection of local anesthetic. His report in 1885 of an injection of cocaine between a patient’s thoracic spinous processes did produce anesthesia, yet he failed to provide any documented evidence of dural puncture (i.e., cerebrospinal fluid return).¹⁴ Some have taken the fact that the morning after the procedure “the only constitutional symptoms complained of were *headache* and slight vertigo” (italics added) as evidence that dural puncture must have occurred. Yet, on careful review of his report, many are doubtful that he was able to accomplish his stated objective.¹⁵ It seems most likely that Corning was able to successfully produce epidural analgesia but probably not spinal anesthesia. Despite Corning’s published observations, attempts at dural puncture would remain relatively uncommon before the introduction of spinal anesthesia in 1899.

The documented technique of dural puncture was independently introduced into clinical practice in 1891, by Wynter, in England, and Quincke, in Germany. Dr. W. Essex Wynter reported his use of lumbar puncture for drainage of cerebrospinal fluid

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in the treatment of tubercular meningitis.¹⁶ While unfortunately being unable to report any survivors among his first 4 patients, he noted that no harm was done and that some temporary relief was observed. Dr. Heinrich Quincke published his experiences with lumbar puncture in the treatment of hydrocephalus and, in addition to describing some clinical benefits, notably reported patients complaining of “pain . . . for several days.”¹⁷ Although he did not elaborate on these symptoms, it seems likely that postdural puncture headaches would have occurred in Quincke’s patients, given his technique of multiple large-bore punctures of the dura and observation that, after 8 days, “edema of the surrounding tissues clearly testified to the continuing escape of cerebrospinal fluid.” A number of other investigations were published throughout the 1890s describing the intrathecal injection of various different agents as well as reporting the utility of lumbar puncture in the diagnosis of bacterial meningitis. So, although not widely practiced, attempts at using lumbar puncture for both diagnostic and therapeutic purposes clearly predated spinal anesthesia and were almost certainly accompanied by PDPHs.

However, it was only with the true clinical introduction of spinal anesthesia by Dr. August Bier that PDPHs were clearly associated with dural puncture and were immediately recognized as an entirely different type of headache than had been previously encountered (Fig 1). In reporting his initial experiences with spinal anesthesia in August 1898, Bier (a student of Quincke’s) described 3 (possibly 4) of his first 6 patients experiencing headaches secondary to the procedure. These intriguing initial clinical experiences led Bier and his assistant, Dr. August Hildebrandt, to subsequently perform the now-famous experiments with spinal anesthesia on themselves. During the course of these experiments, both Bier and Hildebrandt received dural punctures and each subsequently developed a headache. Bier’s entire report makes for fascinating reading but is particularly notable for his seminal and first-hand description of a classic PDPH:

“After performing these experiments on our own bodies we proceeded without feeling any symptoms to dine and drink wine and smoke cigars. I went to bed at 11 p.m., slept the whole night, awoke the next morning hale and hearty and went for an hour’s walk. Towards the end of the walk I developed a slight headache which gradually got worse as I went about my daily business. By 3 p.m. I was looking pale and my pulse was fairly weak, though regular and about 70 beats per minute. In addition, I had a feeling of very strong pressure on my skull and became rather dizzy when I stood up rapidly from my chair. All these symptoms vanished at once when I lay down flat, but returned when I stood up. Towards the evening I was forced to take to bed and remained there



Fig 1. Dr. August Bier (1861-1949).

for nine days, because all the manifestations recurred as soon as I got up. I felt perfectly well as long as I remained horizontal. Appetite and sleep rhythm were unaffected but any prolonged period of reading made me feel dizzy. The symptoms finally resolved nine days after the lumbar puncture.”¹⁸

In his future commentaries regarding spinal anesthesia, Bier would repeatedly stress that the procedure must be improved (especially in reducing the incidence of postdural puncture headache) before it could be considered an established technique. Nevertheless, Bier’s initial report, published in 1899, resulted in widespread attempts at spinal anesthesia. Although the technique quickly proved to be associated with a number of complications, it was the common occurrence of a postural headache that was most apparent. In San Francisco in October 1899, Drs. Dudley Tait and Guido Caglieri were the first to perform spinal anesthesia in the United States and noted “headache” and “vomiting” as the “disagreeable effects.”^{19,20} The first published report of intraspinal cocainization in the United States, by Dr. A. Matas at Charity Hospital in New Orleans in 1899 (performed just 15 days after Tait and Caglieri), stated that the procedure was followed by an “occipital headache.”²¹ By 1900, experiences with spinal anesthesia in small series of patients were being reported, with an incidence of headache commonly around 50%.²² In January 1901, only 2 years after Bier’s original report, a correspondent

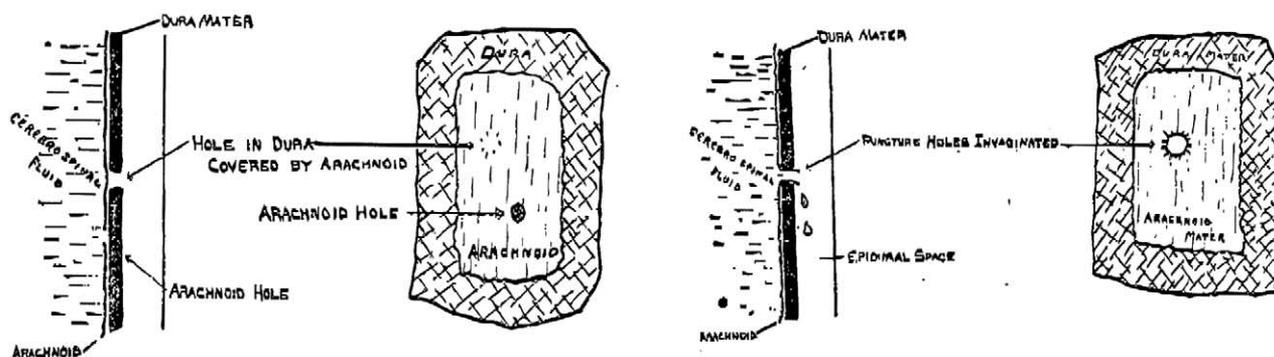


Fig 2. Normal closure of dural puncture hole (left) versus nonclosure with persistent leakage of cerebrospinal fluid (right) (from MacRobert).²⁹ (Reprinted with permission from *JAMA* 1918;70:1350-1353. Copyrighted © [1918], American Medical Association. All rights reserved.)

stated in *The Lancet* that there were already nearly 1,000 published reports of spinal anesthesia.²³ As the use of spinal anesthesia spread, so did reports of PDPH and a quickly growing general awareness of the problem.

Investigations Into the Nature of PDPH

Naturally, clinicians were anxious to determine the cause of these troublesome headaches. Bier, in his original report, attributed the symptoms of headache to "circulatory disturbances (hyperemia or anemia) of the central nervous system."¹⁸ Headaches after dural puncture subsequently came to be attributed to either high or low cerebrospinal fluid pressures.²⁴⁻²⁶ So-called "high-pressure headaches" were believed to be caused by an aseptic meningeal reaction (in response to injected solutions, antiseptics, or blood). Low-pressure headaches were recognized as being much more common and were those that clearly improved upon assuming a head-down position. Such low-pressure headaches were believed to be due to excessive loss of cerebrospinal fluid, the aptly named "leakage theory."

In 1902, J. A. Sicard in France was the first to attribute PDPHs to a loss of cerebrospinal fluid into surrounding tissues after dural puncture.²⁷ Hosemann,²⁸ between 1907 and 1909, repeated dural taps in patients with headaches following spinal anesthesia and found that over 86% had decreased cerebrospinal fluid pressure. Clinicians immediately took special care to minimize cerebrospinal fluid loss during dural puncture procedures, yet continued to note a high incidence of subsequent headaches. Investigators then reasoned that if the loss of cerebrospinal fluid was negligible during the course of a dural puncture and headache symptoms commonly did not begin for days after the procedure, then there must exist a persistent leakage of fluid through the dural puncture site. In 1918, MacRobert²⁹ published his evidence for a persistent hole in

the layered dura as the cause of headache after dural puncture (Fig 2).

Jacobaeus and Frumerie³⁰ in 1923 also showed that cerebrospinal fluid pressures are usually low in patients experiencing PDPH. This observation led them to one of the first successful therapeutic interventions regarding this problem when they injected saline intrathecally to reconstitute normal cerebrospinal fluid pressures (described in detail in Part 3). Ingvar,³¹ also in 1923, showed in cadavers that methylene blue placed into the ventricles leaked into the lumbar epidural space if there had been a previous dural puncture. Heldt,³² in 1929, also reported evidence of a persistent leakage of cerebrospinal fluid after dural puncture. Pool,³³ in 1942, stated that myeloscopic examinations of the epidural space "frequently revealed large collections of epidural fluid when an ordinary lumbar puncture has been done within the previous two to four days." Yet, although individual reports continued to accumulate, solid experimental data regarding the etiology of PDPH were limited for several decades.³⁴

Evidence that the technique of dural puncture itself may be predisposed to cause a persistent leakage of cerebrospinal fluid was provided by Heldt and Moloney in 1928.³⁵ They reported that normal baseline pressures within the epidural space were often negative, a finding later confirmed by others³⁶ (although now actually thought to be artifactual).^{37,38} Awareness of these negative pressures would be useful in the development of the "hanging drop" technique for epidural needle placement and further promote the concept of a continued loss of cerebrospinal fluid through a dural puncture site.

During the 1930s, Masserman³⁹ performed a number of clinical experiments on "cerebrospinal hydrodynamics." He was able to show that only 6% of patients developed headache symptoms if cerebrospinal fluid loss was 15 mL or less but that 93%

developed headaches if the loss was greater than 40 mL. Classic studies by Kunkle et al.,⁴⁰ published in 1943, showed that typical symptoms of PDPH could be consistently produced by drainage of as little as 20 mL cerebrospinal fluid. Thus, although PDPH would continue for many years to be ascribed to both increased and decreased intracranial pressure, the weight of evidence quickly mounted for the latter. This debate was only resolved in the 1950s with the work of Drs. Robert Dripps and Leroy Vandam (described in greater detail in Part 4), in which they convincingly showed the direct relationship between needle size and incidence of PDPH.

Today, it is generally agreed that a persistent loss of cerebrospinal fluid after dural puncture results in a sagging of the brain and traction on pain sensitive structures within the cranium.⁴¹ It seems evident that the development of headache symptoms after dural puncture is also related to a compensatory cerebral vasodilation and vascular congestion.⁴² Yet, even now, the mechanisms of PDPH are only incompletely understood. For example, although there is a correlation between subarachnoid pressures and headaches, not all patients with headaches have decreased pressures.⁴³ Furthermore, it has been shown that the degree of cerebrospinal fluid leakage does not correlate with the severity of the headache.⁴⁴ Further research into the nature of the perennial problem of PDPH is clearly needed.

Part 2: Prevention of PDPH

Even more pressing than the desire to understand PDPHs, practitioners from the earliest days of spinal anesthesia were eager to prevent this “annoying and comparatively frequent by-effect.”⁴⁵ Based on his personal experience, Bier was the first to suggest careful technique to avoid cerebrospinal fluid loss as the primary means of preventing PDPH. He stated that “as I myself can vouch, any escape of cerebrospinal fluid must be avoided if possible.”¹⁸ This advice has been repeated through the years, as generations of clinicians have recommended careful technique as the initial means of preventing or minimizing the occurrence of PDPH. Yet, as previously mentioned, meticulous technique was not the entire solution since headaches continued to be a common problem.

With evidence that typical PDPH symptoms were associated with low cerebrospinal fluid pressures, it was apparent to many that a persistent leakage of cerebrospinal fluid must be prevented. Recognizing that the upright position created a column of cerebrospinal fluid that could promote leakage through a dural hole, Sicard²⁷ was the first to recommend bedrest with the foot of the bed raised after lumbar

Table 1. Example of Reduced Incidence of Postdural Puncture Headache With Smaller Needles

Group*	n	Percentage Incidence of Headache		
		Mild	Marked†	Total
20 gauge	18	11.0	22.3	33.3
22 gauge	108	6.5	3.5	10.0
24 gauge	149	0.7	1.3	2.0
26 gauge	700	0.4	0.0	0.4

*All groups received additional measures to improve hydration.
†A marked headache was defined as one lasting 3 or more days.

Data from Greene.⁶¹

dural puncture. Prolonged bedrest (in Trendelenberg and even prone positions) also came to be commonly advocated.⁴⁶⁻⁵⁰ Strictly avoiding elevation of the head both during surgery as well as for several, or even many, hours afterward became a standard recommendation (although more recent evidence would indicate that such maneuvers are of little benefit in the prevention of PDPH).⁵¹⁻⁵⁴ Although essentially absent of any objective evidence of efficacy, adequate hydration was also among the first recommended prophylactic measures.

The use of smaller-gauge needles was quickly adopted as investigators reasoned that if the technique of dural puncture necessitated leaving a hole in the dura, then ideally that hole should allow as little cerebrospinal fluid loss as possible. The beneficial effects of smaller needles appear to have first been noted in 1914 by Ravaut, in Paris.⁵⁵ A number of investigators were subsequently able to show a dramatic reduction in the incidence of PDPH through the use of smaller needles (Table 1).⁵⁶⁻⁶¹ However, smaller needles proved to be technically more difficult to use and were associated with a slow return of cerebrospinal fluid. The concept of an introducer (referred to as the “double needle” or “needle within a needle” technique) was developed by Hoyt in 1922 and was a tremendous aid in the placement of smaller needles.⁶² Concerns regarding possible increased rates of needle “breakage” with smaller gauge needles were quickly dismissed.⁶³

Needle tip design was also recognized as a potential means of reducing the incidence of PDPH. H. M. Greene, in Oregon, developed the first minimally traumatic needle in 1923.⁶⁴ His needle had a rounded “noncutting” tip, which would spread rather than cut the dural fibers, and was intended to minimize cerebrospinal fluid loss. Hart and Whitacre, using their “pencil-point” needle design in 1951, convincingly showed the utility of this approach.¹²⁴ Their study of 3,489 spinal anesthetics performed with 20-gauge pencil-point needles resulted in only a 2% incidence of PDPH (compared with their usual rate of 5%). Furthermore, the

Table 2. Incidence of Postdural Puncture Headache Comparing 25-Gauge Whitacre (“pencil-point”) and Quincke (“cutting”) Needles

25-Gauge Needle	n	Incidence of Postdural Puncture Headache
Whitacre	200	3%
Quincke	200	8.5%

$P \leq .02$.

Data from Buettner.²⁹²

headaches they observed seemed to be less severe in intensity and shorter in duration than those seen with standard needles. Despite the convincing data, “Whitacre” needles would not become widely used until the 1980s, when they could be inexpensively mass-produced (Table 2). A variety of other “non-cutting” needle designs, which clearly result in a decreased incidence of postdural headache, have subsequently been introduced into clinical practice.⁶⁵ It is notable that a recent scanning electron microscope evaluation of dural lesions after puncture by “cutting” and “pencil-point” needles indicates that the “pencil-point” needles actually appear to cause more trauma (perhaps resulting in a better inflammatory sealing response).⁶⁶

H. M. Greene was also the first to appreciate the important association between dural fiber direction and the potential for leakage of cerebrospinal fluid in 1926.⁶³ He observed that a “cutting needle” (e.g., Quincke point) with the bevel placed perpendicular to the direction of the dural fibers would naturally result in a greater dural rent than a needle placed with the bevel directed parallel. When using needles that are not “minimally traumatic,” the importance of needle bevel direction for puncture of the dura has since been shown for needles of virtually all sizes (Table 3).⁶⁷⁻⁶⁹

Recognizing the fact that the dura consists of several layers that must each allow the leakage of cerebrospinal fluid, investigators also surmised that the angle of needle entrance through the dura may be a significant factor in the subsequent development of PDPH.^{70,71} Experimentally, insertion of a needle with a 30° angle to the dura results in significantly lower rates of leakage than with more acute approaches.⁷² These angle of approach theories appear to be conceptually related to the “unflexed back” techniques originally proposed during the 1950s.⁷³ Overall, it does appear that a paramedian or lateral approach does result in a lower incidence of subsequent headache than a midline technique.

An interesting early attempt to eliminate the chance of a persistent hole in the dura deserves mentioning. Drs. Thomas Heldt (in Michigan) and Marque Nelson (in Minnesota) believed that if a

hole must be made in the dura to perform spinal anesthesia, this hole could be plugged as the spinal needle was withdrawn. In 1928, both investigators independently developed methods for the insertion of catgut across the dura at the time of spinal anesthesia.^{32,74} After the subarachnoid injection of local anesthetic, a small length of dehydrated catgut (of a diameter slightly less than the bore of the spinal needle) was placed through the spinal needle by means of a modified stylet and was left across the site of the dural puncture.⁷⁵ Rapid swelling by the catgut promptly sealed the hole in the dura. This technically difficult measure, although effective at preventing postdural puncture headache, resulted in an unacceptable incidence of arachnoiditis and was quickly abandoned. In his report, Heldt also mentioned his consideration of prophylactically using a “thick solution of celloidin or of gelatin which will coagulate promptly . . . and as a consequence seal the hole in the dura.” Although he believed this approach had promise, it apparently never passed beyond the stage of animal experimentation.

Other notable efforts to prevent postdural puncture headaches involved producing positive pressures within the epidural space, thereby discouraging the development of a persistent leakage of cerebrospinal fluid. In 1952, Kaplan and Arrowood⁷⁶ reported performing spinal anesthesia with a 22-gauge needle and, after administration of the subarachnoid drug, pulled the needle back slowly until no cerebrospinal fluid return was noted. At this point, they presumed the needle to be in the epidural space and injected 10 to 20 mL normal saline solution. Using this prophylactic epidural “saline patch,” they noted only a single headache in their series of 100 patients, with no untoward effects. However, this simple modification of normal spinal technique, although used by others,⁷⁷ does not appear to have been extensively further studied.

Similar to the approach of Kaplan and Arrowood,⁷⁶ in the early 1970s a number of authors began to report their experiences using epidural

Table 3. Incidence of Postspinal Headache With Needle Insertion Perpendicular or Parallel to the Longitudinal Dural Fiber Direction (i.e., long axis of the spine)

Needle	Insertion	n	Incidence of Postdural Puncture Headache
22 gauge	Perpendicular	29	17.24%
22 gauge	Parallel	140	0.71%*
25 gauge	Perpendicular	33	15.15%
25 gauge	Parallel	280	0%†

* $P < .001$ versus 22-gauge perpendicular group.

† $P < .001$ versus 25-gauge perpendicular group.

Data from Mihic.⁶⁷

saline as a means of preventing PDPH after accidental puncture of the dura during attempted epidural needle placement. Craft et al.,⁷⁸ for example, in 1973 placed an epidural catheter 1 level cephalad to an accidental dural puncture and, immediately after delivery and again the next morning, administered a bolus of 60 mL normal saline through the catheter. He reported that patients treated with the prophylactic epidural saline were much less likely to go on to develop headache symptoms (12.5%) than a control group (76.5%).⁷⁸ After initial efforts using bolus injections of epidural saline, investigators soon modified their approach to a continuous infusion, again with encouraging results.^{79,80} Indeed, it does appear that epidural saline is effective in reducing the incidence and severity of PDPH, and such techniques were in common use through the 1980s.⁴¹

It seems safe to say that, of all the measures taken to minimize the incidence of postdural puncture headache, the reduction in needle gauge and attention to atraumatic needle tip design have probably been of the greatest benefit.⁸¹ Over the years, a multitude of other measures have been suggested to aid in the prevention of postdural puncture headaches.⁸² One recent investigation, for example, was able to show that the subarachnoid injection of 10 mL sterile saline at the time of accidental dural puncture with an epidural needle resulted in a significant reduction (32% v 62% in controls) in the subsequent development of PDPH.⁸³ Even today, there seems to be an enduring hope that, with the proper technique, spinal headaches can be prevented entirely. Yet, despite a great deal of progress in reducing the incidence of PDPH, it is unlikely to be eliminated.

Part 3: Management and Treatment of PDPH

Conservative Management of PDPH

Despite all the early measures undertaken to prevent PDPHs, they continued to be a significant problem. Fortunately, most headaches (approximately 85% of cases) were mild to moderate in severity and were adequately treated with common analgesics. Even in the most severe cases, headaches were generally relieved by maintaining the horizontal position.

Gradually, accepted guidelines for the management of PDPHs were developed. Reassurance and placing the suffering patient in a quiet, darkened room was advised. The supine or Trendelenburg position was usually advocated. Readily available analgesics were routinely administered as needed. A wide variety of other pharmacologic agents were used and commonly included contemporary treat-

ments for migraine or other headaches (such as ergotamines).⁸⁴ Adequate hydration, either oral or intravenous (if necessary), was stressed. Hydration was also achieved through the prevention of diuresis (as with vasopressin)⁸⁵ or expansion of intravascular volume (as with corticosteroids).⁸⁶ Abdominal binders were also commonly used.⁸⁷ A number of other measures were recommended and instituted. Yet, because of the generally self-limited nature of such headaches, the mainstay of each of these "treatments" seemed to be the simple passage of time.

Conservative management, which relied so heavily on time, was obviously inadequate. Rapid, direct treatment of PDPHs was desired and vigorously pursued. These approaches to treatment proceeded along 3 not entirely separate lines: the reconstitution of normal cerebrospinal fluid pressures, producing "splinting pressures" in the epidural space, and a mechanical plugging of the dural hole.

Direct Treatment of PDPH

Reconstitution of Normal Cerebrospinal Fluid Pressures. Recognition of the fact that postdural puncture headaches were frequently associated with decreased cerebrospinal fluid pressures naturally led to efforts to reestablish normal cerebrospinal fluid pressures. This was accomplished by either replacing the lost fluid directly or attempting to increase its production.

The first to restore normal cerebrospinal fluid pressures were Jacobaeus and Frumerie³⁰ who, in 1923, reported their experiences with 2 patients. The first, a 15-year-old girl, had a lumbar puncture as a diagnostic evaluation for "fits" resembling epilepsy. She rapidly developed a "very bad headache" that worsened over the course of several days until she was believed to be "very near moribund." A repeat lumbar puncture was performed to determine the cerebrospinal fluid pressure, which was discovered to be extremely low (0.5-1.5 cm). Carefully noting the patient's response, Frumerie then slowly injected a total of 90 mL saline solution through the needle (stopping when the pressure was determined to have returned to normal). They noted: "The patient's condition improved immediately during the salt injection. She got brighter, answered quicker on questions, had a livelier expression on her face and showed considerably improved general tonus. The pulse got quicker and the patient felt afterwards subjectively improved."³⁰

Soon the patient was in her usual state of health and discharged home. They went on to report a similar subarachnoid injection of saline in a second patient experiencing headache after diagnostic lum-

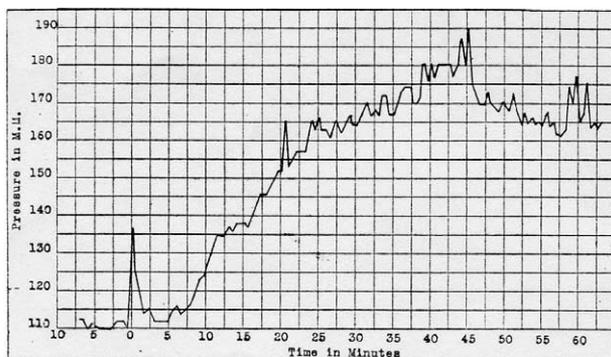


Fig 3. Effect of intramuscular injection of pituitary extract on cerebrospinal fluid pressure (from Solomon).⁹¹ (Reprinted with permission from *JAMA* 1924;82:1512-1515. Copyrighted © [1924], American Medical Association. All rights reserved.)

bar puncture. The second patient's symptoms also rapidly resolved. They concluded in both cases that "the therapeutic effect of the injection was quite indisputable" and that, in the case of the 15-year-old girl, had a "great probability of saving the life." Further attempts at replacing cerebrospinal fluid losses were made by various authors, with some success.^{34,88-90} Generally, however, the subarachnoid injection of saline was perceived as providing only temporary symptomatic relief (while having the very definite disadvantage of creating another dural defect); nevertheless, reports of significant success with this method continued into the 1950s.

Even more attractive than the prospect of another transdural needle insertion to directly replace lost fluid was the concept of promoting the increased production of cerebrospinal fluid. As the physiology and dynamics of cerebrospinal fluid became known, methods to increase its production came to light. These measures primarily consisted of injections intravenous hypotonic saline and intramuscular pituitary extract (Fig 3).^{91,92} Such interventions were quickly adopted for the treatment of PDPH with a modest degree of success.⁹³

Producing "Splinting Pressures" in the Epidural Space. The concept that negative pressure in the epidural space could result in a persistent leakage of cerebrospinal fluid was introduced by Heldt and Maloney³⁵ in 1928. In 1929, Heldt "undertook to interrupt the postlumbar puncture headache by injecting distilled water or normal saline into the epidural space."³² Although he reported that 20 to 30 mL injected in this manner "promptly interrupted the headache," he also noted that the relief obtained was only temporary (from 3 to 48 hours).

In 1948, Drs. Glen Rice and Harwell Dabbs, in Baltimore, "instituted peridural injection of saline

solution to produce a splinting "head of pressure" in the peridural space, attempting to prevent leakage through the subarachnoid-peridural fistula long enough for a fibrin seal to occlude the aperture."⁹⁴ By using a lumbar epidural catheter, they were able to repeatedly inject saline into the epidural space over time, curing headaches using an average total of 82 mL saline. Of the 22 patients described in their report, 21 noted immediate relief and 10 reported complete and lasting relief (with the other 12 reporting a slight return of mild symptoms). Murry et al.,⁹⁵ in 1956, modified the technique of Rice and Dabbs to a caudal injection of saline (an approach recommended because of the reduced likelihood of repuncturing the dura) and reported successful treatment of PDPH in 204 of 243 patients.

Although the epidural injection of saline for the treatment of PDPH gained considerable popularity, it was plagued by a high rate of recurrence of symptoms. The return of symptoms is not surprising considering the very transient nature of the "splinting pressures" produced, with epidural and subarachnoid pressures returning to baseline within minutes of injection.⁹⁶ Given this evidence, permanent cures using epidural saline are thought to be produced by an inversion of a dural flap (the so-called "tin-lid" phenomenon),⁹⁷ thereby preventing further cerebrospinal fluid loss. This proposed mechanism of action helps to explain reports of the utility of epidural saline (used in combination with the blood patch) in situations involving refractory PDPHs.^{98,99}

Obviously, any number of different materials could be used to increase epidural pressures. Even the epidural injection of air has been suggested in an effort to produce a splinting head of pressure that could then allow a fibrin clot to seal the dural rent.⁷⁶ No published studies are available that might show the efficacy of this novel approach.

Plugging the Hole in the Dura. The most direct solution to a dural hole allowing persistent leakage of cerebrospinal fluid would appear to be mechanically plugging the defect. However, because the precise location of the puncture is unknown when treating postdural puncture headaches, efforts in this regard would primarily involve the promotion of clot formation in the epidural space. Robert Danis¹⁰⁰ of Belgium, in 1924, described the injection of artificial serum (developed during World War I) into the epidural space in 10 patients suffering from PDPH. He reported good and permanent relief of symptoms with 80 to 100 mL serum and concluded that such epidural injection "constitutes a surely harmless and likely effective treatment of the consequences resulting from spinal puncture." In Michigan, Thomas Heldt³² stated in 1929 that his group had also considered the epi-

dural injection of an artificial serum (gum acaia solution), but had not done so. Thus, the epidural injection of artificial serum, although initially promising, never seemed to gain clinical favor.

Foreshadowing the intentional modern use of blood for this purpose, Quincke had suggested in 1891 that, despite his efforts to produce sustained cerebrospinal fluid leakage and reductions in intracranial pressure, it appeared that “an opening can become obstructed by arachnoid membrane or clot.”¹⁷ Ingvar, in 1923, also noted that cerebrospinal fluid leakage through dural wounds created during surgery seems to rapidly and spontaneously stop. He stated, “Apparently the bleeding into the canal gives a fibrinous coagulum which blocks the passage.”³¹ Nelson,⁷⁴ in 1930, was also of the impression that bleeding encountered during the course of a lumbar puncture may help to seal the dural rent. More recently, it has even been suggested that the successful treatment of PDPHs using epidural saline administration may be caused in no small part by trauma and bleeding associated with the technique.⁵²

In his exhaustive review, *Neurological Complications After Spinal Anaesthesia*, Gunnar Thorsen in 1947 mentioned the works of Danis and Heldt and concluded, “After removal of the dural puncture needle, sealing of the dural cavity would, perhaps, be possible by first injecting via the introducer a few millilitres of a fibrinogen solution or blood, and then a thrombin solution.”¹⁰¹ Unfortunately, neither Thorsen nor his readers ever appear to have put these thoughts into action and the concept of a true blood patch to stop a dural leak would be delayed until Gormley in 1960.

Part 4: Spinal Anesthesia in the 1950s and Dr. James B. Gormley

Spinal Anesthesia in the 1950s

By 1950, spinal anesthesia was widely recognized as a flawed but acceptable anesthetic choice.^{102,103} The decade to come, however, proved to be a tumultuous era for spinal anesthesia. In 1950, an article published entitled “The grave spinal cord paralysis caused by spinal anesthesia” by Drs. Foster Kennedy (a prominent New York neurologist), Abraham Effron, and Gerald Perry generated considerable widespread critical analysis of the technique.¹⁰⁴ In this article, which reads like a compendium of spinal neurologic complications, they detailed 12 cases of spinal cord paralysis after spinal anesthesia. Although the article was generally deficient in the details of the spinal anesthetics performed, it would certainly give practitioners pause to consider their choice of spinal anesthesia. The authors strongly concluded that “spinal anesthesia

is accompanied by many definite and terrible dangers which are far too little appreciated by surgeons and anesthetists” and that “paralysis below the waist is too large a price for a patient to pay in order that the surgeon should have a fine relaxed field of operation.”

The widespread dissemination of Kennedy’s damning article may well have resulted in the elimination of spinal anesthesia as a reasonable anesthetic choice in the eyes of much of the public. Indeed, it is said that during this time some patients would actually “cringe and withdraw” at the mention of the word “spinal.” Those performing anesthesia also knew, to quote a contemporary anesthesiologist, that “if a patient died under general anesthesia the practitioner could casually and with a straight face coolly inform the family that the patient was “allergic to” or “couldn’t take” the anesthetic . . . reasoning readily accepted by the family at that time. . . . Moreover, they argued that the complications of a spinal anesthetic (meningitis, arachnoiditis, paraplegia, etc.) couldn’t be buried so readily and remained a lifetime reminder of the forever present evils of the technique” (A. J. DiGiovanni, personal communication, February 2001).

Another devastating blow to the reputation of spinal anesthesia at this same time, especially in the United Kingdom, was the well-publicized case of Woolley and Roe versus Ministry of Health and Others.¹⁰⁵ Two patients (Albert Woolley and Cecil Roe) receiving spinal anesthetics on the same day (October 13, 1947) at the same hospital (Chesterfield Royal Hospital, Great Britain) became permanently paraplegic. They jointly sued in 1953, with expert witnesses for the prosecution stating bluntly that, in their opinion, the blame lay with the technique of spinal anesthesia itself. Largely through evidence presented by Professor Robert Macintosh, it was eventually accepted that the adverse outcomes in these cases were almost certainly because of undetected cracks in the local anesthetic ampules, which would have allowed the passage of phenol into the ampules during sterilization. Nevertheless, this high profile case proved to be a near-fatal blow to the use of spinal anesthesia in the United Kingdom and its damaging effects would last for many years.¹⁰⁶

However, it would be the superb and timely analysis of spinal anesthesia by Drs. Robert Dripps and Leroy Vandam that not only shed light on the shadowy concerns of neurologic complications but also summarized and crystallized the knowledge of spinal anesthesia at that time. Their first article on this topic, published in 1954, was entitled “Long-term follow-up of patients who received 10,098 spinal anesthetics. Failure to discover major neurological sequelae.”¹⁰⁷ In this landmark analysis, they failed

to discover any “persistent, progressive, major neurological disease” and concluded that PDPH is for all practical purposes *the* neurologic complication after spinal anesthesia. In their study, they noted an overall incidence of PDPH of 14%. Their follow-up article, “Long-term follow-up of patients who received 10,098 spinal anesthetics. Syndrome of decreased intracranial pressure (headache and ocular and auditory difficulties),” published in 1956, was a timeless evaluation of the demographics of PDPH.¹⁰⁸ Their data strongly indicated that the syndrome resulted from a decrease in cerebrospinal fluid pressure and that this decrease was caused by a leakage of this fluid. Although not failing to emphasize the “seriousness of the complications of spinal anesthesia,” they concluded that such complications are “largely preventable if proper practices are taken.” Furthermore, they proposed that PDPHs could be virtually eliminated through the use of smaller needles (24 gauge or smaller). By putting the sequelae of spinal anesthesia in proper perspective, these 2 publications probably contributed more to salvaging the reputation of spinal anesthesia than any others. Other articles supported the work of Dripps and Vandam, further showing in large case series the safety of properly performed spinal anesthesia.¹⁰⁹⁻¹¹²

Yet, despite its apparent drawbacks, there are a number of reasons why some practitioners might prefer to stay with the technique of spinal anesthesia.¹¹³ The most obvious was the fact that general anesthesia at the time (usually the notoriously flammable ether) was not significantly better in the eyes of many.¹¹⁴ Numerous references were made to the fact that headaches also occurred commonly after general anesthesia, certainly even more commonly than the relatively low incidence of headache seen after spinal anesthesia.^{115,116} Indeed, the headaches after general anesthesia were reported by some to be even more severe than those after spinal anesthesia, which could usually be relieved by simply remaining recumbent. Also, in an era when even healthy patients would expect to be hospitalized for extended periods after relatively minor procedures, the headaches after spinal anesthesia (which rarely lasted longer than a week) were often perceived as more of an inconvenience than a problem. With few trained anesthesia personnel, spinal anesthesia also essentially allowed surgeons to perform both the anesthetic and the surgery. Spinal anesthesia would, in most hands, also have the advantage of a much faster onset of surgical anesthesia than ether.¹¹⁷ Although more of an impression than fact at the time, some clinicians also believed that spinal anesthesia possessed some yet-unproven benefits (such as decreased blood loss) when compared with general.¹¹⁸ Furthermore,

for many procedures spinal anesthesia also provided excellent muscle relaxation (especially attractive in the early days of using neuromuscular blocking drugs, which were introduced clinically in 1942).¹¹⁹

Proponents of regional anesthesia, such as Dr. John Bonica, were of the opinion that these techniques were underutilized during the 1950s.¹²⁰ Although the reasons for this practice would have had many causes, one factor must have been that during this decade spinal anesthesia was generally reported as having a 10% to 15% incidence of PDPH (15%-20% in obstetrics).^{121,122} Furthermore, treatment of PDPH at the time was simply inadequate and was as outlined by Dr. Daniel Moore in *Complications of Regional Anesthesia*.¹²³ In this 1955 text, he recommends an abdominal binder, analgesics, hydration, and other measures (in this case nicotinamide and pituitrin) which, if unsuccessful, can be followed by epidural saline. As a final observation, he notes that to proceed through a full treatment protocol would take about 3 days, the length of time that would be expected for many mild to moderate headaches to resolve spontaneously. Moore concludes his honest commentary by stating, “Nevertheless, the patient feels an attempt to help his problem is being made” (Fig 4). Others at the time would agree that “Time still is the surest cure.”¹²⁴

The “exasperating complication”⁸⁵ of PDPH and its management were essentially as described by Moore when Dr. James B. Gormley began his practice of general surgery in the small town of Berwick, PA, in 1955.

Dr. James B. Gormley

A native Pennsylvanian, Dr. Gormley was born in Hazeltown in 1915 and would spend most of his life in the state. Described as a brilliant “detail man,” Dr. Gormley had originally gravitated toward engineering but, because there were few such jobs in sight during the Great Depression, changed his focus to medicine. He graduated from Lehigh University in 1936 and completed his medical studies at Jefferson Medical College, in Philadelphia, in 1941. He then served during World War II in the Army Medical Corp on a battleship in the Caribbean. His general surgery training was fulfilled at the Flower and Fifth Avenue Hospitals in New York City (Fig 5).

Private practice for a general surgeon in a town the size of Berwick (the population in 1960 was 13,353) was full of challenges in the 1950s and 1960s. Because there were no trained anesthesia personnel in the community until 1970, the options for anesthesia were essentially local, spinal, or gen-

METHODS USED AT THE MASON CLINIC TO
TREAT POSTSPINAL HEADACHES FROM
LOWERED SPINAL FLUID PRESSURE

I. When diagnosis is made the following orders are written:

- A. Apply abdominal binder snugly over 2 bath-towels before patient sits up or is ambulatory. **Rationale:** Decrease epidural space; increase spinal fluid pressure.
- B. Codeine gr. $\frac{1}{2}$ (30 mg.) and Aspirin gr. 10 (600 mg.) p.r.n. for headache. **Rationale:** Symptomatic relief of headache.
- C. Measured oral intake of at least 3,000 cc. of water for 4 days. If this is not met, supplement with 5% dextrose in distilled water intravenously. **Rationale:** Hydration.
- D. Nicotinamide $1\frac{3}{4}$ gr. (100 mg.) intramuscularly 3 times a day for 2 days. **Rationale:** Dilatation of choroid plexus.
- E. Surgical pituitrin (1:2000) 1 cc. intramuscularly 3 times a day for 2 days. **Rationale:** Antidiuretic action.
- N. B. Watch for "pituitrin shock"; if it occurs, stop pituitrin. Do not give drug to patient with heart disease, hypertension, toxemia, pre-eclampsia or eclampsia.

II. If severe headache persists after this treatment, then the placement of a lumbar epidural (peridural) catheter and the injection of normal saline should be evaluated. **Rationale:** Decrease epidural space, increase spinal fluid pressure.

NOTE: Treatment as described takes 3 days—the length of time a mild or moderate postspinal headache of this nature lasts—and therefore its efficacy may be questioned. Nevertheless, the patient feels an attempt to help his problem is being made.

Fig 4. Treatment protocol for postdural puncture headache at the Mason Clinic during the 1950s. (Reprinted with permission.^{1,2,3})

eral. Spinal anesthesia was usually performed by the surgeon with the patient, then attended to by a nurse. Because a surgeon could not perform surgery and administer general anesthesia at the same time, general anesthetics were either provided by a general practitioner or untrained nurses, who were supervised by the surgeon. In addition, the term "general surgeon" was certainly no misnomer, and Dr. Gormley's practice also included frequent obstetric, gynecologic, orthopedic, and otolaryngologic cases.

Under such circumstances, it is easy to understand the appeal of spinal anesthesia. With spinal

anesthesia, after the surgeon had performed the block, nurses were often simply given a syringe of ephedrine and instructed to obtain vital signs, awaiting further instruction. Despite its shortcomings, spinal anesthesia usually permitted surgeons more control over the entire anesthetic process and, of critical importance, allowed patients to control their own airway. For Dr. Gormley, by all accounts an exacting surgeon and described as a man who "walked around like he had a micrometer in his pocket," (T. Cretella, personal communication, March 2001) spinal anesthesia must have been very attractive.

In Berwick, Dr. Gormley had ample experience with spinal anesthesia and, unavoidably, the subsequent headaches. Although some believe that he may have been influenced by certain publications (such as Thorsen¹⁰¹), it seems that 2 of his clinical impressions would most strongly lead him to inject blood to seal the hole left by a puncture of the dura. His first impression was that extravascular blood would result in a local reaction that could result in the sealing of the dural hole, a concept he referred to as "blood irritation." He put this concept into practice, for example, by injecting a small amount of a patient's autologous blood into the region of their lateral epicondyle to treat tennis elbow and



Fig 5. Dr. James B. Gormley (1915-1997).

believed that small amounts of blood should be adequate for most purposes. His second impression was that encountering blood during the conduct of a spinal anesthetic (“bloody taps”) resulted in a lower incidence of headaches. Dr. Gormley’s further observation that bloody taps were relatively common and otherwise inconsequential also led him to act against the prevailing impression of the day: that blood in or near the central neuraxis could only have dire consequences.

Reportedly, Gormley’s first experience with the blood patch was in a female patient who was experiencing a severe PDPH 4 days after spinal anesthesia for varicose vein stripping. In this patient, whose identity remains unknown, he performed a repeat lumbar puncture at the same interspace and then injected 15 mL of sterile saline (after Jacobaeus and Frumerie). The needle was then slowly withdrawn, and, when cerebrospinal fluid return ceased, the needle was assumed to be in the epidural space. At this point, 2 mL of the patient’s freshly drawn blood were injected into the epidural space. During the course of this procedure, 1 L of intravenous fluid was also administered. This patient’s headache was reported to be relieved immediately and permanently.

Under Dr. Gormley’s direction, 6 further cases of PDPH were cured with the epidural injection of 2 to 3 mL autologous blood alone (without intrathecal saline). One of these 6 patients was Dr. Gormley himself, who had developed a severe headache after a myelogram. He subsequently had a colleague, Dr. Thomas Patrick (a general practitioner), perform an epidural blood patch on him, once again with excellent symptomatic relief. Perhaps fitting for such an innovation, Dr. Gormley performed these first procedures with the epidural “blood patch” in the recovery room of the relocated, newly opened Berwick Hospital (Fig 6). Dr. Gormley reported these initial 7 experiences with his epidural blood patch to *Anesthesiology*, and they appeared as an unassuming article in the “Current Comment” section in 1960 (paraphrased by section editor Dr. Stuart Cullen).¹²⁵ It is interesting to note that Dr. Gormley’s report appeared in the same issue in which Dr. John Bonica wrote an editorial in response to the apparently declining use of regional anesthesia in private practice.¹²⁰

Despite subsequent modifications and general acceptance of the epidural blood patch, Dr. Gormley, a surgeon untrained in true epidural techniques, continued to quietly perform his version of the blood patch with good results until his retirement in 1981. In situations at high risk for PDPH such as caesarean deliveries, he would perform the spinal anesthetic with a large-bore needle and then prophylactically inject a 2-mL fresh autologous “epi-

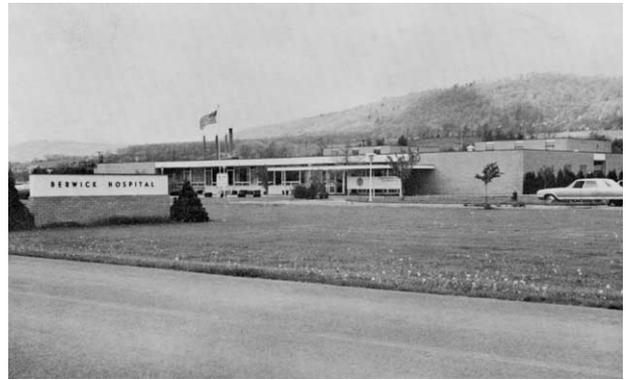


Fig 6. Berwick Hospital, Berwick, PA (ca. 1960). Photograph courtesy of The Berwick Historical Society.

dural” blood patch as the needle was withdrawn. He is said to have performed this prophylactic blood patch several hundred times with only one known failure. Unfortunately, Dr. Gormley never published other than the solitary report in *Anesthesiology* and never received any formal recognition for his part in this landmark development. During his later years in practice, he displayed little interest in the blood patch but instead turned his creative energies toward orthopedic projects, being especially enthusiastic about sliding screw and plate devices that would allow bones the freedom to elongate as they healed. After his retirement he remained active, particularly enjoying his favorite pastime of golf, and died in Florida in 1997 at the age of 82.

Bloody Taps

*Make thick my blood,
Stop up the access and passage . . .*

William Shakespeare
Macbeth, Act I, Scene V

Dr. Gormley’s observation that “the incidence of headache is not as high as anticipated when a bloody tap is produced” naturally invites an investigation of the known data in this regard. Because spinal anesthesia had been in common practice for over half a century by the time Gormley made this statement, it is not surprising that numerous researchers had indeed looked into the issue of whether bloody taps decreased the incidence of postdural puncture headache. As previously mentioned, the idea that blood could plug a dural hole was not a new one and actually dated back to Quincke. Yet an early prevailing opinion was that traumatic taps, with the introduction of blood across the dura, would cause an aseptic meningeal reaction and actually result in increased cerebrospinal fluid pressures, thereby causing headaches.

By 1960, a number of publications had addressed the question of whether “bloody taps” influenced

the risk of developing a PDPH. Nelson, in 1930, based on his pathologic examination of the dura after dural puncture, stated that “if, during puncture, the needle were to strike a vascular portion and rupture a blood vessel, bleeding would take place into the wound and help to plug it.”⁷⁴ He believed that bloody taps should result in a lower incidence of postdural puncture headaches, yet, when he analyzed whether fresh blood was absent or present in the spinal fluid after a tap, he was unable to show any difference in the incidence of “postpuncture reactions.” Koster and Weintrob,²⁵ also in 1930, were of the opposite opinion (i.e., that bloody taps would result in an increased incidence of PDPHs). They reported having paid particular attention to the incidence of headache after bloody taps and found no such increased incidence.²⁵ Emory,¹²⁶ in 1948, reported that the presence of blood in the spinal fluid after dural puncture did not appear to have an appreciable effect on the incidence of spinal headache. In 1953, Harris¹²⁷ again reported that blood in the spinal fluid at the time of the tap appeared to have no influence on the incidence of spinal headache. Notably, Krueger,¹²⁸ also in 1953, reported that traumatic punctures resulted in a remarkably increased incidence of postspinal headaches, but his analysis was marred by the fact that his “traumatic taps” not only included bloody taps but also cases of multiple punctures of the dura.

On the whole, it would seem that Dr. Gormley’s subjective observation that bloody taps resulted in fewer PDPHs was certainly not borne out through the studies of his day. A more recent analysis of accidental dural taps during attempted epidural analgesia for labor was performed from 1976 through 1988. In this study, Khan et al.¹²⁹ also failed to find a statistically significant difference in the incidence of headache in those with (59%) or without (48%) bloody taps and concluded that “it is, perhaps, fortunate that this information was not available to Gormley.”

Part 5: The 1960s, DiGiovanni and Ozdil

Treatment of PDPH Through the 1960s

It seems that Dr. Gormley’s report in *Anesthesiology* may have been either too unassuming or too radical for most practitioners, as it appeared to generate little serious interest. In 1962, what was thought to be the first report of surgical treatment for “post puncture headache” appeared in the medical literature.¹³⁰ A 1964 monograph by Dr. Wallace Tourtellotte and colleagues dedicated to the subject of postlumbar puncture headaches is perhaps most representative of the uncertainty regarding treatment for PDPHs during the 1960s.¹²² In this lengthy

manuscript (120 pages), designed to comprehensively review the “methods of treatment and prevention of post-lumbar puncture headaches,” 49 separate recommendations regarding the treatment or prevention of such headaches are noted. Despite including intrathecal vitamins, x-rays to the skull, sympathetic blocks, and even enemas before a dural tap, the epidural blood patch was not mentioned. To the authors’ credit, they concluded that, “It would appear that future therapeutic trials to prevent post-L.P. headaches should be directed at the site of the L.P. Perhaps, fibrinogen or some other clot promotional agent could be injected into the epidural space as the needle is withdrawn from the subarachnoid space. This would give the best chance of positioning an agent to promote sealing and healing of the dural hole.” They went on to state, “We have not injected fibrinogen because the hepatitis virus may be associated with this blood fraction.”

Gormley’s report was mentioned in passing by several authors during the 1960s. In 1963, Dr. Arthur Tarrow mentions Gormley’s technique in an article entitled “Solution to spinal headaches” published in *International Anesthesiology Clinics*.¹³¹ Yet, despite the promising title, this article actually concentrated on the prevention of spinal headaches, primarily through the use of 26-gauge needles. There is no evidence that Tarrow ever tried the method described by Gormley. Dr. John Bonica also mentions Dr. Gormley’s technique in his *Principles and Practice of Obstetric Analgesia and Anesthesia* in 1967 but concludes by stating that the epidural injection of saline is “a more rational approach.”¹³²

The only report of an attempt to duplicate Gormley’s technique appears to have come from Venezuela in 1963.¹³³ Dr. Edgar Martinez Aguirre, of the opinion that “The prevention of the escape of the cerebrospinal fluid into the peridural space is the logical solution to the problem,” had read in *Anesthesiology* of Gormley’s success and “decided to put it to the test.” His article, published in *Pub Centro Medico de Caracas*, describes the placement of a needle into the peridural space (without dural puncture), rapidly followed by the injection of 2.5 to 3 mL fresh blood. In fine detail, he reports success in 3 of 3 patients treated, comments on the “dramatic nature of the relief produced,” and concludes that the technique clearly warrants further study. Although he reportedly continued to perform and study the procedure, it does not appear that his experiences were further published.

An interesting and often overlooked aspect of the entire issue of PDPHs was the general perception of the problem. It seems that for generations many physicians simply failed to see postlumbar puncture headaches as a legitimate medical problem. It had

long been appreciated that these headaches occurred most commonly in young female patients and most notably in the obstetric subset of this population. In this light, it is significant to note that obstetric patients have been found to have a high incidence of headache complaints with or without anesthetic interventions.¹³⁴ A direct result of these observations was a widely held impression that such headaches were more psychogenic than physiologic. Early reports of headache after unsuccessful spinal anesthesia or attempted lumbar puncture (i.e., failure of cerebrospinal fluid return) were taken by some as evidence of the “hysterical” nature of PDPHs. Not surprisingly given these considerations, “constitutional makeup and disturbed psychogenic influences” were long considered major risk factors for the development of postlumbar puncture headache.^{135,136} Consequently, avoiding spinal anesthesia in patients with a previous history of postdural puncture headache was considered highly desirable by most practitioners (and probably patients alike).¹³⁷ By the 1950s, common suggestions to reduce the risk of PDPH included avoiding spinal anesthesia in neurotics, not telling patients of the risk of headache before performing spinal or epidural anesthesia, and never mentioning the word “headache” in the presence of patients.^{124,138} One small study, published in 1981, indeed found that telling patients that they may experience a headache after lumbar puncture proved to be a “self-fulfilling prophesy.”¹³⁹ Reinforcing the impression that constitution played a major role in the development of postdural puncture symptoms were reports such as that of Levin, who, in 1944, reported an incidence of headache of less than 1% in 2,237 male patients after diagnostic spinal puncture in a military setting.¹⁴⁰ One 1967 study actually compared the incidence of headache after dural puncture with that following a “sham” lumbar puncture.¹⁴¹ This small study of 100 patients noted a high incidence of headache in both groups (28% in the tap group *v* 22% in the sham group) and concluded that “psychogenic factors are of primary importance in the etiology of postpuncture headaches.” It is notable that even more recent studies, Lee in 1991 for example, have continued to remark on a “large psychological . . . component to post-myelogram headache.”¹⁴²

Although psychological factors are almost certainly a component of all headaches, the organic basis of many conditions has only recently come to light. It is interesting to note, for example, that current evidence indicates that PDPHs may be physiologically similar to tension-type headaches and that a preexisting history of tension-type headaches may be predictive of PDPHs.¹⁴³ Although our understanding of PDPH is still far from complete, these

and other recent findings only serve to further support a primarily physiologic nature to this problem. It is easy to imagine how longstanding perceptions of this problem being primarily of psychogenic origin may have contributed to a delay in the development of effective treatments.

Prophylactic Blood Clot Patch

In the early 1960s, Dr. Turan Ozdil, an instructor of anesthesiology at the University of Tennessee in Knoxville, began an interesting independent line of work that would parallel that of Gormley. One day, around 1960, as he was observing a “tubeless tire repair” (in which a piece of rubber is pulled across a hole in an automobile tire using a device resembling a crochet hook), Dr. Ozdil imagined how a hole in the dura could be similarly plugged by a small amount of clotted blood. This simple observation first led to trials using clotted blood to plug needle holes in a plastic bag filled with water and then across the dura in a dog model (Fig 7). These successes led to the injection of clotted blood across the dura in humans at the time of needle withdrawal after subarachnoid local anesthetic administration. In his initial clinical trials, he noted no headaches in a treatment group of 100 subjects compared with a 15% incidence in a control group.¹⁴⁴

As Ozdil and his associate, Dr. W. Forrest Powell, began their research around 1960, they were unaware of Dr. Gormley’s published report. They subsequently “discovered” Gormley’s 1960 article when they went to write their article, published in *Anesthesia & Analgesia* in 1965.¹⁴⁴ This article generated a great deal of interest nationally and internationally, and a National Institutes of Health grant was approved for further related research. This research was never performed, however, primarily because of Dr. Ozdil’s other clinical and teaching obligations.

Ozdil and Powell’s technique, the first prophylactic blood patch, although successful, soon proved to be impractical. In an era when the main focus for reducing the incidence of PDPH was through the use of smaller needles, it was impossible to inject the preclotted blood through needles smaller than 20 gauge. To allow time for the blood to clot, they had drawn their blood approximately 20 minutes before lumbar puncture, greatly adding to the time required to perform a generally quick procedure. It also seemed excessive to prophylactically treat the 85% of patients who would not have gone on to develop a PDPH with such an involved modification of normal spinal technique.

Although their technique was ultimately abandoned, the work of Ozdil and Powell proved to be

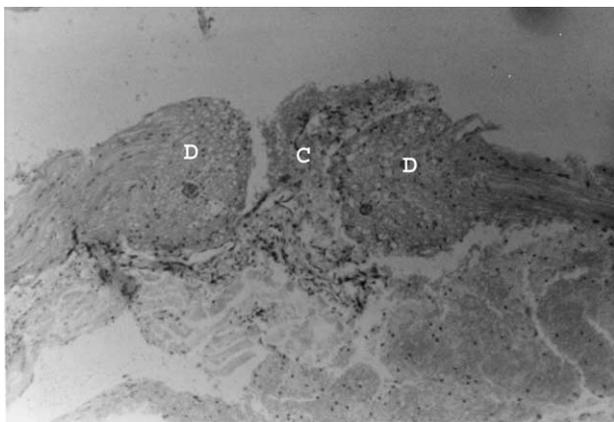


Fig 7. Prophylactic transdural clot patch in a dog model. Photograph shows clot (C), plugging a hole in the dura (D). (Reprinted with permission.¹⁴⁴)

extremely timely. Theirs was the first real study (rather than a simple case report of a few patients) of using blood as a means of plugging a dural hole and was published in a widely read journal. Their work greatly helped to prepare the way for the widespread acceptance of the use of blood to treat symptomatic holes in the dura.

Dr. Anthony DiGiovanni and Development of the Modern Epidural Blood Patch

Obviously, the blood patches as described by Gormley and Ozdil bear little resemblance to the modern epidural blood patch technique. However, Gormley had formally introduced the idea into clinical practice, Ozdil and Powell had more widely disseminated the concept, and it would be Dr. Anthony DiGiovanni who would refine and promote the procedure. Not surprisingly, Dr. DiGiovanni was trained in anesthesiology at the University of Pennsylvania by Drs. Dripps, Vandam, and James E. Eckenhoff during the 1950s, a time (as mentioned previously) when spinal anesthesia was under intense scrutiny. During this period, largely against the trend of the time, he was trained to rely preferentially on regional techniques (Fig 8).

Upon completing his residency, Dr. DiGiovanni went back on active duty in the Air Force and was assigned to Eglin Air Force base in Florida, where he and a nurse comprised the anesthesia staff. He recalls having just returned from Christmas leave in 1960 when he was asked to assist in the care of a patient on the obstetric ward who was complaining of a severe headache after a spinal analgesia given for a vaginal delivery several days previously. Having just read Dr. Gormley's letter in *Anesthesiology*, he was eager to try the new blood patch procedure. However, on examining the patient, he saw that her lumbar region showed evidence of attempted

spinals at several levels, and the obstetrician who had given the analgesic was unable to recall the interspace at which he had eventually punctured the dura. Faced with a cerebrospinal fluid leak at an uncertain level, Dr. DiGiovanni decided to inject a larger volume of blood (10 mL) epidurally to improve spread and increase his chances of success. This initial attempt was indeed successful and encouraged him to offer and provide the blood patch procedure whenever indicated. He continued to perform the epidural blood patch using 10 mL and never tried other volumes of blood because he "couldn't be more satisfied" with the results.

Other than Martinez Aguirre in Venezuela, Dr. DiGiovanni appears to have been the only practitioner to be performing a true epidural blood patch until 1963. In that year, he was transferred to Wilford Hall Air Force Hospital in San Antonio, Texas, as Chief of the Anesthesiology Service and Director of Residency Training. It was there that he was to train many anesthesiologists as well as residents in the blood patch technique. Naturally, Dr. DiGiovanni was concerned about the safety of the novel procedure and stated that he "would quit if there was the least hint" that it might cause any harm. In addition to his own uncomplicated experiences, he contacted both Drs. Gormley and Ozdil, through

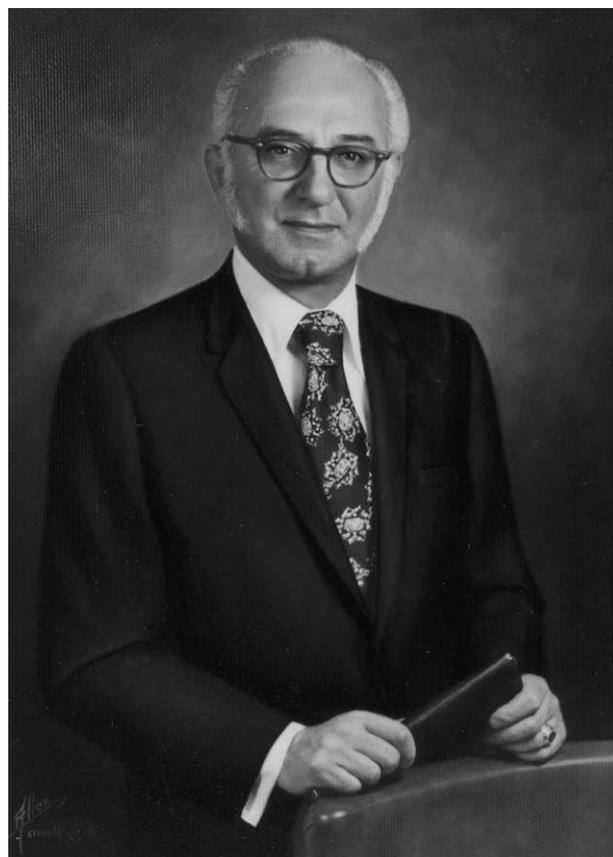


Fig 8. Dr. Anthony DiGiovanni (1918-).

whom his perception of safety in using blood to plug a dural defect was further confirmed. In the late 1960s Dr. Burdett Dunbar, one of Dr. DiGiovanni's staff, suggested that knowledge of the blood patch be more widely disseminated. Drs. DiGiovanni and Dunbar described their technique as well as results in a work entitled "Epidural injections of autologous blood for postlumbar-puncture headache."¹⁴⁵ This study, initially rejected by the journal *Anesthesiology*, was published in *Anesthesia & Analgesia* in 1970.

In this study, DiGiovanni and Dunbar were the first to describe the modern "epidural blood patch" technique using a blood volume of 10 mL. They reported immediate and permanent relief of headaches in 41 of 45 patients as well as no headaches in 5 patients treated prophylactically (immediately after dural puncture). After presenting this data at the American Society of Anesthesiologists Annual Meeting, Dr. DiGiovanni was approached by Dr. John Bonica who, although acknowledging that the procedure had a great deal of potential, had concerns about the safety of the procedure. Noting that blood agar was a great medium for culturing bacteria, he suggested that Dr. DiGiovanni continue to study the blood patch procedure on an animal model. Additionally, a short time after the meeting, Dr. DiGiovanni received a letter from his mentor, Dr. Robert Dripps, who closed it humorously by asking "whom do you think you are, Wild Bill Donovan?" (a general surgeon known for having excised a sebaceous cyst of the scalp under spinal anesthesia). With such reserved encouragement from some of the giants of the profession, DiGiovanni then set out to relieve some of the trepidation associated with the concept of the blood patch through the use of an animal model. Although a multitude of potential complications were possible with the epidural blood patch, the most serious appeared to be those of epidural abscess and arachnoiditis. He reasoned, however, that with proper aseptic technique the incidence of epidural abscess should be no higher than after routine lumbar puncture. Therefore, he sought primarily to address the concerns about arachnoiditis by attempting to determine the fate of blood placed into the epidural space.

With PDPH being such a common and aggravating problem, it seems surprising that a truly effective treatment should have taken so many generations of practitioners. Although there appear to be a number of reasons for this delay in definitive treatment, looming large were widely held and longstanding concerns about the detrimental effects of introducing blood near the central neuraxis (which might then possibly enter the subarachnoid space). At Johns Hopkins University in 1928, Dr. Charles

Bagley evaluated the effects of blood in the cerebrospinal fluid. First in an experimental model using dogs, Bagley attempted to produce "lesions simulating those occurring in human beings when a small amount of blood escapes into the subarachnoid space."¹⁴⁶ He concluded that as little as 0.5 mL blood produced "neurologic disturbance varying from slight difference in behavior to severe convulsive seizures." Bagley went on to report on a number of clinical cases of subarachnoid blood having detrimental effects in humans and recommended treatment in such circumstances by the removal of blood from the cerebrospinal fluid using repeated dural punctures.¹⁴⁷ Research by Bagley and others would have a lasting impression on clinicians' attitudes toward the placement of blood near the central neuraxis.¹⁴⁸ Another impediment to the acceptance of the concept of using blood to treat postdural puncture headaches was the lingering belief that many of these headaches were actually due to meningeal irritation caused by small amounts of subarachnoid blood introduced at the time of dural puncture. In his 1957 text, *Lumbar Puncture and Spinal Analgesia*, for example, Sir Robert Macintosh warns that subarachnoid introduction of as little as "a drop or two of blood" may cause an aseptic inflammatory meningeal reaction.¹⁴⁹ Such serious concerns persisted strongly into the 1950s and continue to be voiced even to this day.¹⁵⁰

To address these grave concerns, Dr. DiGiovanni and his colleagues, Drs. Michael Galbert (an anesthesiologist) and William Wahle (a pathologist), investigated the fate of blood placed into the epidural space of Angora goats (selected for their relatively large epidural spaces). Their results, entitled "Epidural injection of autologous blood for postlumbar-puncture headache. II. Additional clinical experiences and laboratory investigation," were published in *Anesthesia & Analgesia* in 1972.¹⁵¹ Pathologic examination of the goat's epidural spaces at intervals up to 6 months after simulated blood patch showed initial organization of a clot (at 24 hours), followed by fibroblast activity (at 4 days), and a gradual disappearance of evidence of blood (by 2 weeks). They concluded that 2 mL autologous blood placed into the epidural space did not appear to be an irritant and actually resulted in no more tissue reaction than many routine lumbar punctures. In this article, in addition to the encouraging pathologic results, DiGiovanni also reported on further clinical experience with the epidural blood patch. Combining his own experiences with that of several authors (including Gormley) he reported a success rate (defined as relief within 24 hours) of 96.5% in over 200 patients using a variety of blood patch techniques. This report was convincing enough to give the procedure popular momentum, and many

other authors would now join in investigating this new technique. Other than in defense of the procedure, Dr. DiGiovanni did not publish further on the blood patch. He retired from the practice of anesthesiology in 1975, and resides in San Antonio.

It is notable that other than the works of Gormley, Ozdil, and Martinez Aguirre, essentially all early reports of experiences with the epidural blood patch can be traced back directly to DiGiovanni. Drs. Frank DuPont and John Vondrell were residents who had trained under DiGiovanni and shared their experiences using the blood patch with colleagues at their next sites of practice. DuPont published his experience with the blood patch in *Michigan Medicine* in 1972,¹⁵² and Vondrell reported his in the *Wisconsin Medical Journal* in 1973.¹⁵³ The first report to gain widespread attention for the procedure was by Drs. Peter Glass and William Kennedy at the University of Washington. In their brief report, published in the *Journal of the American Medical Association* in January, 1972, they describe “immediate and permanent relief of symptoms” in 47 of 50 patients using 5 to 10 mL of autologous blood.¹⁵⁴ Neither of these authors had ever worked directly with DiGiovanni, but Dr. Glass was an Air Force colleague of Dr. Frank DuPont. During their time together, Dr. DuPont taught the procedure to Dr. Glass, who then introduced it to his department in Seattle where his further investigations were performed.

Part 6: Widespread Experience With the Epidural Blood Patch

Further Studies of Safety, Efficacy, and Other Concerns

The early reports by DiGiovanni and those who had been influenced by him went far to relieve many clinicians’ reservations regarding the blood patch procedure. Yet, despite reassuring experience and data regarding most major concerns, there naturally remained some serious lingering doubts about such a unique procedure. One authoritative commentary on the blood patch in 1972 would state that “the risk-benefit ratio is balanced in the wrong direction to make this a useful technic except in rare circumstances” and that “indications for this procedure are rare.”¹⁵⁵ Even as late as 1980, Dr. J. Selwyn Crawford, the British obstetric anesthesiologist, would note, “There is still some disquiet expressed about this technique, which will be overcome only by publication of its safe and successful application in large numbers of cases.”¹⁵⁶

In 1974, the Society for Obstetric Anesthesia and Perinatology undertook a prospective study of the blood patch.¹⁵⁷ This study of 185 patients, reported on behalf of the society by Dr. Gerald Ostheimer

and colleagues, confirmed the efficacy (98.4% success) and safety (no severe or permanent complications were noted) of the procedure. The following year, Dr. Ezzat Abouleish published his experience with the epidural blood patch in another 118 patients, once again noting the safety and efficacy of the procedure as well as a 95% patient acceptance rate at 2-year follow-up.¹⁵⁸

As successful clinical experience with the blood patch procedure mounted, concerns about infection waned. Crawford,¹⁵⁶ in 1980, recommended taking blood for culture at the time of blood patch, but grew no pathologic organisms from 232 cultures. In a subsequent correspondence, he reported a single case of bacteremia after a blood patch in a patient who had a negative blood culture from the patch blood. He believed that the culture obtained at the time of the patch was valuable in exonerating the blood patch procedure.¹⁵⁹ However, taking blood for culture never gained popular favor, and subsequent studies would indicate that the blood patch is essentially free from significant infectious risk even in immunocompromised patients (such as HIV-positive patients).¹⁶⁰

As mentioned earlier, concerns that small amounts of blood administered during a blood patch could cross the dura and cause arachnoiditis were initially addressed through animal studies.¹⁵¹ DiGiovanni’s work using goats was followed by other animal studies, which continued to be reassuring regarding the concerns about arachnoiditis.¹⁶¹ Clinically, however, such worries were only dispelled through the large numbers of uneventful applications of the technique in humans.

In addition to the serious concerns regarding the possibilities of arachnoiditis and infection, there were also concerns that epidural blood would cause scarring and obliteration of the epidural space and that patients would then be refractory to subsequent epidural anesthesia.¹⁶² A number of clinical reports of successful lumbar epidural, caudal, and spinal anesthesia after blood patch greatly allayed these fears.¹⁶³⁻¹⁶⁶ Effective epidural anesthesia has also been reported within minutes or hours after a blood patch.^{167,168} Although there is some evidence of altered spread of local anesthetic after epidural blood patch,¹⁶⁹ it seems that previous dural puncture itself is probably most responsible for any impairment in subsequent epidural analgesia.¹⁷⁰

Another early concern was that blood placed into the epidural space may result in harmful (and not easily remedied) increases in pressure within the epidural compartment. It had long been appreciated that an epidural hematoma after spinal or epidural anesthesia represented a dire circumstance, and it was only natural to question a procedure that essentially replicated an epidural hematoma.¹⁷¹ Prac-

tioners were also keenly aware of the fact that neurologic complications tend to respond poorly to treatment and must therefore be prevented at all costs. Studies to address these concerns have shown that, although epidural pressure increases do occur with the blood patch, they appear to be temporary and not to a degree that might prove harmful.¹⁷²

As previously mentioned, at the time of DiGiovanni and Dunbar's report in 1970, lumbar epidural or caudal saline was considered to be one of the treatments of choice for refractory PDPHs. Indeed, investigators continued to promote epidural saline as a primary treatment and prevention of such headaches through the early 1980s.⁴¹ It was not until 1978 that a direct comparison between the blood patch and epidural saline was performed. In this study, Bart and Wheeler¹⁷³ were able to convincingly show the superiority of the blood patch (Table 4).

One study, published in 1989 and that does not appear to bear repeating, compared the efficacy of an epidural blood patch to a "sham" procedure for treatment of PDPH.¹⁷⁴ Not surprisingly, in this study using a small number of subjects, they noted a 100% success rate with the blood patch (with 1 patient requiring a second patch) versus 0% for the "sham" treatment.

Further Observations on Volume and Timing of the Epidural Blood Patch

Early experience with the lumbar epidural blood patch usually employed blood volumes of between 5 and 10 mL. Dr. J. S. Crawford, in reporting his experiences with the epidural blood patch at Birmingham Maternity Hospital in England, stated in 1980 that his results were "not always satisfactory" using these small volumes.¹⁵⁶ He advocated using 20 mL of blood "unless, during the course of injection, the patient complains of a feeling of fullness or pain in the back or of pain in a leg. If this occurs the injection is immediately stopped." This suggestion resulted in the common recommendation of injecting up to 20 mL of autologous blood.

It was not until 1986 that investigators began to visualize the spread of blood during the course of blood patch procedures. The first in this regard were Szeinfeld and colleagues,¹⁷⁵ who used a method of imaging red blood cells tagged with Technetium-99, and observed that injecting an average of 14.8 mL blood (range 12-18 mL) resulted in a mean spread, preferentially cephalad, of 9 spinal segments (range 7-14 segments). They concluded that a volume of 12 to 15 mL should generally be sufficient for lumbar epidural blood patch. It is interesting to note at this point that clinically, an epidural blood patch with 10 mL autologous blood has been shown to be

Table 4. Comparison of Epidural Saline Bolus Versus Blood Patch for Treatment of Postdural Puncture Headache Following Dural Puncture With 25-Gauge and 17-Gauge Needles (With Assessment of Relief Made at 24 Hours Following Treatment)

Dural Puncture Needle Size	Epidural Treatment	n	Headaches Relieved After 24 Hours
17 gauge	Saline	6	0%
	Blood	11	72.7%*
25 gauge	Saline	15	60%
	Blood	11	100%†

**P* = .007 versus saline group (17-gauge puncture).

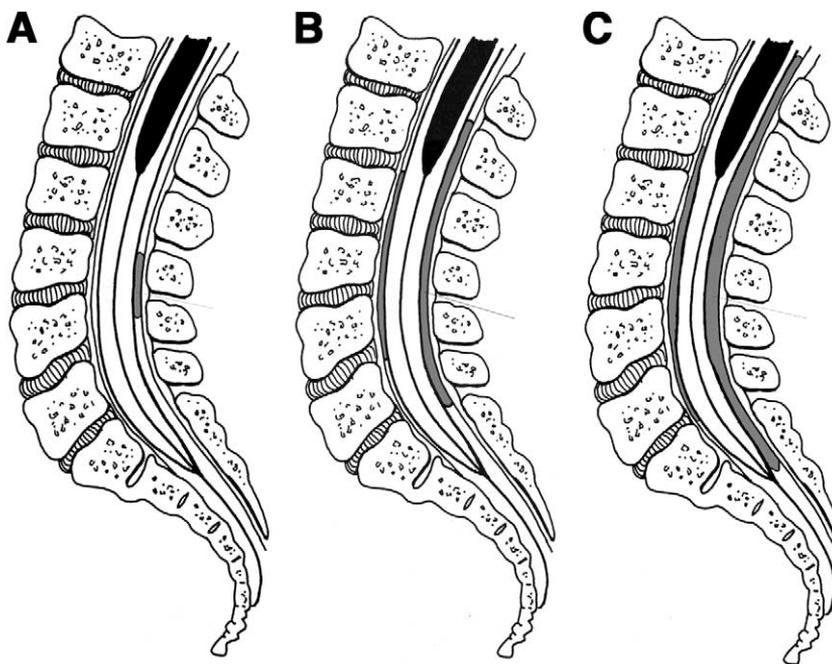
†*P* = .022 versus saline group (25-gauge puncture).

Data from Bart and Wheeler.¹⁷³

as effective as one performed with a height-adjusted volume of 11 to 15 mL.¹⁷⁶ A number of more recent studies (usually using magnetic resonance imaging) seem to indicate that, although spread is extensive even with small amounts of blood, volumes of at least 10 mL are required to result in some degree of dural tamponade.¹⁷⁷⁻¹⁷⁹ It appears that both a tamponade effect (with cephalad displacement of cerebrospinal fluid) and a sealing of the dural hole are important mechanisms for the blood patch procedure (i.e., a "pressure patch") (Fig 9). A "clot-plug," with blood extending across the dura, has commonly been observed both clinically and experimentally and appears to be associated with a successful patch.^{177,178} Although the volume of blood injected generally remains a matter of individual preference, it seems that all practitioners favor stopping the injection of blood when the patient complains of pain or fullness and there appear to be few indications for injection of volumes greater than 20 mL. Furthermore, there is anecdotal evidence that the injection of volumes of blood greater than 20 mL may be associated with complications.¹⁸⁰

Crawford's observation that smaller volumes of blood injected epidurally were not met with satisfactory results leads one to wonder at the efficacy of Gormley's original patch using only 2 to 3 mL. Other than the small number of patients in Dr. Gormley's reported experience and the observation that the presence of cerebrospinal fluid appears to promote blood coagulation,¹⁸¹ one explanation for his apparent success may be in his unusual technique. Because he was untrained in epidural techniques, his approach was to perform another lumbar puncture and then slowly withdraw the needle until cerebrospinal fluid no longer returned. Although at this point he presumed himself to be in the epidural space, it is possible that he was actually in the subdural compartment. Injection into the subdural compartment is probably more common than generally appreciated, and blood placed into

Fig 9. Drawings of the blood patch procedure performed at the L3-4 level. Injected blood is shown in gray. (A) 1960, Gormley repeated a lumbar puncture. The needle was then withdrawn until cerebrospinal fluid return ceased, at which point 2 to 3 mL blood was injected. (B) 1970, modern epidural blood patch as described by DiGiovanni and Dunbar using 10 mL blood. Note the extensive circumferential spread of blood in the epidural space. (C) 1980, epidural “pressure patch” using up to 20 mL blood, as recommended by Crawford. Note the tamponade effect produced.



this space would be expected to stay in a generally central distribution, remaining in the area of the probable dural puncture.^{182,183} Indeed, this approach was used by Shantha and Bisese,¹⁸⁴ who, in 1991, reported a successful radiographically assisted subdural blood patch in a patient after 3 failed epidural blood patch procedures. Racz,¹⁸⁵ in 1997, reported further experience with the subdural blood patch technique in cases in which epidural blood patches had been ineffective.

The issue of the timing of an epidural blood patch has evolved over the years. The use of blood to prophylactically patch a dural hole was originally introduced in 1965 by Ozdil and Powell. The concept was then furthered by DiGiovanni and Dunbar¹⁴⁵ when they included 5 patients in their 1970 article who had received a prophylactic epidural blood patch, none of whom subsequently developed a headache. The role of the epidural blood patch as prophylaxis against PDPH has been repeatedly investigated over the years (although often in more of an observational than scientific manner). Although some studies suggest that prophylaxis may be effective,^{167,186-192} others would indicate that the technique is generally ineffective.¹⁹³⁻¹⁹⁶ One possible explanation for the failure of some early epidural blood patch procedures may be an inhibition of blood coagulation by local anesthetics,¹⁹⁷ and in any case prophylactic blood patch procedures should be delayed until the block has worn off to avoid excessive cephalad spread of local anesthetic.¹⁹⁸ Motivated by concerns about performing a possibly unnecessary procedure that may become more effective with the passage of time, the

current sentiment appears to be against the prophylactic use of the epidural blood patch.^{199,200} Conversely, it seems that it is never too late to perform an epidural blood patch as successful treatments of longstanding PDPHs have been reported many weeks or months after a dural puncture.²⁰¹⁻²⁰⁴

Part 7: Acceptance, Refinement, and Understanding

Acceptance and Further Understanding of the Technique

Despite the accumulation of clinical evidence for the safety and efficacy of the epidural blood patch, the procedure was still not considered the “treatment of choice” for severe PDPH through much of the 1970s.²⁰⁵ By the end of the decade, however, the blood patch was firmly established as an accepted treatment option. In 1981, for the purpose of including “newer procedures,” the American Society of Anesthesiologists Committee on Economics adopted a new procedure code (62273) to the Relative Value Guide for “Injection, Lumbar epidural, of blood or clot patch.”²⁰⁶

Widespread use as well as ongoing clinical investigations of the blood patch technique have advanced the understanding of the procedure and helped to define its ideal practice. It is now appreciated that the blood patch injection should be made at or below the level of dural puncture (due to the preferential cephalad spread of blood and other solutions in the epidural space).^{175,177,207,208} Optimally, it appears that patients should also re-

main recumbent for 2 hours after a blood patch (probably allowing for clot organization and for lost cerebrospinal fluid to be replenished).^{209,210} Recommended activity levels after a blood patch (i.e., avoiding Valsalva maneuvers or air travel), although essentially anecdotal, probably reflect the clinical and experimental reality that blood patches are only able to withstand a certain amount of pressure before being “blown off.”²¹¹⁻²¹⁴

With further study, the true efficacy of the blood patch procedure has also become more fully appreciated. Not surprisingly, both clinical and experimental data would indicate that the epidural blood patch is less effective in sealing large dural holes (such as those seen after accidental dural puncture using an epidural needle).¹⁹⁵ Today, it seems that rather than the 95% to 100% efficacy reported in early studies, the blood patch administered after large-bore needle punctures has a more realistic 90% early (immediate) and 61% to 75% long-term success rate.^{176,215} Given this reality, the necessity of a second epidural blood patch, once thought to be rare, is not unusual. However, if headache symptoms persist after 2 failed blood patches, it is prudent to reconsider the diagnosis.

As previously mentioned, performing a dural puncture is known to be associated with a certain incidence of auditory and visual disturbances. The blood patch appears to be quite effective in treating the auditory disturbances after dural puncture.^{216,217} However, visual disturbances secondary to dural puncture (e.g., cranial nerve palsies) generally appear to resolve slowly after blood patch treatment despite prompt resolution of headache symptoms.^{218,219} This failure of the blood patch to readily reverse such cranial nerve palsies seems to indicate the presence of a neuropraxia in these cases from which patients must gradually recover.

The mysteries of the epidural blood patch (as well as postdural puncture headache itself) remain the subject of intense research. In addition to a multitude of clinical investigations and observations, a number of *in vitro* as well as animal models have also been developed to aid in our further understanding of the blood patch procedure.^{220,221} Although many questions remain to be answered regarding the epidural blood patch, a great deal has been learned during the past few decades and several excellent reviews on the procedure have been published.²²²⁻²²⁴

Contraindications, Complications, and Alternatives

Naturally, the blood patch procedure has always been contraindicated in situations in which epidural cannulation would be inadvisable such as

cutaneous or systemic infections, coagulopathies, or patient refusal. Ethically, the epidural blood patch as usually performed presents a challenge in the Jehovah’s Witness patient. These patients typically require a continuous circuit of blood withdrawal and reinfusion, conditions that are not normally met with the procedure. Although strictly theoretical, concerns have also been expressed regarding the use of the epidural blood patch in the setting of patients having cancerous conditions that may be prone to hematogenous spread.

Extensive practical experience has also shown the epidural blood patch, in addition to having all risks usually associated with epidural needle placement (another dural puncture, infection, bleeding, paresthesias, etc.), to be associated with certain unique complications. Mild symptoms such as transient back pain and radiculitis were immediately noted to be a common consequence of blood patch. Although some low back and leg pain may be seen in over one third of patients, these symptoms generally appear to be self-limited.^{225,226} Other relatively common and benign after effects of the blood patch include transient bradycardia,²²⁷ modest temperature elevations,¹⁵⁸ and neckache.¹⁵⁷

More serious complications of the blood patch procedure have generally been rare, often consist of individual case reports, sometimes involve significant deviations from standard practice, and commonly fail to show cause and effect. In this regard, the procedure has been implicated in the development of spinal subdural hematoma,^{183,228} abducens and facial nerve paresis,^{229,230} permanent spastic paraparesis with cauda equina syndrome,¹⁸⁰ significant lumbovertebral and radicular pain,²³¹⁻²³⁴ seizures,²³⁵ pneumocephalus,²³⁶ persistent meningismus,²³⁷ aseptic meningitis/arachnoiditis,^{150,238} and even exacerbation of postdural puncture headache.²³⁹ The performance of an epidural blood patch has also been associated with acute neurologic deterioration in patients having elevated intracranial pressure.^{240,241}

Despite the widespread and successful use of the epidural blood patch procedure over the past several decades, a number of alternatives have been proposed and promoted since its introduction into clinical practice. Because the blood patch is an invasive procedure, it is not surprising that a variety of pharmacologic treatments have been proposed as viable alternatives.²⁴² Some notable pharmacologic measures include sumatriptan, intravenous caffeine,^{243,244} theophylline, and adrenocorticotrophic hormone (ACTH).^{245,246} Because of concerns about the use of blood itself, the epidural placement of a number of nonblood materials, including dextran,²⁴⁷⁻²⁵² albumin, fibrin glue,²⁵³⁻²⁵⁵ and gelatin²⁵⁶ have also been reported. Even so-called “al-

ternative" treatments, such as acupuncture, have been proposed.²⁵⁷ Although many of these newer treatments have been reported to be successful, none have been extensively studied or been shown to be equal or superior to the epidural blood patch, and each has its own potentially serious side effects (such as anaphylaxis with the use of dextran and cardiac arrhythmias with caffeine).

Expanded Indications for the Epidural Blood Patch

Since the clinical acceptance of the epidural blood patch, indications for the procedure have also gradually expanded beyond that of postdural puncture headache. These applications now include the treatment of surgical cerebrospinal fluid leaks (pseudomeningoceles),²⁵⁸⁻²⁶¹ durocutaneous fistulas,²⁶²⁻²⁶⁶ and even chronic "low-pressure" headaches in the absence of known dural puncture (known as "spontaneous intracranial hypotension" or Schaltenbrand syndrome).²⁶⁷⁻²⁷¹ The epidural blood patch has also been used to seal persistent dural leaks around implanted intrathecal catheters.^{272,273} In exceptional circumstances, the blood patch technique has also been refined to include both computed tomography-guided and fluoroscopic techniques.²⁷⁴

Naturally, the vast majority of dural holes necessitating blood patching continue to be in the lumbar spine, but the epidural blood patch procedure has also been successfully performed beyond the lumbar region to include caudal,^{262,275,276} thoracic,²⁶⁹ and even cervical²⁷⁷ applications. The American Society of Anesthesiologists Relative Value Guide reflects this expanded scope of practice and no longer uses the word "lumbar" in the description of procedure code 62273.

Although lumbar puncture is commonly performed in children, PDPH appears to be rare in the pediatric population (and is perhaps underreported). However, classic PDPHs and other problems secondary to cerebrospinal fluid loss do occur in this population, particularly in children older than 12 years of age.²⁷⁸⁻²⁸⁰ Use of the blood patch was expanded to include the pediatric population in 1984,²⁸¹ with this initial report being followed by a number of subsequent reports of successful performance of the procedure in children as young as 4 years old.^{262,275,282-284}

Over the years, dural puncture itself has been noted to be associated with several rare but serious neurologic complications. Dural puncture has long been recognized as an uncommon cause of subdural hematoma, and the procedure has also been implicated in the precipitation of seizures,²⁸⁵ intracerebral bleeding,²⁸⁶ and aneurysmal rupture.²⁸⁷ It now appears that PDPH and subdural hematoma

share a common etiology: cerebral vasodilation and traction on bridging cerebral vasculature (with vascular disruption in the case of subdural hematoma). Because most serious complications after dural puncture are accompanied by the symptom of headache, some authors are now of the opinion that the prompt treatment of severe PDPH with the epidural blood patch may play an important role in the prevention of serious neurologic sequelae secondary to dural puncture.²⁸⁸⁻²⁹¹

Conclusion

The problem of PDPH has been a clinical reality since the first reports of puncturing the dura over a century ago. The first detailed description of a PDPH, by Dr. August Bier in 1899, is still echoed by those who suffer from this complication today. However, for a number of reasons, effective treatment for this relatively common problem remained undiscovered for many decades.

As the nature of such headaches became more fully understood, a great deal of progress was made in the prevention of this complication. Primarily through decreasing needle size, redesigning needle points, and attention to needle bevel direction, the incidence of these headaches has been dramatically reduced. Yet, despite a persistent hope that PDPHs might be able to be prevented entirely, it seems clear that there will remain a need for definitive treatment in refractory situations.

Largely against the prevailing sentiment of his time, Dr. James Gormley, a general surgeon in Pennsylvania, was the first to use blood as a means of sealing a persistent dural hole in the setting of PDPH. Dr. Gormley's concept was then adapted and refined by an anesthesiologist, Dr. Anthony DiGiovanni, into the procedure now recognized as the epidural blood patch.

The epidural blood patch procedure and its full role in clinical practice continue to become more clarified over time, but further study is clearly needed. The epidural blood patch, although still not without its risks or concerns, is generally regarded as safe and effective. Today, the procedure is commonly performed and has become the "gold standard" for the treatment of severe PDPH unresponsive to conservative measures.^{4,5,11}

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An Announcement from the Wood Library-Museum of Anesthesiology

Donald Caton, M.D., has been named the 2004 Wood Library-Museum Laureate Historian of Anesthesia. The Laureate of Anesthesia program was established in 1994 to increased recognition of the rich history and importance of anesthesiology by honoring the work of scholars who have made singular contributions to the field. The honor is awarded every four years to an individual who has made outstanding contributions of original materials related to the history of our specialty, as reflected by articles published in peer-reviewed journals or in monographs.

Dr. Caton has published extensively on the history of anesthesia. Eleven of his 67 peer-reviewed articles have focused on historical topics, and in 2000, he won the Anesthesia History Association's David M. Little Award and the British Medical Association Commended Award for his book *What a Blessing She Had Chloroform*. In addition, Dr. Caton has given 62 lectures on the history of anesthesiology and has mentored 39 medical students in the study of the history of medicine.

The Laureate Historian of Anesthesia is elected by an international panel of judges who are known historians and are active contributors to the history of medicine.

For more information about the Wood Library-Museum of Anesthesiology, call (847) 825-5586, or send an e-mail to wlm@asahq.org.