tive, the test could be repeated in 6 to 8 weeks and if still no reaction occurs, the vaccination can be repeated. It has often been recommended that persons planning on working in an infectious environment be rendered positive prior to commencing such work. This is considered important for the protection of the present reputation of B.C.G. as a valuable weapon in the fight against tuberculosis.

**Discussion**

Many factors are important in considering the use of B.C.G. vaccine. Every effort should be made to avoid indiscriminate use of the vaccine, since the method of vaccination is inexpensive and would appeal to the public generally. Little would be gained, and much of the present public health prevention and control measures might be lost. The preparation of the vaccine should be limited to the well established laboratories already supplying it, for use in man. A seminar on B.C.G. in June, 1947 (U.S.A.) presented many interesting facts. The following are statements of the moderator, H. C. Sweany, at the beginning and conclusion of the meeting.

"Certain facts have been learned: It has been proved beyond doubt that B.C.G. is harmless, that it is feasible to administer the vaccine to human beings, and that it offers some degree of protection against a later infection with virulent tubercle bacilli. The unfavourable features are the dangers of contamination or mixing cultures with virulent strains, the difficulty of applying it to great masses of people, the false security that may be engendered by its use, and that it does not afford the complete protection that smallpox vaccination does. It seems clear that, if rigidly supervised, B.C.G. has a place in anti-tuberculosis work."

**Summary and Conclusions**

1. B.C.G. vaccine is innocuous and is of definite value in the prevention of tuberculosis.

2. It is recommended for use among those negative reactors destined to live or work in a tuberculous or potentially tuberculous environment. Infants and children whose parents or other members of the family are stricken with the disease; our young women planning or already training as nurses; and any group working in a sanatorium environment, are among those recommended for vaccination.

**References**


**Résumé**

Description du B.C.G. et des réactions qui suivent son emploi, chez l'animal et chez l'homme. Discussion des résultats obtenus et exposé des diverses méthodes de vaccination. La méthode intradermique paraît la plus simple mais il semble que la méthode par scarification sera la plus employée. Le B.C.G. est inoffensif et les résultats obtenus à la suite de son emploi sont indéniables. On l'emploiera chez les individus Mantoux-négatifs qui doivent séjourner où il y a des tuberculeux, notamment chez les infirmières; chez ceux qui vivent dans les sanatoria et auprès de tuberculeux. Jean Saucier

**Infectious Polynéuritis of Unknown Etiology (Guillain-Barré Syndrome) in Childhood**

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The polynéuritides have for several decades been described under many headings. In 1916, Guillain, Barré and Strohl 1 separated from the polyradicular neuritides, a group of patients who had, besides the symptoms common to polynéuritis, an increase in the cerebrospinal fluid proteins but without cellular reaction. In subsequent years more of these cases were recognized and many case reports and excellent monographs on this condition appeared in the literature, stressing the more important findings and adding new ones. 2, 3

For more than 20 years all the cases reported were in adults; indeed it was considered a disease of adult life until Ford 4 and Hecht 5 in 1937 reported cases in children. Hecht described 7 cases of "acute infective polynéuritis" in children between the ages of 2 and 10 years, which illustrate the clinical picture. In 1941, Casamajor and Alpert 6 reviewed the English and French literature and found 38 cases reported in children under 12 years of age. They described three additional cases bringing the total to 41. Since then several reports have appeared

* Resident in Medicine.

† Such as infective polynéuritis, acute febrile polynéuritis, Landry's ascending paralysis, polyradiculo-neuritis, polineuritis, acute infectious neuritis, Guillain-Barré syndrome, Guillain-Barré-Strohl syndrome, radiculo-neuritis and polynéuritis of unknown etiology.
commenting on the relative frequency and importance of this condition in childhood.\textsuperscript{5, 8}

The etiology of this disease still remains obscure. It is the modern consensus that the disease is caused by a neurotropic virus closely related to that of poliomyelitis. Some authors have even considered this condition an abortive form of poliomyelitis but no proof in support of this view has been advanced. Attempts to reproduce the disease in mice, guinea pigs, rabbits and monkeys have been unsuccessful.\textsuperscript{9}

Lewy,\textsuperscript{10} in 1945, studied the histopathological changes in 2 specimens of sacral roots and found an almost identical picture in both, a "peracute radiculopathy". Macroscopically, there is always oedema of the brain, spinal cord and peripheral nerves, with congestion of the meninges. Microscopically, the peripheral nerves show edema, infiltration of inflammatory cells and vascular congestion of the bundles. There is swelling of the myelin sheaths and fragmentation of the axis cylinders. The anterior horn cells also show changes, principally chromatolysis and vacuolization. The changes are most marked in the peripheral and cranial nerves but it is of interest to note that all the changes are reversible. Post-mortem examinations have revealed that the disease affects the viscera but clinically no systemic manifestations are found.\textsuperscript{9}

Although the etiology of this syndrome is not known and the pathological lesions are not characteristic, it is interesting that the clinical picture is so well defined. Generally, there is a history of a slight upper respiratory infection 8 to 10 days prior to the onset of symptoms. The mildness of these preceding infections makes it difficult to evaluate the real rôle played by them in the general picture of this syndrome. Shortly after the onset of these mild symptoms, signs of peripheral neuritis develop symmetrically in the lower extremities and muscle tenderness may be noted as well as sensory changes. All patients later develop a symmetrical flaccid paralysis of the lower extremities. In some patients the paralysis continues to advance upward and may result in a complete quadriplegia. The rate of spread, as well as the degree of involvement, is quite varied; the paresis may be only manifested by awkwardness in the performance of voluntary movements, while in others it may be complete. There is no special wasting or atrophy of muscle groups other than that referable to disuse. At other times the sensory symptoms may be the first to appear. When they do occur they are generally limited to the distal portions of the extremities. The other neurological manifestations that have been described in this condition are not characteristic. Cranial nerve involvement is seen frequently.\textsuperscript{11, 12} Ford and Walsh\textsuperscript{13} reported a case with increased cranial pressure and papillöœdema.

The findings in the cerebrospinal fluid are of great importance in this condition. The most characteristic change is the so-called albumino-cytological dissociation, that is, an elevation in the protein content without a cellular increase. In 1936, Guillain\textsuperscript{14} postulated that a protein level of over 300 mgm. % was an essential finding for the diagnosis. This figure is definitely too high, since typical cases have been reported with protein values close to 100 mgm. %. In some instances the increase in proteins does not occur at the onset of the motor changes but appears later in the course of the disease.\textsuperscript{15} The rest of the cerebrospinal fluid findings are generally normal, although xanthochromia may at times be found.

The most important point in differential diagnosis is to separate it from acute anterior poliomyelitis, and because of the different prognosis the establishment of the correct diagnosis is of more than academic interest. The important points to consider in differentiating these two conditions are: the seasonal epidemic incidence of poliomyelitis, the absence of fever or of constitutional signs at the onset of the motor disturbances and the marked symmetry of the lesions in the Guillain-Barré syndrome. If albumino-cytologic dissociation is present, this is in favour of the latter, although it is sometimes seen in poliomyelitis.\textsuperscript{17, 18, 19} Again, if the recovery is complete, without muscular atrophy or contractures, the diagnosis of Guillain-Barré syndrome would appear more logical. We have seen several borderline cases in which accurate diagnosis was impossible, and until further knowledge of the etiology of the Guillain-Barré syndrome is achieved, they will remain unclassifiable. The second important differential diagnosis that should be considered is lead poisoning, but x-ray, haematological and clinical studies should simplify the differentiation. The other polyneuritides, such as those due to diphtheria, syphilis or vitamin B deficiency, usually offer little difficulty in the differential diagnosis, but when doubt persists, laboratory examinations
are of aid. In the early phase of the neuro-
logical form of acute porphyria the symptoms
may simulate a Guillain-Barré syndrome, and
differentiation here is aided by spectrographic
studies and the fact that the urine when exposed
to light, darkens. Pseudo-hypertrophic muscular
dystrophy at its onset may also simulate the
Guillain-Barré syndrome.

The prognosis of this condition in childhood
is good, as compared to an approximate 20% 
mortality in adults.16 The duration of the symp-
toms is quite variable and they may last from
2 weeks to 2 to 3 years. Recovery of function
follows in reverse order in which they were lost.

One of the main purposes of this report is to
present the case histories of 4 children with
Guillain-Barré syndrome. We present our his-
tories in detail so that comparison with the dis-
ease in the adult may more easily be made. We
feel that the group, though small, gives the
average picture of the disease in children.

CASE 1

J.J., a 5-year old boy, was admitted to the Children’s
Memorial Hospital on May 17, 1941, with the chief
complaint of inability to walk for 2 days. His past
history was irrelevant. He was in apparent good health
9 days prior to admission when he developed a slight
headache and somewhat irritable. He could stand up,
but on attempting to walk, after the first step or two, would
fall. There was indefinite weakness of arms and legs
which appeared to be more marked in the proximal segment
of the arm and leg. Strength from the lower limb was
adequately performed but not maintained
for any length of time. Ankle, knee and radial reflexes
could not be elicited. The remaining reflexes were
normal and there was no cranial nerve involvement.
There was slight tenderness in the popliteal spaces but
no definite joint or muscle tenderness. Sensory responses
to touch, pin prick and position were also normal.
White blood count showed a leukocytosis of 13,000 cells
with a slight neutrophilia. Repeated urinalyses were
normal. Blood Wassermann and Mantoux of 0.1 mgm.
O.T. were negative. Throat culture showed a slight growth of M. catarrhalis. X-ray studies of the long bones showed no lesion. On the day following admission a lumbar puncture revealed a crystal clear fluid, with an
initial pressure of 130 mm. of water. On microscopic
examination 4 cells per c.mm. were found. The bio-
chemical examination revealed: proteins 175 mgm.%,
chlornide 435 mmg.%, glucose 68 mmg.%. He
was given 5 mgm. of thiamine chloride by mouth
daily from the time of admission. During the first week
he became progressively weaker. A second lumbar tap on
the 5th hospital day showed an increase in the cerebro-
spinal fluid proteins to 230 mgm. % with only 3 cells.
During the second hospital week the child became pro-
gressively weaker and was unable to lift his arms
or raise his head without assistance. He also complained of
pain behind both knees. By the third hospital week
he had begun to improve and could hold his arms up
while the muscular tenderness was less marked and his
general disposition was excellent. A third lumbar puncture on the 23rd hospital day showed a mildly positive
pleocytosis and no cells. At this time the routine of
administration of the thiamine chloride was changed to
daily intramuscular injections. He improved rapidly,
and was discharged from hospital 6 weeks after adm-
ission with nearly normal muscular power. He was
followed by the Physiotherapy Department and dis-
charged as cured on August 15, 3 months after admission to
the hospital.

CASE 2

M.N., a 21/2-year old boy, was admitted to the Chil-
dren’s Memorial Hospital on April 4, 1944, with the
complaints of refusing to sit, stand or walk for the last
3 days. Past history was irrelevant.

The child was well until approximately 10 days prior
to admission, when the parents noted that he was holding
himself very stiffly, and had some difficulty on mictu-
rition accompanied by dysuria, voiding only once a day.
He was seen at that time by his family physician who
advised circumcision. Two days before admission he
refused to sit or stand and appeared to have pain when
his legs were touched. He did not seem ill and his
temperature was normal, but as these symptoms con-
tinued, he was brought to the hospital.

Examination showed a pale, well developed and well
nourished child who was fretful and unco-operative,
preventing to lie on his side. He would not sit or stand.
There was no frank paralysis and he could move his legs
somewhat, especially to withdraw them from painful
stimuli. Power in the upper extremities was better.
When he cried, the umbilicus moved upward suggesting
relative weakness of the recti below the umbilical level.
Ankle and knee reflexes could not be obtained, radial
reflexes were greatly diminished. Response to painful
stimuli was fair, there was no cranial nerve involvement
and the fundi were normal. Hemoglobin, white blood
count and differential showed normal values for his age.
Mantoux 0.1 mgm. O.T. and blood Wassermann were
negative. Throat culture showed a slight growth of a
hemolytic streptococcus. Urinalysis and x-ray studies
of the long bones were normal. Lumbar puncture on
the day of admission revealed a slightly xanthochromic fluid with a few crenated red blood cells, 3 lymphocytes
per c.mm., a positive Pandy reaction and on biochemical
examination a protein level of 251 mgm. %.

In the hospital, the general weakness became more
marked, he could not withdraw his legs to painful
stimuli and weakness of the depressors of the lower lip,
especially on the right, was noticed. There was no
hyperesthesia on stroking the leg, but the calf muscles
were tender on pressure and passive movements of the
legs became very painful. He became incontinent of both
urine and feces. On admission because of pain, he was
started on a daily intramuscular injection of 25 mgm.
of thiamine chloride. The pain disappeared after 10
days of therapy.

A second lumbar puncture done on the 12th hospital
day revealed a very faintly xanthochromic fluid with a
protein content of 420 mgm. % and no cells. At this
time his general picture was the same, with the exception
that the superficial reflexes were now very difficult to
elicit. On the 20th hospital day he developed measles
and was transferred to the isolation pavilion. Following
this episode, he began to recover gradually and 5 weeks
after admission he was able to sit up in bed. He con-
tinued to progress favourably and 11 weeks after ad-
mission was discharged from the hospital. He was fol-
lowed by the Physiotherapy Department and 3 months
after the onset of symptoms was discharged with normal
muscular power.

CASE 3

P.S., a 2-year old girl, was admitted to the Children’s
Memorial Hospital for the first time on April 17, 1946.
Her past history was irrelevant. One month prior to
admission it was noted that she sat and stood up with

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difficulty. This weakness increased steadily and 2 weeks before admission she began to complain of abdominal pain and of pain in her knees. One week before admission she began to have difficulty in swallowing and her voice became hoarse.

Physical examination showed a well developed, well nourished child, lying comfortably on her back. During examination she became fretful, cried with a weak voice, and it was noticed that she had a somewhat nasal quality of speech. She refused to walk and could not maintain the sitting position, but trunk and neck muscles were obviously weak as she could easily be tipped off balance. Arms and legs were generally weak but there was no bulbar or cranial involvement. The pupils dilated poorly but she appeared to swallow without regurgitation, although choking and coughing after swallowing. No deep reflexes could be obtained, and no pain or gross loss of pain sensation were noticed. The white blood count was 10,000 with a normal differential count; hemoglobin was 10.6 gm. %. Repeated urinalyses were normal. Mantoux 0.1 mgm. O.T. and blood Wassermann were negative. A throat culture revealed no pathogenic organisms present. X-rays of the skull, chest and long bones were essentially normal. A lumbar puncture done the day after admission showed a slightly xanthochromic fluid with 16 cells per c.mm., a positive Pandy reaction and a protein content of 453 mgm. %. At this time, daily intramuscular injections of thiamine chloride, 20 mgm., were instituted.

Eight days after admission a 2nd lumbar puncture resulted in clear fluid with 10 cells, a positive Pandy reaction, and on chemical analysis, proteins 221 mgm. %, sugar 97 mgm. % and chloride 446 mgm. %. Two weeks after admission, her weakness became very marked and tenderness, mainly distal to the knees, was elicited. This marked weakness lasted for a period of 5 to 8 days when a remarkable improvement was noted. One month after admission, the greater part of the motor function of her lower extremities had returned and 3 weeks after admission she was able to walk unaided. During her 6th week in the hospital, she developed scarlet fever and was moved to the isolation hospital. At this time examination showed absent knee reflexes and a slight weakness of both lower extremities. On discharge from the isolation hospital, 2 weeks later, her recovery was complete.

CASE 4

S.M., a 10½-year-old boy was admitted to the Children's Memorial Hospital on May 29, 1946, with complaints of difficulty in standing up and in walking for 10 days prior to admission and pain in the thighs for 2 days at the onset of symptoms. His past history was irrelevant.

He was in apparent good health until 12 days before admission when he awoke complaining of pain in his thighs and calves. These pains persisted for 2 days and were relieved by massage. Ten days prior to admission, while walking, he fell several times and his parents noted that he walked in a very "disjointed" manner. This progressed until he was unable to walk.

Examination showed a well developed, well nourished boy, lying comfortably in bed, without apparent distress. Temperature, pulse and respirations were normal. General physical examination was essentially normal, positive objective findings being limited to the neurological examination. There was no pain or muscle tenderness. He had marked symmetrical weakness of all muscles of the lower extremities. On standing he was very unstable and he was unable to rise on his toes. There was a slight weakness of the biceps, triceps and handgrip. There was some difficulty in sitting up or turning over, indicating some trunk involvement. No facial or bulbar involvement was present, and respiratory movements were normal. All deep reflexes were abolished. Sensation and superficial reflexes were normal. White blood count was 7,800, with a normal differential. Urinalyses were normal. Mantoux test with 0.1 mgm. O.T. and blood Wassermann were negative.

Several throat cultures revealed non-pathogenic organisms. X-rays of the long bones were normal. Lumbar puncture the day after admission revealed a xanthochromic fluid under normal pressure. On microscopical examination no cells could be seen, and the chemical analysis showed an elevated protein content of 221 mgm. %. Daily intramuscular injections of 20 mgm. of thiamine chloride were instituted.

During the first month of hospitalization there was a marked progression of the weakness, the lower extremities became completely paralyzed and he lost about 75% power of the upper extremities. He could not hold his arms in the outstretched position and could not lift his head off the pillow. However, he could turn his head and elevate his shoulders well, indicating good function of the sternocleidomastoids and the trapezi. Inter-costal movements were good, but his cough was feeble, indicating some weakness of the respiratory muscles.

No cranial nerve involvement was noted. Complete areflexia persisted. Cerebrospinal fluid protein was still very elevated. (For details of spinal fluid see Table I.) By the middle of July, 2 months after the onset of symptoms, he showed wasting of the extremities, and despite a rigid regimen of physiotherapy, contractions of the hands were developing.

**Table I.**

<table>
<thead>
<tr>
<th>Cells</th>
<th>Colour</th>
<th>Protein</th>
<th>Chloride</th>
<th>Glucose</th>
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<tr>
<td>mgm. %</td>
<td>mgm. %</td>
<td>mgm. %</td>
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<td></td>
</tr>
<tr>
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<td>0</td>
<td>xanthochromic</td>
<td>221</td>
<td></td>
</tr>
<tr>
<td>June 21</td>
<td>2</td>
<td>clear</td>
<td>230</td>
<td></td>
</tr>
<tr>
<td>Aug. 6</td>
<td>0</td>
<td>xanthochromic</td>
<td>650</td>
<td></td>
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<tr>
<td>Aug. 29</td>
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<td>xanthochromic</td>
<td>424</td>
<td></td>
</tr>
<tr>
<td>Sept. 21</td>
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<td>clear</td>
<td>412</td>
<td></td>
</tr>
<tr>
<td>Dec. 11</td>
<td>0</td>
<td>xanthochromic</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Feb. 13</td>
<td>0</td>
<td>clear</td>
<td>415</td>
<td></td>
</tr>
</tbody>
</table>

Ten weeks after admission active movements of the upper extremities were first noted, he stated he felt better and his apathy disappeared. He then slowly and gradually began to improve. Four months after admission he began to have movements in his lower extremities and the contractions of the hands disappeared. At this time a bilateral foot drop was noted. Coincidental with his clinical improvement a decrease in the cerebrospinal fluid proteins was noted. Six months after admission he began to walk unaided although his gait was far from normal. He continued to progress and at present, 1 year after onset of illness, his muscular power is normal.

**DISCUSSION**

Although the Guillain-Barré syndrome is not common in childhood, we have encountered 4 typical cases in the past 5 years. During the same period this general pediatric hospital had a total of 19,560 admissions, giving a ratio of 1 to approximately 5,000 admissions. We have also had the opportunity of studying 5 other patients, who, although they had a peripheral neuritis and showed various other features of the Guillain-Barré syndrome, failed to present the complete association of symptoms necessary to this diagnosis. The exact nature of these 5 cases, which we have not included, has not been definitely established, and it is possible that these represent atypical forms of the syndrome.

From the study of these cases, the following differential points may be considered in com-
parison with cases described in adults. Apparently there is wide variation in age, the youngest of our series being 2 years, the oldest 10½ years of age. The onset is generally quite rapid. In 3 of our patients the exact moment of appearance of the symptoms could be determined, viz., 3, 10 and 12 days before admission. One child, case 3, had a long prodromal period of approximately 1 month, simulating the onset as seen in the adult.

The most frequent complaint was difficulty in walking, which was a major feature in our 4 cases. The preponderance of the motor signs over the sensory was striking in all cases. In fact, in 2 patients (3 and 4), no changes in sensation could be elicited, and only 1 patient (2) were they of such degree as to make the child uncomfortable. The three youngest patients in our series appeared quite contented and even euphoric, while the oldest (4) showed the apathy usually seen in adult patients with this condition. Susman and Maddox20 also commented on the apparent euphoria of these children which, we think, could be in part explained by the absence of pain.

Among adults, 35 to 50% of all cases show some involvement of cranial nerves, particularly of the 7th or facial nerve. The only evidence of facial nerve involvement among our cases was in patient No. 2 who showed a transient weakness of the depressors of the lower lip. In patient No. 3, cranial nerve involvement was manifested by difficulty in swallowing and by a hoarse voice with a distinct nasal quality. A notable feature of our series was the complete absence of respiratory involvement. It is well known that the prognosis in childhood is almost uniformly excellent, as contrasted with the mortality of approximately 20% found in adults. Since the deaths among adult patients are, for the most part, secondary to a paralysis of the respiratory muscles, the sparing of these muscles in children is probably the major factor in the usually favourable outcome. In all 4 patients of our series, recovery was complete.

As in adults, the course of this disease in childhood is variable. Our case 1 is an example of a short course. This 2½ year old child showed complete recovery after only 2 months. In contrast with this, in case 4, the child began to show signs of recovery only after 11 weeks and was not considered cured until 1 year after onset.

Throughout our series, we obtained the impression of a correlation between clinical and biochemical findings. This was shown most strikingly by patient No. 4 (see Table 1), in whom a definite clinical improvement was found to coincide with a definite decrease in cerebrospinal fluid proteins.

Since this syndrome tends to be self-limited and in children eventual complete recovery is the general rule, it is difficult to estimate the efficacy of the various treatments that have been proposed. There is no specific treatment beyond symptomatic relief and general nursing care. During the convalescent stage, physiotherapy in the form of hydrotherapy, massage and exercise, has proved of benefit. The use of thiamine chloride intramuscularly in large doses has been reported as beneficial in relieving the sensory symptoms; as in children the sensory symptoms are of secondary importance we may argue that its routine use is probably unwarranted. This argument is supported by our own experience in these 4 cases. The Kenney method too has been used, but without particular success.24 More recently Shaeffer22 and Test23 have reported on the use of neostigmine methyl sulphate and in their cases the authors thought it contributed greatly to the rapidity of the patient’s recovery. We have had no experience with either of these last methods.

**Summary**

Four case histories of the Guillain-Barré syndrome in childhood have been presented. They illustrate the differential features of the disease in children in comparison with the disease in the adult, namely, the wide range of age, rapid onset, great preponderance of motor signs over sensory symptoms, the variable course and the good prognosis. In this small series a suggested impression of some correlation between clinical and biochemical findings has been obtained.

**References**

THE treatment of strictures of the common bile duct has always been complicated, and followed by results which are relatively poor, except that during the past few years results have improved. It is true that with almost any type of repair, results will be good for a short time, up to one or two years following repair. Because of this tendency for the stricture to reform, decision cannot be made regarding the final result until two or two and a half years have elapsed since repair. This feature has been one of the factors in the confusion in following the results in various types of operations.

ETIOLOGY OF STRICTURES OF THE COMMON DUCT

During the past 11 years (1936-1947), we have encountered 39 patients with stricture of the common duct at Illinois Research and Educational Hospital. The relative incidence of causative factors in this group is shown in Table I.

<table>
<thead>
<tr>
<th>Cause of stricture</th>
<th>No. of cases</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>Operative trauma</td>
<td>25</td>
<td>64</td>
</tr>
<tr>
<td>Inflammation</td>
<td>8</td>
<td>20</td>
</tr>
<tr>
<td>Chronic fibrosing pancreatitis</td>
<td>5</td>
<td>13</td>
</tr>
<tr>
<td>Pancreatic cyst</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
<td>100</td>
</tr>
</tbody>
</table>

N.B. Only 3 patients, excluding the pancreatic group, were jaundiced before cholecystectomy.


Numerous mechanisms may produce strictures of the common duct (see Fig. 1). Unfortunately, carelessness, haste, and inadequate knowledge of anatomy (including particularly the anomalies) are responsible for most of the strictures produced by operative trauma. Too commonly, serious hemorrhage from the cystic artery is allowed to develop, and is stopped by blind stabbing with an artery forceps which may have included part of the common duct, with damage of that structure by the ligatures controlling the bleeding point. Occasionally a stricture is produced by the scarring resulting from the healing of an ulceration in the wall of the duct produced by a gallstone. Very rarely indeed does a stricture result from the trauma or effect of cholecchochostomy.

PREVENTION OF STRICTURES

Since the operative treatment of the common duct is so difficult and so commonly followed by poor results, it is obvious that great attention must be paid to prevention of strictures. Careful observance of the precautions listed below will minimize stricture formation.

1. Obtain good exposure.—Since it is so essential to see and identify structures during operations on the biliary tract, the incision must be made long enough to obtain good exposure and retraction must otherwise be effective. Good anesthesia with proper relaxation is essential in biliary surgery.