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Post–Dural Puncture Bacterial Meningitis

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A fatal case of viridans streptococcus meningitis is reported, which occurred as a complication of epidural anesthesia. One hundred seventy-nine reported cases of post–dural puncture meningitis are reviewed. Evidence suggests that most cases are probably caused by contamination of the puncture site by aerosolized mouth commensals from medical personnel, some are caused from contamination by skin bacteria, and, less frequently, other cases are caused directly or hematogenously by spread from an endogenous infectious site. Controversy exists regarding prevention, surveillance, incidence, and treatment of this serious complication.

> ". . . there are precautions so imperative that even their universal disregard will not excuse their omission."
>
> —Learned Hand, Circuit Judge
> THE T.J. HOOPER, 60 F.2d 737 (2nd Cir. 1932)

THIS review begins with a case report of post–dural puncture meningitis (PDPM), due to *Streptococcus salivarius*, which occurred approximately 8 h after inadvertent dural puncture associated with epidural anesthesia for labor analgesia. Central diabetes insipidus (CDI), a rare complication of meningitis, adds complexity to the case. The connection between PDPM and lumbar puncture (LP) was not considered by a team of treating specialists, and the patient died 2 days after delivery.

Among 179 reported cases of PDPM, two additional mortalities are found. All three cases are obstetric cases involving healthy young primiparas. Various strains of viridans streptococcus (a mouth commensal) are the dominant causative organism in PDPM, accounting for 49% of the 179 reported cases. Viridans streptococcus is becoming antimicrobial resistant— emphasizing a potential serious hazard of PDPM. Other causal organisms found in PDPM include *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Enterococcus faecalis* along with six other less commonly encountered organisms. However, in 64 cases (36%), no organism was isolated or reported.

Evidence that most cases of PDPM are caused by droplet contamination (by medical personnel) during dural puncture includes the clustering of some cases and the matching of DNA fragments of the involved organisms in the patients' cerebrospinal fluid (CSF) with the operators' nasopharyngeal swabs. Other cases may result from needle contamination from incompletely sterilized skin. Those cases that are likely endogenous (hematogenous) in origin represent a minority of the reported cases.

Incidence statistics for PDPM are uncertain for the United States. The Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia, in the United States collects data on nosocomial infections. However, its definition of such an infection excludes PDPM from this classification—the typical case appears within 6 and 36 h after dural puncture—too soon for the CDC's definition. Perhaps this explains why only one case of PDPM from the United States has been reported since 1996.

There is no national standard for dural puncture–related infection control, but guidelines do exist for prevention of intravascular central catheter–related infection (1% incidence). Extrapolation of standards for central line infection control to dural puncture–related infections is not ideal, but the recommendation for similar maximal precautions (caps, gowns, gloves, masks) might be considered for neuraxial instrumentation.

Post–dural puncture meningitis is a serious complication of dural puncture. The case presented below suggests a lack of familiarity with this condition among the many medical and surgical specialties involved in its recognition and treatment.

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Case Presentation

A 28-yr-old primigravida was hospitalized at term for delivery. There were no unusual findings on the initial history and physical examination. Epidural anesthesia was attempted shortly after admission but was complicated by accidental dural puncture. The original epidural catheter was removed and replaced with another, one interspace above. The patient delivered a healthy male

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infant. Later that evening, she developed fever (101.6°F), headache, and neck pain. She had a seizure and became disoriented. A presumptive diagnosis of migraine or chemical meningitis was made, and she was transferred to the intensive care unit on the second hospital day. There, she required mechanical ventilation and support for cardiorespiratory collapse. Medications included cephtriaxone, vancomycin, meropenem, dexamethasone, diphenylhydantoin, dopamine, calcium, magnesium, and phenylephrine.

An LP was performed approximately 15 h after admission. The CSF was cloudy; the pressure was not measured— but the fluid is described as having "shot out." CSF glucose was less than 10 mg/dl, protein 550 mg/dl, leukocyte count 12,400/dl with 94% segmented neutrophils, 2% lymphocytes, 2% monocytes, 2% band cells. Gram-positive cocci in chains and pairs were seen in the CSF, but initial culture was reported as "no growth." *Staphylococcus simulans*, isolated from the CSF, grew from broth only. *Streptococcus salivarius/vestibularis* was isolated from blood. Both organisms were sensitive to all antibiotics on the standard panel. Head computerized tomography (CT) scan was performed shortly after the LP. It showed cerebral edema. The clinical course was rapidly downhill— complicated by hypernatremia. The initial serum sodium was 136 mM. On the day of death, the highest reading was 174 mM. Serum osmolalities on the second hospital day ranged from 283 to 365 mOsm/kg (reference range, 275–295 mOsm/kg). On the same day, urine osmolality was 77 mOsm/kg (reference range, 500–800 mOsm/kg). Urinary output on the second hospital day was 6 l. Diabetes insipidus was recognized. An attempt to match fluid input with this output failed. On the third hospital day, a cerebral perfusion study was performed, which showed good perfusion through the level of the circle of Willis without intracerebral perfusion. Life support systems were withdrawn, and the patient died 60 h after admission.

The final diagnosis on the medical chart reads: "1. Massive cerebral edema with brain death secondary to acute meningitis of unclear etiology. It is not clear whether this was bacterial, nonbacterial, or chemical meningitis. 2. Postpartum."

The autopsy was signed out as follows: "I. Acute and organizing bacterial meningoencephalitis. II. Status post normal vaginal delivery of term male infant." The brainstem was compressed against the cerebellum.

Case Discussion

In neither the medical chart nor the autopsy report is there mention of an organism, or connection between meningitis and recent LP, although mention is made (in the autopsy report) of a recent needle-puncture mark that continues into a hemorrhagic track leading to epidural and subdural blood staining of the lumbar cord. There were two punctures, but only one is described. Symptoms appeared approximately 10 h after the first epidural attempt. The endocrinologist diagnosed diabetes insipidus, but the relation to meningitis was not mentioned. Central diabetes insipidus—as opposed to nephrogenic—a rare complication of meningitis, has been reported associated with viral,[1] meningococcal,[2] pneumococcal,[3] tuberculous,[3] listeria,[4] and fungal[5] meningitis. Hypopituitarism associated with meningoencephalitis is typed as either primary-pituitary or hypothalamic. Both could have existed here. Brain herniation could explain the appearance of CDI. The diagnosis of CDI could have been confirmed by the patient's response to 5 μg arginine vasopressin. The case above may be the first-reported case of CDI associated with meningitis due to viridans streptococcus.

The laboratory report of the blood and CSF cultures were not available until 3 days after the patient died. DNA bacterial probes and commercial kits for rapid (hours *vs.* days) identification of organisms (down to the species level) are available. Their use in the clinical setting is urged in future cases of meningitis, not only to provide quick identification but also to pinpoint a possible break in sterile technique.

The patient was cared for by qualified specialists in anesthesiology, obstetrics, neurology, neurosurgery, endocrinology, nephrology, internal medicine, intensive care, and pathology. None mention the diagnosis of PDPM. Pertinent here is the question: How prevalent is the apparent lack of familiarity with PDPM in the medical community?

Analysis of the Reported Cases

Table 1 summarizes 179 cases[6–66] of (bacterial) PDPM found in the medical literature reported between 1952 and 2005. This collection of reported cases may not be complete, and its analysis is subject to factors common to meta-analyses. There are two mortalities[47,53] among these cases.

The Mortality Case from Singapore

One of the patient deaths (an obstetric case from Singapore) involves a 22-yr-old primigravida who requested epidural analgesia. The first attempt at catheter placement (by a second-year resident) at the L3–L4 intervertebral space failed. The catheter was then sited at the L2–L3 interspace by a senior staff member. Full aseptic technique is reported to have been followed. A healthy infant was delivered vaginally. The catheter was removed 2 h after insertion. The next day, the patient reported perineal pain and headache, but she was discharged without further assessment. She was readmitted 2 days later with vomiting, fever, and

Table 1. Some Characteristics of 179 Reported Cases of Dural Puncture–related Meningitis since 1952

Reference	Year	No. of Cases	Reason for Dural Puncture	Organism	Comment	Country
Baumann and Koch[6]	1952	1	Diagnostic lumbar puncture	*S. micro-aerophilia*	Baumann's case	United States
		15	Diagnostic lumbar puncture	1 case *pyocyaneus*; organism not available for others	Cases referenced by Baumann (in German)	Germany
Berman and Eiselem[7]	1978	1	Spinal anesthesia	Group D streptococcus	Renal calculus	United States
Schlesinger *et al.*[8]	1982	11	Myelography	Viridans streptococcus, *S. salivarius, S. bovis*	—	United States
		2	Pneumoencephalography	*S. salivarius*		
Kilpatrick and Girgis[9]	1983	17	Spinal anesthesia	1 each *S. mitis*, *S. aureus*; 8 cases *Pseudomonas aeruginosa*	17 cases over 5 year period; 7 culture-negative	Egypt
Schelkun *et al.*[10]	1985	1	Myelography	*S. sanguis*	—	United States
Ready and Helfer[11]	1989	2	Epidural: obstetric	*S. uberis*, *S. faecalis*	Manual removal of placenta in case 1	United States
Berga and Trierweiler[12]	1989	1	Epidural: obstetric	*S. sanguis*	Extradural blood patch	United States
Roberts and Petts[13]	1990	1	Spinal: manual removal of retained placenta	No growth (lumbar puncture after antibiotics)	"Use of face masks and gown debatable"	Great Britain
Vukmirovits *et al.*[14]	1991	1	Spinal anesthesia	*Corynebacterium xerosis*	Article in Hungarian	Hungary
Lee and Parry[15]	1991	1	Obstetric: spinal cesarean delivery	None, but spinal fluid glucose low	3 attempts needed for lumbar puncture at 2 interspaces	Great Britain
de Jong and Barrs[16]	1992	2	Myelography	*S. salivarius*	*	The Netherlands
Watanakunakorm and Stahl[17]	1992	1	Myelography	*S. salivarius*	Facemask not worn	United States
Linnemann and Bulow[18]	1993	1	Epidural	Unknown	Article in Danish8 attempts to insert catheter	Denmark
Torres *et al.*[19]	1993	1	Diagnostic lumbar puncture	*S. salivarius*	"Difficult" tap	Spain
Blackmore *et al.*[20]	1993	1	Spinal anesthesia	*S. mitis*	Facemask worn	Australia
Durand *et al.*[21]	1993	1	Epidural catheter	Not stated	1 case among of 493 meningitis cases reviewed	United States
Newton *et al.*[22]	1994	1	Obstetric spinal	*S. salivarius*	—	United States
Domingo[23]	1994	1	Myelography	Viridans streptococcus	—	Spain
Garlicki *et al.*[24]	1994	4	Epidurals in all 4 cases	Not known	Article in Polish	Poland
Harding *et al.*[25]	1994	1	CSE: obstetrics	*Staph epidermidis*	Facemask worn; extradural blood patch	Great Britain
Pegues *et al.*[26]	1994	1	Epidural	*S. aureus*	Analgesia for metastatic breast cancer	United States
Stallard and Barry[27]	1995	1	CSE: obstetrics—cesarean	No growth (lumbar puncture after antibiotics)	2 tries; 3 sticks	Great Britain
Gelfand and Abolnik[28]	1995	3	Myelography	Viridans streptococcus	Cluster of 2 cases†	United States
Veringa *et al.*[29]	1995	1	Myelography	*S. salivarius*	Facemasks recommended‡	The Netherlands
Aldebert and Sleth[30]	1996	1	CSE	Gram+ cocci; nonhemolytic strep	—	France
Baird[31]	1996	2	Myelography	Viridans streptococcus	—	Australia
Schneeberger *et al.*[32]	1996	4	Spinal anesthesia; 2 arthroscopy, 2 varicose vein stripping	*S. sanguis*, *S. mitis*, *S. salivarius*, *S. cremoris*	§	The Netherlands
Cascio and Heath[33]	1996	1	CSE: obstetrics	*S. salivarius*	See text for discussion	United States
Cooper and Sharpe[34]	1996	1	Epidural steroid injection	*S. aureus*	Chronic back pain	Canada
Liu and Pope[35]	1996	1	CSE: lithotripsy	No growth	Gram+ diplococci	United States
Kaiser *et al.*[36]	1997	1	Spinal anesthesia	*S. salivarius*	Use of facemask urged	France
Aromaa *et al.*[37]	1997	4	Epidural or spinal	Purulent meningitis	Study based on patient insurance claims	Finland
Kaiser and Suppini[38]	1997	2	Myelography	Viridans streptococcus	Use of facemask urged	France
Molinier *et al.*[39]	1998	1	Spinal anesthesia: hysteroscopy	*S. salivarius*	Use of facemask urged	France
Laurila *et al.*[40]	1998	1	Spinal: arthroscopy of knee	*S. salivarius*	—	Finland
Bouhemad *et al.*[41]	1998	1	CSE: obstetrics	*S. salivarius*	—	France
Duflo *et al.*[42]	1998	1	CSE: obstetrics	Viridans streptococcus	Article in French	France
Schlegel *et al.*[43]	1999	1	Myelography	Abiotrophia defective	Mask not worn	France
Cabellos *et al.*[44]	1999	2	Reasons for lumbar puncture not specified	*S. salivarius*, viridans streptococcus	Spinal fluid culture +, blood culture −	Spain
Lurie *et al.*[45]	1999	1	Spinal: obstetrics	Viridans streptococcus	No need for facemask	Israel
Fernandez *et al.*[46]	1999	1	Spinal anesthesia	*S. mitis*	Article in Spanish	Spain
Choy[47]‖	2000	1	Epidural: obstetric	No growth	See text for discussion; 2 attempts at epidural	Singapore
Tortosa and Hernandez-Palazon[48]	2000	1	Spinal: herniorrhaphy	*E. faecalis*	—	Spain
Yaniv and Potasman[49]	2000	1	Spinal: lithotripsy	*S. salivarius*	Facemask worn	Israel
Villevieille *et al.*[50]	2000	1	Spinal: hydrocele repair	*S. mitis*	Facemask worn	France
Varlet *et al.*[51]	2000	1	Spinal: ortho material removal	*S. sanguis*	—	France
Idigoras *et al.*[52]	2001	1	Epidural: meniscectomy	*S. salivarius*	Genetic methods used for species identification	Spain
Thomas and Cooper[53]‖	2002	1	Spinal: cesarean	None (lumbar puncture after antibiotics)	Case 16 in reference; see text for discussion	Great Britain
Trautman *et al.*[54]	2002	3	Spinal: 2 cases knee arthroscopic surgery#	Both *S. salivarius*	—	Germany
			Case 3: epidural: obstetrics	*S. aureus*	Epidural abscess**	

(*continued*)

Table 1. Continued

Reference	Year	No. of Cases	Reason for Dural Puncture	Organism	Comment	Country
Videira *et al.*[55]	2002	3	Spinal anesthesia	Streptococcus 2 cases, no growth in 1 case	—	Brazil
Couzigou *et al.*[56]	2003	2	Spinal: knee arthroscopySpinal: meniscal surgery	*S. salivarius* both cases	Poor fit of mask"Change q3h" urged	France
Kocamanoglu *et al.*[57]	2003	1	Spinal: inguinal herniorrhaphy	*S. salivarius*	Mask worn; used PI solution from multiuse bottle	Turkey
Pinderand Dresner[58]	2003	1	CSE: obstetrics	*N. meningitidis*	No growth††	Great Britain
Pandian *et al.*[59]	2004	19	Spinal anesthesia	3 cases: *S. aureus*; 1 case: *P. aeruginosa*; 1 *Acinetobacter* species	No growth in 78% of 27 patients	India
		4	Myelography	1 *M. tuberculosis*	11 cases: cesareans	
Moen *et al.*[60]	2004	29	Spinals and epidurals; myelography: 1 case	11 α-hemolytic strep, 1 *S. aureus*, 17 cases undetermined	See text for discussion‡‡	Sweden
Conangla *et al.*[61]	2004	1	Spinal: inguinal herniorrhaphy	*S. salivarius*	Article in Spanish	Spain
Salazar *et al.*[62]	2004	1	Spinal: obstetrics	*S. oralis*	Article in Spanish	Spain
Vernis *et al.*[63]	2004	1	CSE: obstetrics	*S. mitis*	Inadvertent dural puncture: 2 blood patches	France
Hooten *et al.*[64]	2004	1	Epidural steroid injection for back pain	*S. aureus*	Inadvertent dural puncture; patient immunocompromised	United States
Siman-Tov and Gadoth[65]	2004	1	Epidural: knee arthroscopy	*E. faecalis*	Delayed diagnosis	Israel
Bussink *et al.*[66]	2005	1	Spinal: knee arthroscopy	*S. sanguis*	Delayed onset—10 days after spinal	The Netherlands

* Two cases from same neurologist. † Two cases from same neurosurgeon. Mask not worn. ‡ DNA fingerprinting found patient isolate matched neurologist's throat swab isolate. § Four cases from a single anesthesiologist. Facemask not worn. ‖ This reference contains report of a mortality from post–dural puncture meningitis. # Cases 1 and 2: same operating room sequentially. ** Pulsed-field electrophoresis showed match with anesthetist's throat swab and patient's spinal fluid *S. aureus* isolate. †† Diagnosis of community acquired meningococcal meningitis made using polymerized chain reaction, then analyzing DNA fragments. See text for discussion. ‡‡ Cases include one cluster of four within one department.

CSE = combined spinal–extradural anesthesia; PI = povidone iodine.

severe headache. She was febrile (38.2°C) and alert and denied neck pain. The uterus was well contracted; the abdomen was soft. There was an odorous vaginal discharge, not obviously purulent. The blood leukocyte count was 12,300/dl with 83.2% neutrophils. A provisional diagnosis of endometritis was made and treated with intravenous ceftriaxone and metronidazole. Six hours later, she developed mental confusion, nuchal rigidity, and right sixth nerve palsy. Head CT revealed mild hydrocephalus and minimal cerebral edema. Intravenous dexamethasone was started, but the neurologic status continued to deteriorate. Forty-eight hours after admission, an LP was performed. CSF analysis revealed an increased CSF protein (2.75 g/l), a CSF/blood glucose ratio of 0.34, and a leukocyte count of 459/dl (11% polymorphs, 85% lymphocytes). Cultures of CSF, blood, urine, and vaginal swabs were negative. Treatment was changed to include ceftazidime, isoniazid, rifampin, and pyrazinamide (to cover mycobacterium tuberculosis) and acyclovir (to cover herpes simplex). The patient's neurologic status continued to worsen, and she died 4 weeks later. An autopsy was declined.

The Mortality Case from England

The second mortality case[53] in table 1 occurred in a primipara after cesarean delivery (because of preeclampsia), during spinal anesthesia. The patient received antibiotics on the first postpartum day because of fever. Hours later, she developed severe headache, vomiting, hypertension, and coma. A head CT scan showed cerebral edema. The report does not state whether an LP was performed. Despite supportive care, her condition deteriorated, and she died a few days after delivery. An autopsy showed acute purulent meningitis with sagittal sinus thrombosis. The report, from a committee that periodically reviews all maternal mortalities in England, states that the postmortem finding of meningitis was a surprise because bacterial cultures premortem and postmortem did not grow an organism. They were unable to say whether the illness was related to the spinal or was a coincidence.

Discussion of the Three Mortalities

All three mortalities are obstetric cases—all young, healthy women without risk factors. Two delivered vaginally with epidurals; one required a cesarean delivery during spinal anesthesia. Both epidural cases required a second attempt to place the catheter. In case 1 (presented here), there was clinical evidence of inadvertent dural puncture. In all three cases, the diagnosis of PDPM was not considered at the onset of headache and fever after LP. Migraine and then chemical meningitis were considered in the first case; endometritis was considered in the Singapore case. The report of the English case does not mention the premortem diagnosis. Treatment

with antibiotics was delayed in the American and Singapore cases and not specified in the English case. In the American case, an LP was done before a head CT scan despite the presence of coma and seizures. This LP could have caused brain herniation and hastened the patient's death. In the Singapore case, LP was delayed; in the English case, LP was unmentioned.

In the Singapore case, it is stated that CSF lymphocytosis suggested viral etiology, whereas the low CSF glucose suggested bacterial etiology. However, by day 4 of a case of partially treated meningitis, CSF lymphocytes would have been expected to have replaced CSF neutrophils. The author of the case report correctly notes that delayed LP and pretreatment with antibiotics probably rendered the CSF cultures negative. The American case is the only one in which organisms were isolated—but they were dismissed as contaminants. The blood isolate was *Streptococcus salivarius*. The CSF isolate was *Staphylococcus simulans*.

In a recent report[60] of serious complications after neuraxial blockades between 1990 and 1999 (in Sweden), there were cases of meningitis found among the surgical cases but none among the obstetric patients, so it is unusual that the three mortalities reported here are obstetric.

Further Analysis of Cases

Case 27[33] in table 1 illustrates a common misconception. Here, a 28-yr-old primipara delivered vaginally 4 h after combined spinal-extradural anesthesia. She developed—16 h after delivery—fever, chills, headache, photophobia, and nuchal rigidity. A diagnostic LP—25 h after delivery—yielded cloudy fluid, low CSF glucose (29 mg/dl), and an increased leukocyte count (5,800/dl); Gram staining did not demonstrate bacteria. A diagnosis of bacterial meningitis was made, and she was treated with ceftriaxone and vancomycin and promptly recovered. All cultures of CSF remained negative until 72 h, when a single broth culture grew *Streptococcus salivarius* with no growth on the culture plates. Blood and urine cultures were also negative. The authors of this case report state that it is unlikely that the meningitis was caused by the bacteria (*S. salivarius*) isolated from the patient's CSF. It was thought to be a contaminant because it grew only in a single broth culture, it did not grow on culture plates, and it was not a bacterial pathogen that commonly causes meningitis. The dismissal of *S. salivarius* as a nonpathogen contaminant echoes that of the clinicians in the American mortality case discussed above.

Of the 179 cases in table 1, 29 (17%) occurred after myelography. The absence of reports of such cases since 1999 may be explained by the current use of enhanced magnetic resonance imaging as a replacement for more invasive myelography.

Table 2. Distribution of Table 1 Cases of PDPM by Country of Origin

Country	No. of Cases
Sweden	29
United States*	29
India	27
Germany	18
Egypt	17
France	13
Spain	8
The Netherlands	8
Finland	5
United Kingdom	7
Poland	4
Australia	3
Brazil	3
Israel	3
Canada	1
Turkey	1
Singapore	1
Denmark	1
Hungary	1

* All but one of these cases predate 1997.

PDPM = post–dural puncture meningitis.

It is more difficult to explain the absence of reported cases from the United States since 1996. The one exception is a 2004 case from the Mayo Clinic.[64] In this case, a 70-yr-old man received epidural steroid injections for intractable hip and back pain. Inadvertent dural puncture occurred. He developed an epidural abscess and meningitis due to *Staphylococcus aureus*, which was promptly diagnosed and responded to medical treatment.

Table 2 shows the distribution of table 1 cases among the reporting countries. This distribution of cases is not proportional to the countries' population. Possible reasons for the diminishing reports from the United States include underdiagnosis, underreporting, and fear of litigation.

Table 3 shows the spectrum of procedures among the 179 cases in table 1. The cases are categorized as obstetric *versus* nonobstetric. Fifty-four percent of the cases of PDPM occur after spinal anesthesia (50% of these are nonobstetric; 4% are obstetric). Nine percent of the

Table 3. Reasons for LP among the 179 PDPM Cases in Table 1*

Procedure	Obstetric, n (% n)	Nonobstetric, n (% n)
Spinal anesthesia	7 (4)	89 (50)
Epidural	4 (2)	17 (9)
Combined spinal–extradural	8 (4)	2 (1)
Myelography		29 (16)
Diagnostic		17 (9)
Pneumoencephalography		2 (1)
Epidural steroid injection		2 (1)
Not specified		2 (1)

* Sum of percentages = 98% due to rounding.

LP = lumbar puncture; PDPM = post–dural puncture meningitis.

cases reported after epidural anesthesia/analgesia are nonobstetric; 2% are obstetric. Meningitis after combined spinal–extradural anesthesia is more frequent among the obstetric cases (4%) *versus* only 1% among the nonobstetric cases. Moen *et al.*[60] suggest that it is important to subgroup the obstetric cases because they found a much lower incidence of neurologic complications related to neuraxial anesthesia in obstetrics.

Table 3 also tabulates the nonanesthetic cases of PDPM—myelography (29 cases; 16%); diagnostic LP (17 cases; 9%). Epidural steroid injection,[34,64] pneumoencephalography,[8] and an unknown category[44] each account for 2 cases (1% each). Sixteen of the 17 reported diagnostic cases are from Baumann and Koch's 1952 article.[6] There is only one other,[19] a case reported in 1993. Of note is the absence of therapeutic taps. The categories are without denominators, and percentages are for cases reported over several decades— unlikely to reflect present-day data. But it is still likely that spinal anesthesia is presently involved in the majority of cases of PDPM—as expected, because it is likely the most common reason for a dural puncture.

Returning to table 1—among the epidural anesthesias, one case[12] required an epidural blood patch; epidural abscess was associated with another.[54] There are two instances[25,63] of extradural blood patching in the combined spinal–extradural group and none among the spinals. Mechanical difficulties play a role in at least three of the cases and may be a risk factor for PDPM. In the first,[15] spinal anesthesia required three attempts at two interspaces, the second case[18] required eight attempts to insert an epidural catheter, and the third[19]—a diagnostic LP—was described as a "difficult tap."

Neurologic Sequelae

Information about neurologic sequelae in table 1, patients who survived, is not present in the case reports. In a recent prospective study[67] (in The Netherlands) of community-acquired meningitis in adults, the overall mortality rate was 21%. There were 696 episodes of meningitis during the study period (October 1998 to April 2002). Pneumococcal meningitis carried a higher mortality rate than meningococcal meningitis (30% *vs.* 7%; $P < 0.001$). At discharge from the hospital, 26% of the cases had neurologic findings: cranial nerve palsies in 19%, of which 14% were eighth nerve. Hemiparesis was present in 4% of the survivors, quadriparesis was present in 1%, and aphasia was present in 2%. Long-term sequelae (such as seizures or impaired cognition) were not studied. Drawing conclusions (by extrapolation) about mortality or sequelae for the 179 cases that comprise this review is inappropriate. But despite the fact that the cases here are hospital acquired, have a different pathogenesis, have different causative organisms, and lack statistical power, the relatively much lower mortality rate suggests underreporting or underdiagnosis of PDPM or both.

Table 4. Occurrence of Causative Organisms Tabulated in Table 1

Organism*	n
No growth	46
S. salivarius	30
Viridans streptococcus	29
Not specified	12
α-Hemolytic strep	11
Staphylococcus aureus	9
Pseudomonas aeruginosa	8
S. mitis	6
S. sanguis	5
Purulent meningitis	4
None (LP after antibiotics)	3
E. faecalis	3
Streptococcus	2
Gram+ nonhemolytic strep	1
S. uberis	1
S. cremoris	1
S. micro-aerophilia	1
S. oralis	1
Group D streptococcus	1
Staph epidermidis	1
Corynebacterium xerosis	1
Abiotrophia defectiva	1
Acinetobacter species	1
N. meningitidis	1

* Organism designations are those used by the authors of the case reports.

The distribution of organisms isolated in table 1 cases is shown in table 4. The organisms are listed with the name designation as given by the authors of the various case reports. Various strains of viridans streptococci—the dominant isolate— occur in 88 instances (49% of the 179 cases). In 65 cases (36%), no organism was isolated (or specified). *Staphylococcus aureus* (5%) and *Pseudomonas aeruginosa* (4%) were, respectively, the second and third most frequently occurring pathogens. Three cases are caused by *Enterococcus faecalis* (2%)—the third most frequent organism found. Six other organisms are found once, in each of 6 cases.

Among the table 4 isolates, there is one occurrence of *Neisseria meningitidis*.[58] In this case, the combined spinal— extradural technique was used for labor analgesia. The anesthetist wore a hat, gown, and gloves, but not a mask. Hours later (after a normal delivery), the patient reported headache and became irritable and difficult to rouse. A neurologist diagnosed postpartum migraine. Later, the patient became febrile and was having difficulty with speech. A head CT scan was performed, which showed prominent temporal horns but no hydrocephalus. Urine culture was consistent with a urinary tract infection, and the neurologist diagnosed migraine with dysphagia and prescribed antibiotics for the concomitant urinary tract infection. LP revealed that the CSF was cloudy, and CSF glucose was 1.1 mM. No organisms

were seen on CSF Gram stain or culture. Studies on microbial fragments (using polymerase chain reaction and probes to detect microbial DNA) were positive for *N. meningitidis*. The patient was treated and recovered without neurologic sequelae. A diagnosis of coincidental community-acquired bacterial meningitis was made. However, the possibility that this case is a case of inoculation meningitis is not ruled out. Asymptomatic carriers of *N. meningitidis* are not infrequent. To pinpoint the source, nasal or nasopharyngeal swabs of the patient and medical personnel should have been done to compare these isolates with the patient's CSF isolate.

Viridans Streptococci Are Becoming Antimicrobial Resistant

Several studies of bacteremia in immunocompromised patients[68–70] have identified viridans streptococcus among the isolates. Penicillin-resistant viridans streptococcus was encountered—the highest rate of resistance (47%) was found for the United States. It is not known whether there are differences in antimicrobial susceptibility among the various organisms within this group, at the species level.[71] The need for prevention of PDPM is emphasized because of the probability that future cases may involve a resistant organism. Thus far, no such case has been reported for PDPM.

Pathogenesis of PDPM

Quagliarello and Scheld[72] have reviewed the pathogenesis of meningitis. They describe how ". . . the successful meningeal pathogen must sequentially colonize the host mucosal epithelium, invade the intravascular space, cross the blood-brain barrier, and survive in the cerebrospinal fluid . . ." The first two of these antibacterial obstacles involve immunoglobulin A proteases, bacterial pili, binding epitopes, and the molecular basis for complement evasion. The third obstacle, crossing the blood–brain barrier and entry into the CSF, is the least understood step. However, it is only the fourth obstacle that the PDPM organism must overcome: The meningeal pathogen circumvents the first three obstacles in those cases where it is inoculated directly into the CSF (once there, it thrives). In experimental meningitis, there is a 2- to 3-h delay between bacterial inoculation into the CSF and onset of disease.[72] At onset, tumor necrosis factor, and other host cytokines (various interleukins) can be found in the CSF. There is strong experimental evidence that these inflammatory cytokines induce inflammation and break down the blood–brain barrier. Other mechanisms allow entrance into the CSF of leukocytes which, upon breakdown (along with lysis of bacterial pathogens), emit toxic metabolites that can lead to vasogenic brain edema. There is a resulting loss of cerebrovascular autoregulation, subjecting the brain to the risk of hyperperfusion or hypoperfusion. An understanding of these events has important implications on the type and timing of treatment and is discussed in the section on treatment below.

How then, in PDPM, does the organism gain entry into the CSF? Because PDPM is rare, the fact that there are several reports of clustering of cases—two patients with the same neurologist,[16] two from the same neurosurgeon,[28] four from a single anesthesiologist,[32] and more recently, two cases occurring sequentially from the same operating room after arthroscopic surgery during spinal anesthesia[54]—suggests an operator-related role in its pathogenesis.

Second, Veringa *et al.*[29] identified the source of a case of *S. salivarius* meningitis that occurred after myelography: They compared isolates from the throats of the operating neurologist and his assistant with that of the patient's CSF isolate. Using polymerase chain reaction amplification of the extracted DNA and fatty acid profiles of the organisms, they discovered that only the neurologist's isolate matched the patient's. Similarly, Trautman *et al.*[54] report a case of *S. aureus* meningitis and epidural abscess after epidural anesthesia for delivery of twins. They performed nasal swabs on the anesthesiologist and nine midwives and two gynecologists who attended the case. The pulsed-field gel electrophoretic patterns of the various *S. aureus* isolates of the medical personnel were compared with that of the patient's CSF isolate. Only that of the anesthesiologist matched that of the patient.

Third, most of the organisms that cause PDPM are commensals of the mouth and upper airway. These observations support the droplet mechanism for the pathogenesis of most cases of PDPM. That is, the aerosolized organism that enters the CSF during dural puncture originates in the upper airway of medical personnel.

This droplet mechanism of pathogenesis of PDPM is not universally recognized. The fact that not all cases of PDPM are acquired in the same way contributes to this circumstance. Exactly how the organism gains entrance into the CSF *via* the puncture—whether from the patient's skin, the operator's contaminated hands, or medicinals or instruments or directly from aerosolized organisms— has not been determined but is less important in explaining the pathogenesis.

Post–dural puncture meningitis is distinct from epidural abscess, another serious complication of needling which, in turn, is sometimes complicated by meningitis. The mechanism here may be rupture of the abscess into the CSF or inadvertent dural puncture (during epidural catheter placement). Mouth commensals (*e.g.*, viridans streptococcus) are the most frequent organisms associated with PDPM; skin commensals (*e.g.*, *S. aureus*) are the prominent organisms found in epidural abscess. Epidural abscess is more frequent after epidural block; PDPM is more frequent after spinal block.

Risk factors for epidural abscess after central neuraxial blockade[73,74] include long-term catheter placement (> 2 days), diabetes mellitus, old age, immunosuppression, and thromboprophylaxis (with low-dose or low-molecular-weight heparin). (The magnetic resonance imaging distinction between epidural abscess and a degrading infected epidural hematoma can be difficult.[73]) By contrast with epidural abscess, in inoculation-type PDPM, the organism deposited into the CSF multiplies rapidly and within hours results in fulminant meningitis, often in healthy individuals.

Post–dural puncture meningitis can occur by a mechanism other than inoculation of an exogenous organism. In 1920, Weed *et al.*[75] performed LP on experimental animals, into which virulent organisms had been recently injected intravenously. This resulted in meningitis due to the injected organism. A similar experiment was performed by Carp[76] in 1992 using rats with *E. coli*—induced bacteremia. Those animals with a circulating colony count of 50 colony-forming units/ml or more developed meningitis only after LP. If the animals were given a single dose of gentamicin before the puncture, the risk of meningitis was eliminated. Lee and Parry[15] state that the rate of bacteremia after normal delivery is reported as high as 7% and suggest that manipulation of the uterus could be associated with an even higher rate. In table 1, the first case described by Ready and Helfer[11] involves an organism (*Streptococcus uberis*) that was isolated from the patient's vaginal secretions, blood, and urine, as well as spinal fluid. This suggests endogenous origin in this case (and perhaps similar cases).

The risk of performing central neuraxial block in the presence of bacteremia has not been determined. In the animal studies mentioned above, meningitis did not occur in the absence of a critical number of colony-forming units per milliliter. A retrospective study of bacteremic children[77] (comparing one group that underwent LP with another group that did not) showed an association between LP and the subsequent development of meningitis in the subgroup of those younger than 1 yr. The authors of this study caution that the two cohorts may not have been equally matched with respect to the likelihood of meningitis. In a more recent review of spinal and regional anesthesia,[78] the authors conclude that the presence of bacteremia is a relative but not an absolute contraindication for central neuraxial blocks. Antibiotic prophylaxis (in bacteremic patients) is advised, before the puncture, and those patients should be closely followed up postoperatively.

Based on analysis of the reported cases, it seems that most cases of PDPM result from exogenous inoculation; the source of the organism most likely from droplets originating from medical personnel. An organism was identified in only 114 instances (63%) of the 179 cases reported (table 4). Of these, there was only one case (1%) of endogenous origin. Eighty-seven cases (76%) involve mouth commensals; 26 cases (23%) are due to organisms commonly found on skin.

The older medical literature contains references to reusable needles, syringes, and multidose vials of anesthetics, contrast media, and antimicrobials—and in one case[57] a multiple-dose povidone iodine vial—as a suspected source of CSF contamination. One would assume that modern packaging has more or less eliminated this issue.

Evidence That Viral URI Increases the Load of Mouth Commensals

Further support for the droplet mechanism is provided by the following study. *Staphylococcus aureus*, including its methicillin-resistant variant, is another commensal of the upper airway. Its transmission by droplets (and skin scales) has been found responsible for several outbreaks of disease in nurseries, intensive care, and burn units. The history of these outbreaks is reviewed by Sheretz *et al.*[79] They describe their investigation of an outbreak of a cluster of methicillin-resistant *S. aureus* colonization among 8 of 43 patients in a surgical intensive care unit during a 3-week period in 1994. Five of the 8 patients developed methicillin-resistant *S. aureus* pneumonia, and another developed methicillin-resistant *S. aureus* bacteremia. Molecular typing of the isolates from the patients and from hospital personnel was performed. The analysis traced the probable source of the outbreak to one physician who had an upper respiratory tract infection (URI) during the time of the outbreak. They later found that, in this physician, there was little dispersal of the organism in the absence of a URI. In the presence of a URI—which (later) was purposely induced, using a strain of rhinovirus of type 39, administered through intranasal drops as part of the study—a surgical mask was effective in significantly reducing dispersal of the organism. These authors suggest that other bacterial pathogens such as *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Haemophilus influenzae*, and *Neisseria meningitidis* may also exhibit this "viral-bacterial interaction phenomenon." Viridans streptococcus, another mouth commensal, should be added to this list. These authors also point out that the observed increase in organism dispersal accompanying the URI may be due to the increase in nasal exudate with coughing and sneezing rather than a true viral–bacterial interaction. In addition to URIs, dental caries also increase the bacterial load of mouth commensals[80] and could increase the risk of organism dispersal. Although not a contraindication, judgment is important when considering LP in the presence of a URI. Those who perform LP should refrain from talking into the field.

Other Sources of Possible Contamination

Reinsertion of the stylet was shown by Strupp and Brandt[81] to reduce the incidence of post–dural puncture headache. After the LP needle is introduced (with stylet in place), the stylet is removed and presumably placed back on a sterile tray. In the interval between the needle's introduction and removal, the stylet lies on the tray exposed to ambient air, which can be the source of droplet contamination. Attention to sterile technique requires that the needle be placed under a barrier (a sterile cloth would do) during this interval. The same precaution holds in case the initial attempt at LP is unsuccessful and the needle is temporarily put back on the tray while landmarks are palpated.

Repetitive removal and insertion of syringes at the distal portion of the catheter, without gloves, should also be avoided.

Caps, Gowns, Gloves, Facemasks: Are They Necessary for Neuraxial Instrumentation?

In 1982, Seigel *et al.*,[82] using scanning electron microscopy, demonstrated contamination of the stylet tip from a spinal needle after it was lightly brushed against unwashed surgical gloves. After a sterile solution of povidone iodine was injected through the spinal needle, variously sized particles (mostly 5–10 μm) were found (using energy dispersive x-ray analysis). These particles were identified as starch, talc, glass particles from the vials, and plastic particles from the plastic syringe and tubing used in drawing up the povidone iodine. The use of an introducer for small-gauge blunt needles is likely to circumvent this problem.

A good surgical mask—as opposed to a plain paper mask—has been shown[83] to eliminate spray from the mouths of surgical staff while they talk. That is, oral flora could not be recovered from an agar plate placed at a distance of 30 cm from the mouth of persons who were wearing such a mask. When the same subjects talked without a mask, organisms grew on more than 50% of the plates. Excellent protection from a surgical mask lasts for only approximately 15 min. A proper large, soft, pleated, pliable mask (as opposed to a simple cloth or paper mask) remains a good bacterial filter for up to 8 h, but it may be prudent to change masks between cases.

There is controversy regarding the need for a mask for LP. The case for facemasks is made in several references.[84–88] The opposite position is taken by others.[89–92] Some call for studies to prove efficacy and cost-effectiveness. Because PDPM is rare, such a study would take an unlikely global effort. Several of the naysayers inappropriately quote Orr,[91] who describes an experiment at Severalls Surgical Unit, Colchester, England, where masks were not worn in the operating room for a period of 6 months, during which time the incidence of surgical wound infections did not increase (in fact, it significantly decreased). Because viridans streptococci are ordinarily not wound pathogens in immunocompetent subjects, an opinion about the wearing of masks in the operating room is not applicable to LP. In the case of LP, the CSF provides an excellent culture medium for these mouth and skin commensals, whereas a surgical wound does not.

The gown controversy parallels the mask controversy. Puzniak *et al.*[92] compare the use of gowns and gloves *versus* gloves alone in a medical intensive care unit with respect to their effect on horizontal transmission of vancomycin-resistant enterococci. They found that gowns were protective in reducing vancomycin-resistant enterococci acquisition in patients in the intensive care unit. Whether the gown's protective mechanism is *via* provision of a physical barrier to vancomycin-resistant enterococci transmission or *via* an indirect mechanism by enhancing compliance with other infection-control procedures (*e.g.*, hand washing) was not determined. Other studies,[93,94] in pediatric settings, do not show that gowns have a beneficial effect.

There is no national standard for dural puncture–related infection control, but guidelines do exist for prevention of intravascular central catheter–related infection.[95] These guidelines state that maximal sterile barrier precautions (cap, mask, sterile gown, sterile gloves, and large sterile drape) are applicable. There are two studies[96,97] that demonstrate reduced rate of infection using maximal precautions *versus* standard precautions (sterile gloves and small drapes). One hundred seventy-six patients who had a central venous catheter inserted using maximal sterile barrier precautions were compared with 167 control patients who had their catheters inserted using standard precautions. These matched patients were followed for 3 months after insertion or until the catheter was removed, whichever came first. There were a total of four catheter-related infections in the test group *versus* 12 in the control group ($P = 0.03$, chi-square test). The catheter-related septicemia rate was 6.3 times higher in the control group ($P = 0.6$, Fisher exact test).[96] Recommendations for the prevention of central line infections (which have a higher incidence rate than PDPM) cannot be automatically applied for LP. But because there are no similar infection-control studies for neuraxial techniques, these results should be noted.

Differential Diagnosis

Post–dural puncture meningitis, despite its rarity, should be on the list of differential diagnosis for any patient who develops headache, neck pain, or both after dural puncture. Similar findings are more frequently due post–dural puncture headache, but the seriousness of the former diagnosis requires a high index of suspicion.

The presence of fever, altered sensorium, or other neurologic signs can differentiate PDPM from post–dural puncture headache (clinically). Chemical meningitis, aseptic meningitis, and meningitis associated with certain drugs should also be considered if CSF findings do not suggest bacterial meningitis.

Management and Treatment

Management and treatment recommendations, given below, are in accord with the 2004 Bacterial Meningitis Guidelines of the Infectious Diseases Society of America (IDSA Guidelines).[98] Early recognition is critical.

The suspicion of meningitis should immediately trigger collection of blood and CSF for culture and analysis. But treatment should not be delayed while waiting for results. Empiric broad spectrum antimicrobial treatment and adjunctive therapy should be given promptly. In those cases where a head CT scan is indicated before LP, empiric treatment need not await the result. The scan is used to rule out signs of a mass lesion or signs of increased intracranial pressure. Under these circumstances, the possibility for brain herniation renders LP contraindicated. The IDSA Guidelines for adults recommend an LP (after start of adjunctive—where indicated—and empirical antimicrobial therapy) in all patients except in those who are immunocompromised, have a history of central nervous system disease, or have new-onset seizures, papilledema, altered consciousness, or focal neurologic deficit (based on the study of Hasbun *et al.*[99]). Under any of these conditions, LP should not be performed before a CT scan.

Early inflammatory response contributes to a series of events that are responsible for increased morbidity and mortality in animal models. The results of a prospective, randomized, double-blind, multicenter (European) study[100] (of adults with bacterial meningitis) suggest that dexamethasone, administered with or within 15–20 min of the first injection of antibiotic, significantly reduces the risks of both an unfavorable outcome and death. The beneficial effect of steroids is most evident in the subgroup of patients with pneumococcal meningitis.[100,101] The IDSA Guidelines recommend that adjunctive dexamethasone be continued for 2–4 days only if CSF Gram stain reveals gram-positive diplococci in blood or if CSF cultures are positive for *S. pneumoniae*. The IDSA Guidelines also recommend withholding steroids if the 15- to 20-min window is not met.[98] Both of these recommendations are at odds with a more recent report on community-acquired meningitis. Here, van de Beek *et al.*[102] would not restrict steroid use to only the *S. pneumoniae* group, and they are less rigid about defining the window of steroid usefulness at 20 min. They call for further study of these recommendations. No official treatment recommendation exists for the types of meningitis specifically related to LP. Because approximately 50% (or more, today) of cases of PDPM are caused by strains of viridans streptococci (a gram-positive diplococcus), it is reasonable that initial empiric treatment be as specified for *S. pneumoniae* in the IDSA Guidelines for both adjuvant and antimicrobial therapy. Other anti-inflammatory agents have been studied, but only in animals.

The concomitant administration of steroids and vancomycin probably results in lower CSF levels of vancomycin, because the increased antiinflammatory effect of the steroids lessens the penetration of vancomycin into the CSF.[98] Vancomycin is not recommended as the sole antimicrobial but rather in combination.

Empiric antimicrobial therapy recommendations for purulent meningitis are outlined in the IDSA Guidelines.[98] They are dependent on the patient's age and the most likely bacterial pathogen. Pending specific bacterial identification and sensitivity studies, vancomycin (to cover β-lactam resistance) plus a third-generation cephalosporin is recommended with the addition of ampicillin in patients older than 50 yr (to cover possible listeria).

Incidence of PDPM

The incidence of this condition is not known for the United States. The CDC requires meningitis surveillance for only *Neisseria meningitidis*. In the United States, national statistics for reportable diseases are dependent on local and state health departments, which, in turn, require notification by medical providers. There is no uniformity in the reportable-disease lists among the various states.[103] This system is acknowledged, by epidemiologists, to be inefficient and results in underreporting.[104] There are two elements to completeness of disease reporting: The condition must be diagnosed, and it must be reported. Underreporting has been observed to be disease specific. Completeness of reporting is especially important for infrequently occurring diseases.[105]

The National Bacterial Meningitis System (within the CDC) publishes results of periodic surveillance studies. One such report[106] states that the incidence of meningitis found in its study suggests that only 30–40% of all cases of bacterial meningitis in the participating states were reported through the surveillance system.

Not only is PDPM not reportable nationally as part of the CDC's meningitis statistics, it is also excluded as a nosocomial disease. The CDC collects data on hospital-acquired infection through its National Nosocomial Infection Surveillance System. However, its inclusion of a case of nosocomial bloodstream infection requires that ". . . one or more positive blood cultures be drawn at least 72 h after hospital admission, in asso-

ciation with clinical signs of sepsis . . ."[106] This requirement would exclude almost all the reported cases in table 1 from the official nosocomial classification—the typical case appears within 6 and 36 h after dural puncture.

A rough estimate of the number of cases of PDPM in the United States might be derived by extrapolating from estimates from other countries:

Incidents of serious injury to patients are reported to the National Board of Health and Welfare in Sweden (population 8.8 million). Using data contained in both the national administrative files and questionnaires completed by anesthesiologists, Moen *et al.*[60] report on certain severe neurologic complications that occurred after neuraxial blockades between 1990 and 1999 (in Sweden). Meningitis (purulent, not aseptic) after spinals and epidurals is among the various complications considered. The overall incidence of meningitis after spinal block was 1 case of meningitis in 53,000 (0.2/10,000) performed spinals. Of the 29 cases of meningitis found, 28 were due to viridans streptococcus. This would correspond to approximately 800 US cases during this 10-yr period, because the population of the United States is approximately 28 times that of Sweden.

In France, the SOS Regional Hotline Service solicited 8,150 French anesthesiologists to report complications of regional anesthesia occurring between August 1998 and May 1999.[107] The participants reported adverse events by calling a hotline as soon as the event occurred. The incidence was found to be 1 case of meningitis in 35,439 (0.3/10,000) performed spinals and 1 case of meningitis in 5,561 performed epidurals (1.8/10,000). Obstetric cases were not included. There was no reported incidence of meningitis in 5,640 performed spinals and 29,732 epidurals related to regional anesthesia in obstetrics.

After the occurrence of two cases of PDPM at a hospital in Brazil, the records of the Hospital Commission for Infection Control in Brazil were reviewed for cases of meningitis associated with spinal anesthesia between 1997 and 2000.[55] There, three cases after 38,128 spinal anesthesias (1.3/10,000) were identified.

Although it is difficult to estimate US incidence of PDPM from these varied sources, it seems likely that hundreds of such cases have gone unrecognized or unreported.

Toward Safer Health Care

Amalberti *et al.*[108] describe difficulties in improving patient care and offer some thoughtful solutions. The current medical mind-set with respect to safety can be improved. Achieving a safer system requires some modification of physician autonomy (which may require a hard sell). This would include more consultation between specialties for problem solving and a uniform, more efficient system for surveillance and monitoring.

It is not known what proportion of those who perform LP wear a proper mask (or take other maximal sterile barrier precautions) and, if they do, whether they change masks between cases. Nor is it known whether dural punctures are performed while the operator has a URI. It *is* known that wearing a mask is not the standard of care in many communities.[89] Black and Weinstein[90] describe their informal survey: Neurologists, internists, neurosurgeons and radiologists reported that they do not wear masks regularly when performing LPs on the ward, under fluoroscopic guidance, or during myelography; anesthesiologists more commonly wore masks for LP's and often noted that this is routine procedure in or out of the operating room. A well-designed survey among the various specialties, on both their mask-wearing conventions and their awareness of PDPM, would be useful.

The quotation under this article's title refers to a 1928 litigation that involved the T.J. Hooper, one of two tugboats that pulled three coal barges from Norfolk, Virginia, bound for New York. The tugs sank off the Jersey Coast in what is described as the perfect storm. At the time, such tugs were not required to be equipped with radio receivers. Had they had receivers, the crew would have been alerted to the coming storm in time to make safe haven at the Delaware breakwater until the storm blew over. The owners of the tugs and the barges were sued by the owners of the coal companies for their loss. The tug owners were found liable for damages, and they appealed. The appeals court upheld the lower court, ruling that "there are precautions so imperative that even their universal disregard will not excuse their omission." So it should be for the standard-of-care defense with respect to all aspects of sterile technique and awareness of potential harm when performing dural puncture.

References

1. Lichtenstein MJ, Tilley WS, Sandler MP: The syndrome of hypothalamic hypopituitarism complicating viral meningoencephalitis. J Endocrinol Invest 1982; 5:111–5

2. Christensen C, Bank A: Meningococcal meningitis and diabetes insipidus. Scand J Infect Dis 1988; 20:341–3

3. Franco-Paredes C, Evans J, Jurado R: Diabetes insipidus due to *Streptococcus pneumonia* meningitis. Arch Intern Med 2001; 161:1114–5

4. Sloane AE: Transient diabetes insipidus following listeria meningitis. Ir Med J 1989; 82:132–4

5. Juffermans NP, Verbon A, Van der Poll T: Diabetes insipidus as a complication of cryptococcal meningitis in an HIV-infected patient. Scand J Infect Dis 2002; 34:397–8

6. Baumann DP, Koch LC: Streptococcal meningitis following diagnostic lumbar puncture. Ann Int Med 1952; 36:1090–92

7. Berman RS, Eiselem JH: Bacteremia, spinal anesthesia and development of meningitis. ANESTHESIOLOGY 1978; 48:376–7

8. Schlesinger JJ, Salit IE, McCormick G: Streptococcal meningitis after myelography. Arch Neurol 1982; 39:576–7

9. Kilpatrick ME, Girgis NI: Meningitis: A complication of spinal anesthesia. Anesth Analg 1983; 62:513–5

10. Schelkun SR, Wagner KF, Blanks JA, Reinert CM: Bacterial meningitis following Pantopaque myelography. Orthopedics 1985; 8:73–76

11. Ready LB, Helfer D: Bacterial meningitis in parturients after epidural anesthesia. ANESTHESIOLOGY 1989; 71:988–90
12. Berga S, Trierweiler MW: Bacterial meningitis following epidural anesthesia for vaginal delivery: A case report. Obstet Gynecol 1989; 74:437–9
13. Roberts SP, Petts HV: Meningitis after obstetric spinal anaesthesia. Anaesthesia 1990; 45:376–7
14. Vukmirovits G, Todorova R, Aranyi Z, Kali G: Purulent meningitis, caused by Corynebacterium xerosis, after spinal anesthesia. Orv Hetil 1991; 132:2911–2, 2917–8
15. Lee JJ, Parry H: Bacterial meningitis following spinal anesthesia for caesarian section. Br J Anaesth 1991; 66:383–6
16. de Jong J, Barrs ACM: Lumbar myelography followed by meningitis (letter). Infect Control Hosp Epidemiol 1992; 13:74–75
17. Watanakunakorn C, Stahl C: Streptococcus salivarius meningitis following myelography (letter). Infect Control Hosp Epidemiol 1992; 13:454
18. Linnemann MU, Bulow HH: Infections after insertion of epidural catheters. Ugeskr Laeger 1993; 155:2350–2
19. Torres E, Alba D, Frank A, Diez-Tejedor E: Iatrogenic meningitis due to *Streptococcus* salivarius following a spinal tap (letter). Clin Infect Dis 1993; 17:525–6
20. Blackmore TK, Morley HR, Gordon DL: *Streptococcus mitis*–induced bacteremia and meningitis after spinal anesthesia. ANESTHESIOLOGY 1993; 78:592–4
21. Durand ML, Calderwood SB, Weber DJ, Miller SI, Southwick FS, Caviness VS, Swatz MN: Acute bacterial meningitis in adults: A review of 493 episodes. N Engl J Med 1993; 328:21–8
22. Newton JA, Lesnik IK, Kennedt CA: *Streptococcus* salivarius meningitis following spinal anesthesia (letter). Clin Infect Dis 1994; 18:840–1
23. Domingo P, Mancebo J, Blanch L, Coll P, Martinez E: Iatrogenic streptococcal meningitis. Clin Infect Dis 1994; 19:356–7
24. Garlicki S, Caban J, Krukowiecki J, Kluba-Wojewoda U: Four cases of bacterial meningitis after epidural anesthesia. Przegl Lek 1994; 51:268–9
25. Harding SA, Collis RE, Morgan BM: Meningitis after combined spinal-epidural anesthesia in obstetrics. Br J Anaesth 1994; 73:545–7
26. Pegues DA, Carr DB, Hopkins CC: Infectious complications associated with temporary epidural catheters. Clin Infect Dis 1994; 19:970–2
27. Stallard N, Barry P: Another complication of the combined extradural-subarachnoid technique (letter). Br J Anaesth 1995; 75:370–1
28. Gelfand MS, Abolnik IZ: Streptococcal meningitis complicating diagnostic myelography: Three cases and review. Clin Infect Dis 1995; 20:582–7
29. Veringa E, van Belkum A, Schellekens H: Iatrogenic meningitis by *Streptococcus* salivarius following lumbar puncture (letter). J Hosp Infect 1995; 29: 316–8
30. Aldebert S, Sleth JC: Bacterial meningitis after combined spinal and epidural anesthesia in obstetrics. Ann Fr Anesth Reanim 1996; 15:687–8
31. Baird RW: Streptococcal meningitis complicating myelography: Report of additional cases (letter). Clin Infect Dis 1996; 22:395
32. Schneeberger PM, Janssen M, Voss A: Alpha-hemolytic streptococci: A major pathogen of iatrogenic meningitis following lumbar puncture. Case reports and a review of the literature. Infection 1996; 24:29–33
33. Cascio M, Heath G: Meningitis following a combined spinal-epidural technique in a labouring term parturient. Can J Anaesth 1996; 43:399–402
34. Cooper AB, Sharpe MD: Bacterial meningitis and cauda equina syndrome after epidural steroid injections. Can J Anaesth 1996; 43(pt 1):471–4
35. Liu S, Pope A: Spinal meningitis masquerading as postdural puncture headache (letter). ANESTHESIOLOGY 1996; 85:1493–4
36. Kaiser E, Colineau X, Tourrette JH: Prevention of iatrogenic meningitis after myelography. J Radiol 1997; 78:15
37. Aromaa U, Lahdensuu M, Cozantitis DA: Severe complications associated with epidural and spinal anaesthesias in Finland 1987-1993: A study based on patient insurance claims. Acta Anaesthesiol Scand 1997; 41:445–52
38. Kaiser E, Suppini A, de Jaureguiberry JP, Paris JF, Quinot JF: Acute *Streptococcus* salivarius meningitis after spinal anesthesia. Ann Fr Anesth Reanim 1997; 16:47–9
39. Molinier S, Paris JF, Brisou P, Amah Y, Morand JJ, Alla P, Carli P: 2 cases of iatrogenic oral streptococcal infection: Meningitis and spondylodiscitis. Rev Med Interne 1998; 8:568–70
40. Laurila JJ, Kostamovaara PA, Alahuhta S: *Streptococcus salivarius* meningitis after spinal anesthesia. ANESTHESIOLOGY 1998; 89:1579–80
41. Bouhemad B, Dounas M, Mercier FJ, Benhamou dD: Bacterial meningitis following combined spinal-epidural analgesia for labor. Anaesthesia 1998; 53: 292–5
42. Duflo F, Allaouchiche B, Mathon L, Chassard D: Bacterial meningitis following obstetric combined spinal and peridural anesthesia. Ann Fr Anesth Reanim 1998; 17:1286
43. Schlegel C, Merlet JM, Fremaux A, Geslin P: Iatrogenic meningitis due to *Abiotrophia defectiva* after myelography. Clin Infect Dis 1999; 28:155–6
44. Cabellos C, Viladrich PF, Corredoira J, Verdaguer R, Ariza J, Gudiol F: Streptococcal meningitis in adult patients: Epidemiology and clinical spectrum. Clin Infect Dis 1999; 28:1104–8
45. Lurie S, Feinstein M, Hiefetz C, Manet Y: Iatrogenic bacterial meningitis after spinal anesthesia for pain relief during labor (letter). J Clin Anesth 1999; 11:438–9
46. Fernandez R, Paz I, Pazo C, del Rio D, Fernandez O: Meningitis produced by *Streptococcus mitis* after intradural anesthesia (letter). Enferm Infecc Microbiol Clin 1999; 17:150
47. Choy JC: Mortality from peripartum meningitis. Anaesth Intensive Care 2000; 28:328–30
48. Tortosa JA, Hernandez-Palazon J: Enterococcus faecalis meningitis after spinal anesthesia (letter). ANESTHESIOLOGY 2000; 92:909
49. Yaniv LG, Potasman I: Iatrogenic meningitis: An increasing role for resistant Viridans streptococci? Case report and review of the last 20 years. Scand J Infect Dis 2000; 32:693–6
50. Villevieille T, Vincenti-Rouquette I, Petitjeans F, Koulmann P, Legulluche Y, Rousseau JM, Diraison Y, Brinquin L: *Streptococcus* mitis-induced meningitis after spinal anesthesia. Anesth Analg 2000; 90:500–1
51. Varlet C, Gorce P, Ouaknine B, Pourriat JL: Meningitis after spinal anesthesia. Ann Fr Anesth Reanim 2000; 19:360–2
52. Idigoras P, Valiente A, Iglesias L, Trieu-Cuot P, Poyart C: Meningitis due to *Streptococcus salivarius* (letter). J Clin Microbiol 2001; 39:3017
53. Thomas TA, Cooper GM: Maternal deaths from anaesthesia: An extract from *Why Mothers Die 1997–1999*, The Confidential Enquiries into Maternal Deaths in the United Kingdom. Br J Anaesth 2002; 89:499–508
54. Trautman M, Lepper PM, Schmiyz FJ: Three cases of bacterial meningitis after spinal and epidural anesthesia. J Clin Microbiol Infect Dis 2002; 21:43–5
55. Videira RL, Ruiz-Neto PP, Brandao-Neto M: Post spinal meningitis and asepsis. Acta Anaesthesiol Scand 2002; 46:639–46
56. Couzigou C, Vuong TK, Botheral AH, Aggoune M, Astagneau P: Iatrogenic *Streptococcus salivarius* meningitis after spinal anesthesia: Need for strict application of standard precautions. J Hosp Infect 2003; 53:313–4
57. Kocamanoglu IS, Sener EB, Tur A, Ustin E, Sahinoglu H: Streptococcal meningitis after spinal anesthesia: Report of a case (letter). Can J Anesth 2003; 50:314–5
58. Pinder AJ, Dresner M: Meningococcal meningitis after combined spinal-epidural analgesia. Int J Obstet Anesth 2003; 12:183–7
59. Pandian JD, Sarada C, Radhakrishnan VV, Kishore A: Iatrogenic meningitis after lumbar puncture: A preventable health hazard. J Hosp Infect 2004; 56: 119–24
60. Moen V, Dahlgren N, Irestedt L: Severe neurological complications after neuraxial blockades in Sweden 1990–1999. ANESTHESIOLOGY 2004; 101:950–59
61. Conangla G, Rodriquez L, Alonzo-Tarres C, Avila A, de la Campa AG: *Streptococcus salivarius* meningitis after spinal anesthesia. Neurologia 2004; 19:331–3
62. Salazar MP, Perez JCR, Lopez G, Abella L, Maranes I, Linares M: Bacterial meningitis after intradural anesthesia. Rev Neurol 2004; 39:398
63. Vernis L, Duale C, Storme B, Mission JP, Roi B, Schoeffler P: Perispinal analgesia for labour followed by patient-controlled infusion with bupivacaine and sufentanil: Combined spinal-epidural *versus* epidural analgesia alone. Eur J Anaesthesiol 2004; 21:186–92
64. Hooten WM, Kinney MO, Huntoon MA: Epidural abscess and meningitis after epidural corticosteroid injection. Mayo Clin Proc 2004; 79:682–6
65. Siman-Tov T, Gadoth N: Enterococcal meningoencephalitis following epidural anesthesia. Isr Med Assoc J 2004; 6:780–1
66. Bussink M, Gramke H, van Kleef M, Marcus M: Bacterial meningitis ten days after spinal anesthesia (letter). Reg Anesth Pain Med 2005; 30:210–1
67. van de Beek D, de Gans J, Spanjaard L, Weisfelt M, Reitsma JB, Vermeulen M: Clinical and prognostic factors in adults with bacterial meningitis. N Engl J Med 2004; 351:1849–59
68. Lyytikainen O, Rautio M, Carlson P, Anttila V, Vuento R, Sarkkinen H, Kostiala A, Vaisanen ML, Kanervo A, Ruutu P: Nosocomial bloodstream infections due to viridans streptococci in haematological and non-haematological patients: Species distribution and antimicrobial resistance. J Antimicrob Chemother 2004; 53:631–4
69. Wisplinghoff H, Reinert R, Cornely O, Seifert H: Molecular relationships and antimicrobial susceptibilities of viridans group streptococci isolated from blood of neutropenic cancer patients. J Clin Microb 1999; 37:1876–80
70. Pfaller MA, Jones RN, Doern GV, Sader HS, Kugler KC, Beach ML: Survey of blood stream infections attributable to gram-positive cocci: Frequency of occurrence and antimicrobial susceptibility to isolates collected in 1997 in the United States, Canada, and Latin America from the SENTRY Antimicrobial Surveillance Program. SENTRY Participants Group. Diagn Microbiol Infect Dis 1999; 33:283–97
71. Facklam R: What happened to the streptococci: Overview of taxonomic and nomenclature changes. Clin Microbiol Rev 2002; 15:613–30
72. Quagliarello VJ, Scheld WM: Bacterial meningitis: Pathogenesis, pathophysiology, and progress. N Engl J Med 1992; 327:864–72
73. Wang LP, Hauerberg J, Schmidt JF: Incidence of spinal epidural abscess after epidural analgesia: A national 1-year survey. ANESTHESIOLOGY 1999; 91:1928–36
74. Schroeder TH, Krueger WA, Neeser E, Hahn U, Unertl K: Spinal epidural abscess: A rare complication after epidural analgesia for labour and delivery. Br J Anaesth 2004; 92:896–8
75. Weed LH, Wegeforth P, Ayer J, Felton L: Influence of certain experimental procedures upon the production of meningitis by intravenous inoculation. Monogr Rockefeller Inst Med Res 1920; 12:57–112
76. Carp H, Bailey S: The association between meningitis and dural puncture in bacteremic rats. ANESTHESIOLOGY 1992; 75:667–9

77. Teele DW, Dashefsky B, Rakusan T, Klein JO: Meningitis after lumbar puncture in children with bacteremia. N Engl J Med 1981; 305:1079–81
78. Beland B, Prien T, Van Aken H: Spinal and regional anesthesia in bacteremia. Anesthesist 2000; 46:536–47
79. Sheretz RJ, Reagan DR, Hampton KD, Robertson KL, Streed SA, Hoen HM, Thomas R, Gwaltney JM Jr: A cloud adult: The Staphylococcus aureus-virus interaction revisited. Ann Intern Med 1996; 124:537–47
80. Dajani AS, Taubert KA, Wilson W, Bolger AF, Bayer A, Bayer A, Ferrieri P, Gewitz MH, Shulman ST, Nouri S, Newburger JW, Hutto C, Pallasch TJ, Gage TW, Levison ME, Peter G, Zuccaro G: Prevention of bacterial endocarditis: Recommendations by the American Heart Association. Circulation 1997; 96:358–66
81. Strupp M, Brandt T: Should one reinsert the stylet during lumbar puncture? N Engl J Med 1997; 336:1190
82. Seigel RS, Williams AG, Waterman RE: Potential complications in myelography: I. Technical considerations. Am J Roentgenol 1982; 138:705–8
83. Philips BJ, Fergusson S, Armstrong P, Anderson FM, Wildsmith JAW: Surgical face masks are effective in reducing bacterial contamination caused by dispersal from the upper airway. Br J Anaesth 1992; 69:407–8
84. Baer ET: Iatrogenic meningitis: The case for face masks. Clin Infect Dis 2000; 31:519–21
85. Moen V: Meningitis is a rare complication of spinal anesthesia: Good hygiene and face masks are simple preventative measures. Lakartidningen 1998; 95:628, 631–2, 635
86. Jorbeck H: Good hygiene prevents meningitis after lumbar puncture: The best insurance is using face masks. Lakartidningen 1998; 95:596–7
87. McLure HA, Talboys CA, Yentis SM, Azadian BS: Surgical face masks and downward dispersal of bacteria. Anesthesia 1998; 53:624–6
88. Dolinski S, Gerancher JC: Unmasked mischief (letter). Anesth Analg 2001; 92:280–1
89. Gelfand MS, Cook DM: Streptococcal meningitis as a complication of diagnostic myelography: Medicolegal aspects. Clin Infect Dis 1996; 22:130–2
90. Black SR, Weinstein RA: The case for face masks: Zorro or zero? Clin Infect Dis 2000; 31:522–3
91. Orr NWM: Is a mask necessary in the operating theatre? Ann R Coll Surg England 1981; 63:390–92
92. Puzniak LA, Leet T, Mayfield J, Kollef M, Mundy LM: To gown or not to gown: The effect on acquisition of vancomycin-resistant enterococci. Clin Infect Dis 2002; 35:18–25
93. Renaud MT: Effects of discontinuing cover gowns on a postpartal ward upon cord colonization of the newborn. JOGN Nurs 1993; 12:399–410
94. Donowitz LG: Failure of the overgown to prevent nosocomial infection in a pediatric intensive care unit. Pediatrics 1986; 77:35–8
95. O'Grady NP, Alexander M, Dellinger EP, Gerberding JL, Heard SO, Maki DG, Masur H, McCormick RD, Mermel LA, Pearson ML, Raad II, Randolph A, Weinstein A: Guidelines for the prevention of intravascular catheter-related infections. Clin Infect Dis 2002; 35:1281–307
96. Mermel LA, Mc Cormick RD, Springman SR, Maki DG: The pathology and epidemiology of catheter-related infection with pulmonary artery Swan-Ganz catheters: A prospective study utilizing molecular subtyping. Am J Med 1991; 91 (suppl 3B):S197–205
97. Raad II, Hohn DC, Gilbreath BJ, Suleiman N, Hill LA, Bruso PA, Marts K, Mansfield PF, Bodey GP: Prevention of central venous catheter-related infections by using maximal sterile barrier precautions during insertion. Infect Control Hosp Epidemiol 1994; 15:231–8
98. Tunkel AR, Hartman BJ, Kaplan SL, Kaufman BA, Roos KL, Scheld M, Whitley RJ: Practice guidelines for the management of bacterial meningitis. Clin Infect Dis 2004; 39:1267–84
99. Hasbun R, Abrahams J, Jekel J, Quagliarello VJ: Computed tomography of the head before lumbar puncture in adults with suspected meningitis. N Engl J Med 2001; 345:1727–33
100. de Gans J, van de Beek D: Dexamethasone in adults with bacterial meningitis. N Engl J Med 2002; 347:1549–56
101. van de Beek D, de Gans J, McIntyre O, Prasad K: Steroids in adults with bacterial meningitis: A systematic review. Lancet Infect Dis 2004; 4:139–43
102. van de Beek D, de Gans J, Tunkel AR, Wijdicks WFM: Community-acquired bacterial meningitis in adults. N Engl J Med 2006; 354:44–52
103. Roush S, Birkhead G, Koo D, Cobb A, Fleming D: Mandatory reporting of diseases and conditions by health care professionals and laboratories. JAMA 1999; 282:164–70
104. Marier R: The reporting of communicable diseases. Am J Epidemiol 1977; 105:587–90
105. Doyle TJ, Glynn KG, Groseclose SL: Completeness of notifiable disease reporting in the United States: An analytical literature review. Am J Epidemiol 2002; 155:866–74
106. Pittet D, Li N, Woolson F: Microbiological factors influencing the outcome of nosocomial bloodstream infections: A 6-year validated, population-based model. Clin Infect Dis 1997; 24:1068–78
107. Auroy Y, Benhamou D, Bargues L, Ecoffey C, Failissard, Mercier F, Bouaziz H, Sami K: Major complications of regional anesthesia in France: The SOS Regional Anesthesia Hotline Service. ANESTHESIOLOGY 2002; 97:1274–80
108. Amalberti R, Auroy Y, Berwick D, Barach P: Five system barriers to achieving ultrasafe health care. Ann Intern Med 2005; 142:756–64