Neurological Infections After Neuraxial Anesthesia

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Nowadays various forms of neuraxial analgesia and anesthesia are widely used in obstetrics. Just over a century ago spinal analgesia was first applied in late labor [1]; it continued to be used by enthusiasts until the practice of continuous lumbar epidural analgesia and then the local anesthetic bupivacaine took the stage in the 1960s and 1970s. The reemergence of atraumatic spinal needles in the latter part of the twentieth century then opened the way for the wider use of spinal and combined spinal-epidural blockade. Over the years the complications associated with the various techniques have emerged and been tackled, so that currently serious complications of neuraxial blockade are extremely rare, particularly among the obstetric population, who are usually young and fit. Among these serious complications, however, neuraxial infection, comprising meningitis and epidural and para-epidural infection, is probably the most important, for two reasons. First, infection is the now commonest cause of neuraxial injury claims among obstetric patients in the United States [2]. Second, by our own actions we may affect its occurrence. Every case of infection following neuraxial blockade therefore deserves our close attention.

Although both epidural abscess and meningitis are complications of neuraxial anesthesia, their causation and risk factors are disparate. While epidural abscess is primarily a complication of epidural catheter use, the route of infection via the catheter entry point and the causative organism, the *Staphylococcus*, nosocomial meningitis is a complication of dural puncture (for whatever purpose) and is usually caused by streptococci of the viridans type, commonly found in the upper air passages and the vagina.

The rarity of both types of complication makes incidence, relative risk, and efficacy of preventive measures impossible to establish using high-quality
evidence. Reliance therefore has to be placed on case reports, indirect
evidence, and common sense.

**Incidence**

Expectant mothers often ask about the incidence of complications of
neuraxial anesthesia, but there is, in truth, no such thing as “an incidence”
of meningitis or epidural abscess, accurate or otherwise. The incidence of
problems varies widely, depending on the skill and training of the practi-
tioners concerned, as well as on the risk factors in the population. The fre-
quent occurrence of case clusters gives the lie to any attempt to measure
a true incidence. Anesthesiologists, nevertheless, have a duty to inform
patients of the complications associated with a proposed procedure and
are expected to give some figure for the level of risk, however futile such
estimates may be.

A number of surveys have been published that attempted to establish the
frequency of serious complications of neuraxial blockade, but the findings
vary widely, for many reasons. Some old surveys, although they may be
based on accurate local records, relate to a time when practices and prepa-
ration of equipment and drugs were less safe than they are today. Local
audit is likely to be more accurate than any multicenter survey, but now,
with the incidence of complications so much reduced, small numbers lack
the power to estimate it. Moreover, level of skill can still have a major
impact. For example, a Danish national survey of the incidence of epidural
abscess after epidural analgesia demonstrated an incidence of 1:5661 in
university hospitals and of 1:796 in community hospitals [3]. The problems
associated with all such surveys are summarized in Box 1.

Surveys of regional block complications that include both surgical and
obstetric patients indicate that complications arise less frequently among
the latter. Moen and colleagues [4] detected 29 cases of meningitis (albeit
clustered) in a Swedish national survey involving 1,260,000 spinal anes-
thetics for surgical patients, but none among a possible 55,000 spinals for
cesarean section, and 12 epidural abscesses among 450,000 surgical epidu-
rals and one among 200,000 for labor. A French national survey also found
fewer complications among obstetric than surgical patients [5]. The sterility
of the delivery suite may not be so scrupulously controlled as that of the
operating room (OR), but the parturient, being usually young and fit, seems
to come off relatively lightly.

The numbers of infectious complications found in surveys of neuraxial
blocks in obstetric patients are given in Table 1 [6–13]. Surveys that were
not designed to detect such complications, or those that were prompted
by the occurrence of a case or case series, as reflecting an inevitable bias,
are excluded. The included surveys, given in chronologic order, initially fo-
cused on epidural analgesia, but with increased use of spinal and combined
spinal-epidural (CSE) blocks, such limitations became appropriate. Little
reliance should be placed on a recent attempt to collate the findings of surveys of epidural complications in obstetric patients [14], as it overlooked the wide use of spinal and hybrid techniques and used faulty denominators. The studies included in Table 1 provide denominators of 116,987 spinals and CSEs and 908,270 epidurals. There were only 629 spinals in the regional survey by Holdcroft and colleagues, [10] too few for inclusion. The prospective multicenter study by Scott and Tunstall [9] mentioned two unconfirmed cases of meningitis. If these are included and presumed to fall among the spinal anesthetics, this gives an aggregated incidence of meningitis among the spinals in all these surveys of 1:39,000 or 25.6 per million (95% confidence interval [CI]: 5.3 to 74.9 per million) and of epidural abscesses among the epidurals of 1:303,000 or 3.3 per million (95% CI: 0.7 to 9.7 per million). I would not vouch for either the accuracy or the validity of these figures nor rely on their confidence intervals!

Information from surveys involving surgical patients emphasizes the idiosyncratic nature of infectious complications. In a US retrospective review of 4767 spinal anesthetics from a single institution, there were no infectious complications [15]; similarly in a Swedish follow-up study of 8501 spinals and 9232 epidurals, there were no instances of meningitis or abscess [16], whereas in a Danish survey of 17,372 epidural anesthetics, there were nine epidural abscesses, admittedly all in elderly people, eight of whom were immunocompromised [3]. In a Brazilian survey there were three cases of meningitis following 38,128 spinal anesthetics, and none in 12,822

### Box 1. Reasons for inaccuracy in surveys of infective complications of neuraxial blockade in obstetric patients

1. Many questionnaire surveys suffer from a poor response rate, and potentially, therefore, a reporting bias.
2. Clinical practice has changed radically since the older surveys were conducted.
3. Small local audit may be more precise than larger multicenter surveys, but with improvement in practice, smaller studies lack power to assess incidence of rare disorders with accuracy.
4. Level of skill and training vary from place to place.
5. Denominator data are inaccurate, while numerators may lack diagnostic precision.
6. Cases arising late, in the community, may be missed.
7. Surveys that are prompted by the occurrence of a case or cluster of cases have a built-in positive reporting bias.
8. Infectious complications usually arise for a specific reason; they are not natural events with “an incidence” as such.
patients who received other types of anesthesia [17]. Interestingly, because there were too few cases to attain statistical significance, the authors stated “The incidence of meningitis was similar in patients subjected to spinal anesthesia and in those subjected to another anesthetic technique.” Oh the mockery of statistics!

It is sometimes supposed that neuraxial infection may be a chance event in relation to neuraxial anesthesia. Indeed, in an excellent review of neurologic complications of neuraxial anesthesia in obstetrics, Loo and colleagues [18] highlight the numbers of case reports of epidural abscess in obstetric patients that occurred in the absence of anesthesia. In the case of meningitis, the causative agents in nosocomial infection are quite different from those in community-acquired disease [18], so the two are readily distinguished.

**Meningitis**

Meningitis may follow diagnostic lumbar puncture and myelography as well as neuraxial anesthesia. Despite the paucity of cases that are detected...
in surveys of neuraxial anesthesia, case reports abound. Thirty-eight con-
cerning obstetric patients are summarized in Table 2 [19–46]. There is one
case of viral meningitis (case 8 [24]) and one of community-acquired disease
(case 29 [43]) but neuraxial anesthesia is implicated in the rest. The first seven
cases listed followed saddle-block spinal anesthesia using nondisposable
equipment and tetracaine, a practice then popular in the United States for
the second stage of labor. Cases 1 to 3 were from a US army hospital in Ger-
many where spinals were given by obstetricians, and all occurred within 3
weeks [21]. They were ascribed to chemical meningitis attributed to detergent
used to wash equipment. This poses the question: why the low cerebrospinal
fluid (CSF) glucose? This is, of course, a diagnostic feature of bacterial men-
ingitis. In these cases, as in case 4 [22], undetected bacterial infection would
seem more likely. Cases 5 to 7, another case cluster, resulted from extraor-
dinary practice by a single anesthesia provider [23]. Thereafter, causation is
more subtle. Two cases that were reported as meningitis (cases 11 and 28)
[24,40] rather resembled epidural infection or abscess.

Clinical features and management

Among the patients listed in Table 2, symptoms appeared hours or a few
days after anesthesia, the exception being among the cases of *Aspergillus*
meningitis, in whom the onset time was up to 1 month [45,46].

The initial clinical picture is of headache and fever, often with backache
and emetic symptoms, classical signs of meningism, drowsiness, and leth-
argy. Some cases have been confused initially with dural puncture headache,
one receiving two blood patches [23]. The condition is usually benign when
treated promptly. In severe and untreated cases, the patient may become
unrousable, with diabetes insipidus and other signs of cerebral edema
[44–46]. Among the 38 cases in Table 2, there were six deaths, three due
to the *Aspergillus* (see below under “Causative organisms”). There can be
no cause for complaisance in this condition, particularly as resistant organ-
isms are emerging even among the meek viridans *Streptococcus* [47].

The CSF is often cloudy and shows a raised white cell count, predomin-
antly neutrophils, raised protein, and low glucose concentrations. CSF
culture may yield no growth, as in cases 1 to 4, 12 to 15, 19, 21, 24, and
27 (see Table 2), even when bacteria are visible on microscopy. Such cases
are sometimes diagnosed as chemical meningitis, but previous antibiotic
treatment may contribute to this finding, while the white cell count and still
more the low CSF glucose suggest a bacterial origin.

Lumbar puncture aids diagnosis, but it should be remembered that it is risky
in the presence of raised intracranial pressure [44] and, indeed, epidural ab-
scess. Imaging may therefore take precedence in doubtful cases. It is clearly im-
portant to give appropriate antibiotics early, which will usually be before the
causative organism or its sensitivity is established, recognizing that antibiotics
may impede its culture. Vancomycin, with third-generation cephalosporins,
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<th>Organism and findings</th>
<th>Treatment</th>
<th>Comments and outcome</th>
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<tbody>
<tr>
<td>Gibbons [19] US</td>
<td>1. Spinal for labor, forceps delivery</td>
<td>2 h</td>
<td>In all cases, CSF typical for bacterial meningitis, including low glucose, but no growth. Ascribed to “chemical meningitis” due to detergent</td>
<td>Ampicillin</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td>2. Spinal for labor, forceps delivery</td>
<td>6 h</td>
<td></td>
<td>Penicillin, chloramphenicol</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td>3. Spinal for labor, forceps delivery</td>
<td>3 h</td>
<td></td>
<td>Penicillin, chloramphenicol</td>
<td>Recovered</td>
</tr>
<tr>
<td>Phillips [20] USA</td>
<td>4. Spinal for labor</td>
<td>2 h</td>
<td>2nd LP consistent with bacterial meningitis</td>
<td>Colistin, cefalotin, hydrocortisone</td>
<td>Recovered</td>
</tr>
<tr>
<td>Corbett and Rosenstein [21] USA</td>
<td>5. Spinal for labor</td>
<td>36 h</td>
<td>Case cluster: <em>Pseudomonas aeruginosa</em> in all cases, also grown from multiuse bottle of saline used to rinse stylet before insertion</td>
<td>Colistin, polymyxin B</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td>6. Spinal for labor</td>
<td>3 days</td>
<td></td>
<td>Colistin, polymyxin B</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td>7. Spinal for labor</td>
<td>4 days</td>
<td></td>
<td>Colistin, polymyxin B</td>
<td>Recovered</td>
</tr>
<tr>
<td>Neumark et al [22] Austria</td>
<td>8. Uncomplicated epidural for labor</td>
<td>8 days</td>
<td>Coxsackie B (unrelated)</td>
<td>Intensive care, no mention of antibiotics</td>
<td>h/o TB; stormy passage, recovered</td>
</tr>
<tr>
<td>Berga and Trierweiler [23] USA</td>
<td>9. Accidental dural puncture in labor Epidural blood patch × 2</td>
<td>1 day</td>
<td><em>Streptococcus sanguis</em></td>
<td>Oxacillin, cefotaxime</td>
<td>Recovered</td>
</tr>
<tr>
<td>Ready and Helfer [24] USA</td>
<td>10. Uncomplicated epidural for labor PI to skin</td>
<td>1 day</td>
<td><em>Streptococcus uberis</em> <em>(α-hemolytic)</em></td>
<td>Ceftriaxone, vancomycin, then ampicillin</td>
<td>Recovered</td>
</tr>
<tr>
<td>#</td>
<td>Source</td>
<td>Description</td>
<td>Duration</td>
<td>Pathogens/ Antimicrobials</td>
<td>Outcome</td>
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<td>11</td>
<td>Roberts and Petts [25] UK</td>
<td>Uncomplicated epidural for CS. PCEA hydromorphone for 48 h</td>
<td>5 days</td>
<td><em>Streptococcus faecalis</em>; epidural inflammation; not drowsy</td>
<td>Recovered</td>
</tr>
<tr>
<td>12</td>
<td>Sansome et al [26] UK</td>
<td>Spinal for retained placenta</td>
<td>18 h</td>
<td>CSF typical for bacterial meningitis but no growth</td>
<td>Recovered</td>
</tr>
<tr>
<td>13</td>
<td>Bugedo [27] Chile</td>
<td>Accidental dural puncture; chlorhexidine; epidural replaced</td>
<td>3 days</td>
<td>CSF typical for bacterial meningitis but glucose near normal and no growth</td>
<td>Recovered</td>
</tr>
<tr>
<td>14</td>
<td>Lee and Parry [28] UK</td>
<td>Spinal for CS</td>
<td>5 h</td>
<td>Signs of bacterial meningitis</td>
<td>Recovered</td>
</tr>
<tr>
<td>15</td>
<td>Davis et al [29] UK</td>
<td>Early labor, cesarean for genital herpes, chlorhexidine, spinal three attempts. Mask not worn as of doubtful value (sic)</td>
<td>16 h</td>
<td>CSF typical for bacterial meningitis but no growth</td>
<td>Recovered</td>
</tr>
<tr>
<td>16</td>
<td>Newton et al [30] USA</td>
<td>Uncomplicated epidural for labor “full aseptic technique” no mask!</td>
<td>2 days</td>
<td>Group B <em>Streptococcus</em> from blood and vagina</td>
<td>Recovered</td>
</tr>
<tr>
<td>17</td>
<td>Lurie et al [31] Israel</td>
<td>Spinal for labor; mask worn, PI to skin; vaginal delivery</td>
<td>12 h</td>
<td><em>Streptococcus salivarius</em></td>
<td>Recovered</td>
</tr>
<tr>
<td>18</td>
<td>Harding et al [32] UK</td>
<td>Spinal for labor; chlorhexidine</td>
<td>12 h</td>
<td><em>Streptococcus viridans</em></td>
<td>Recovered</td>
</tr>
<tr>
<td>19</td>
<td>CSE for labor, sterile gown and gloves, no mask assumed; chlorhexidine; forceps delivery</td>
<td>3 days</td>
<td>Bacterial meningitis picture except normal glucose, no growth</td>
<td>Recovered</td>
<td></td>
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<table>
<thead>
<tr>
<th>Author</th>
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<th>Treatment</th>
<th>Comments and outcome</th>
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<tbody>
<tr>
<td><strong>20.</strong> Stallard and Barry [33] UK</td>
<td>CSE, early labor, epidural re-sited, spinal for emergency CS; blood patch</td>
<td>21 h</td>
<td><em>Staphlococcus epidermidis</em></td>
<td>Vancomycin, cefotaxime</td>
<td>Recovered</td>
</tr>
<tr>
<td><strong>21.</strong> Stallard and Barry [33] UK</td>
<td>Epidural × 2 for labor; mask and chlorhexidine; spinal for cesarean same space</td>
<td>18 h</td>
<td>Bacterial (no growth)</td>
<td>IV antibiotics</td>
<td>Recovered but had PDPH</td>
</tr>
<tr>
<td><strong>22.</strong> Goldstein et al [34] USA</td>
<td>Epidural for labor and CS</td>
<td>6 days</td>
<td>Group B <em>Streptococcus</em></td>
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<tr>
<td><strong>23.</strong> Cascio and Heath [35] USA</td>
<td>CSE for labor</td>
<td>16 h</td>
<td><em>S. salivarius</em> (dismissed as contaminant)</td>
<td>Vancomycin, ceftriaxone</td>
<td>Recovered</td>
</tr>
<tr>
<td><strong>24.</strong> Donnelly et al [36] UK</td>
<td>Membranes ruptured; spinal for CS; chlorhexidine; no mask</td>
<td>4 days</td>
<td>CSF no growth, glucose not measured; bacterial meningitis diagnosed</td>
<td>Ampicillin, cefotaxime</td>
<td>Recovered</td>
</tr>
<tr>
<td><strong>25.</strong> Bouhemad et al [37] France</td>
<td>CSE for labor; wore a mask, iodine skin prep, vaginal delivery</td>
<td>14 h</td>
<td><em>S. salivarius</em></td>
<td>Amoxicillin, cefotaxime, fosfomycin</td>
<td>Recovered</td>
</tr>
<tr>
<td><strong>26.</strong> Duflo et al [38] France</td>
<td>CSE for labor (“all hygienic measures taken” but sufentanil ampoule shared), vaginal delivery</td>
<td>8 days</td>
<td><em>S. viridans</em></td>
<td>Ceftriaxone</td>
<td>Recovered</td>
</tr>
<tr>
<td><strong>27.</strong> Choy [39] Singapore</td>
<td>Epidural for labor; first attempt failed; mask worn; PI skin prep</td>
<td>3 days</td>
<td>Delayed diagnosis of meningitis, no growth but low CSF glucose</td>
<td>Ceftriaxone, metronidazole then ceftazidine, isoniazid, rifampicin, acyclovir</td>
<td>Died</td>
</tr>
<tr>
<td>Authors</td>
<td>Case Details</td>
<td>Incubation</td>
<td>Pathogen(s)</td>
<td>Treatment</td>
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<tr>
<td>Trautman et al [40]</td>
<td>28. Epidural for twin delivery</td>
<td>2 days</td>
<td><em>Staphylococcus aureus</em> from anesthesiologist’s nose; called meningitis but actually abscess</td>
<td>Ceftriaxone, netilmicin</td>
<td>Recovered</td>
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<tr>
<td>Pinder and Dresner [41] UK</td>
<td>29. CSE for labor</td>
<td>1 day</td>
<td><em>Neisseria meningitidis</em> (ie, chance event)</td>
<td>Cefotaxime, amoxicillin</td>
<td>Recovered</td>
</tr>
<tr>
<td>Vernis et al [42] France</td>
<td>30. CSE for labor</td>
<td></td>
<td>One case of meningitis in the course of a randomized trial</td>
<td></td>
<td>Recovered</td>
</tr>
<tr>
<td>Thomas and Cooper [43] UK</td>
<td>31. Spinal for CS in preeclampsia; no mention of mask or antibiotic cover</td>
<td></td>
<td>CSF findings not mentioned</td>
<td></td>
<td>Died</td>
</tr>
<tr>
<td>Baer [44] USA</td>
<td>32. Accidental dural puncture, epidural replaced, delivered vaginally</td>
<td>&lt;1 day</td>
<td><em>Staphylococcus simulans</em> from CS, <em>S salivarius</em> from blood</td>
<td>Ceftriaxone, vancomycin, meropenem, dexamethasone</td>
<td>Died</td>
</tr>
<tr>
<td>Rodrigo et al [45] Sri Lanka</td>
<td>33. Spinal for elective CS</td>
<td>7 days</td>
<td><em>Aspergillus fumigatus</em></td>
<td>Broad-spectrum antibiotics, fluconazole</td>
<td>Died</td>
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<td></td>
<td></td>
<td>Antibiotics, amphotericin</td>
<td>Died</td>
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<tr>
<td></td>
<td>34. Spinal for emergency CS</td>
<td>10 days</td>
<td><em>A fumigatus</em></td>
<td></td>
<td>Died</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>Antibiotics, amphotericin</td>
<td>Died</td>
</tr>
<tr>
<td></td>
<td>35. Spinal for elective CS</td>
<td>23 days</td>
<td><em>A fumigatus</em></td>
<td></td>
<td>Survived</td>
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<td></td>
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<td></td>
<td>Itraconazole, amphotericin, voriconazole</td>
<td>Survived</td>
</tr>
<tr>
<td></td>
<td>36. Spinal for elective CS</td>
<td>11 days</td>
<td><em>A fumigatus</em></td>
<td></td>
<td>Died</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Antibiotics, late antifungal therapy</td>
<td>Died</td>
</tr>
<tr>
<td></td>
<td>37. Spinal for elective CS</td>
<td>2 days</td>
<td><em>Pseudomonas, A fumigatus</em></td>
<td></td>
<td>Survived</td>
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<tr>
<td></td>
<td>38. Spinal for elective CS, previous double valve replacement</td>
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</table>

**Abbreviations:** CS, cesarean section; CSE, combined spinal-epidural; CSF, cerebrospinal fluid; h/o TB, history of tuberculosis; IV, intravenous; LP, lumbar puncture; PCEA, patient-controlled epidural analgesia; PDPH, post-dural puncture headache; PI, povidone iodine.
should be given pending further information. The use of steroids is debatable, but usually recommended for community-acquired meningitis [44].

Causative organisms

Taking into account these and the numerous reported cases of post–spinal meningitis in surgical patients, in the great majority the causative organism is a viridans-type or α-hemolytic Streptococcus, often identified as Streptococcus salivarius [4,30,31,35,37,38,44]. These organisms are normally of low virulence and, as the name implies, live harmlessly in the upper air passages, as well as the vagina, but they like a watery environment and the cerebrospinal fluid provides a suitable culture medium. By the same token they are disinclined to grow on culture plates, but prefer broth, hence the frequency with which they are apparently missed, or dismissed as contaminants merely because they are commensals [35,44]. Nevertheless, once carried to the subarachnoid space by special conveyance, cause meningitis they certainly do.

In some cases, β-hemolytic Streptococcus or Pseudomonas have been isolated; the latter also loves a watery medium. A recent case was described of Herpes simplex meningitis following cesarean section [48], but the type of anesthesia was not reported and the authors failed to respond to a written request for clarification.

A cluster of cases of meningoencephalitis occurring in July 2005 in previously fit women who had undergone cesarean section in Sri Lanka, half of whom died, caused great concern and some initial perplexity. The quality of anesthesia and the aseptic technique were immaculate and it was not until a fungal infection was confirmed at the first autopsy that subsequent lives could be saved by antifungal treatment [45,46]. The Aspergillus does not normally cause meningitis except in the immunocompromised; like Streptococcus viridans it requires a means of entry to the CSF. The source of the infection turned out to be syringes used for spinal anesthesia that had been donated following the tsunami and stored in an unsuitable warehouse, at 41°C and 75% humidity.

There is a clear distinction between the range of organisms causing nosocomial meningitis and those causing community-acquired meningitis (Neisseria meningitidis, Streptococcus pneumoniae, or Haemophilus influenzae). Among the cases in Table 2, the odd exception [22,41] proves the rule.

Risk factors

Among the 36 cases of anesthesia-related meningitis described here, 21 followed spinals, 6 followed CSEs, and 3 followed accidental dural puncture. Thus 30 of 36 followed known dural puncture. Of the six cases following epidural blockade, two probably had primarily epidural infection (cases 11 and 28), two insertions were difficult and probably involved undetected
dural damage (a well-recognized possibility, even with “normal” epidurals), and two were caused by a Group B *Streptococcus* (cases 16 and 22), a more virulent organism also to be found in the patient’s bloodstream and vagina. Nevertheless, in an Iowa study of 73 parturients with β-hemolytic streptococcal infection, the only one to develop meningitis had been given spinal anesthesia [49]. Survey findings [4,17,49] as well as case reports [44] would support dural puncture as a prerequisite for nosocomial meningitis, substantiating the view that, in the cases following apparently normal epidural analgesia, there was undetected dural puncture.

Clearly a contaminated needle may carry organisms from the operator’s unmasked upper air passages [40,44] or from the patient’s skin, into the CSF, but as dural puncture may be associated with the entry of blood into the cerebrospinal fluid, the source of organisms may also be the patient’s bloodstream [24,29,30]. Bacteremia is therefore a contributory factor.

It is noticeable that the great majority of parturients with nosocomial meningitis have labored. Despite the occurrence of meningitis among surgical patients, no obstetric cases were found in the Swedish survey, where spinal and CSE anesthesia are rarely used during labor [4]. Meningitis appears surprisingly rare when spinals are used for elective caesarean section. There are five possible reasons.

1. The spinal is sited in the OR, a cleaner environment than the delivery suite.
2. The patient is not thrashing about in an amniotic fluid-soaked bed.
3. In the OR the anesthesiologist is more likely to wear a mask.
4. Unlike vaginal delivery, elective cesarean section is not normally associated with streptococcal bacteremia.
5. An antibiotic is always given immediately after delivery by cesarean section.

As among surgical patients [4], immunocompromise is a theoretic risk factor, although the parturients in the cases cited in Table 2 were apparently fit beforehand.

There is no evidence that vaginal delivery per se, even with genital tract sepsis, but without neuraxial anesthesia, is a sufficient risk factor for meningitis [49,50]. The implication would seem to be that the dura should not be punctured during labor if an epidural would do instead.

**Epidural abscess and related infection**

Epidural abscess is a recognized complication of epidural catheterization, but it may also occur spontaneously [18]. It arises infrequently among obstetric patients, as it is seen with greatest frequency among the elderly and immunocompromised [3,4]. Cases have been reported sporadically following neuraxial blockade in obstetric patients [40,51–65]; they are summarized in Table 3. One was reported as meningitis, but was actually an
<table>
<thead>
<tr>
<th>Author</th>
<th>Procedure</th>
<th>Catheter duration</th>
<th>Onset</th>
<th>Findings</th>
<th>Treatment</th>
<th>Comments and outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crawford 1975 [51] UK</td>
<td>Epidural for labor</td>
<td>Labor only</td>
<td>16 days</td>
<td>Bacteremic infection of hematoma β hemolytic <em>Streptococcus</em></td>
<td>Laminectomy</td>
<td>Developed subacute bacterial endocarditis</td>
</tr>
<tr>
<td>Ngan Kee et al [52] New Zealand</td>
<td>Epidural, early labor, twins, for CS. Difficult insertion</td>
<td>52 h</td>
<td>5 days</td>
<td>Neuro deficit 6 days. MRI <em>Staphylococcus aureus</em> from vagina and abscess</td>
<td>Cefuroxime; then cefotaxime + rifampicin; laminectomy 8 days</td>
<td>Recovered gradually</td>
</tr>
<tr>
<td>Borum et al [53] USA</td>
<td>Epidural for labor and tubal ligation</td>
<td>27 h</td>
<td>4 days</td>
<td>Local inflammation; mild deficit; MRI: abscess, <em>S aureus</em></td>
<td>Surgery and drainage 6 days</td>
<td>Recovered</td>
</tr>
<tr>
<td>Kindler et al 1996 [54] Switzerland</td>
<td>Epidural for labor, difficult insertion, severe preeclampsia</td>
<td>88 h</td>
<td>10 days</td>
<td>Local inflammation; CT; Mass; <em>S aureus</em></td>
<td>Immediate surgery flucloxacillin</td>
<td>Recovered</td>
</tr>
<tr>
<td>Jenkin et al [55] Australia</td>
<td>Epidural for CS; diabetic</td>
<td>?Operation only</td>
<td>6 days</td>
<td>Group B <em>Streptococcus</em>, present in vagina <em>S aureus</em> in blood. Mild neurological deficit.</td>
<td>Surgery; penicillin, gentamycin Ceftriaxone, gentamycin and metronidazole; then flucloxacillin and rifampicin</td>
<td>Minimal residual leg weakness</td>
</tr>
<tr>
<td>Dysart and Balakrishnan [56] New Zealand</td>
<td>CSE for CS; good asepsis; no antibiotic</td>
<td>48 h</td>
<td>9 days</td>
<td><em>S aureus</em></td>
<td>Gradually recovered with conservative treatment</td>
<td>Gradually recovered</td>
</tr>
<tr>
<td>Study (and authors)</td>
<td>Region</td>
<td>Intervention (time)</td>
<td>Infection Details</td>
<td>Treatment</td>
<td>Outcome</td>
<td></td>
</tr>
<tr>
<td>--------------------------------------------</td>
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<td>---------------------</td>
<td>----------------------------------------------------------------------------------</td>
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<td></td>
</tr>
<tr>
<td>Dhillon and Russell [57] UK</td>
<td>UK</td>
<td>Epidural for labor; good asepsis; blood in catheter 6.5 h</td>
<td>Local swelling, mild neuro deficit; pus extending from subcutaneous to epidural space; <em>Streptococcus pneumoniae</em></td>
<td>Surgery 9 days; flucloxacillin, penicillin</td>
<td>Recovered</td>
<td></td>
</tr>
<tr>
<td>Collier and Gatt [58] Australia</td>
<td>Australia</td>
<td>Epidural for labor; CS, postop analgesia 84 h</td>
<td>Lumbar puncture failed; MRI: abscess; no neurological deficit. No organism identified</td>
<td>Cefalothin, ceftriaxone, metronidazole</td>
<td>Slowly recovered</td>
<td></td>
</tr>
<tr>
<td>Rathmell et al [59] USA</td>
<td>USA</td>
<td>CSE for labor after car accident, vaginal delivery 72 h</td>
<td>Local inflammation, slight neurological deficit, MRI abscess; <em>S aureus</em></td>
<td>Laminectomy 10 days; cephalaxin</td>
<td>Recovered</td>
<td></td>
</tr>
<tr>
<td>Unseld and Eisinger [60] Germany</td>
<td>Germany</td>
<td>Epidural for labor, two insertions Brief</td>
<td>Neurological deficit. MRI: abscess; <em>Pseudomonas aeruginosa</em></td>
<td>Abscess drained after 14 days</td>
<td>Recovered despite delay</td>
<td></td>
</tr>
<tr>
<td>Rohrbach and Plotz [61] Germany</td>
<td>Germany</td>
<td>Epidural for labor Immunological impairment? Labor</td>
<td>MRI: abscess</td>
<td>Immediate surgical drainage; prolonged antibiotics</td>
<td>Recovered</td>
<td></td>
</tr>
<tr>
<td>Trautman et al [40] Germany</td>
<td>Germany</td>
<td>Epidural for twin labor and delivery; mask worn 24 h</td>
<td>Neurological deficit; LP grew <em>S aureus</em> (from anesthesiologist’s nose); MRI: abscess</td>
<td>Repeated surgical drainage 4 days; ceftriaxone, netilmicin, ofloxacin</td>
<td>Paraparesis unchanged</td>
<td></td>
</tr>
</tbody>
</table>

(continued on next page)
### Table 3 (continued)

<table>
<thead>
<tr>
<th>Author</th>
<th>Procedure</th>
<th>Catheter duration</th>
<th>Onset</th>
<th>Findings</th>
<th>Treatment</th>
<th>Comments and outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evans and Misra [62] UK</td>
<td>Epidural for twin labor, vaginal delivery; mask not worn</td>
<td>12 + h</td>
<td>1 week</td>
<td>Eventual neuro deficit. MRI: abscess. <em>S aureus</em></td>
<td>Laminectomy after 12 days. Flucloxacillin &gt; 6 weeks</td>
<td>Recovery slow and incomplete</td>
</tr>
<tr>
<td>Veiga Sanchez [63] Uruguay</td>
<td>Epidural CS</td>
<td>24 h</td>
<td>5 + days</td>
<td>Radiculitis. MRI: osteomyelitis and epidural abscess</td>
<td>Ceftriaxone</td>
<td>Gradually improved without drainage</td>
</tr>
<tr>
<td>Schroeder et al [64] Germany</td>
<td>Epidural for labor. Good asepsis</td>
<td>6 h</td>
<td>5 days</td>
<td>CT inconclusive; MRI abscess; <em>S aureus</em>. Mild sensory deficit</td>
<td>Laminectomy 6 days, ceftriaxone, rifampicin, metronidazole. Later clindamycin</td>
<td>Quick recovery</td>
</tr>
<tr>
<td>Chiang et al [65] Taiwan</td>
<td>CSE for CS, PCEA</td>
<td>72 h</td>
<td>3 days</td>
<td>Local inflammation + abscess on MRI. Presumed MRSA</td>
<td>Oxacillin, vancomycin, fusidic acid</td>
<td>Recovered without surgery</td>
</tr>
</tbody>
</table>

**Abbreviations:** CS, cesarean section; CSE, combined spinal epidural; LP, lumbar puncture; MRSA, methicillin-resistant *Staphylococcus aureus*; PCEA, patient-controlled epidural analgesia.

* see Table 2.
epidural abscess [40]. All followed epidural catheterization, in three cases as part of a CSE. None followed spinal anesthesia alone.

**Clinical features and management**

In the assembled cases the catheters remained in situ for a median of 24 hours (see Table 3) and the median time to presentation was 6 days, with the range of 2 to 16 days. Reviewing the world literature including non-obstetric cases, onset times have ranged from 1 to 60 days with the majority less than 5 days [66]. The presenting symptom is backache, usually extremely severe, with marked local tenderness, and sometimes radiating root pain. There may be evidence of inflammation with fluid leak at the insertion site. A blood count reveals raised C-reactive protein and white count [66]. Neurologic deficit may follow, in the form of leg weakness, paresthesiae, bladder disturbance, and other evidence of cauda equina syndrome. Fever and signs of inflammation serve to differentiate epidural abscess from hematoma. MRI allows early diagnosis, while blood culture may identify the organism before or without surgical interference. Diagnostic lumbar puncture is contraindicated.

Full neurologic recovery is dependent on age and also on early detection and treatment [62,66]. Once neurologic changes are present, surgical intervention is usually considered essential to recovery, although four of the women featured in Table 3 [56,58,64,65], with admittedly mild neurologic deficit, recovered fully with conservative treatment only. In three cases, recovery was incomplete despite surgical intervention [40,55,62]. Successful percutaneous needle drainage of an epidural abscess has been reported [67], although only laminectomy can ensure that all loculations are drained under direct vision. Antibiotic treatment needs to be continued for 2 to 4 weeks [68].

**Epidural-related infection**

Inflammation at the site of catheter insertion, along the track and adjacent to, but not apparently involving, the epidural space, is also described. Eight such reports involving various sites [24,69–75], some including many patients [73], are summarized in Table 4. Catheterization had usually been prolonged to allow postoperative patient-controlled analgesia. A recently published report described both a subdural abscess after a CSE and infection in the subcutaneous tissues of an apparently misplaced blood patch [74].

All such conditions are associated with back pain and signs of inflammation, and presumably pose a threat of spread to the epidural space. Moreover, paraspinal abscess may itself be associated with neurologic deficit [69,70].

**Causative organisms**

The great majority of epidural abscesses is caused by *Staphylococcus aureus* [66], with the occasional *Streptococcus* and *Pseudomonas* (see Table 3).
<table>
<thead>
<tr>
<th>Author</th>
<th>Procedure</th>
<th>Catheter duration</th>
<th>Onset</th>
<th>Findings and organism</th>
<th>Treatment</th>
<th>Comments and outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ready and Helfer a</td>
<td>Epidural for CS</td>
<td>48 h</td>
<td>5 days</td>
<td><em>Streptococcus faecalis</em>; diagnosed as meningitis but actually epidural “inflammation”</td>
<td>Gentamycin, ceftazidime, vancomycin, penicillin G</td>
<td>Recovered</td>
</tr>
<tr>
<td>Kinahan and Douglas. 1995</td>
<td>Epidural analgesia for labor, re-sited due to blood in catheter; vaginal delivery</td>
<td>10 h</td>
<td>4 days</td>
<td>MRI: Piriformis pyomyositis; vagina grew group B <em>Streptococcus</em> and <em>Enterococcus</em>. Neurologic deficit h/o pelvic arthropathy; neurologic deficit; paraspinal abscess grew <em>Mycobacterium tuberculosis</em></td>
<td>Ampicillin; “IV antibiotics”</td>
<td>Recovered</td>
</tr>
<tr>
<td>Raj and Foy 1998</td>
<td>Epidural analgesia for labor, instrumental vaginal delivery</td>
<td>Labor</td>
<td>19 days</td>
<td>Discitis; biopsy grew <em>Staphylococcus aureus</em> paraspinal abscess</td>
<td>Surgical drainage and decompression. Tuberculosis therapy</td>
<td>Probably coincidental. Recuperated</td>
</tr>
<tr>
<td>Hill et al 2001</td>
<td>Epidural analgesia for labor</td>
<td>24 h</td>
<td>2–4 days</td>
<td>Discitis; biopsy grew <em>Streptococcus bovis</em></td>
<td>Surgery 2 days</td>
<td>Recovered</td>
</tr>
<tr>
<td>Bajwa et al 2002</td>
<td>Spinal for CS</td>
<td>None</td>
<td>7 days</td>
<td>Discitis; biopsy grew <em>Streptococcus bovis</em></td>
<td>Vancomycin</td>
<td>Possibly unrelated</td>
</tr>
<tr>
<td>Authors</td>
<td>Procedures</td>
<td>Time to Infection</td>
<td>Site/Infection</td>
<td>Management</td>
<td>Outcome</td>
<td></td>
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<td>-----------------------------------------------------------------------------</td>
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<td></td>
</tr>
<tr>
<td>Cohen et al 2003</td>
<td>PCEA ropivacaine, epinephrine, fentanyl</td>
<td>n/k</td>
<td>2–3 days</td>
<td>12 cases of epidural site infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collis et al 2005</td>
<td>Epidural 3 attempts, for labor</td>
<td>4 h</td>
<td>2–6 days</td>
<td>Superficial discharge; MRI: subdural abscess, \textit{S aureus}</td>
<td>Laminectomy and decompression. Vancomycin, fusidate, cefotaxime</td>
<td>Recovered</td>
</tr>
<tr>
<td></td>
<td>Epidural 2 attempts, for labor</td>
<td>6.5 h</td>
<td>&lt;1 day</td>
<td>Head and backache and inflamed buttock; MRI: subcutaneous infected blood patch, \textit{Escherichia coli} in blood</td>
<td>Blood patch, apparently misplaced, Vancomycin, ciprofloxacin</td>
<td>Persistent back pain</td>
</tr>
<tr>
<td>Huang et al 2005</td>
<td>Spinal for CS. Separate epidural for PCEA. Mask but no hand wash, PI</td>
<td>58 h</td>
<td>Gradual onset; presented 20 days</td>
<td>Local inflammation; MRI: paraspinous abscess</td>
<td>Gentamycin, teicoplanin, oxacillin. Surgical drainage</td>
<td>Recovered</td>
</tr>
</tbody>
</table>

\textit{Abbreviations:} CS, cesarean section; h/o, history of; IV, intravenous; n/k, not known; PCEA, patient-controlled epidural analgesia; PI, povidone iodine.

\(^a\) see Table 2.
Epidural-related infections have been caused by a variety of organisms (see Table 4).

**Potential routes of infection**

**The catheter track**

The catheter track appears to be the most important route of entry. Inflammation may be noted at the catheter entry point before the onset of more serious symptoms, implicating the catheter track rather than its contents [54,59]. Organisms may come from the patient’s own skin. Bacteria, particularly *Staphylococcus epidermidis*, reside in large numbers in the deeper recesses of hair follicles, and are hard to eradicate by antiseptic skin preparation [76,77].

**The bloodstream**

Cases such as those involving a β-hemolytic *Streptococcus* [51,55] may have resulted from blood-borne infection alighting on a nidus in the form of a catheter or hematoma in the epidural space.

**Equipment**

Infection following a brief period of catheterization [57,60,64] suggests that epidural equipment may have contaminated the epidural space. The strain of *S aureus* may show the source to be the nose of the operator [40].

**The injectate**

The injectate is a potential route of infection if it does not contain a bacteriostatic local anesthetic such as racemic bupivacaine. Although rarely implicated as the source of infection, infusions of opioid alone or of ropivacaine or levobupivacaine, which have less antibacterial activity than bupivacaine [78–81], have been used for postoperative analgesia in many reported cases [24,52,54,58,59,64,73].

**Possible risk factors**

**Prolonged catheterization**

This is a self-evident risk for epidural site infection [3,18,52,66,82]. It is hardly surprising that catheters are progressively more likely to become contaminated over time.

**Immunocompromise**

Diabetes, steroid treatment and malignancy feature regularly in surveys of epidural infection and abscess [3,4,52,66,82,83], although less commonly among parturients. This topic is covered more fully by an excellent review [83].

**Traumatic catheter insertion**

Difficult insertion with multiple attempts, breaks in sterile technique, and subsequent need for manipulation, are frequently mentioned [3,52,54,57,60].
It is easy to see how multiple attempts can disturb both the aseptic technique and organisms deep in the skin.

**Blood in the epidural space**

A hematoma is a potential nidus for infection but seems to be implicated infrequently [51]. The risk of infecting the epidural space with a blood patch is considered by some so great as to necessitate prior blood culture. Concern about the danger of introducing infection with an autologous blood patch in the presence of maternal fever led Cesur and colleagues [84] to use allogeneic blood to treat a post–dural puncture headache in a pyrexial patient. Nevertheless, a Medline search for *epidural abscess AND epidural blood patch* yields zero at the time of writing, while the only recorded instance of an infected blood patch is one that appears to have been misplaced or to have leaked into the subcutaneous fat [74]. It has been mooted that the presence of HIV infection might contraindicate blood patch, but the virus is so prevalent throughout the body that this fear appears groundless [85,86].

**Infection elsewhere**

After dural puncture, the presence of bacteremia is a risk factor for meningitis [87] and, as would seem intuitive, adjacent infection is a risk for epidural abscess [51,52]. Among surgical patients, inflammation at the epidural entry point has been found to be increased in frequency when there is an infected wound elsewhere in the body [88,89], although Jakobsen and colleagues [88] conclude that a distant abscess or infected wound should not contraindicate epidural analgesia. Chorioamnionitis might be considered a risk for meningitis rather than abscess. Although a common threat, chorioamnionitis may be accompanied by bacteremia in only 2.5% of sufferers [88] and has not apparently been implicated in neuraxial infection [90–92].

**Lying in a wet contaminated bed**

Lying in a wet contaminated bed is generally considered a risk for parturients [44,54,93], and in a similar vein, hyperhidrosis has been cited in cases of epidural abscess [65,94].

**Unsuitable dressing on an indwelling epidural catheter entry point**

The prevalence of lying in a wet contaminated bed would suggest the advisability of an occlusive dressing. Doubt has been cast on the wisdom of such a policy, however [54,73], as it may be associated with an increase in sepsis [95]. A chlorhexidine-impregnated patch dressing may be more suitable [96]. The subject is further addressed in the recommendations that follow.
Absence of antibacterial local anesthetic agent

It has been postulated that the comparatively low infection rate associated with prolonged epidural compared with intravenous catheterization may in part be attributable to the use of antibacterial local anesthetics in the former [97]. However, opioids alone and single-enantiomer local anesthetics, which have little antibacterial action, are now used increasingly commonly (see Potential routes of infection). For prolonged epidural catheterization it would seem logical to add a local anesthetic with antibacterial activity to an opioid.

The etiology and risk factors for meningitis and epidural abscess are summarized and compared in Table 5.

Measures to prevent neuraxial infection

Measures to prevent neuraxial infection have now happily become the focus of increased attention, with several reviews in recent years that repay attention [77,83,96–98]. It is frequently mourned that measures we are asked to use are not evidence-based. Unlike infection related to surgical wounds or central venous catheterization, neuraxial infection is too rare for evidence about its prevention to be obtainable from randomized trials. Extrapolation from other fields (often misleading), indirect evidence, logic, and common sense may be the best available tools, while oft despised case reports are frequently quoted and indeed may be the only “evidence” about some risk factors.

Table 5
Summary of etiologic factors for meningitis and epidural abscess

<table>
<thead>
<tr>
<th>Entry</th>
<th>Meningitis</th>
<th>Epidural abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Via dural puncture, from contaminated equipment or the patient’s blood</td>
<td>• Along the catheter track or, less so, down its lumen</td>
</tr>
<tr>
<td>Usual causative organism</td>
<td>• Viridans type <em>Streptococcus</em></td>
<td>• <em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>Possible source of infection</td>
<td>• Operator’s mouth</td>
<td>• Patient’s skin and bed</td>
</tr>
<tr>
<td></td>
<td>• Talking without a mask</td>
<td>• Contaminated epidural equipment</td>
</tr>
<tr>
<td></td>
<td>• Blood-borne</td>
<td>•Injectate</td>
</tr>
<tr>
<td></td>
<td>• Vagina</td>
<td>• Rarely blood-borne</td>
</tr>
<tr>
<td>Risk factors</td>
<td>• Dural puncture + labor ± vaginal delivery</td>
<td>• Prolonged catheterization</td>
</tr>
<tr>
<td></td>
<td>• No face mask</td>
<td>• Immunocompromised: steroids, diabetes, AIDS</td>
</tr>
<tr>
<td></td>
<td>• Vaginal infection</td>
<td>• Multiple attempts at insertion</td>
</tr>
<tr>
<td></td>
<td>• Bacteremia</td>
<td>• Poor aseptic technique</td>
</tr>
<tr>
<td></td>
<td>• Poor aseptic technique</td>
<td>• Lying in a wet contaminated bed</td>
</tr>
<tr>
<td></td>
<td>• Immunocompromise?</td>
<td>• Absence of bactericidal local anesthetic</td>
</tr>
</tbody>
</table>
The following, in the order in which they must be practiced, are candidates for inclusion in the armamentarium against neuraxial infection:

*Wear hat and mask*

The value of masks (sometimes redundantly called face masks, whether for giving inhalational anesthesia or as worn by staff) is commonly misunderstood. Randomized trials have demonstrated that the omission of masks in the OR does not increase wound infection, but this has no relevance to the insertion of neuraxial blocks. Moreover, the authors of a reliable study [99] did not dare include prosthetic surgery, which should tell us something. Reviews of the need to wear masks may concentrate on protection for staff or surgical wound infection, but often overlook completely their place in anesthetic procedures [100]. That many anesthesiologists fail to wear masks and believe them to be useless [17,101,102] is not evidence of their ineffectiveness in preventing neuraxial infection. Among case reports of nosocomial meningitis, a mask is not mentioned, not worn (“as it is of doubtful value” [28] or because it “contributes little to prevent infection during spinal or epidural anesthesia” [31] or is not considered part of a “full aseptic technique” [29]) or mentioned in the wrong order (“Following surgical scrub, the anesthesiologist put on sterilized gloves, a surgical gown, and a mask.” [103]—such an order of events defies the imagination).

Commensals in the upper air passages *do not cause wound infection*, but as well as endangering prostheses, they repeatedly cause nosocomial meningitis, particularly when a mask is not worn by the anesthesiologist [4,18,44,77,104]. Such a complication is normally too rare to designate as an outcome of a randomized trial. This is where both indirect evidence and common sense come in. A mask can readily and efficiently diminish the dispersion of bacteria from the mouth and nose [105,106]. Not talking during insertion is not an option for the compassionate obstetric anesthesiologist.

Hubble and colleagues [107] found that, during dummy operations, bacterial counts in laminar flow theaters increased fourfold when hats were omitted, 15-fold when masks were omitted, and 22-fold when neither were worn. There is therefore indirect evidence to support wearing a hat as well as a mask.

In the face of all the evidence, it is clearly mandatory to wear a mask for spinal insertion, and because of the possibility of accidental dural puncture, not to mention Staphylococci in the nose, for epidural insertion also. Masks must be correctly applied, of good quality, and changed regularly, at least between patients [108]. The cost of a mask is a fraction of the cost of dural puncture, let alone that of a case of meningitis [44].

*Remove prostheses and baubles from the hands*

It belies belief that anyone would wear jewelry and false fingernails when preparing to conduct a sterile procedure, yet they apparently do [102]. Not surprisingly, such addenda make cleaning the hands less efficient [109].
**Wash hands in alcohol or carry out a surgical scrub**

This procedure is considered more important than wearing sterile gloves [77,98]. Studies have shown that an alcohol-based gel is more effective at eliminating microflora than an antimicrobial soap [109], that various alcoholic preparations are most effective at eliminating *Escherichia coli* but that chlorhexidine has a longer lasting effect [110] and that 4% chlorhexidine is more effective and longer lasting than 10% povidone iodine [111].

**Don sterile gloves without touching the outside with bare hands**

A sterile gown, although part of the surgical ritual of “full aseptic precautions,” is rarely worn for spinal insertion, but usually worn for epidural siting in the United Kingdom to avoid contaminating a catheter that is destined to remain some time in the patient. This is an expensive addition to the ritual, and impossible to evaluate, but can only be safer than not doing so. There is little dissent about sterile gloves [102], although they are no substitute for hand washing [77,96].

**Clean the patient’s back widely, twice, with chlorhexidine in alcohol**

Many studies have addressed how best to clean the patient’s skin and keep it clean; they are comprehensively reviewed elsewhere [96]. Guidelines developed for skin preparation for surgery and for central venous and arterial catheterization [112] have been adapted, in part, as recommendations for neuraxial anesthesia [96].

As with hand disinfection, chlorhexidine outperforms povidone iodine consistently. Sakuragi and colleagues [113] showed that 0.5% chlorhexidine in ethanol, unlike 10% povidone iodine, could completely inhibit growth of methicillin-resistant *Staphylococcus aureus* in vitro. A study from France comparing 0.25% chlorhexidine (with benzalkonium and 4% benzyl alcohol) and 10% povidone iodine [114] and another from the United States comparing 2% aqueous chlorhexidine and 10% povidone iodine [115] showed the superiority of chlorhexidine in preventing central venous and arterial catheter-related sepsis and bacteremia.

In the field of epidural insertion results are similar. Thus, Kinirons and colleagues [116] showed that 0.5% chlorhexidine in alcohol was more efficient and longer lasting than 10% povidone iodine in inhibiting catheter colonization in prolonged use, although Kasuda and colleagues [117] found no difference between the agents for short-term catheterization. Organisms residing deep in the hair follicles are a potential source of epidural infection [76]. Sato and colleagues [76] found that, with its greater penetrative power, 0.5% chlorhexidine in alcohol was significantly better than 10% povidone iodine at inhibiting growth of organisms in human skin sampled at lumbar laminectomy. Yentur and colleagues [118] confirmed that skin preparation with 10% povidone iodine cannot prevent contamination of epidural
needles and catheters during insertion. Moreover, open bottles of 10% povidone iodine do not maintain their sterility [119]. Of direct clinical relevance is the observation by Cameron and colleagues [120] that switching between povidone iodine and chlorhexidine for skin preparation was associated with remarkable changes in epidural insertion–site infection (Fig. 1), while site infection was associated with epidural abscess. Fig. 1 suggests that even alcoholic iodine is less efficient than alcoholic chlorhexidine.

Alcohol is rapid and effective and improves the efficacy of both chlorhexidine and iodine, whether as Duraprep [93] or the old-fashioned iodine tincture [121], but chlorhexidine has consistently superior residual activity. Whether given as 0.5% alcoholic solution or by aqueous spray, reduced skin colonization is still observed hours later at epidural catheter removal [122].

Antiseptic solutions reduce bacterial counts exponentially, thus if the count is high before skin preparation, it will still be relatively high after a single application; two applications are therefore not only more effective, they may also be strongly indicated [123].

It is therefore important to spray or paint at least twice, allowing the solution to dry between applications, a routine that is widely practiced [76,108,110,122,124,125]. Alcoholic preparations dry rapidly, so if the first coat is applied by the anesthesiologist before preparing the sterile equipment, dual application need not delay proceedings for a mother in pain. Moreover, if a spray is used it may be applied by an assistant with further time saving.

Neither povidone iodine nor chlorhexidine is licensed for skin preparation before neuraxial anesthesia [96]. Yet because chlorhexidine injected directly into the anterior chamber of a rat’s eye has been associated with degeneration of adrenergic nerves [126], it is supposed that minute amounts of skin prep passing into a patient’s subarachnoid space could pose a material threat. Thus, despite its numerous glaring disadvantages (Table 6), iodine is still preferred to chlorhexidine in some quarters. The best expert

![Fig. 1. Annual incidence of epidural insertion site infection, showing skin preparation solution used before epidural insertion. (Reproduced from Cameron CM, Scott DA, McDonald WM, et al. A review of neuraxial epidural morbidity. Anesthesiology 2007;106:997–1002; with permission.)](image-url)
opinion recommends otherwise [96], while it would seem wise to keep any skin prep solution off the cart on which the sterile equipment is prepared. Avoid contaminating equipment that will enter the patient

Common sense suggests it is wise to apply sterile drapes securely to the area and, if possible, to use a no-touch technique for any elements that will enter the subarachnoid space.

Apply a suitable dressing to the catheter entry point

Evidence-based guidelines relating to dressing central and peripheral venous catheters [112] are probably, in the main, applicable to prolonged epidural catheterization. A meta-analysis suggests that application of a transparent polyurethane film as an intravenous catheter dressing is associated with an increase in local sepsis and bacteremia [95], although the guidelines [112] appear to misquote these findings. Moreover, no amount of occlusion can exclude the bacteria already resident in the patient’s skin [76]. More suitable would appear to be a chlorhexidine-impregnated patch dressing, which not only reduces skin colonization, it also absorbs blood and exudate, which would otherwise provide a favorable culture medium [96].

In a high-risk situation, avoid prolonged catheterization and prescribe antibiotics

The risk of epidural abscess is increased by prolonged catheterization, by immunocompromise, and by multiple attempts at insertion. It would seem

<table>
<thead>
<tr>
<th>Speed of onset</th>
<th>Chlorhexidine</th>
<th>Povidone iodine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accelerated by alcohol</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Effect extended</td>
<td>“several hours”</td>
<td>“Limited…requires reapplication every 24 h”</td>
</tr>
<tr>
<td>Skin reaction</td>
<td>Rare</td>
<td>Yes, erythema, urticaria, vesicular lesions</td>
</tr>
<tr>
<td>Effective in presence of blood or pus</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Bacterial resistance</td>
<td>Rare</td>
<td>Yes, particularly Staphylococcus aureus</td>
</tr>
<tr>
<td>Approved for skin prep before neuraxial block</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>CNS adverse events described</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Stays sterile in container</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

^a Studies consistently demonstrate a more prolonged effect with chlorhexidine. The explanation of these apparently contradictory statements has been sought but is unknown.

logical, therefore, to avoid prolonged epidural catheterization when other risk factors are present. If it is felt that the benefits of epidural analgesia outweigh the risks, then there is circumstantial evidence that suitable antibiotics reduce the chances of infection, although early administration may be crucial [3,77,97]. Inclusion of racemic bupivacaine would be an added safeguard (see Possible risk factors).

A more cautious approach suggests: “No amount of preparation … can maintain sterility throughout the time that an epidural catheter may remain in situ. No skin dressing, occlusive or otherwise, can prevent entry via a catheter site of bacteria resident in the patient’s skin. If there is the slightest contraindication to prolonged catheterization, the moral is simple: take it out”[50].

In relation to the risk of nosocomial meningitis, an approach of extreme caution would be, where possible, to avoid dural puncture during labor without good reason, particularly in the presence of genital tract infection or other risks. Antibiotics may also play a role in preventing meningitis after dural puncture.

Recommendations for the prevention of neuraxial infection are summarized in Box 2.

References


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