That subdural effusions commonly complicate purulent meningitis, particularly in infants, is now generally appreciated, but some confusion and concern remains as to their prognostic significance and management. The present study is an attempt to evaluate final prognosis in relation to these subdural collections in a consecutive and unselected group of patients under the age of 3 years, treated by reasonably standard regimens, and followed for intervals of 6 months or more thereafter. The concern we now all share over the eventual fate of such patients is clearly related to improved survival rates; more babies now survive to have more complications.

PATHOGENESIS

The mechanisms by which subdural collections develop are not here our immediate concern, but some of the various speculations will be briefly enumerated only to emphasize that by several of them—alone or in combination—additional damage other than that producing the subdural collection might be expected in other parts of the central nervous system. In other words, the same factors which might be responsible for producing subdural effusions could logically be invoked to explain a number of other complications and neurologic sequelae which develop during or after recovery from meningitis; by the same reasoning, the presence of subdural collections might simply reflect accompanying neurologic damage.

Briefly, these mechanisms include: embolic phenomena; septic thromboses and rupture of bridging veins; direct extension or simple transudation through the arachnoid or its villi ("leaks"); thromboses of dural sinuses or their tributaries with retrograde extension to the subdural space by way of the pachionian granulations; simultaneous blood-borne infections; sudden changes in intracranial tension related either to the meningitis alone or to early diagnostic procedures; previous or concurrent subdural bleeding incident to convulsions or other trauma; alterations in capillary permeability or in clotting mechanisms related to associated diseases or deficiencies; the effects of asphyxia; and perhaps some collections resulting from local bleed-
ing incident to initially negative subdural taps.

Regardless of which cause or combination of causes obtains in the individual case, subdural collections are most commonly encountered in the youngest patients during the phase of rapid head growth, presumably while cranial structures are most expandable. Also, they are apt to occur in those patients who are recognized and treated for meningitis at relatively late stages, in those who exhibit the most conspicuous clinical symptoms and signs of early high intracranial pressure, or in those whose early response to conventional therapy is unfavorable or atypical.

CLINICAL MATERIAL FOR ANALYSIS

Patients

The present analysis is concerned with 343 patients, newly born to 36 months of age, being consecutive admissions for acute purulent meningitis over a 4-year period ending in 1955. Fifty-one (14.9%) of the patients died, and 14 of these had major underlying neurologic defects for which the meningeal infection might be legitimately considered only as a terminal event. These 14 patients had such preceding conditions as congenital hydrocephalus, hydrancephaly, meningomyelocele, encephalocele, porencephaly, communicating dermal sinus, astroblastoma, ependymoma, infantile coarctation and severe compounded skull fractures. In each of the 14, death seemed more logically related to the preceding condition than to the meningeal infection. Thus we might claim a "corrected" total fatality rate of 10.8% for all 343 patients.

Among the 292 survivors, there were 55 who had early subdural effusions, 32 who had mild neurologic sequelae, and 25 who were left with severe handicaps. In Tables I and II, the total group is distributed by age, etiology and case-fatality rates.

Treatment

During the period of this study, patients were treated by a reasonably uniform plan. Immediately after prompt collection of all pertinent diagnostic specimens, and without delay for establishing a definitive etiologic agent, the patients were "flooded" with a priming combination of agents designed to combat effectively the commonest causes for meningitis in this age and at that time. Initial doses were given parenterally and quickly, with proper attention to renal function and state of hydration. The priming combination ("blitz") consisted of sulfadiazine 100 mg/kg, chloramphenicol 100 mg/kg and penicillin 600,000 units.

Continuing therapy usually included these three same agents for 8 days but with permissible variations in dosage and duration and substitutions, according to the organism recovered, its in-vitro sensitivity tests and the clinical response. The continuing dosages were: for sulfadiazine, from 100 to 200 mg/kg daily divided into three or four parts and given at 6- to 8-hour intervals; for chloramphenicol, a total dosage of 50 to 100 mg/kg/day divided into three or four equal parts and given at these same intervals; and for penicillin, the original dosage of 600,000 units was given every 2 to 6 hours.

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>H.I.*</th>
<th>Pn.*</th>
<th>Men.*</th>
<th>&quot;Others&quot;†</th>
<th>Unknown**</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 6</td>
<td>4/40</td>
<td>2/13</td>
<td>1/2</td>
<td>13/26</td>
<td>8/30</td>
<td>28/131</td>
</tr>
<tr>
<td>6-12</td>
<td>3/34</td>
<td>1/8</td>
<td>1/2</td>
<td>2/5</td>
<td>2/33</td>
<td>9/82</td>
</tr>
<tr>
<td>Over 12</td>
<td>3/44</td>
<td>0/10</td>
<td>2/12</td>
<td>3/7</td>
<td>6/32</td>
<td>14/130</td>
</tr>
<tr>
<td>Totals</td>
<td>10/123</td>
<td>3/31</td>
<td>4/16</td>
<td>18/38</td>
<td>16/135</td>
<td>51/348</td>
</tr>
</tbody>
</table>

* Hemophilus influenzae, Pneumococcus, Meningococcus.
† See text for components.
** Cultures negative; previously treated.
Parenthetically, it speaks well for the diligence and care furnished by the many house officers directly concerned with treatment of these patients that toxicity referable to the initial dosage of sulfadiazine was encountered in only 3 of the 343 patients treated. In all 3, eventual outcome seemed entirely satisfactory following diuresis and reduction of drug dosage. It seemed to us that the calculated risk of occasional undesirable side-effects of such therapy was more than favorably offset by the urgent need to obtain high levels in the tissues for each of the therapeutic agents, promptly and effectively.

RESULTS OF ANALYSIS

Subdural Collections

Although subdural collections were expected (particularly in young infants who were admitted to the hospital late) for those with conspicuous early symptoms of intracranial hypertension or those whose response to therapy was atypical or unfavorable, more specific features which led to diagnostic subdural punctures included: convulsions, focal neurologic signs, persistent fever, bulging fontanelle, rapidly increasing head size, projectile vomiting and opisthotonus. When any of these features were present, alone or in varying combinations, punctures were promptly done at the lateral angles of the fontanelles (or in older infants slightly beyond) through the coronal sutures.

Among the survivors, early collections were demonstrated in 55 patients, and suspected by the same criteria but not found in 33. For lack of any suspicious symptoms or signs the subdural spaces in the remaining 204 patients were not tapped. When a collection was suspected, bilateral taps were routinely made, and in 24 of the 55 cases with subdural collections these were present on both sides. The distribution of these collections according to age of the patients and cause of meningitis is displayed in Tables III and IV.

Interestingly, both fatality rates and frequency of subdural collections were greatest among patients who had meningitis due to "mixed and other" causes. These included mostly the very young infants with meningitis due to a variety of organisms—coliform, staphylococci, streptococci, listeria, mimeae, pseudomonas, and salmonellas—with dual etiology in several cases and difficulties in attaining bacteriologic control for many.

Most patients for whom etiologic diag-

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>H.I.</th>
<th>Pn.</th>
<th>Men.</th>
<th>&quot;Other&quot;</th>
<th>Unknown</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 6</td>
<td>15/86</td>
<td>5/11</td>
<td>0/1</td>
<td>7/13</td>
<td>11/14</td>
<td>38/103</td>
</tr>
<tr>
<td>6-12</td>
<td>7/31</td>
<td>1/7</td>
<td>0/1</td>
<td>1/3</td>
<td>3/31</td>
<td>10/73</td>
</tr>
<tr>
<td>Over 12</td>
<td>8/46</td>
<td>1/10</td>
<td>1/10</td>
<td>0/4</td>
<td>1/46</td>
<td>5/116</td>
</tr>
<tr>
<td>Totals</td>
<td>24/113</td>
<td>7/28</td>
<td>1/12</td>
<td>8/30</td>
<td>15/119</td>
<td>55/292</td>
</tr>
</tbody>
</table>
TABLE IV

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>HI.</th>
<th>Pn.</th>
<th>Men.</th>
<th>“Others”</th>
<th>Unknown</th>
<th>Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 6</td>
<td>41.7</td>
<td>45.4</td>
<td>—</td>
<td>53.8</td>
<td>26.2</td>
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<td>6-12</td>
<td>22.6</td>
<td>14.3</td>
<td>—</td>
<td>33.3</td>
<td>9.7</td>
<td>16.4</td>
</tr>
<tr>
<td>Over 12</td>
<td>4.3</td>
<td>10.0</td>
<td>10.0</td>
<td>0.0</td>
<td>2.2</td>
<td>4.3</td>
</tr>
<tr>
<td>Totals</td>
<td>21.2</td>
<td>25.0</td>
<td>8.3</td>
<td>40.0</td>
<td>12.6</td>
<td>18.8</td>
</tr>
</tbody>
</table>

nosis remained “unknown” or “questionable” had been treated by various persons and means before they were admitted to our hospital, and for only a few had previous therapy been guided by any preliminary examination of cerebrospinal fluid. In a large number, penicillin or other antibacterial agents had been given without suspicion of meningitis or before development of signs or symptoms of meningeal irritation. It is gratifying to note, perhaps as a reflection of the very efficacy of even indiscriminate therapy, that both fatality rate and the incidence of subdural collections in this group compared so well with those of patients with meningitis of specified etiology.

The character, amount, timing, laterality and clinical features accompanying the subdural collections were critically examined to establish the validity of criteria for expecting them or for relating their characteristics to the likelihood of additional neurologic sequelae. Though the general criteria for recognition of these collections seemed to be borne out, there were enough exceptions so that we could not establish statistically the validity of any one circumstance—or of several combinations. On the average, those with proved subdural collections received therapy about 3 days later than did those who had no evidence to suggest such collections. The initial pressures of the cerebrospinal fluid, measured at lumbar level, were not significantly different among those who did or did not develop collections; the means and medians for both groups were about 300 mm. One might argue, but would have difficulty in proving, that those without collections were apt to be more robust, to struggle more, and thus to destroy the validity of such comparisons.

There were no apparent differences in the frequency of subdural collections or other complications related to race or sex of patients, etiology of the meningitis, frequency of positive blood cultures, and characteristics encountered in the routine examinations of the original cerebrospinal fluid secured at lumbar taps. In only 8 of the 55 patients was an initial diagnostic subdural tap negative and a later tap positive; in other words, significant collections (1 ml or more, obtained promptly) were encountered at the first tap in 47 of the 55 patients who had these demonstrated at any time.

The typical subdural fluid was xanthochromic and cloudy, often slightly bloodtinged; in only three instances was frank pus encountered, and only five collections were definitely or grossly bloody. From 10 of the 49 collections cultured, the same organism was recovered as had been secured from the initial specimen of cerebrospinal fluid. The pressures within the collections were not routinely measured, but seemed usually increased. At the time these patients were being treated, many initial subdural collections were completely drained, not merely sampled; and the amounts varied from as little as 1 ml to as much as 54 ml from either side. A 5-month-old patient had the largest collection in our experience and it cleared completely; the infant has had no recognized neurologic defects.

**Sequelae**

Sequelae which may follow purulent meningitis (whether or not there is an effusion) are numerous, reasonably well-
TABLE V

SEVERITY OF SEQUELAE AMONG 292 SURVIVORS (AS JUDGED 6 MONTHS OR MORE AFTER ACUTE MENINGITIS)

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Status</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>0*</td>
<td>Considered or known to be intact. 235</td>
<td></td>
</tr>
<tr>
<td>+ †</td>
<td>Behavior problems, emotional instability, poor gains in weight and height. 21</td>
<td></td>
</tr>
<tr>
<td>++</td>
<td>Localized defects, but functional. 11</td>
<td></td>
</tr>
<tr>
<td>+++</td>
<td>Retarded development and convulsions. 19</td>
<td></td>
</tr>
<tr>
<td>++++</td>
<td>Invalids; &quot;decerebrate,&quot; hydrocephalus, opisthotonus, etc. 13</td>
<td></td>
</tr>
</tbody>
</table>

* "Negative."
† "Positive."

known, and too variable for detailed description here. For our present purposes, we have considered that those we encountered could be divided into four groups, as indicated in Table V. Neither the parents nor the physicians who have examined the 235 individuals that are indicated as "intact" have had concern as to any handicap whatsoever, though data are incomplete for some of these patients. Twenty-one of the patients are said to have, or are known to have had, behavior disturbances, emotional instability, poor weight or height gains, and questionable delays in developmental schedules, which might be attributed either to the meningitis or to other preceding and environmental causes. In other words, these mildest residua in 21 patients are only questionably attributable to the preceding meningitis—"post hoc, ergo propter hoc." A second group of 11 patients have localized defects of relatively minor nature, but are not seriously incapacitated. These included palsies of the extra-ocular muscles, hearing defects, mild monoplegia, hemiparesis, etc. For our present purposes we have designated these first two groups, including 33 patients in all, as "mild." The third and fourth groups, comprising 25 patients, have unmistakably severe subsequent neurologic handicaps; in group 3, there are 12 patients who are definitely retarded and have serious convulsive disorders, while in group 4 there are 13 patients who present the pitiful picture of chronic basilar meningitis with fixed opisthotonus and hydrocephalus. In Table VI, sequelae are arranged according to age and by etiology of the purulent meningitis; nearly a fifth of all survivors had residua of concern, and slightly less than half of these were severe and disabling.

Though there seems no doubt that sequelae are significantly more common among those who had early subdural collections, specific significance as to frequency or severity of sequelae could not be attached to the time at which these occurred, their amount, their character or their duration. Both acute collections and later sequelae were commonest in the youngest patients and in those who were treated late—but in the older patients, where the collections were less frequent, they were ominous by their more consistent association with later handicaps. Among 10 patients with subdural collections from which cultures were positive for the same organism that caused the meningitis, only 3 escaped with no residua; 3 had severe sequelae, and 4 had mild sequelae. Distribution of sequelae was al-

TABLE VI

TOTAL SEQUELAE AMONG ALL SURVIVORS, BY AGE AND CAUSE

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>H.1.</th>
<th>Pn.</th>
<th>Men.</th>
<th>&quot;Others&quot;</th>
<th>Unknown</th>
<th>Total</th>
<th>Per Cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 6</td>
<td>7/36</td>
<td>3/11</td>
<td>0/1</td>
<td>5/13</td>
<td>9/42</td>
<td>24/103</td>
<td>23.3</td>
</tr>
<tr>
<td>6-12</td>
<td>3/31</td>
<td>2/7</td>
<td>0/1</td>
<td>2/3</td>
<td>5/31</td>
<td>14/73</td>
<td>16.4</td>
</tr>
<tr>
<td>Over 12</td>
<td>9/46</td>
<td>4/10</td>
<td>1/10</td>
<td>1/4</td>
<td>6/46</td>
<td>21/116</td>
<td>18.1</td>
</tr>
<tr>
<td>Totals</td>
<td>19/113</td>
<td>9/28</td>
<td>1/12</td>
<td>8/40</td>
<td>20/119</td>
<td>57/292</td>
<td>19.2</td>
</tr>
</tbody>
</table>
most identical when related to the time or number of taps before the collections cleared.

**Pertinent Relationships**

In Figures 1-4, results are portrayed by age for each of the four major etiologic groups; the number of infections due to meningococci was too small for such analysis. In the etiologic groups, we have considered only those patients for whom the eventual fate as to sequelae has been established, and for whom subdural collections were clearly demonstrated (“positive”) or not suspected (“negative”). Thus, from the point of view of prognosis, the figures shown could be considered as at least conservative estimates as to frequency or severity of sequelae. Suspected, but unproved collections, and any uncertain follow-ups are excluded from the numerical comparisons.

Among those 135 patients who had meningitis due to unknown causes (Fig. 1), most having been treated before admission, 88% survived. In this figure both deaths and residua among survivors are distributed by age. Half of 12 babies with proven early subdural collections were left with severe neurologic handicaps, while among 66 patients for whom no collection was suspected there were only 2 such, and in one of these the handicap might well have been suspected before the meningitis occurred. In all, there are 20 residua among 119 survivors.

Among the 123 patients with meningitis due to Hemophilus influenzae, 93% survived and about 16% of the survivors are known to have residua of some degree (Fig. 2). In this group serious disabilities were also clearly more common among those who had proven early subdural collections (3 of 19) than they were among the patients for whom such were not suspected (2 of 54).

As expected, poorest results as to survival were obtained with the group of 38 patients who had the mixed or “other” infections previously discussed (Fig. 3). Here we were dealing with the youngest infants,

![Graph](image)

**Fig. 1.** Number of deaths, sequelae and recoveries in each 6-month period, by age at which purulent meningitis due to “unknown” organisms occurred.
almost half having serious underlying congenital defects involving the central nervous system. Even in this small group, though numerically far from impressive, the frequency of serious sequelae was slightly greater among the survivors who had early proven subdural collections (two of five compared to two of seven).

Survival rates among the 31 with pneumococcal meningitis were better than we would have expected, though nearly a third of the survivors are considered to have residual handicaps of some degree. Here too (Fig. 4) severe sequelae appeared to be clearly related to the presence of early subdural collections, being obvious in three of seven with effusions, and in none of 20 without such.
In Tables VII and VIII* known sequelae are related to subdural collections which were demonstrated ("positive"), to those which were suspected but could not be proved ("negative"), and to a third group in which there were no evidences of subdural collections for which diagnostic punctures were indicated ("negative"). Among these three groups the differences in incidence of sequelae are large; there is sufficient evidence that the frequency of both the total and severe residual effects differ significantly among the three groups (p < .0005). Obviously, the observed gradations suggest that the features indicative of subdural effusions simply reflect underlying brain damage or are causally related.

Finally, distribution of effusions and sequelae were considered among the survivors of three age-groups (Table IX). Among those under 6 months of age at the time meningitis occurred, 37% had "positive" subdural taps, and 63% had no collection suspected or demonstrated. In this group of 103 babies, 23% had sequelae, 14% falling among those with subdural collections and 9% in those without. In this age group, then,

* (P<.0005.)

* Data were adequate for final satisfactory evaluation in only 223 of 292 survivors; 69 were reported or considered to be normal, but are not included in these tables.
about a third of those with subdural collections, and roughly a sixth of those without such, had later sequelae of varying degrees. Among 73 infants who survived purulent meningitis during the second 6 months of life, 12 (about 16%) had definite subdural collections. Here again, about a third of those with early subdural collections, and about a seventh of those without such, had later handicaps attributable to their meningitis.

Among 116 survivors between 12 and 36 months of age, only about 4% had “positive” subdural collections and all of these were left with sequelae; while only one-seventh of those for whom no collection was suspected or proved had similar damage.

**DISCUSSION**

These studies leave little doubt that subdural collections in acute and purulent meningitis have ominous prognostic significance. It is difficult to believe that associated sequelae are due to the diagnostic maneuvers necessary to demonstrate these collections or to their commonly accepted treatment by repeated taps according to pressure symptoms until “dry.”

We have the impression that the best explanation for the observed correlation is that the subdural collections simply reflect the occurrence of variously localized or diffuse neurologic and vascular involvement in acute bacterial meningitides.

Whether or not the over-all prognosis could be improved with more aggressive surgical attack on these collections at an earlier date remains a controversial question. So far, it has been our practice to interfere surgically for removal of subdural membranes only when collections persist despite repeated taps or when they have at the initial tap or later the characteristics typical of subdural hematoma. In only one of the present cases under discussion was there evidence to suggest that a traumatic subdural hematoma had antedated the occurrence of meningitis. In only two others was the initial subdural fluid grossly bloody; in these three the residual effects were severe despite concerted medical and neurosurgical measures.

Because most of these collections are recognized after reasonably adequate control of the infection, and because we believe that most of them are due to inflammatory vascular changes in the very expandable immature meninges, adrenal steroids may be justified or even indicated in their treatment for an effect in ameliorating the inflammation that is perhaps responsible both for the subdural collection and some of the associated neurologic damage. Judged from results observed in the present series, effects of such a trial would have to be remarkably consistent or comparable in a considerable number of patients before any final conclusion could be reached or a firm recommendation made.

Certainly, with the improved survival rates now obtaining for most types of purulent meningitis, further studies are urgently indicated to elucidate the specific mechanisms for production of these collections, how best to treat them, and how to prevent more of the many handicaps among survivors.

**CONCLUSIONS**

In a group of 343 consecutive patients under 36 months of age with acute purulent meningitis, survival rates were encouraging but neurologic sequelae were disturbingly frequent. Fifty-five infants had subdural...

---

**TABLE IX**

PERCENT ACUTE SUBDURAL EFFUSIONS AND LATER RESIDUA, BY AGE

<table>
<thead>
<tr>
<th>Age (mo)</th>
<th>Under 6</th>
<th>7-12</th>
<th>Over 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>With effusions Residua, %</td>
<td>36.9</td>
<td>16.4</td>
<td>4.3</td>
</tr>
<tr>
<td>Without effusions* Residua, %</td>
<td>63.1</td>
<td>88.6</td>
<td>95.7</td>
</tr>
</tbody>
</table>

* (Not found, or tap not indicated.)

---

970 SUBDURAL EFFUSIONS

---

DISCUSSION

These studies leave little doubt that subdural collections in acute and purulent meningitis have ominous prognostic significance. It is difficult to believe that associated sequelae are due to the diagnostic maneuvers necessary to demonstrate these collections or to their commonly accepted treatment by repeated taps according to pressure symptoms until “dry.”

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Because most of these collections are recognized after reasonably adequate control of the infection, and because we believe that most of them are due to inflammatory vascular changes in the very expandable immature meninges, adrenal steroids may be justified or even indicated in their treatment for an effect in ameliorating the inflammation that is perhaps responsible both for the subdural collection and some of the associated neurologic damage. Judged from results observed in the present series, effects of such a trial would have to be remarkably consistent or comparable in a considerable number of patients before any final conclusion could be reached or a firm recommendation made.

Certainly, with the improved survival rates now obtaining for most types of purulent meningitis, further studies are urgently indicated to elucidate the specific mechanisms for production of these collections, how best to treat them, and how to prevent more of the many handicaps among survivors.

**CONCLUSIONS**

In a group of 343 consecutive patients under 36 months of age with acute purulent meningitis, survival rates were encouraging but neurologic sequelae were disturbingly frequent. Fifty-five infants had subdural...
collections during the acute phase of their disease. An attempt was made to evaluate the prognostic significance of these.

It appears clear that the frequency and severity of neurologic sequelae are related to the presence of early subdural collections, but these could not be specifically correlated as to volume, character, or time at which they occurred. Subdural collections are most frequent in the youngest patients, for whom early recognition of meningitis may be difficult or delayed.

It is assumed, but not proved, that factors similar to those responsible for production of the subdural collections also caused many of the evidences of brain damage that appeared concomitantly or developed later.

Acknowledgment

We are indebted to Dr. Lila Elvebach and Miss Ethel Eaton for advice and help with the statistics involved in this study, to Drs. W. A. Williams, Jr. and Crawford W. Long for help with analyses of hospital records, to Mrs. Scott Wilson for securing many of the follow-up observations, and to Miss Vera Morel for preparation of the figures.

REFERENCES

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ACUTE SUBDURAL EFFUSIONS AND LATE SEQUELAE OF MENINGITIS
Ralph V. Platou, Andrew Rinker and Joseph Derrick

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