

MEDICO-SURGICAL SUGGESTIONS

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MEDICO-SURGICAL SUGGESTIONS

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(Established 1932.)

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TYPHOID FEVER IN CHILDREN*

K. M. SUKHTANKAR, M.B., D.C.H., D.PED.

Bombay.

The subject of my talk in to-day's meeting is, "Typhoid Fever in Children." There is much to talk on the subject. I am not going into the book picture of this condition, but will restrict myself as far as possible to the pictures I have seen in the Byramjee Jeejeebhoy Hospital for Children in Bombay, from September 1947 to September 1948. I shall describe the signs, symptoms and complications which I have seen, the investigations which were done and the treatment which was followed in this hospital. This subject was selected by me after reading in the year book of Pediatrics 1945, about the special treatment of typhoid fever and that is with penicillin and sulphathiazole. I have studied during this short period about 25 cases of typhoid fever, out of which twenty-two cases were treated with penicillin and sulphathiazole. I am glad to tell you that I am very fortunate to do this short work under the able direction and advice of my teacher, Dr. George Coelho.

AETIOLOGY

Incidence—Typhoid fever prevails especially in temperate climates. It is widely distributed throughout the world. In India it is far from uncommon. Twining pointed out, that in 1882, a fever often prevailed in Bengal, which proved fatal under typhoid symptoms and in which the small intestines were found ulcerated after death. Ewart and Peet have recorded numerous cases of fever occurring in various parts of Bengal, Madras and Bombay Presidencies. It presents everywhere the same essential characteristics and is an index of sanitary intelligence of a community. To-day it is more prevalent in districts than in cities because of the contaminated water supply and imperfect sewage. Thus the propagation of the disease is largely from the country to the city. In Bombay and in suburbs, typhoid fever is in endemic form. From the statistics drawn by the Bombay Municipality it is seen that the incidence is very high in overcrowded localities. This year, the prevalence of typhoid in epidemic form collaborates with the Municipal report of 1944.

*Read at the Scientific Section of the Silver Jubilee Session of the All-India Medical Conference, held in Calcutta, Dec. 1948.

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1945 and 1946, as shown against the areas affected. From the beginning of August to the end of October 1948, there were 1,391 attacks and 455 deaths.

Children who share the same food as adults suffer from the infection as frequently as adults, if not more. The predisposition to enteric fever is much influenced by age, the disease being chiefly met with in youth and adolescence. It is frequent in childhood but infants are attacked rarely. Dr. Coelho had diagnosed typhoid fever in a child 6 months old. Dr. Sanzgiri has reported one in a case 5 months old. Youngest child in the B. J. Hospital, Bombay, was found to be one year old. Thus we can say that the disease may affect any age. Incidence of typhoid fever in children is less as compared with bacillary infections.

Age Incidence in 25 cases—0-4 years—Nil, 4 years old—1 case, 5 years old—6 cases, 6 years old—1 case, 7 years old—4 cases, 8 years old—1 case, 9 years old—2 cases, 10 years old—6 cases, 11 years old—1 case, 12 years old—3 cases.

Bombay City Fever Hospital showed the following age incidence:—

In 207 cases: 1 year—1 case, 2 years—4 cases, 3 years—3 cases, 4 years—9 cases, 5 years—9 cases, 6 years—5 cases, 7 years—15 cases, 8 years—24 cases, 9 years—23 cases, 10 years—45 cases, 11 years—23 cases, 12 years—47 cases.

From the above figures it is seen that younger children, and particularly infants, seldom acquire typhoid fever. The infrequency of infantile typhoid is due to limited opportunity of contacts with the organism rather than to any other degree of immunity. Unless the mother, nurse or aya happens to be a carrier, infants are not likely to be exposed. Majority of the cases are above 7 years in both the hospitals. It is common at this age because this is a school-going age and majority of us know that at this age we used to buy food at the school gates. This food is the common source of infection. Ice fruits, fruit salads prepared from over-ripe fruits, Bhel prepared from cheap vegetables and other oily things and the ice *sharbat* which is well mixed with road dust are the common things seen popular at the school gates.

Sex—Typhoid bacillus has got equal affection for both sexes. This may be true in adults, but in children, males are more affected because boys eat the above infected things at the school gates. In our hospital record, there are 16 males out of the total of 25 patients.

Seasonal incidence—In Bombay, typhoid fever is endemic. The incidence goes on rising from May reaching its peak in August. In our hospital, out of 25 cases, 5 cases were admitted from January to April, 15 cases from May to October, 5 in November and December. The City Infectious Hospital of Bombay shows the seasonal incidence as given below:—Out of 208 cases in the last 5 years, 26 cases were admitted from January to April, 148 from May to October and 34 cases in November and December. Osler says, "Typhoid fever is essentially an autumnal fever and more than half his cases were admitted in August

September and October." Our figures show the same seasonal incidence. From the figures drawn by the Bombay Municipality from 1940-1947, the number of attacks and deaths of typhoid fever is increasing year after year, and from 1948 to 1946, the number is quite high. This may be due to over-crowding, poor diet, scarcity of water, etc. Dr. Das Gupta, the Health Officer of the Bombay Municipality, is of opinion that typhoid fever in Bombay may be frequently contracted from infected milk. Convalescent and healthy carriers are frequent sources of infection. Out of 25 cases, history of typhoid fever in the family was available in two patients only. It has been variously estimated that after recovery from typhoid fever, 1 to 5 per cent of the patients remain carriers for a period varying from a few weeks, months, to many years.

CLINICAL PICTURE

Mode of Onset—In the majority of our cases, we do not know the mode of onset, as these children are brought in the hospitals usually in the second week or later. Out of the 25 cases 11 cases were admitted at the end of first week, 9 patients after 15 days, 4 after 20 days and 1 patient was admitted about three weeks after the first attack of typhoid fever. Majority of the patients were brought for continuous fever. 8 cases were acutely ill and toxic when admitted, 2 cases were semi-conscious, 1 had continuous fever and 2-3 attacks of epistaxis in the first week, one had vomiting at the onset of the fever, 3 had cough and continuous fever, 2 had rigors and intermittent temperature. The rest had remittent type of fever.

The symptoms preceding the actual onset of the disease are very difficult to elicit in infants and young children. With abrupt onset there is sudden rise of temperature and vomiting. Convulsions rarely occur. Chill or chilliness was frequently complained of by the older children at the onset and headache was more frequently noted than any other single symptom except fever.

Physiognomy—It is very difficult for me to describe the change in the countenance in typhoid patients. I had eight patients who were looking acutely toxic and ill when admitted. Two patients were semi-conscious.

General condition was very poor in three cases and fair in the rest. Four patients gradually became emaciated. Out of these four, one died of perforation later on, and one died of peripheral circulatory failure. In this latter case, the condition of the patient from the beginning was not good. He was very toxic from the time of his admission. He was given nasal feedings. His temperature touched normal in about 2 weeks' time and remained normal for three days. His general condition was good then and he was taking food by mouth. On the fourth day, temperature again went up to 103° and 104° F, and was continuously high. He was toxic and the general condition began to deteriorate. He died in the fifth week.

Temperature—In almost all cases, temperature at the time of admission was 101°-104° F. Majority showed later on the remittent type of temperature. Two showed intermittent temperature. Such a temperature with an enlarged spleen often suggests malaria. One of the two cases showed malaria parasites in the blood film. This case responded to paludrine. Widal reaction was strongly positive showing clearly the possible association of these two fevers. The typical temperature curve is not unusually seen in young children but is well observed in grown up children. In this series of 25 cases, not a single case showed plateau in temperature. In 8 cases, the temperature was irregular. In children it is quite common to get rigors. Three patients had rigors at the onset. Two cases were getting rigors in the hospital. In one, as I have already said, M.P. were found in the blood film. In two cases, extremities were cold even at the height of the fever. 4 cases were perspiring on the face and forehead in the 3rd and 4th weeks. In 14 cases the temperature came down to some extent with the administration of penicillin and sulphathiazole. In two cases temperature came down by crisis—in one there was a leaking perforation of the intestine, proved later on by operation, while the cause in the other is not known. Other intestinal complications, such as diarrhoea, constipation, tympanitis etc. had no effect whatsoever on temperature. Secondary skin infections like abscess, boils, bedsores and impetigo do affect the temperature. In our series, 2 patients had bedsores—one on the scalp and gluteal regions. One had boil in the axillary region, one had impetigo. Some of the children looked ashy pale at the beginning of the rise of temperature.

Alimentary System—TONGUE—In the majority of our cases, the tongue was thinly furred in the beginning, while its tip and margin were unusually red. In some cases it remained in this state throughout the attack. In other cases it became thickly coated in the centre with the margin and tip clear. During the period of convalescence, tongue of 17 patients became clean and moist. 3 patients had red raw tongue with granular tip. One patient's tongue was magenta coloured. One had atrophied papillae and few transverse fissures. The tongue of two patients was almost clean and moist throughout the attack. A peculiarity of the tongue is its unusual redness, which may be confined to the tip and edges or extended over the entire surface. Another character of the tongue is the existence of transverse fissures often deep and painful. In one case tremors of the tongue were marked in the 3rd week. This patient was very much emaciated. He used to have irregular loose motions and therefore was kept for a long time on protein milk and banana. Tremors disappeared gradually as the patient's general condition began to improve. In the majority of toxic cases, the tongue was thickly coated and there was offensive smell in the month. Lips were cracked in three cases. Cheilosis was present in 4 cases. Sordes collected on the teeth of 8 patients who were in the typhoid state. Catarrhal stomatitis and gingivitis were present in two cases only. This was

producing great distress. There was excessive flow of saliva in these cases. Signs of vitamin B deficiency were marked in the majority of the cases who were on liquid diet. Other deficiencies commonly observed were: (1) vitamin A deficiency showing marked phrynodema, (2) iron deficiency leading to anæmias. Blood count report showed this in 48 blood counts—19 had hypochromic and 17 patients had hyperchromic anæmias. Thirst was marked in 4 cases. This is an important sign in adults. Children take large amounts of fluid and so thirst is not common in them. Dehydration was present in 3 cases. Appetite is usually lost. It was good in 6 cases throughout their stay in the hospital. Moderate appetite in one case and poor appetite in 11 cases. 4 cases were given nasal feeds. Nausea and retching was present in four cases. Vomiting was present in one case at the onset of fever. Three patients had vomiting in the ward. Out of these, two had vomiting after taking milk, while in one, it was present after perforation. In two out of the eight toxic cases, vomiting was present during the toxic stage.

ABDOMEN—In the majority of the cases, the abdomen was doughy from the day of admission. In 3 patients it was soft on palpation in the beginning, but soon it became doughy. Moderate distension was present in most of the cases who were on full diet. In one case abdomen was retracted. This patient showed signs of meningitis but the C.S.F. was normal chemically and bacteriologically even after repeated examinations, thus excluding T. B. meningitis. Widal reaction confirms the diagnosis of typhoid fever. In nearly all cases peristalsis was well heard on auscultation. In the case of perforation, they were absent when perforation took place. In another case, they were absent and in addition to this sign, patient complained of pain in the abdomen. Operation revealed nothing. He however became all right after the operation. Six patients complained of pain in the abdomen, especially round about the umbilicus. Out of these 6, one had passed round worms one showed round worm ova when the rectal swab was examined and one had perforation of the intestine. Other causes may be tender intestine, constipation, cholecystitis. The liver was palpable in 14 cases, and the spleen in 8. Slight icterus was observed in 4 cases.

BOWEL MOVEMENT—Regular in seven cases, constipated in 13 cases, mild diarrhoea in one case and severe diarrhoea in one case. The record of infectious disease hospital of Bombay shows diarrhoea in six cases out of 75. Constipation was present in nearly all cases. Obstinate constipation was not present in a single case. The above figures clearly show that diarrhoea is less frequent than constipation though the infection is located primarily in the intestine. Constipation is welcomed in children, but obstinate constipation is not good as hard faecal masses cause ulceration of the bowels. Stools were generally offensive. They were fairly well formed to soft in consistency. Usually the bowels were opened once a day or on alternate days. Diarrhoea stools were offensive; large

offensive flatus was passed irrespective of diarrhoea or constipation. Rectal swab examination gave the following findings:

18 cases showed nothing abnormal pathologically; round worm ova in two cases, cyst of giardia lamblia in one case, Balantidium coli in one case, few pus cells in three cases,

Respiratory System—Epistaxis is more common in the early stage. I had one case who had epistaxis in the first week. Throat congestion in 13 cases. 15 cases showed abnormal respiratory signs and symptoms, bronchitis being the commonest. The record of the City Fever Hospital collaborates with our readings. Lobar pneumonia in 2 cases, broncho-pneumonia in 2 cases, pleurisy in one case, catarrh of the lungs 3 cases, enlarged hilar glands one case, pulmonary oedema in one case. Out of the 75 cases of the Bombay City Fever Hospital, lobar pneumonia in 7 cases, bronchopneumonia in 14 cases, catarrh in one case, pulmonary oedema in one case. The physical signs improved with penicillin and sulphathiazole administration.

Cardiovascular System—Pulse values more than Widal reaction. During the early stage pulse rate is increased but not in proportion to the fever. In older children pulse is like adult pulse and is relatively slow. In infants and younger children pulse rate frequently reaches 150-180 p.m. which in older patients would certainly be considered as an unfavourable sign. In the late stages it is more rapid, feeble and small. Dicrotic pulse which is very common in typhoid fever in adults, was found in 2 cases in my series. Volume and tension was good in the majority of the cases. In the case of perforation, pulse was poor in volume and tension. Blood pressure—only in two cases showed subnormal reading. **HEART**—The size was normal in all cases.

HEART SOUNDS—Systolic murmur was heard in one case in the mitral area for about 5-6 days only.

ROSE SPOTS—In two cases, 8-10 spots, about 1.2 mm. in diameter disappearing on pressure, macular in character were found in the abdominal wall on the eighth day of the fever.

Genito-urinary System—In nearly all cases urine was passed freely. In the present typhoid epidemic in Bombay, there is oliguria in many patients. These patients soon become oedematous and urine reports show increased albumin, presence of pus cells, R.B.C.s and casts. Thus we see a nephritic picture in association with the typhoid infection.

Out of my 25 cases the chemical examination of urine showed trace of albumin in 10 cases, bile pigment in 2 cases. Microscopic examination showed epithelial casts in 3 cases and 3-4 pus cells in nearly every case.

Central Nervous System—As far as the nervous system is concerned individual and constitutional peculiarities make themselves strongly felt here. Thus there are children who dream away or sleep away their typhoid fever, others again behave peculiarly at the beginning and often become very restless, delirious, have fantasies and keep in constant

activities. These children require strict and constant supervision and guard. I had 3 cases of this type. In severe cases especially in older children with long high fastigia and under-nourishment there develops the picture of status typhosus. These cases often show the signs of encephalitis. Out of 25 cases 8 were toxic and acutely ill at the time of admission. 6 patients were drowsy, 2 unconscious, 4 very irritable and restless.

All these toxic patients showed signs of meningo-encephalism (namely, heaviness of the head, rigidity of the neck, sluggish or absence of pupillary reaction, squint, absence of superficial reflexes and brisk deep reflexes). Kernig's sign was present in 3 out of 8 cases. Lumbar puncture was done in all these cases. Fluid was clear, of moderate to increased tension; proteins normal or slightly increased; cells 8-10, lymphocytes predominating. Other findings are normal. Post typhoid insanity was observed in one case. This patient showed signs of encephalitis during the attack. He became mentally normal as the nutrition improved.

Complications—The Bombay City Infectious Hospital record of 75 cases showed complications in 48 cases. I have already mentioned complications in the lungs, C.N.S. and C.V.S., alimentary system that were observed in B. J. Hospital. Other complications were as follows:—

Parotitis in 3 cases, bilateral in one case.

Deafness in 2 cases.

Otitis media in 2 cases.

INVESTIGATIONS

I have spoken about the reports of urine, stools and cerebrospinal fluid.

Blood Examination—Total W.B.C. count shows leucopenia in 24 counts, moderate leucocytosis in 18 counts, 19 counts showed relative lymphocytosis. There is a general tendency of labelling any continuous fever with a furred tongue, slight abdominal distension or a few gastrointestinal symptoms, which do not yield to sulpha drugs or a diaphoretic mixture as typhoid and if the temperature is moderate it is labelled as paratyphoid. This diagnosis is not at all justifiable. However suggestive, the signs and symptoms may be, no fever may be labelled as typhoid without a positive blood or stool culture or failing that a positive agglutination test, keeping in mind previous antityphoid inoculation. In our hospital out of the total of 66 blood reports for agglutination tests 51 gave positive findings. The first week had 6 reports, second week 18, third week 10, fifth week 5 and sixth week 3 reports.

Blood cultures—In all, 4 blood cultures were positive. One in 1st week, 2 in the second week and one in 3rd week.

Stool and urine cultures were not successful.

DIFFERENTIAL DIAGNOSIS

In children there are other conditions which stimulate typhoid fever clinically. Widal reaction helps in diagnosis. I shall put before you

those conditions which we at first diagnosed as typhoid but later on blood agglutination test and other investigations gave us real diagnosis.

(1) Heat fever—This is more common in infants from 6 months to about 36 months in the months of April, May, June and October. There is diurnal fluctuation in temperature and because of restricted diet, this condition is usually accompanied by diarrhoea or constipation. Tongue is furred and there is loss of appetite; Widal reaction is negative. We came across such cases in the out-patient's department.

(2) Primary tubercular complex with or without evident enlargement of mediastinal glands

(3) Upper respiratory tract infections.

(4) Otitis media.

(5) Interlobar pleurisy.

(6) Pneumonia—2 cases.

(7) Rheumatic carditis—1 case.

(8) Paratyphoid—2 cases.

(9) Unknown pyrexia—1 case.

In the out patient's department I came across one case of round worm infestation. There was definite history of passing round worms. Other signs were similar to typhoid fever. Stool examination showed plenty of round worm ova. Widal reaction was negative. Oleum chenopodium made him all right.

I have already dealt with one more condition that is malaria. One case at first from the history of fever with rigors and intermittent temperature I thought of malaria. Paludrine was given. Blood film was examined twice. No M. P. detected. The temperature did not come to normal even after 6 days. Blood was sent for Widal test. It was positive.

Associated Conditions—Malaria and typhoid in 2 cases. (2) T. B. infiltration and typhoid in one case. (3) Enlarged mediastinal glands and typhoid in 2 cases. (4) Upper respiratory tract infection and typhoid in 13 cases. (5) Round worm infestation and typhoid in 2 cases.

PROGNOSIS

In typhoid fever in children except for bronchopneumonia the prognosis in the principal complications, namely perforation and haemorrhage has not been greatly improved by modern treatment. Another important complication namely toxæmia is much less now a days than before and this is because of treating the patient with full diet and penicillin injections along with oral administration of sulphathiazole in full doses. Haemorrhage can be prevented or ameliorated by large doses of Vitamin C and Calcium injections. Our hospital record shows that prognosis is brought to a better state by keeping the patient on full diet as far as possible. From September 1947 we have gone one step ahead by starting penicillin and sulphathiazole treatment. The following figures will give an idea of how the mortality rate is becoming less. Side by side I am giving you figures of attacks and deaths in the City Infections Hospital where soft diet in the form of pudding, bread and butter, eggs (boiled),

banana, rice, etc. is given but penicillin and sulphathiazole treatment is not followed.

Year	B. J. HOSPITAL		I. D. HOSPITAL	
	Attacks	Deaths	Attacks	Deaths
1942	31	4	26	6
1943	44	4	16	5
1944	25	7	36	6
1945	8	2	Not available	
1946	38	9	82	11
1947	42	2	48	1
1948 upto Sept	20	2	Not available	

These figures give us at least some idea of the change in prognosis from high mortality to low by giving the patient full diet from the very beginning and penicillin and cibazol treatment after the diagnosis is confirmed by either blood culture or Widal reaction. Poorly nourished children or those who have been debilitated by some constitutional or acute disease have a less favourable prognosis than those who are robust. Today we can say that prognosis is somewhat better than before. Prognosis depends upon (1) virulence of the epidemic, (2) severity of the disease, (3) previous health, (4) resistance of the child to the infection. It is seen from the hospital records that the younger the child lesser the complications and prognosis is better. Out of 25 B. J. Hospital cases 2 patients of about 10 years age died. City Hospital record showed upto 8 years out of 69 attacks 7 deaths. 9-12 years out of 139 attacks 22 deaths.

TREATMENT

General Management—It is strictly seen that good nursing and correct hygienic management which are still the most important points in the treatment of typhoid fever are rigidly followed in our hospital. Rest is bed is very essential. But this cannot be observed very strictly in children. They move about when the temperature goes down, go to lavatory, squat for a stool rather than taking a bed pan, so far this did not meet with danger. This shows that a certain amount of latitude may be tolerated. Older children are placed in bed and kept there constantly. The use of bed pan and urinal is insisted upon. Their beds are kept smooth to prevent the occurrence of bed sores. From my record you have seen that there are only 2 cases of bed sores. These were treated with sulphathiazole ointment. Mothers are given instructions that the position of the child in bed must be changed from time to time. This prevents hypostatic congestion of the lungs and occurrence of bed sores. The mouth, tongue and teeth of the little patient are kept scrupulously clean by the use of a mild antiseptic wash. This precaution prevented stomatitis and other infection of the mouth. 3 patients were very toxic and irritable. Their mouths were not properly clean. In these patients later on a swelling in the peritoid region was seen. This was treated with sulphadiazine orally, and with the application of glycerin balladonna.

locally in the parotid region. Mouth is cleaned with antiseptic wash. Skin is kept healthy by regular daily sponging and application of antiseptic powder especially over the pressure points. Only one patient had developed boils in the mid-axillary region and one developed impetigo. Boils were treated by injecting penicillin 1-1000 into them while for impetigo we used 1 per cent gentian violet in water. Enema was given to those who could not pass stools on their own. We used simple soap water when there was no distention. In the presence of distention we used turpentine enema and turpentine stupes. In one of my patients, I had to carry on hydrotherapy. The patient's temperature went up to 106°F. He was restless and irritable. We wrapped the child in a sheet soaked in the tap water with an ice bag on his head. Sheets were removed soon after the fall of temperature to 100°F.

Nutrition of the Child—Till 1942-43 the diet for typhoid fever patients in B. J. Hospital used to be skimmed milk with tea or coffee and butter milk. This diet was continued till the child had no fever. These patients suffered from abdominal distension and discomfort. Occurrence of faecal impaction was very common especially in older children. Ema- ciation anaemia and other deficiency ailments were very common in these patients. Since the last 3-4 years Dr. Coelho introduced the following diet for the patients from the day of admission. Bread, butter, banana, milk with tea or coffee, rice, minced meat and liver, ice cream etc. Experience taught us that this diet makes the patient look brighter and less toxic. Dr. Coelho was the supporter of the liquid diet upto 1944-45. One day a child who was in a dying condition asked for Khima and Dr. Coelho had prescribed it to satisfy the patient's last desire on this earth. On the next day he saw the patient somewhat brighter and looking at him with a smiling face. Dr. Coelho added bread, rice to Khima. Like that with daily addition he came to full diet with surprisingly good results. He tried the same full diet since then for all patients, and now we see undoubtedly good results. Patients should be given as much as they want. The administration of such a diet had no deleterious effect on the patient. The range of temperature, the frequency of stools and toxæmia were not affected badly. Patients did not complain of abdominal pain or discomfort after the food was given; 3 patients had complained of pain. We have given them mild carminative and continued the same diet. Complications like diarrhoea and perforation were seen to be more common in those to be kept on liquid diet, such as glucose water, milk, or butter milk. The full diet treatment is definitely beneficial to the course of illness and the prognosis is much better. Full diet should include adequate carbohydrates and limited amounts of protein. Because by giving a large amount of proteins, products of nitrogenous wastes or added to those from destruction of the nitrogenous tissues. This increase throws strain upon the kidneys, adds to toxæmia and raises the temperature. We usually give 3-4 small pieces of meat or liver, or 2-3 tablespoonfuls of minced meat or minced liver. Even in the presence of

loose bowels, we gave banana protein milk and minced cooked liver. Banana upto 10 per day, white of about 10-12 eggs and 2-3 teaspoonfuls of meat can be given safely. In younger children boiled milk preferably protein milk was given in the same quantity as in the child's accustomed diet. On account of its rapid utilisation and protein sparing property sugar is given liberal amounts. The old idea which is still held by many and which is based on the pathology of the intestine that a diseased intestine cannot assimilate and digest solid food is wrong. By the time the food reaches the diseased part of the intestine it is well digested and is in the assimilable form. By supplying full diet, protein is supplied in sufficient amount to make up for the loss of body protein. There is great increase in the destruction of the nitrogen containing tissues while the main carbonaceous components of the body such as fat are affected in a much smaller degree. Nitrogenous tissues are destroyed in fever by the action of toxin and by starvation. We cannot give a large amount of proteins because it increases the products of nitrogenous wastes and flood the circulation with these products. This throws the strain upon the kidneys and also adds to toxæmia. Therefore protein spacers and preferably carbohydrates are used. When the temperature comes to normal protein supply should be increased with advantage. From the experience of our hospital records and that of the City Fever Hospital I have marked that the patients who were brought late in the hospital are generally toxic because they are all on liquid diet and the fluid administration is also insufficient. Mortality also is very high in these cases.

Special Treatment with Penicillin and Cibazol—Joseph Bigger reports that in broth freed from substances antagonistic to sulphanilamide drugs, sulphathiazole inhibits the growth of *E. typhus* to a considerable extent. It has also some bactericidal effect. But only on a small inoculum, Penicillin in concentration upto 8 units per c.c. reduced but did not prevent the growth of the same in broth. However the combination of penicillin and sulphathiazole had beneficial effect on the bacillus. Even if a patient is not benefitted by the treatment it certainly does no harm. He suggested sulphathiazole in full doses according to age, 4-hourly and penicillin about 50,000-60,000 units 3-hourly for 5 days and preferably for 7 days. Fever may occur during this time as the result of release of toxic substances from the killed bacteria. Both drugs are discontinued at the same time and the patient is observed for the presence of any more bacilli. If more are found treatment is continued for another four days. According to McSweeney, the treatment should be started during the later stages of illness when the symptoms of the disease are fully developed. We have started Joseph Bigger's line of treatment after seeing the blood culture or Widal reaction as positive. 25 patients are treated on this line. 8 patients were very toxic. Toxicity disappeared in 7 cases at the end of the course of 5-7 days. The 8th case did not respond so much at the end of the first course. After 4 days rest, again another course for 7 days was given and the patient became all right. Another advantage of this treatment

is that it is helpful in treating and preventing lung complications. As for the effect on temperature it was seen in our cases, that it came to normal in 11 cases out of 25. In 3 patients it came down to 100°F. from 104–103°F. In one case we have tried procain penicillin 1 c.c. daily for 10 days. This patient was very toxic and was getting convulsions. He had lung signs and was being given nasal feeds. He was discharged completely cured.

Symptomatic treatment—Diaphoretic mixture for fever.

For toxæmia and dehydration 5 per cent glucose saline I.V. or subcutaneously—150–200 c.c. In the presence of toxæmia, pallor, refusal to take feeds, poor general condition and running of high temperature blood transfusion is badly needed. 3 patients were given B.T. in our cases. 200–250 c.c. is usually given. If blood pressure is low cortical extract of adrenal gland is given, 1 c.c. of doca I.V., with normal saline or 12½ per cent glucose saline, twice a day preferably. For high temperature hydrotherapy is done.

Treatment of complications—Lung complications were treated with sulphadiazine and penicillin; diarrhoea with mist. bismuth cum creta for a day or two and then mist. acid hydrochlor dilute. Treatment of complication is already described at different places.

Treatment during convalescence—For loss of appetite acid hydrochlor dilute with glycerin pepsin mixture and yeast tablets.

In case of stomatitis or for other vitamin B deficiency signs—Vitamin B complex given by injections or orally.

For anaemia—Ferrous sulphate orally. Liver extract injections or by mouth.

This then is the outline of treatment. There is nothing original about what I have said. The methods are gleaned from here and there and smoothed out into a routine after buffetings and some bitter experiences I shall feel I have done something useful if a few of you, particularly colleagues of my age, derive some benefit from a point here and there.

I like to conclude by saying that typhoid fever must be treated with full diet, penicillin injections and sulphathiazole tablets by mouth.

Journal I.M.A. November 1949.

INTESTINAL OBSTRUCTION*

PHILIP THOREK, M.D.

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All who come in contact with the problem of intestinal obstruction realize that it still presents a diagnostic and therapeutic challenge. The mortality continues to remain high despite the many recent advances in electrolyte balance, intestinal siphonage, caloric requirements and surgical technic. Any plan which aids in the early diagnosis and treatment of the obstructing lesion helps further to reduce the number of fatalities. Wangensteen, Haden, Orr, Collier and many others have contributed monumental stepping stones which enable us to understand the pathologic physiology of this condition.

Intestinal obstruction is a symptom complex and not a disease, hence, it is not enough to make a diagnosis of "just intestinal obstruction." In attacking this problem we have devised a plan whereby we can make an earlier and more thorough diagnosis, thus enabling proper therapy to be instituted more rapidly. To correctly diagnose the condition it is necessary to ask and answer the following four questions:

1. Is this an intestinal obstruction?
2. Is it a large or small bowel obstruction?
3. Is it strangulated or non-strangulated?
4. Is the obstruction complete or incomplete?

In answer to question number one: "Is this an intestinal obstruction?" we expect to find the obstructive triad, namely, distention, constipation and vomiting. Even though the triad may be present wholly or in part, its individual parts call for clarification. In regard to distention, one must define what he means by the term. Since we have no standard for measuring the distended abdomen, we have decided to utilize the anatomic relationship of the umbilicus to the xiphoid process. We believe that the normal abdomen is scaphoid and not flat, hence, the umbilicus is normally placed below the xiphoid. When the umbilicus is on a level with the xiphoid, the abdomen is called flat, and when the umbilicus is above the xiphoid, the abdomen is described as being distended. Therefore, when the umbilicus is on a level with, or above the xiphoid, some pathologic condition exists. When such an abnormally placed umbilicus is found we consider the differential diagnosis of the seven "F's," namely, Fat, Faces, Fluid, Flatus, Fetus, Fibroids and "Ph"antom tumors. In almost every case one of the "F's" has been found to be the underlying cause. It is important to record the position of the umbilicus when the patient enters the hospital, and to re-check this every hour thereafter. If the umbilicus is below the xiphoid when the patient is first seen, and one hour later is found on a level with the xiphoid, this signifies early distention. In this way we can avoid the development of a late perterminal distention that so many neglected intestinal obstructions present. Regarding obstipation, we

know that the average intestinal obstruction passes neither feces nor flatus, but we also recall that this may be lacking in incomplete obstruction, as for example in Richter's hernia, in which only part of the circumference of the bowel is incarcerated. In such cases the resulting irritation and hyperperistalsis may even lead to a diarrhea which can be most misleading when one makes a diagnosis of intestinal obstruction. Vomiting will be more thoroughly discussed under question number two. Regardless of the absence or presence of the obstructive triad, it is far more important to elicit the one pathognomonic finding of intestinal obstruction, namely, that pain and intestinal sounds appear at the same time. This synchronization of sound with pain differentiates intestinal colic from any other type of intermittent pain. The physician should place his stethoscope upon the patient's abdomen when he states that he is getting his pain, and if it is of an intestinal nature he will hear the rushing bowel sounds at this time.

Question number two, namely, "Is this a large or small bowel obstruction?" The most important differentiating factor to this question is whether or not vomiting is present or absent. Patients with large bowel obstructions do not vomit, but those with small bowel obstructions do. We all have seen late cases of large bowel obstruction where vomiting has been present as a late and not too distressing symptom, but in the small bowel lesions vomiting appears very early. The higher the obstruction the more fulminating the vomiting. Utilizing this one fact, we can usually differentiate the small from the large bowel obstructions. To use the word "fecal" vomiting as being descriptive of intestinal obstruction is incorrect. The term "feculent" is more descriptive since fecal vomiting refers to a gastrocolic fistula or some similar lesion. The flat x-ray film is used to further differentiate the small from the large bowel obstruction. It is unnecessary to stand or turn the patient or to give him any contract media. A flat x-ray picture, which can be taken with a portable machine, will usually give the desired information. If the obstruction is a large bowel lesion, the x-ray film usually reveals a large distended colon which appears as a horse-shoe or inverted "U." The rectosigmoid is the most common location for these lesions. If, on the other hand, the obstruction is small bowel in nature, the typical paralleling or step-ladder pattern will be present. The history also aids in differentiating the two types of obstructions. A slow, progressive, chronic, increasing constipation speaks for a large bowel lesion, but a sudden violent attack signifies small bowel pathology. Patients with intestinal obstruction who have had previous surgery are small bowel obstructions until proved otherwise. The large bowel obstruction resulting from postoperative adhesions is a rarity. A two quart diagnostic enema is also of help. The large bowel can usually retain two quarts of fluid plus its usual contents. If the bowel cannot take the two quarts, this speaks for a large bowel lesion. There are many other ways of

differentiating the two, but time nor space do not permit extending this discussion.

Question number three, "Is this a strangulated or non-strangulated intestinal obstruction?" can usually be answered by the presence or absence of tenderness. Patients with intestinal obstructions do complain of colicky pain, but the strangulated lesion has pain plus localized tenderness. This tenderness is best found by the patient, who will usually locate the exact point of the pathology. The classical example of this is a strangulated inguinal hernia. The patient has diffuse pain over his entire abdomen, but will permit one to palpate it; however, he resents having pressure made over a strangulated mass because of its exquisite tenderness. Our incision is usually determined by the location of the patient's tenderness. Another differentiating point between the strangulated and non-strangulated obstruction is the appearance of the patient. A patient who has a strangulated intestinal obstruction is acutely and violently ill and usually is in shock or impending shock, whereas the patient with an intestinal obstruction without strangulation does not present such a dramatic picture. The flat x-ray film may aid in the differentiation of a strangulated from a non-strangulated small bowel obstruction. If a small bowel, non-strangulated, intestinal obstruction is present the typical step ladder pattern is observed and the valvulae conniventes are readily seen. If, on the other hand, a small bowel strangulated obstruction is present, no characteristic bowel pattern is assumed since the distended loops arrange themselves in whatever portion of the abdomen that the obstruction occurs. The valvulae conniventes are not easily detected or seen because of the extravasation of blood into the strangulated loop of bowel and into the abdominal cavity.

Question number four states: "Is this a complete or incomplete obstruction?" As has been mentioned, a patient with a complete intestinal obstruction passes neither flatus nor feces per rectum, but if the obstruction is incomplete some flatus and feces may be expelled, especially with repeated enemas. It is important not to be misled by the results of the first enema, since a copious movement and flatus may be expelled following its administration. This, however, is material which is distal to the lesion. If repeated enemas bring flatus and feces then we assume that the lesion is incomplete; if the returns of the repeated washings are clear, we conclude that the obstruction is a complete one. A "scout" film of the abdomen should be taken when the patient arrives. This immediately reveals the bowel pattern and also determines whether or not flatus is present in the region of the hollow of the sacrum. If the flatus over the sacrum is absent following repeated enemas, we consider the condition a complete obstruction, but if the flatus continues to come down and appear over the sacral region, the lesion is an incomplete one. A patient with a complete obstruction will appear more ill than one with an incomplete lesion; therefore, the clinical appearance and impression is of importance.

Based on these four questions, one may make a proper diagnosis instead of just "intestinal obstruction." The case, therefore, may be diagnosed as a large bowel, non-strangulated, incomplete intestinal obstruction, or a strangulated, small bowel, complete intestinal obstruction, depending upon the findings.

Treatment

When one labors through the voluminous literature on the subject of the treatment of intestinal obstruction, it becomes difficult to apply this maze of material. It is wise, therefore, to have a plan based upon a simple summary. We have devised a plan based on the six "S's," since we state that the treatment of intestinal obstruction consists of Suction, Saline, Sanguine, Surgery, Sulfa and the "Stir-'em" technic.

Suction, or gastro-intestinal siphonage, has done much to lower the mortality of this condition. It has its pitfalls, however, and these must be kept in mind. It has no place in large bowel obstructions nor should it be used when strangulation is present. On the other hand, it may be curative in postoperative ileus, non-strangulated adhesive obstruction, or in obstructions associated with peritonitis; these are usually small bowel lesions. Its value as a pre- or postoperative adjunct needs no emphasis. To keep a patient with carcinoma of the rectosigmoid and a large bowel intestinal obstruction on continuous siphonage is to court disaster. Hence, its uses and abuses must be thoroughly understood.

Saline can prolong the life of a patient with an intestinal obstruction; however, it cannot cure the condition. It is an excellent form of supportive therapy. Chloride ions have been lost in the patient who has manifested a great deal of vomiting or in whom continuous gastro-intestinal siphonage has been instituted. These must be replaced, and it is mainly by the use of physiological saline that the patient's chloride balance may be maintained. By restoring this electrolyte balance one is able to put his patient into better condition to withstand surgery, and in this way also to lower the mortality. Saline, however, is not the only supportive therapy that the patient needs; this will be discussed subsequently.

Sanguine is the word used to refer to blood and its derivatives. We feel that the only place for the use of whole blood is in the replacement of lost red cells. We prefer to keep the protein balance of the patient normal with plasma, serum or amino acid therapy. If the obstruction is associated with blood loss, we feel that the fluid of choice is then whole blood. In many cases of strangulated obstructions, or in cases which might necessitate extensive bowel resection, whole blood is preferred. Maintaining a normal protein level permits a patient to properly seal because of his good fibrin content. Hypoproteinemia and hyperchloridemia are two conditions which must be avoided in the case of intestinal obstruction as well as in all other surgical emergencies. Too little protein and too much chloride both produce tissue edema and permit the patient to "drown" in his own body juices. It is because of hypoproteinemia

and hyperchloridemia that sutures pull out of edematous tissue. Faulty suturing is the cause of intestinal leakage; this is due to poor pre- and postoperative care. The patient's vitamin needs must be maintained, especially the water soluble vitamins B and C which he loses readily. Vitamin C is truly the "surgeon's" vitamin because this is the one which is essential to sound wound healing.

Surgery is a subject which cannot be discussed adequately in a few minutes or a few pages, and I will only have time to touch upon the surgical highlights as they pertain to the patient with an obstruction. If a patient has a strangulation he should have immediate surgery. As has been stated, the patient will tell us where to make the incision if we just permit him to reveal his most tender spot. Complete large bowel, non-strangulated lesions require immediate colostomy for the release of intra-colonic pressure. We prefer the so-called "blind" cecostomy in such conditions. This is made through an exaggerated Mc Burney's incision which hugs the anterior superior iliac spine. If the cecum is distended, and it surely should be in an obstructed colon, then it bulges into the wound. It is held in place by two hemostats and an iodoform pack is placed between the cecum and the perietal peritoneum. Following this stitchless procedure, the patient is returned to bed and the cecum is opened some six hours later and after it has had a chance to seal off. Since the bowel wall is edematous and will not retain sutures it is unwise to directly attack an obstructed colonic lesion. It is for this reason that we leave the primary pathology alone and do a preliminary cecostomy away from the site of the lesion. For the following ten days or two weeks the patient may be deflated, prepared and then re-operated. It is at this time that a true evaluation of the pathology can be made and a resection done. The cecostomy acts as a vent in the event that an intestinal anastomosis is performed. In strangulated lesions we may be confronted with the question: "Is the bowel which has been freed viable or not?" It seems impractical to stand about placing hot towels on a segment or intestine and watch it color. Viability is readily determined if one merely flicks the bowel with the finger and watches for peristaltic waves. If it is able to contract, regardless of the color of the intestine, it is viable. Intestinal obstruction is usually associated with a transudate which is present in the peritoneal cavity; if this is bloody a strangulation is present. Therefore, if a blind cecostomy is done and a sanguinous fluid noted, we must abandon the cecostomy and explore for the presence of a strangulated lesion. The type of anastomosis performed is purely a personal one; however, we feel that a lateral anastomosis is the safest in the hands of the occasional operator. If time is a factor, one should be familiar with the technic of the so-called quick "aseptic" end to end anastomosis.

Sulfa drugs have taken their place among the chemotherapeutic agents used in the treatment of intestinal obstruction. There is also a place for such allied drugs as penicillin and streptomycin. Following the

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surgeon, we place three to four grams of sulfadiazine in the peritoneal cavity and follow this with 100,000 units of penicillin every three or four hours intramuscularly. We do know that penicillin will not affect the coliform group of organisms but it will attack streptococci and staphylococci. Sulfadiazine is administered intravenously following the first postoperative day and streptomycin is coming into its own as the main chemotherapeutic agent against the gram negative rods. Sulfasuxidine and sulfathaladine will keep the bacterial count low in the intestinal tract if these drugs can be taken by mouth.

By "stir 'em" technic we mean early ambulation, active and passive movements and breathing exercises. The beneficial effects brought about by getting patients out of bed as soon as possible have been well proved. We do not wish to infer that early ambulation should be carried to an extreme. It is our plan to have our major surgical cases out of bed on the first postoperative day; however, each case presents an individual problem. Having the patient move about, having him take a few deep breaths every hour, and encouraging arm and leg movements all play their part in lowering the incidence of phlebothrombosis, pulmonary complications and their sequelae.

Only the surface has been scratched in this discussion of the vast subject of intestinal obstruction; however, we feel that if we approach the problem with the "Four Questions," make a diagnosis based upon these, and then summarize the treatment with our "Six S's," we should have a logical approach to a given case.

Rocky Mountain Medical Journal August 1949.

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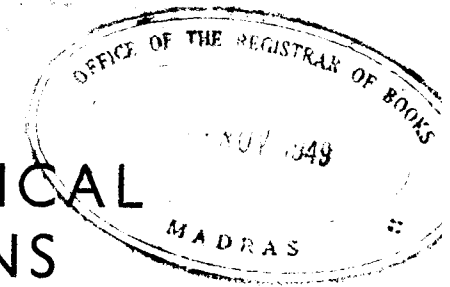
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NEPHRITIS IN CHILDREN.*

G. COELHO, M.B., B.S., B.HY., M.R.C.P., F.C.P.S.,

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CLASSIFICATION

Many have attempted a classification of nephritis. Blackfan (1926) divided it into (1) the acute glomerular and (2) acute tubular. Ellis (1942) divided them into two types—type I with an acute onset and type II with an insidious onset. The correct classification would be the pathological one based on the changes found at autopsy. But a large number do not reach autopsy. As the acute cases recover and the picture of a chronic case may be different, during life one has to be guided by the clinical findings and biochemical investigations.

Clinically the author's series of 38 cases can be divided into three groups: (1) where the first attack has a sudden beginning, 16 out of 38 belong to this group, (2) where the picture develops gradually in the first attack, 7 cases, (3) where the patient comes with a history of previous attacks mostly of the second group—15 out of 22 cases.

When all cases of nephritis are studied the first group is seen largely in children. Ellis (*loc. cit.*) dividing his cases into two types found that of 173 cases of the first type 60 per cent fell within the first two decades and 46 of them were below ten years of age. In my series of 38 cases there were none below 2 years, 12 between 2 and 5, 14 between 5 and 10 and 12 between 10 and 18 years.

ETIOLOGY

The exact pathogenesis is still not definite. Because of the high incidence of a previous infection, it has been long considered that this is a casual factor. But actual bacterial invasion of the kidneys has not been proved. It is said that the damage is due to the excretion by the kidneys of toxins produced by the streptococci. Farr produced nephritis in rats by injecting nephrotoxic sera. At first these animals presented a picture of nephrosis, but later when the urea excreting power had been diminished by about 50 per cent, hypertension became an important factor. Histologically these kidneys showed a variety of lesions. There is a period between the acute infection and the nephritis that still

*Read at the Scientific Section of the Silver Jubilee Session of the All India Medical Conference, Calcutta, December 1948.

needs explanation. Therefore it has been suggested that nephritis is an allergic phenomenon. Schick said this in 1907 and later Press, Longcope and O'Brien demonstrated that a large proportion of cases of acute nephritis gave a marked allergic reaction to intradermally injected filtrates from cultures of streptococci.

HISTOLOGY

Pathologically the description of the kidney lesions has varied. Volhard said that the tufts were bloodless in the early stages. Osler said that they were filled with leucocytes and red blood cells. They also described changes in the tubules and in the interstitial tissues. In the initial phases there is swelling of the intercapillary substance of the glomerular thickening of the basement membrane and tubular degeneration. Aldrich (1945) reported a complete lack of conformity in his observations; one case showed hyalinisation of the glomeruli even as early as three weeks, another extensive tubular degeneration and hyperaemia in the medullary portions, while in a third the changes in the kidney were negligible. In summarising, he writes, "there is great variation in the extent of renal involvement, the glomeruli show changes which may be generalised and of a highly destructive nature, or localised in a few areas and insignificant in degree. The tubular changes are not severe as a rule." Seven of Ellis' patients died in this acute stage and of these, three died in uraemia, their kidneys showed severe glomerular and tubular changes.

CLINICAL FINDINGS

ACUTE ONSET.

The child is brought with any one or more of the following complaints:

1. Scanty urine, 2. Red urine, 3. Puffiness of face, especially round the eyes in the morning.

With these basic symptoms there may be 1. Fever, 2. Headache, 3. Vomitting, 4. Drowsiness, 5. Convulsions, 6. Loss of vision.

On examination the child may show:

1. *Oedema*—Oedema of face, oedema of lower extremities, generalised anasarca of a variable degree, ascites. The first one is present in 100 per cent of cases, the second is in 80 per cent and the remaining in less than half. Ascites is very rare. When oedema is generalised it does not pit on pressure but is firm to the feel. This is best seen on the thighs and back. A certain degree of such oedema may not be detected by the eye but can be detected by the daily gain in weight. "The brain in acute glomerular nephritis is firm and tense and the convulsions flattened" (Blackfan, 1926).

2. *Infection*—Many writers have described an infection which precedes an attack of haemorrhagic Bright's disease. Addis writes, "a clinician's haemorrhagic Bright's Disease is a disease within a disease," as the manifestations of the involvement of the kidney appear even during the period of convalescence. Ellis (*loc. cit.*) recorded a history

of previous infection in 84 per cent of his cases, and these suffered from tonsillitis, sore throat, cold, influenza. Blackfan (*loc. cit.*) recovered a haemolytic streptococcus from the rhinopharynx of 14 out of his 24 patients. Besides the infections of throat, infection of the skin like impetigo and boils may also precede the nephritis. In our cases six had clinical signs of infection of the upper respiratory tract and five had skin infection, scabies with secondary infection. This skin infection was also seen in those children who came with a previous history of oedema, and who during the attack for which they sought admission had extensive oedema—five cases.

3. *Urine*—(a) No urine may be passed for over 24 hours. Most of the children pass very much less urine than normal. It may be frank blood—two cases. It may be smoky—(three cases) in appearance or it may be dark or coffee colour—three cases. As diuresis sets in the thick blood appearance may change to light pink.

(b) Albumin is a constant finding. It may be little, due to the cells in the urine or it may be in excess.

(c) Red blood cells, white blood cells and casts are essential for a diagnosis of acute nephritis. They are present in abundance in all acute cases. Casts of the varied types are always present, cellular and granular predominating.

4. *Blood Pressure*—An increase in the systolic and diastolic pressure is a constant finding in these cases. Hypertension, haematuria and hard oedema form the diagnostic triad in this condition. A systolic pressure above 115 in a child of six years is considered significant. I have seen systolic pressure as high as 250 mm. of mercury in a girl of 4 years. The rise in the pressure is sometimes dramatic. Pressures about 150 mm. of mercury are average for all ages. Even with systolic pressures ranging between 150 and 200 mm. the children play about and do not show any discomfort. But a high blood pressure may be associated with the following manifestations of encephalopathy.

(a) Vomitting, which may be persistent, projectile, and have no relation to food.

(b) Headache.

(c) Drowsiness.

(d) A cloudy mind with loss of correct appreciation of surroundings.

(e) Coma.

(f) Convulsions.

These and a rising blood pressure are danger signals. They may arise at any stage of the illness and have no relationship to the haematuria or anuria. Rarely there may be blindness with a blood urine and a high blood pressure. I have seen only one case in twenty years.

A rising blood pressure precedes or is closely associated with the development of cerebral symptoms. Volhard and Fahr were not certain as to which was primary and which secondary. Marriott (cited by

Blackfan, 1926) suggested that the arterial hypertension is the result of capillary constriction caused by a toxin, and this toxin was responsible for the uræmia. Kylin (cited by Blackfan, 1926) was also of the opinion that the hypertension was due to capillary constriction. But Boas (cited by Blackfan, 1926) found capillary pressure normal in a number of cases of acute glomerular nephritis. Cushing experimenting with dogs found that the arterial pressure rose as the intracranial pressure increased. Blackfan (*loc. cit.*) reported a case where an œdematous brain was found incarcerated in the cranial vault. In the cases reported by him he observed that the decrease of œdema and lowering of the blood pressure were coincident with the disappearance of cerebral symptoms, and that the blood pressure fell with the fading away of the œdema. He writes "it is not to be denied that alterations in the capillaries may be the primary cause of the œdema," but the cerebral symptoms may be explained on the basis of cerebral œdema and increased intracranial tension. In support of this suggestion he cites the therapeutic results obtained by injections of hypertonic saline and one per cent solution of magnesium sulphate in these cases. Hypertonic saline intravenously reduces the volume of the brain. Ellis (*loc. cit.*) did not observe spasm of the retinal vessels in his cases of hypertensive encephalopathy. Neither did he find any increased intracranial pressure. But Blackfan (*loc. cit.*) suggests that the absence of increased intracranial pressure when a lumbar puncture is done may be due to the œdematous brain interfering with the free circulation of the cerebrospinal fluid.

5. *Fever* may or may not be present. Six children had fever. It is found when there is an acute infection of the throat, or an infected condition of the skin, though a number of children with multiple abscesses and heavy secondary infection after scabies do not have any pyrexia. There may be a frank acute hæmaturia without any temperature.

6. *Blood chemistry, retention of nitrogenous products*—There is an increase in the blood urea which ranges between 30 and 80 mg. per 100 cc. in the majority cases. Still neither the œdema nor the oliguria, nor the general symptoms are always parallel with the degree of retention of nitrogenous products. The blood cholesterol is not affected. The plasma proteins are generally not diminished. Ellis (*loc. cit.*) found that the plasma proteins were less than 6 per cent in one-third of his cases. Blackfan (*loc. cit.*) did not find them reduced, neither did Lyttle (1938).

7. *Hæmopoietic changes*—In the early stages there is no change in the red blood cells nor in the Hb. but with the persistence of the loss of blood in the urine there is a fall in the red blood cells and Hb. Two of my cases were saved with blood transfusions. The white cells count reflects the infection if present anywhere.

8. *Heart*—Toxic myocarditis is one of the complications of this condition. Enlarged heart has been found in some cases on screening.

Enlargement of the heart and alteration of the heart sounds are the first signs. This is a grave complication and may cause death.

DIAGNOSIS

I shall only mention a few conditions with bloody or smoky urine that should be remembered these days:

1. After sulfa drugs hæmaturia may occur due to the damage done by the crystals. But under the microscope one sees only red blood cells. There are no casts. The blood urea is not raised.

2. Blackwater fever. This is very rare in children in Bombay. Cases that were referred to the hospital as blackwater fever turned out to be acute nephritis. The diagnosis should be made on the paucity of red blood cells and the presence of methæmoglobin in the urine. This combined with the fever with rigors clinches the diagnosis. The blood pressure is not raised in blackwater fever.

3. Stone in the kidney, tubercular kidney may cause a sudden hæmaturia. The diagnosis is built with further observation.

4. Administration of urotropine. To-day this drug is not used as much as before. But there are still old practitioners who are conservative and use it in urinary infections. Excessive use of urotropine may cause hæmaturia.

5. Angioneurotic œdema. This comes on suddenly but the urine is free.

6. To decide whether it is an acute or chronic glomerulo nephritis is sometimes difficult. It is true history is a guide, but our patients do not give a correct one very often. An acute attack may be seen during a chronic stage. Following the patient for some days one notices an improvement in volume of urine excreted, a fall in the blood pressure, a decrease in the red cells in the urine. After that the progress is at a standstill. The œdema becomes chronic. The other is the differentiation between nephrosis. In a straightforward case the urine would not contain any red blood cells, there would be no rise in blood pressure nor blood urea and the blood cholesterol would be raised and the serum albumin would be low. The œdema is very marked in nephrosis. Sometimes acute glomerulo-nephritis may appear as a complication of nephrosis. Then there will be a hæmaturia, hypertension and an increase in blood urea.

The other condition that is intriguing is benign albuminuria, orthostatic, physiologic, etc. Though Lyttle (*loc. cit.*) says that if urine is examined repeatedly albuminuria is found in 12.68 per cent of all children, my experience has been otherwise. I find it is rare, barring the febrile albumin, though I have not examined the urine repeatedly.

PROGRESS

With hypertension the first sign of improvement is a fall in the blood pressure. A diuresis sets in when the blood pressure comes down to about 120. With the diuresis the œdema disappears gradually. A

fall in weight is the best guide. It takes on an average eight days for it to disappear.

But the blood and casts take a long time to disappear. Our patients seldom stay in the hospital for more than three weeks if they have no oedema but when they leave they are not cured. Their blood urea comes down, but their urine still contains red cells and casts. Our follow-up is very poor and I am sorry I cannot say much about them.

These incompletely cured children are candidates for chronic nephritis in adults. Hence a complete cure should be the aim. Still the treatment is symptomatic and not quite effective. Our knowledge of its pathogenicity is limited.

PROGNOSIS

The hæmaturic nephritis with hypertension following an acute infection has a better prognosis as regards recovery than the other types.

During the course of the illness, myocarditis and a rapidly rising blood pressure are bad prognostic signs.

Though naked eye hæmaturia may disappear in two weeks time yet traces of albumin and a few red cells may persist as long as six months.

TREATMENT

1. *Rest in bed*—This is considered by all as the most important part of the treatment. Aldrich, Ellis both lay great stress on rest. My difficulty has been in enforcing this rest. Our children, whether in hospital or at home, are seldom in bed.

2. *Magnesium sulphate*—In all cases with hypertension magnesium sulphate is the sheet anchor of treatment. It is given orally, intramuscularly, intravenously. Orally a saturated solution is used containing four drachms of magnesium sulphate in an ounce of water. This must be appropriately sweetened and coloured to make it agreeable. The solvent should contain at least three drachms of syrup to each ounce. This is the route in all cases when the patient is conscious and the blood pressure is below 150. When the blood pressure is over 150 and is fast approaching 200, when there are signs of encephalopathy the intravenous route is desirable as it brings down the blood pressure promptly. For this either 1 per cent solution in 5 per cent glucose or 4 per cent in 5 per cent glucose is used. If the former, 5 cc. for each pound weight and if the latter, 1.25 cc. per pound weight is the dose for each injection. The injections must be given slowly, not more than 2 cc. per minute and when the stronger solution is given blood pressure should be measured on the other arm, as there is a danger of its falling very rapidly. If facilities are available it may be given as a drip; otherwise it may be given by a 20 cc. syringe and a two way stopcock. The effects of intravenous magnesium sulphate on a patient with symptoms of encephalopathy are dramatic. There should be no hesitation in using this as the first line in treatment.

When the veins are collapsed or there is a difficulty in getting them magnesium sulphate may be injected intramuscularly in concentration of

25 per cent solution. These solutions are readily available. The intravenous and intramuscular injections may be repeated every 8 hours till the blood pressure comes below 150 mm. Hg.

The action of magnesium sulphate may be due to its sedative action, releasing the capillary spasm or to a change of the osmotic relationship between the blood and tissues of the brain. Blackfan (*loc. cit.*) discussing these possibilities does not favour the sedative action as he thinks that the dose administered is too small to exert this physiological action. He suggests that the beneficial action may be due "to an increase in the total concentration of the electrolytes in the blood which thus alters the osmotic relationship between the blood and tissues." Though Blackfan writes of the cathartic action of magnesium sulphate when administered by mouth, our experience has been different. Half an ounce of saturated magnesium sulphate administered four hourly to children 6 to 10 years of age has not resulted in more than two stools a day so long as the blood pressure has been high.

3. *Control of Infection*—To combat any infection, be it of the upper respiratory tract or the skin sulfa drugs and penicillin are used. My use of the sulfa drugs has been from the days of proseptasine. Since then I have passed from sulfanilamide to sulfapyridine and now to sulfadiazine. Though crystalluria and hæmaturia have been mentioned by various authors I have been fortunate in not having met with a single case. Penicillin is of recent introduction. All these antiseptics and antibiotics are given for 4 days at least. Diaphoretics, diuretics electric cradles, hot air baths and any method that increases fluid loss through the skin is contraindicated as it increases the concentration of toxins.

4. *Fluids*—This is still a matter of controversy. There are yet physicians who want to save the kidney all possible work and rest it. Fluids have been restricted and are being restricted even now. Ellis (*loc. cit.*) is in favour of restricting the fluid intake to one pint of fruit juice and even Christian (1934) is in favour of fruit juices, carbohydrates with restricted fluids in the first week of a very acute nephritis. But Neuburgh (1936) wrote "it has been universally taught that blockage of the kidneys is the cause of nephritic oedema. According to that doctrine to get rid of the oedema one must remove the water by other routes and at the same time permit as little water as possible to enter the body. Only a few experiments were needed to show the utter fallacy of these earlier beliefs and to indicate the shocking amount of harm that their acceptance has worked. Examination of data shows that the kidneys of the oedematous subject were able to secrete a very large amount of urine containing a normal amount of solids as soon as he was permitted to drink a sufficient amount of water." From the year 1933 I have not restricted the fluid intake. I started it as the lesser of the two evils as it was impossible to restrict the fluid intake in the hospital patients who either helped themselves liberally from a water tap or were helped by the relatives. This unrestricted fluid intake did not do them any harm.

Since then there is full freedom to drink as much as they like; in fact, they are coaxed to take at least $2\frac{1}{2}$ ounces per pound body weight per day, irrespective of the state of the oedema.

Aldrich (*loc. cit.*) who is a strong supporter of liberal fluids, writes "since that time (when Breunemann drew his attention to it) water has been denied to no patient and we have usually urged fluids. No patient so treated has died of acute post-infectious haemorrhagic nephritis in this period."

What should be the nature of fluids? Most of the children like fruit juices or coloured aerated water and these I place on high priority. With this rice congee water may be allowed.

5. *Diet*—Christian (*loc. cit.*) writing on the types of nephritis and their management says, "Ideas as to the management of a developed nephritis of any type have been considerably modified in recent years. We recognise that the disease is by no means limited to the kidney. Now practitioners are more influenced in their plan of treatment by consideration of the patient as a whole than in the past. Now they consider less dietary constituents that theoretically increase the work of the kidney and are more concerned with the nutrition of the patient at a high level. . . . rapidly the diet should be changed in amount and variety with only moderate restriction of protein, salt and fluid, a diet not very different actually from a normal diet, free from any excesses." Once fluids were allowed, it did not do away entirely with the problem of feeding. Most of these children are not acutely ill. They still have an appetite and hunger for food. Here again the time-honoured custom is to ban solids, particularly proteins for a long time.

The next item that was introduced in the dietary by me was fruits. Bananas, papaya, chikoos, guavas, water-melons were next allowed. These have no proteins, and so there could be no objection. I am talking retrospectively. These children did well. Then we added diluted milk and rice and vegetables. This too made no difference to the course. If protein could be given as milk why not protein in other forms. Children, some of them have an aversion to milk. So I introduced meat in the dietary. For the last two years a full diet is prescribed, without restricting the quality or the quantity. The full diet at the hospital consists of egg, bread and butter, milky coffee, bananas in the morning, bone soup, a meat dish, two vegetables, a custard pudding, a banana or orange at noon, milky tea in the afternoon, rice and dal or rice and chapatis at night. They made no worse recovery than the others.

Boyd (1937) writes "our therapeutic approach is hampered because of ignorance as to the pathologic physiology involved in its causation. Often empirical therapy has been based on a misinterpretation of physical or laboratory signs. For example, when abnormality of the kidney or of the urinary constituents has been apparent, it has been rationalised that the kidney should be put at rest; that substances requiring elimination through the kidney should be withheld from the

diet. This has served as the rational for the limitation of protein foods, salt and water in the treatment of albuminuria, oedema, haematuria or manifest renal disease of any type."

Aldrich (*loc. cit.*) writes that "a careful check of results does not show that those restricted did any better than those who were allowed protein."

6. *Other Treatment*—For encephalopathy magnesium sulphate is the primary treatment. Neither venesection nor a lumbar puncture is done these days. For the control of convulsions any sedative is used. I have found the quick acting sedative Seconal Sodium particularly useful. Cupping and sweating have no place in the treatment of nephritis to-day.

CHRONIC NEPHRITIS.

Children of group two are of two types. One, the chronic glomerulo—nephritis, come with a story that without any recent infection their feet got swollen first and then the whole body. They have no fever. The quantity of urine is diminished and is in inverse proportion to the oedema. The oedema pits on pressure. When extreme the child may add three pounds to its weight daily. The scrotum or vulva are very swollen, and the skin over these areas and in the feet is translucent. The child is unable to open the eyes. Such a child looks miserable.

The urine is loaded with albumin. There are red blood cells, white blood cells and casts, but these are fewer than in the previous condition.

The blood pressure does not rise above normal, vomiting, headache and other signs of encephalopathy are absent.

With increasing oedema, fluids accumulate in the abdomen. While ascites has been common in this condition, hydrothorax and fluid in the pericardium have been rare in my experience.

With the ascites the child complains of pain in the abdomen and when there is pyrexia with it, the possibility of peritonitis must be borne in mind.

When the skin is tense, there may be an erysipelas infection of the skin.

The blood urea is not raised, but the blood cholesterol may be. There is always a certain degree of anaemia. The plasma proteins are diminished.

Progress:

The course may extend over three to four weeks. The severe oedema may last well over ten days.

During this period there may be a rise of temperature and then there may be haematuria and hypertension, or this period of pyrexia may be followed by improvement in the general condition and disappearance of oedema.

Even after the disappearance of oedema, the urine contains albumin in a good quantity and epithelial cells and casts.

Sometimes the improvement occurs, and no reason can be ascribed to it.

Similarly there may be a relapse, without any apparent reason. In some cases, of course, there is an intercurrent infection.

Prognosis is unsatisfactory, as the course is one of progressive renal destruction.

In spite of voluminous literature, I do not think we have come to any conclusion regarding nephrosis. Though Lyttle and Aldrich considered nephrosis a distinct entity yet while Aldrich discards a diagnosis of nephrosis if hypertension and haematuria are present, Lyttle states that moderate hypertension and microscopic or even gross haematuria are seen with anatomically proved nephrosis. Ellis does not think that this concept of nephrosis as a separate identity is justified as "if the patient with so-called 'nephrosis' does not die in the early stages and does not recover, he ultimately becomes indistinguishable from a patient with Type II nephritis. This effort to diagnose this condition is mainly because of the prognosis, as nephrosis with albuminuria, oedema, non-retention of nitrogenous products and a high cholesterol content of the blood, are said to have a better prognosis as some of them recover.

The treatment of the chronic patient is unsatisfactory. Though sometimes an upper respiratory infection may flare up an acute state, yet it is not always so; on the contrary, sometimes an acute infection with a high temperature has brought about a diuresis and cleared the oedema. This has tempted me to try pyrexial therapy in these cases, but the results have not been encouraging.

In the treatment of the chronic case magnesium sulphate has no place. Administered by any route it does not help the clinical condition. I have tried large doses of alkalies with no success. When the scrotum or vulva is oedematous to a bursting point one is naturally anxious. 25 per cent solution of glucose intravenously and calcium chloride orally sometimes help. Then suddenly diuresis occurs and everything seems brighter.

These patients are given a full diet, and plenty of vitamins. This diet should contain more of protein. This is given as milk, curds, meat and fish. The optimum quantity of protein is 8 Gm. per kilogram weight of body per day. This diet should in the earlier stages be salt free. This does not mean the complete absence of salt, but a minimal amount. If the quantity of salt be tapered down in about a fortnight there is not much opposition from the child to such a diet. Fluids should not be restricted.

Blood transfusions and concentrated plasma intravenously have not helped.

Recently amino acids and protein hydrolysates are available. In spite of all attempts they are not palatable. If a child can take meat, fish and curds I prefer him to take these rather than these manufactured products. My experience with intravenous administration of protein

hydrolysate has been disastrous. One patient died, and three nearly died.

It is rational to search for foci of infection and remove them. The tonsils and adenoids are freely removed. The benefits of this operation do not come up to expectations in the majority of cases. In our patients an infection of the skin preceded the nephritis oftener than infection of the upper respiratory tract.

There is no method of prevention of acute nephritis. Tonsillectomy was once recommended as a prophylactic measure but Illingworth showed that tonsillectomy does not prevent nephritis, but may predispose to it and that it does not cure nor arrest its progress to a chronic stage. To-day with the sulfa drugs and penicillin we have powerful means of combating infection. Yet the incidence of nephritis is about the same.

(*Journal, I.M.A Aug. '49.*)

THE DEVELOPMENT OF HUMAN PERSONALITY DURING THE STAGE OF PUBERTY

BY LAKSHMI CHANDRA, M.A.

(*Delhi University*).

The age at which puberty sets in ranges from the age of 12-15 years. Variations in the age of onset of puberty are due to differences in climate and the build of the body. In the male the onset of puberty is marked by the appearance of facial hair and changes in the voice. In the female the changes are much more marked and are obviously sexual such as the development of breasts, onset of menstruation and changes in the body contour.

These physical changes evoke certain psychical reactions in both the sexes. Both the boy and the girl become self aware and self conscious, modest and shy. Modesty is much more marked in the girls and is associated with a feeling of shame and particularly of uncleanness on account of menstruation.

The attitude of the society towards menstruation is unhealthy and certain taboos and restrictions are placed upon the persons during menses. Women and girls are isolated and are regarded as some sort of untouchables especially among the Hindus. They are not allowed to cook or to serve food and are forbidden many social activities. This attitude of the society should be discouraged and menstruation should be regarded as a healthy sign of womanhood and the phenomena should be accepted open-heartedly otherwise it may lead to very bad consequences. The parents especially the mother should explain to the girl that menstruation is a natural and normal phenomenon and is a badge of womanhood.

In the boys the sexual phenomena of erections and nocturnal emissions wrongly called "pollutions" are common during puberty and these give them great anxiety and worry.

The boy may come to regard these emissions as a sign of disease and may suffer from anxiety and feeling of inferiority. In India quacks and their advertisements in newspapers aggravate these psychological upsets very much. The parent, teacher or the doctor should explain the true physiological nature of these changes to the boys. In India the general attitude is to repress all knowledge about these things and thereby lead the young boys in to wrong channels for knowledge. Some boys acquire the habit of masturbation as erection's and emissions are pleasurable.

The external signs of puberty are often associated with feelings of guilt, as if these disclosed to the world at large the secrets of the mind and soul. Acne is common and the false notion that it is connected with masturbation increases the feeling of guilt and produces a feeling of shame. Anxiety and phobias are common. The children may imagine that they have got some disease like tuberculosis, cancer, insanity or heart disease. They may have the phobias of the other sex. The girl may start having anxiety at the level of phantasy or dream, of being pursued by a man or an animal or being run over by a horse.

Psychology of Sex. The common view regarding sex is the copulation of male sex organ with female sex organ and the aim of sex is regarded as reproduction. Freud has given a very wide meaning to sex and has divided sexuality into three phases :—

- (1) The sexual feeling.
- (2) The sexual aim and
- (3) The sexual object.

A scientific theory of sex, should not only explain the normal phenomena of sex but should also explain, the abnormal and the perverse sexual activities as sometimes observed in perverts and abnormal persons. It is by understanding the morbid forms of sexuality and relating it to normal sexuality that we understand the normal sexuality.

1. *Sexual feeling.* Sexual feeling is the feeling of pleasure in companionship, in love and in sexual acts with the sexual object.

2. *Sexual aim* consists in the actual embrace, copulation and other acts of a sexual nature or the aim towards which the instinct strives.

3. *Sexual object* is the object with reference to which the feeling and aim develop or it is the person from which the sexual attraction emanates.

Sexual perversion or sexual abnormal behaviour may take place in reference to both sexual object and sexual aim.

(1) Deviation in reference to the sexual object. The normal phenomenon is striving for the opposite sex but there are persons of either sex who strive for persons of their own sex. Such persons are called inverters and there are various degrees of inversion. Some may be occasionally inverted or amphigenously inverted (*i.e.* the sexual object may be of either sex) but some are absolutely inverted (*i.e.* their sexual object must be always of the same sex). Under this class also come

those perverts who have substituted some other organ or part of the body for the normal organ of sexual gratification (*i.e.* mouth or anus for vagina). Then there are the so-called fetishists who substitute for the sexual object a part of the body but little adapted for sexual purposes, such as the foot or hair or some inanimate object (clothing) related to the sexual person.

(2) Deviation may take place in reference to sexual aim and in this group are those perverts whose sexual desires are confined to the performance of an act which is normally an introductory or preparatory to sexual act. Such persons seek sexual gratification in kissing, in looking and touching, in seeing the sexual organs of others or by exhibiting their own.

A special class of perversion comes under sadism and masochism.

Sadism is characterised with the tendency to cause pain to the sexual object for gratification while masochism is the tendency to receive suffering and pains at the hands of the sexual object for gratification.

Sexual perversion is not a sign of degeneration or innate behaviour. It is an acquired characteristic manifested in every race from the primitive to the highly civilised and at all times

Sexuality of Neurotics.

Psychoanalysis has thrown much light on the sexual instinct in neurosis (hysteria, obsessions, dementia precox and paranoia). The symptoms of these diseases are chiefly caused by sexual instincts. Hysteria is characterised by an immense sexual desire and a very exaggerated form of sexual repression. The symptoms are the substitutes for a series of emotionally accentuated psychic processes, wishes and desires to which a path of discharge through the conscious psychic activities has been closed by repression. These mental formations strive for expression and through a process of conversion into somatic phenomena appear as symptoms. In other forms of neurosis the symptoms result not only at the expense of the so-called normal sexual instinct but also mainly at the expense of perverse sexual instinct. In all neurotics there is without exception in the unconscious psychic life feelings of inversion and fixation on persons of the same sex.

Infantile Sexuality.

Infantile sexuality and perversions are closely related together. The infantile sexuality is of perverted type since the child is lacking in all that which transforms sexuality into the reproductive function. Freud has named it pleomorphic perverse *i.e.* perverse of many types. It is only children and perverts who do not regard sexuality as the reproductive function and hence they are closely related to one another. In normal sexuality various components of sex instinct are subordinated to the purposes of reproduction and everything that occurs before this conversion has taken place is called perversion. But one should remember that in all perverse satisfactions, the act still terminates usually in a complete orgasm. Some of the features of perversion are present in

the sexual life of a normal man. For example the kiss is a normal sexual phenomenon but it becomes perversion when one starts getting discharged only by kissing. For here the kiss has become the sole aim of sexuality. According to Freud perversions do not consist newly stepping of the sexual aim nor the replacement of genitalia nor even in the variations in the sexual object but solely in the exclusiveness with which these deviations are maintained, so that the sexual act which serves the reproductive process is rejected altogether. Sexuality comprises several component impulses and normal sexuality consists in discarding some of the components as useless and in combining the others in such a way as to subordinate them for reproduction. In perversion one component impulse is supreme while the others are subjected for its own purpose. Normal sexuality consists in concentrating and organising the various component impulses but this organisation is lacking in perversions as well as in the infantile sexuality. Each stage of sexual development in a child as discussed in earlier articles, has its own sexual component impulses which are directed to an object which is changing in all the stages of development.

Phenomena of Sexual Abnormality and its causes :—

¶ The Libido can behave in three different ways

(1) If unhindered by any obstruction, it progresses easily from one stage of sexual development to another.

(2) If hindered, it may either take to inhibition or to (3) regression. Libido inhibition takes place when all preparatory phases are not passed through with success, some of the functions remain arrested at these early stages, with the result that with the general development there goes a certain amount of inhibited development as well.

Both inhibition and regression always go together. Stronger the inhibition of the libido, more are the chances for it to go back to those fixations which were formed in childhood.

There are two kinds of regression of libido :—

(1) Return to the earlier sexual objects invested with libido which are always incestuous in character.

(2) Return of the whole sexual organisation to earlier stages.

In hysteria there is always a return to earlier sexual objects while in obsessional neurosis there is a return of the whole sexual organisation to earlier stages. Regression of libido without repression would never give rise to neurosis but would result in a perversion. The manifestations of progressing libido, if blocked, can return to an earlier stage by the process of regression and find satisfaction there. But if all manifestations of the libido are repressed then the libidinal impulses find expression in the form of symptoms of some neurosis.

Causes of Neurosis :—

(1) People suffer from neurosis when the possibility of satisfaction for the libido is far removed from them i.e., they become subject to

neurosis in consequence of a frustration and the symptoms are substituted for missing satisfaction.

But frustration alone does not cause neurosis. Moreover the component impulses of sex are so plastic that they can get some satisfaction even when one of the components gets satisfied. Further libido can take to sublimation which is always and in every case fails to discharge more than a certain proportion of the libido. The power of sublimation in man is present to a restricted limit.

(2) Then the "adhesiveness" of the libido, turns men into perverts, in whom the perversion which always carries feelings of shame and disgust with it causes internal conflict, a conflict of the ego and the libido. There is no neurosis without such conflict. Conflict is produced by frustration when the unsatisfied libido is urged to seek other paths and other objects. But these other objects and parts cause disfavour in part of the personality. Then the rejected libido (which has been rejected by the part of the personality called ego) manages to get partial satisfaction by taking to devious paths and so forms symptoms which are substitutive satisfaction necessitated by the fact of the frustration. The conflict is always a conflict between the sex instincts and the ego instincts which try to block the expression of sex instinct.

Libido and ego development always go hand in hand and if one is blocked the other also remains blocked.

Perversion occurs when the ego approves of fixation, but when the ego does not approve and is against the fixation of libido then neurosis will occur because the ego will repress this fixation of libido.

Symptom Formation. Several symptoms comprising a disease are caused by conflict which results when a new form of satisfaction is sought. Symptom is a compromise between ego instincts and sex instincts and it is maintained by both. In all symptoms regression of libido to earlier objects or organization is always apparent. The libido is drawn into the path of regression by the fixation it has left behind it at these places in its development. "Now the path of perversion branches off sharply from that of neurosis. If these regressions do not call forth a prohibition on the part of the ego, no neurosis results, the libido succeeds in obtaining a real, although not a normal satisfaction. But if the ego, which controls not merely consciousness but also the approaches to motor innervation and hence the realization in actuality of mental impulses, is not in agreement with these regressions, conflict ensues." So says Freud while distinguishing between neurosis and perversion. When thus the libido is blocked, then it is bound to seek expression in one way or the other by misguiding the ego. The libido is dictated by the ego so long as it finds expression but no sooner it is blocked, it takes to symptoms through which it gets some sort of expression. The fixations are revived and the libido after throwing away the influence of the ego finds its cathaxis through the unconscious system and is subjected to special characteristics of the unconscious system namely, condensation and displacement. In this process repressions and fixation of childhood sexuality are aroused and form the contents of the symptoms of the neurosis. Analytical analysis shows that the libido of the neurotics is attached to their infantile sexual experiences.

Symptoms provide satisfaction in place of one lacking in reality, which are formed by means of regression of the libido to a previous time of life, i.e., (a reversion to earlier phases in the object choice or in the

organization). The neutrotic is tied to a period in the past life. Symptoms reproduce the early infantile way of satisfaction differing from that infantile way of satisfaction because of consorship which is implied in conflict. Now in symptoms the scenes of infancy which are revived are not always true because phantasies formed in infancy are also revived in symptoms. By phantasy is meant those childhood experiences which did not happen actually but which were there in the imagination of child. The symptoms some times take a physical form and at others a mental form. When it takes a physical form then the neurosis is called conversion neurosis and in all conversion neurosis some part of the body expresses the symptoms by doing some movement.

Impotence and Frigidity :—

Cause

- (1) Fear of disapproval or punishment.
 - (a) Fear of criticism or ridicule.
 - (b) Fear of bodily injury from some disapproving person other than the partner.
 - (c) Fear of pregnancy.
- (2) Hostility towards the partner :—
 - (a) A general resentment towards members of opposite sex desire to do them harm.
 - (b) The woman resents what she considers domination by man.
 - (c) The man is envious of the woman and her role in life refuses to give her pleasure because of this envy.
 - (d) A fear by one of the partners of injuring the genital or of the other. This fear is based upon the castration complex.
- (3) Conflicting loves (usually unconscious).
 - (a) The man loves some other woman and is in unconscious conflict (mother). This is based upon oedipus complex. The woman loves some other man (father) and this is based upon the Kalpetra complex.
 - (b) Latent homo-sexuality which is a condition of emotional immaturity.
 - (c) Too much self love which is a condition of emotional immaturity.

Treatment Since cases of impotency and frigidity are caused by disturbances in emotions and ideation, the cure lies in modifying feelings present in such people towards one another and modifying their knowledge of sexual functioning. Those physicians and doctors who think that these diseases are physiological and with this assumption doing operation in sexual organs are doing it at their own risk because operations have met with limited success. The causes of impotency and frigidity which have been mentioned above are emotional and psychological in nature and therefore the treatment should also be psychological, rather than physiological. The treatment should consist of changing the emotional make up of the impotent man and the frigid woman.

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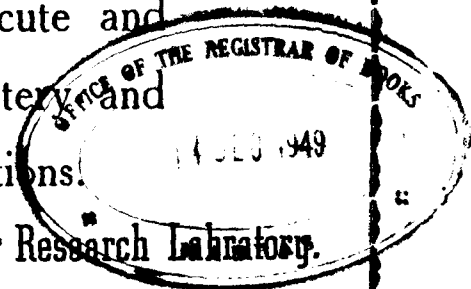
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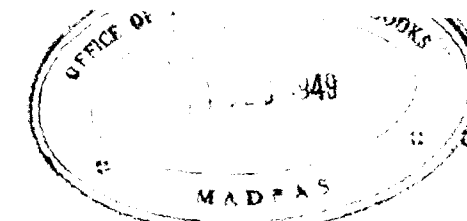
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PURULENT MENINGITIS IN CHILDHOOD*

F. HOWELL WRIGHT, M. D.

Chicago, Illinois

A quarter of a century ago when Dr. Josephine Neal made her classic analysis of the age incidence of bacterial meningitis in New York City, the topics of prognosis and treatment were scarcely worthy of discussion. Invasion of the meninges by bacteria other than the meningococcus resulted in disease, the prognosis of which was so uniformly hopeless that recoveries were regarded as miracles or errors in diagnosis. Depending upon the epidemic strain of organism, meningococcus meningitis mortality varied from 80 to 75 per cent. Survivors, particularly infants, ran an additional risk of permanent damage to the central nervous system. Therapy was then confined to spinal drainage and to the administration of horse antiserum, practices which had a modest effect upon meningococcus meningitis but none whatsoever upon other types. Today meningitis is still a serious disease, but antisera and the antibiotic drugs when properly used can cure about 90 per cent of those suffering from meningococcus meningitis, about 70 per cent of those whose meningitis is caused by other pyogenic organisms, and even an occasional victim of tuberculous meningitis.

Just as the physician's power to alter the course of meningitis is improved by these new products, so must he bear a greater measure of responsibility, for now his skill in prompt and accurate diagnosis and treatment has become the crucial determinant of the outcome for his patient. Particularly disconcerting are those Pyrrhic victories in which bacteriologic cure is obtained but irreparable damage to the nervous system remains. They leave the annoying suspicion that complete recovery might have followed earlier on more adequate treatment. Full realization of the potential benefits of modern methods will always depend upon the practicing physician whose alertness determines the length of delay before a lumbar puncture reveals the true character and gravity of the illness.

*Presented before the 78th Annual Session of the Colorado State Medical Society at Glenwood Springs, September 23, 1948. From the Department of Pediatrics, University of Chicago.

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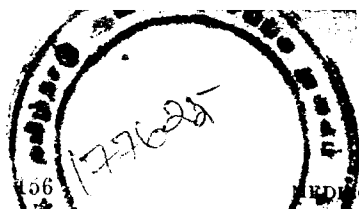
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Q. As they are, Dr. Neal's figures' give us a picture of the incidence of bacterial meningitis which is probably still valid. They show first that it is a relatively infrequent disorder. During the period from 1910 to 1924, which the study covers, there were roughly 100 cases reported annually in New York City with its 5-6 000,000 population. A similar order of frequency may be inferred from recent figures which are available only for meningococcus meningitis, but which undoubtedly constitute more than half the total. For the past fifteen years, between 2,000 and 8,000 cases have been reported each year in the United States except for two epidemic years during mobilization when the figure was doubled. It seems clear, that the average practitioner will have little opportunity to exercise his diagnostic skill.

A second feature of meningitis emphasized by Dr. Neal's study is its relative frequency among children. Within her 1,500 cases, two out of five were less than 10 years old, and one in five was under a year. This concentration of cases within the pediatric age has important connotations for diagnosis and prognosis. Modern treatment has been least successful in reducing mortality and neurologic residua among infants less than 2 years of age. Unfortunately, this is most often retarded by the absence of distinctive clues. Whereas the adult or older child may present subjective complaints of fever, chills, headache, nausea, vertigo, pains in the back or neck, or objective leads such as delirium, coma, stiff neck or back or positive Kernig's sign, the infant is more prone to an indefinite and insidious onset. His illness may be announced by fever, vomiting, anorexia, diarrhea, irritability or generalized convulsions, but these events do not necessarily attract attention to the nervous system for they are common symptoms of many infantile disorders. Suspicion of infantile meningitis should always be aroused by retraction of the head, abnormal tenseness of the fontanelle, a high-pitched cry, persistent or localized muscular twitching, a vacant stare, ocular palsy, bizarre respiratory rhythm or a positive Brudzinski response. Absence of the classical signs of meningeal irritation can provide no reassurance in infancy, and were the slightest doubt exists a diagnostic lumbar puncture should be done. In some instances the clinical findings are so indefinite that discovery depends upon a systematic search for infection, instituted because the degree of illness seems out of proportion to the physical findings.

Dr. Neal's data also show the relative frequency with which the various bacteria invade the meninges. In her series the meningococcus and tubercle bacillus were found in approximately 85 per cent of the cases, occurring about equally. Most of the remaining 15 per cent were caused by pneumococci, hemolytic streptococci and influenza bacilli, each constituting about 5 per cent of the total. Less than 1 per cent were produced by staphylococci and by the colon bacillus group of organisms. Judging from the volume of cases reported in the literature the same general relations obtain today except for a decline in the

importance of tuberculous and hemolytic streptococcus meningitis. The diagnosis and treatment of each type must be considered separately.

Meningococcus Meningitis

Meningococcus infections always appear as primary diseases spread by respiratory contact. The factors which determine susceptibility are poorly understood. The robust are affected as well as the debilitated, young infants and septuagenarians. The meningococci enter through the respiratory tract and pass by way of the blood stream to the meninges. Toxins which they produce damage the endothelium of capillaries and produce petechiae and ecchymoses in the skin and mucous membranes. The extent of these clinical manifestations of meningococcemia is quite variable. Small infants seldom present any skin lesions at all; in older individuals the lesions may be few and small, particularly if the infection is mild; but in severe or fulminating cases, the number and size of the hemorrhages may be so great that the patient's life is threatened more by vascular collapse than by the meningitis itself. The combination of hemorrhagic manifestations and meningeal irritation can be taken as presumptive evidence of meningococcus meningitis, but definite confirmation should always be sought through blood culture and careful spinal fluid examination.

Isolation and bacteriologic identification of meningococci from the spinal fluid is the only conclusive etiologic proof. Proper handling of the first specimen is important. Meningococci are less hardy than other organisms which may be encountered in spinal fluid, and inoculation upon warm media containing blood should be completed before the fluid cools. If possible a set of media should also be incubated at reduced oxygen tension. The chorio-allantoic fluid of fertile eggs offers a highly successful means of isolation to those familiar with the technic. Even when isolation is successful, the result cannot be known for eight to thirty-six hours and consequently a more immediate confirmation of the diagnosis must be sought through direct examination of centrifuged spinal fluid stained by the Gram method. If the examiner has patience and a little experience he can usually find the typical Gram negative biscuit-shaped diplococci either within or apart from leucocytes unless invasion of the meninges is a very recent event.

Accessory examinations of the spinal fluid lend support to the nonspecific diagnosis of bacterial meningitis and give a rough measure of its severity. The pressure is regularly elevated except in neglected cases where the circulation of spinal fluid has been blocked by thick exudate in the subarachnoid space. With increasing severity of the infection, polymorphonuclear leucocytes range from a few hundred to many thousand per cubic millimeter, the protein content from 50 to several hundred milligrams percent, and the sugar content declines from 80 milligrams per cent toward zero.

In vitro, meningococci are sensitive to all the common sulfonamide drugs and to penicillin. Extensive clinical trials have shown

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that sulfanilamide, sulfapyridine, sulfathiazole, sulfadiazine or sulfamerazine all give results which are superior to serum therapy alone. In municipal contagious disease hospitals which must accept all the neglected and complicated cases, the gross mortality rate has been reduced from levels of 25.35 per cent to around 10-20 percent or even lower. In units of the Armed Services where the patients are in optimal physical condition and diagnosis is hastened by continuous medical surveillance, the mortality has been reduced as low as 3 per cent. General preference among the sulfonamides now favors sulfadiazine because it is easy to administer, diffuses readily into the spinal fluid and has a low incidence of toxic reactions. Blood levels of 8-12 mgm. per cent should be established and maintained until clinical cure is assured. In most cases this provides an adequate concentration in the spinal fluid and at the same time avoids the danger of hematuria if an adequate urine flow is maintained. For rapid initial dosage, 0.1 gm. of sodium sulfadiazine per kilogram of body weight diluted to a 5 per cent solution or less can be injected subcutaneously or intravenously. Similar parenteral dosage should be repeated two or three times a day until the patient is able to accept the drug orally. Injections should be made slowly and should be controlled by determinations of the sulfadiazine blood levels. Additional insurance against hematuria and renal blockage may be derived from scrupulous attention to the daily fluid intake which in infants should be about 150-175 c.c. per kilogram of body weight; in older children proportionately a little less. The urine pH should be kept at 7 or above and can be tested easily with nitrazene paper. Alkalinization may be initiated either by the oral administration of sodium bicarbonate in doses equal to the amount of sulfadiazine given, or by including within the daily parenteral fluids from one to three injections of 1/8 molar sodium lactate solution in individual doses of 10 c.c. per kilogram of body weight. The urine should be examined at least daily to assess the degree of microscopic hematuria and through it the degree of renal irritation.

Although penicillin alone is less reliable than sulfadiazine, it is an important adjuvant in therapy. It does not antagonize the action of the sulfonamides, has practically no important toxicity, and is now generally available. Where septicemic manifestations are prominent, the growth of organisms must be stopped as rapidly as possible. Intramuscular or intravenous injections of penicillin should be given every three hours in doses of 20,000 to 100,000 units according to age and the gravity of infection. Combination of penicillin with procaine should not be used since the procaine molecule interferes with the action of sulfonamides and confuses the chemical determination of their concentration in blood or spinal fluid. In cases of average severity, the adjuvant use of penicillin is less urgent but equally logical. The possible penalty for insufficient therapy is too severe to justify its omission.

If the patient is sensitive to sulfonamides and penicillin must be used alone, it should be given intrathecally as well as intramuscularly.

because its rate of diffusion into the spinal fluid is insufficient to produce adequate levels. Doses of 5,000 to 20,000 units in a concentration of 1,000 to 2,000 units per c.c. in saline should be injected two or three times a day. The volume injected ought not to exceed the quantity of spinal fluid removed.

The routine use of antiserum in the treatment of meningococcus meningitis has been abandoned by most authorities because it appears to contribute little more than serum disease. A notable exception is Toomey who still advises routine administration of 100,000 to 200,000 units of antitoxin.

Vascular collapse may appear during the late stages of neglected meningitis or during any stage when endothelial damage is marked. If initial treatment with antibiotics, intravenous fluids, warmth and spinal drainage fails to improve the peripheral circulation within two to three hours, adrenal insufficiency must be considered. Except where unusual facilities are available for rapid determination of the blood concentrations of sodium, potassium, chloride and sugar, the diagnosis must be assumed and the usual treatment supplemented with intravenous saline, glucose, adrenalin and cortical extract until the blood pressure and general appearance of the patient indicate that he is responding. If the clinical manifestations are actually due to adrenal insufficiency, recovery is unlikely. Reports of survival from the Waterhouse-Friderichsen syndrome are few and the diagnosis is usually open to question.

Patients with meningococcus meningitis of average severity usually respond rapidly to penicillin and sulfonamides. Improvement can be expected within twelve to twenty-four hours and in many instances the temperature reaches normal and symptoms begin to subside within two days. Termination of treatment may be considered after the temperature has been normal for two days provided there are no complications and the spinal fluid is sterile with falling cell count and rising sugar content. Relapses which follow too early withdrawal of treatment and late complications, such as arthritis, ordinarily yield in a satisfactory manner to the resumption of therapy. In young infants the danger of adhesive interference with spinal fluid circulation demands a more cautious withdrawal of therapy.

Influenza Bacillus Meningitis.

Influenza bacillus meningitis selects a rather narrow age range between 6 months and 3 years. A sprinkling of cases occurs among younger infants and older children, but the disease is quite rare in adults. Ordinarily it appears as the first manifestation of illness. Demonstrable infection of the blood stream is commonly present. Among infants it is almost universal and is occasionally associated with other localizations such as otitis media, pneumonia, empyema or arthritis.

The influenza bacillus is Gram negative and pleomorphic. In artificial media it appears as long and short thin rods, but in spinal fluid a short bacillus, almost coccoid in form, usually predominates and may be easily

confused with the meningococcus. It withstands temperature changes poorly so that prompt inoculation upon chocolate agar is essential for successful isolation. Biologically the influenza bacillus resembles the pneumococcus in that its capsule contains a polysaccharide which determines its type specificity. With few exceptions the influenza bacilli which produce meningitis in man belong to Pitman's type b. If a sufficient concentration of organisms is present in the spinal fluid, direct typing and immediate identification can be accomplished by using the Neufeld technic, for the organisms demonstrate capsular swelling when placed in homologous rabbit antiserum. With light infections, such positive identification must await cultural isolation.

The bacilli exude specific carbohydrate into the body fluids where it is presumed to serve some function in facilitating their invasion of the tissues. The carbohydrate is excreted in the urine.

Treatment of influenzal meningitis, of course, aims to kill all the bacteria present within the body and to eliminate their products. Infection of the subarachnoid space which remains unchecked for several days forms adhesions which may localize pockets of infected pus and bacteria and shield the latter from therapeutic agents. Even if the infection is eradicated, the remaining adhesions sometimes interfere permanently with the circulation of spinal fluid. Therapy should be prompt and vigorous in order to avoid such complications which are all too common in this form of meningitis.

No less than three specific types of treatment must be considered—sulfadiazine, type specific anti-influenzal rabbit serum, and streptomycin. A fourth, penicillin, is reported to be effective in very large doses but it should never be used as the sole agent in treatment unless the penicillin sensitivity of the strain isolated is definitely known.

Mouse protection tests and clinical studies leave no doubt that sulfadiazine alone, streptomycin alone, or sulfadiazine combined with rabbit serum are all forms of therapy which will save some of the 90-95 per cent of patients who would otherwise die. Evaluation of the relative effectiveness of these methods is hampered by the necessity of making allowances for age, duration and severity of infection and for drug resistance of individual strains. In expert hands the mortality has been depressed below 10 per cent by the use of sulfadiazine combined with rabbit serum and almost as low with streptomycin alone. Alexander correctly recommends that cases which are not under special study should receive the benefit of all three agents in the initial treatment. This provides the maximum insurance against unforeseen difficulties.

In such a program, sulfadiazine forms the basic and prolonged element of treatment. The principles of its administration have already been described. Blood levels of 8-12 mgm. per cent should be maintained until two weeks after clinical and bacteriologic cure seems complete.

Anti-influenzal rabbit serum is standardized in terms of milligrams of antibody nitrogen which is determined chemically from the precipitate formed by the union of antibody and specific carbohydrate. Antibody, of course, combines with the carbohydrate whether it is attached to a bacillus or freely floating in body fluids. The initial therapeutic dose of serum ranges from 25 to 150 mgm. of nitrogen depending upon the severity of the infection. Alexandar uses a graded scale of dosage, giving 25 mgm. when the spinal fluid sugar concentration is 40 mgm. per cent or over, and 100-150 mgm. of nitrogen if the spinal fluid sugar has fallen to 15 mgm. per cent or less. If intradermal and conjunctival tests for sensitivity are negative, the calculated dose, well diluted in saline, should be administered by intramuscular or intravenous drip over a one to two-hour period. The rabbit antibody molecules are sufficiently small to permeate into the spinal fluid where they agglutinate the organisms and combine with soluble carbohydrate. Direct intrathecal injection is not advisable early in treatment for the patient is usually made worse by the sudden combination of large amounts of antibody and antigen in the subarachnoid space. Its use should be reserved for desperate cases which have failed to respond to the usual methods. The initial intravenous or intramuscular dose of serum should be repeated daily until the patient's serum in a dilution of 1:10 can produce capsular swelling of the organisms originally isolated from his spinal fluid. Four injections or less usually suffice, but the number should be controlled with daily tests for excess of circulating antibody. Immediate serum reactions are not uncommon, but are readily controlled with adrenalin. Serum disease appears in about half the patients after one to three weeks. The anti-histamine drugs are useful in prevention and treatment of both the immediate and the delayed actions. There is logic also in the practice of giving at least small daily injections of serum until it is certain that no more will have to be used. This avoids the possibility of anaphylactic reactions if serum therapy is interrupted for several days and then has to be resumed because of a relapse.

Streptomycin is the most potent destroyer of *Hemophilus influenzae* but it has unfortunate toxic reactions and is useless against the resistant strains of organisms which sometimes emerge during treatment. The chief toxic reactions of deafness and ataxia seem to be a function of the total dosage and length of administration of the drug. If large doses of streptomycin are given for a brief time, the risk of toxicity will be minimized but the major lethal action of the drug against susceptible organisms may still be utilized. There is no point in continuing its use if the presence of resistant strains has been recognized. The drug should be given intramuscularly every three hours in a daily dose of 40 mgm. per kilogram of weight. The rate of its diffusion into the spinal fluid is insufficient to maintain adequate concentrations so that a daily intrathecal dose of 25 to 50 mgm. is also required. Both routes of administration

should be terminated after five days to a week. Persistence of a mild pleocytosis is not necessarily an indication of continuing infection since streptomycin stimulates some increase in spinal fluid cell count when used intrathecally.

To summarize, the therapy which seems to offer the maximum chance for full recovery consists of (1) sulfadiazine with adequate fluid and alkalinizing agents given from the start and continued until recovery has been assured for at least a week; (2) anti-influenzal rabbit serum in initial dosage sufficient to provide an excess of circulating antibody and maintained at this level by supplementary injections as necessary; and (3) streptomycin in heavy dosage intramuscularly and intrathecally for a brief period at the onset.

Pneumococcus Meningitis

Pneumococci may reach the meninges in the same fashion as the meningococci—via the blood stream after initial entry through the upper or lower respiratory tract. Sometimes there is evidence of pneumonia or infection of the middle ear. In other instances, the pathway to the meninges appears to be more direct and not necessarily mediated by general bacteremia. Direct entry may occur through trauma or operative disturbance of the cribriform plate of the ethmoid bone, after internal extension of a necrotic focus of mastoiditis, or by extension of purulent labyrinthitis through the internal auditory meatus. When such a pathway is clinically apparent, the existence of meningitis due to some organism other than the meningococcus may be suspected. However, the clinical picture is not a reliable guide to bacteriologic differentiation and the organisms must be identified in the spinal fluid.

The spinal fluid displays the general features of increased pressure, pleocytosis, increased protein and decreased sugar described for meningococcus meningitis. Usually, however, even when the fluid is not very cloudy and the cell count is relatively low, pneumococci appear in great profusion and are easily discovered in strained smears although the inexperienced may confuse them with meningococci. Often the concentration of organisms in the initial spinal fluid is sufficient to permit immediate identification and typing through the appearance of capsular swelling in type specific serum using the Neufeld technic. When this is impossible the organisms will grow out readily upon blood agar or in blood broth under ordinary incubator conditions. Ultimate typing of pneumococci is essential to the appropriate use of specific serum.

Untreated pneumococcus meningitis is almost universally fatal. The use of sulfonamides alone, or with specific anti-pneumococci rabbit serum may save from one-third to two-thirds of patients over 2 years. Penicillin alone is about equally effective, but the results are somewhat better if both sulfadiazine and penicillin are given. Infants who survive are commonly left with permanent cerebral damage. Alexander reports complete recovery in six out of eight small infants who received combined treatment with sulfadiazine, penicillin and type specific

antipneumococcus rabbit serum. When the pneumococcus is typable and the corresponding serum can be obtained, patients at any age ought to receive all three agents.

The general principles of sulfadiazine and penicillin administration have been described above. Because of the less favorable prognosis, the level of sulfadiazine in the blood should be maintained at 20 mgm. per cent or as high as microscopic hematuria will permit. Penicillin is very effective against most strains of pneumococci in vitro and maximum doses should be administered intramuscularly with a daily intrathecal dose of 5,000 to 20,000 units, depending upon age. When type-specific rabbit serum is available, the patient should be tested for sensitivity by intradermal and conjunctival injection before administration. The usual dose for a child is 20,000 to 40,000 units given intravenously, well diluted in saline. Such serum is now scarce because the commercial houses have stopped producing it in quantity. Termination of the sulfadiazine and penicillin should be delayed for at least a week after consistently negative spinal fluid cultures are obtained. Pneumococcus meningitis is notorious in its propensity to relapse after apparent cure. Repeated examination of spinal fluid is advisable for two or three weeks after the cessation of treatment to be sure that infection has been permanently controlled.

The therapy used to control meningitis is amply sufficient for complications such as pneumonia and for the simpler varieties of upper respiratory infection. If mastoid infection demands operative intervention, it should be deferred until there is some assurance that the meningitis is responding.

Hemolytic Streptococcus and Staphylococcus Meningitis

Treatment of these less common forms of meningitis is similar to that described for pneumococcal meningitis. In both varieties the organisms are usually found on direct smear of the spinal fluid and are easily cultivated in blood broth or upon blood agar plates. Streptococcus meningitis occurs among small infants with general bacteremia, and in older children as a result of cranial extension of streptococcus mastoiditis. All strains of beta hemolytic streptococci which are pathogenic for man are highly susceptible to penicillin, which should be used as the main therapeutic agent. Transfusion for anemia and ultimate surgical attention to suppurative mastoiditis should be included in the management.

Meningitis produced by staphylococci is most commonly associated with bacteremia and metastatic abscesses. In small infants it may follow direct penetration through meningoceles or congenital dermal sinuses.

Strains of staphylococci vary so much in their sensitivity to sulfonamides and penicillin that in vitro testing of individual organisms is highly desirable in order to guide treatment. If such testing is not possible, sulfadiazine should be given to the limit of renal tolerance combined with penicillin in maximum doses intramuscularly and intrathecally.

Meningitis due to other Bacteria

The colon bacillus and other enteric organisms produce meningitis in newborn infants and may invade the spinal fluid of older children and adults during septicemia or urinary tract infections. Sulfadiazine and streptomycin are generally effective against such organisms, but each individual strain should be subjected to an in vitro test for sensitivity to the antibiotics which are available to provide the best guide for therapy. When these organisms enter the subarachnoid space directly through leaking meningoceles or the less common congenital dermal sinuses, permanent cure cannot be expected unless the portal of entry can be extirpated surgically.

Numerous other bacteria may be found in isolated cases of meningitis. Treatment and prognosis depend upon bacteriologic definition and the determination of antibiotic susceptibility in each instance.

Tuberculous meningitis presents a separate and more difficult problem. Since 1945 occasional complete cures have been reported from prolonged and vigorous streptomycin therapy given intramuscularly and intrathecally. The results are not predictable. Prolongation of life is usual, but survival is nearly always at the expense of serious neurologic residua or represents a temporary remission preceding fatal relapse. The hazards of streptomycin toxicity should not deter treatment if the diagnosis is assured.

The preceding discussion has attempted to set forth the technic of diagnosis and treatment of bacterial meningitis under optimal conditions. Bacteriologic facilities sufficient to carry out all the tests mentioned are found in relatively few hospitals and even under the best circumstances some therapeutic decisions must be made without accurate bacterial diagnosis. As soon as the lumbar puncture reveals purulent spinal fluid, immediate therapy must be considered. While the needle is still in place, 3,000 to 10,000 units of crystalline penicillin may be instilled into the spinal canal. A blood culture should be obtained rapidly thereafter and injection of parenteral sulfadiazine, penicillin, fluids and alkalinizing substances started. Such measures initiate proper therapy against meningococci, pneumococci, streptococci and staphylococci, but may be insufficient for *Hemophilus influenzae* and other gram-negative bacilli. The spinal fluid should be immediately cultured upon blood agar, blood broth and two chocolate agar plates or slants. To favour the growth of meningococci, one chocolate agar preparation and a blood culture flask should be incubated at reduced oxygen tensions in a vacuum jar or in a dessicator in which a candle is burned to exhaust the oxygen supply. If inoculation is made quickly onto warm media of this sort, there is an excellent chance that the offending organism will be recovered. If the spinal fluid smears clearly define the infecting agent, therapy can be rapidly shifted in favour of the appropriate substances. If, however, neither bacteriologic diagnosis nor clinical improvement is available within twenty-four to forty-eight hours at most, streptomycin and

anti-influenzal rabbit serum should be added to the therapeutic program, particularly if the child is under 3 years of age. Such an unscientific approach may subject the patient to unnecessary treatment, but if bacteriologic guidance cannot be obtained there is no other way to protect him against the dangers of chronic meningitis and its usual residua.

In conclusion it should be emphasized that treatment is more often made difficult and unsatisfactory by delayed diagnosis than by exceptional severity of the infection. The practitioner alone has the power to minimize this all-important obstacle to full recovery from bacterial meningitis.

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Extracts.

SPECIFIC THERAPY OF BACTERIAL INFECTIONS OF CENTRAL NERVOUS SYSTEM.

BY H. F. DOWLING ET AL.

Proper treatment of acute disease of the central nervous system depends upon early diagnosis, determination of the causative agent and selection of the appropriate drug or antibiotic.

Meningitis caused by gram negative cocci

Meningococcic meningitis. This is the commonest form of meningitis. Sulfadiazine or sulfamerazine is the drug of choice. Dose for adult, 6 gms. at once followed by one gram every four hours. Dose for child, $\frac{1}{2}$ gr. per pound of body weight immediately followed by 1 gr. per pound for each 24 hours in divided doses at four hour intervals.

Comatose patients should be given an initial intravenous dose of 0.5% solution of the sodium salt in 1/6 molar lactate or normal saline solution. Subsequent doses are given by mouth or stomach tube. Sodium bicarbonate 6 gm. at the start and 3 gm. every 4 hours should be used concomitantly in order to minimize the possibility of the formation of renal calculi. Penicillin is needed only in fulminating cases or in those who do not respond to sulfonamides within 24-48 hours. The best way to give penicillin to these cases is in doses of 10 lac units every two hours. Treatment with sulfonamide compounds and penicillin may be discontinued one week after the temperature drops if the patient is doing well in all other respect.

(ii) *Gonococcic meningitis.* It is very rare. Same treatment as for meningococcic meningitis is carried out.

Meningitis caused by gram positive cocci.

(i) *Pneumococcic meningitis.* The orthodox treatment is daily injections of 10,000-20,000 units of penicillin intrathecally plus sulfonamide and 5 lac-20 lacs units of penicillin systemically. The authors treated 22 patients with pneumococcic meningitis with doses of 10 lac units every two hours. Three patients received a single intrathecal injection of Penicillin and 12 received sulfonamides. Out of these 8 cases died.

This gives a fatality rate of 36 percent as against 62% in cases treated on the orthodox lines. Patient in coma or in extremes is given a single initial intrathecal injection as well. Sulfa-drugs are not needed with these big doses.

(ii) Meningitis caused by staphylococci and B. streptococci. 10 lac units of penicillin are given every two hours intramuscularly. No intrathecal therapy is needed.

Meningitis caused by gram Negative Rods.

(i) *Himophilus Influenzae meningitis*. Infants and young children are given streptomycin in daily doses of 1.5 g. m. in divided doses every 6 hours intramuscularly. Adults and older children are given 3 gm. per day. This should be combined with sulfadiazine. If this combined therapy shows no effect in 48 hours of treatment specific rabbit antiserum should be given in doses of 100-200 mg. of antibody nitrogen.

(ii) Meningitis caused by other gram negative bacilli as B. coli, B. pyocyaneus, Friendlanders' bacillus and aerogenes is treated on the same lines as influenzal meningitis except that specific serum is not needed.

Tuberculous Meningitis

In early study of streptomycin treatment in tuberculous meningitis there was clinical arrest in 25% of cases. But complete recovery is said to occur only in 5-10% of cases. Streptomycin combined with promizole is the ideal treatment. Streptomycin is given in intramuscular dosage of 9 mg. per kilogram of body weight per day, up to a maximum of 2 gms. at 6 hour intervals. Intrathecal doses of 1 mg. per kilogram up to a maximum of 50 mg. are also given, at first at daily intervals and after two weeks on alternate days. This treatment is continued for four months.

Cerebral Abscess. This is treated on the same lines as meningitis. If needed surgical drainage must be done.

(Abstracted from the J. A. M. A 19-3-48.)

GONORRHEA AND THE SULFONAMIDES

By E. M. C. DUNLOP

Earlier workers gave a very high percentage of cure of acute gonorrhea with sulfa drugs. King (1939) after treating 300 cases of acute gonorrhea with sulfapyridine stated that by the third week 90 per cent of the cases had no symptoms and no signs of infection.

Sommerville (1941) treated 300 cases with 2 g of sulfapyridine daily for 7 days, followed by irrigation for two weeks. After nine weeks' observation a cure rate of 92 per cent was seen, with complications only in 1.66 per cent of cases.

Laird (1942) treated 764 cases with sulfapyridine using three schemes of dosage, 6 g daily for 4 days, 4 g daily for 6 days or 6 gm daily for 3 days. He stated a cure rate of 81-91 per cent. Jefferiss and McElligot (1943) treated 219 cases with 18-20 g of sulphathiazole spread over

8 days. After three months of observation he obtained a cure rate of 90 per cent. Complication was seen in 1 per cent of cases.

Williams et al (1944) treated 540 cases for 5 days with 4.5 gm of sulphapyridine daily. 45 per cent of these were cured by one course alone a further 30 per cent were cured by the additional minor therapy soon after the course was finished. There was a relapse in 5 per cent of these with incidence of complications in 0.25 per cent by the end of three months. He suggested 25 g of sulphathiazole in five days and this treatment gave somewhat better results.

The present series consisted of 408 male patients treated with two standard courses of sulphathiazole as follows:—

- (1) 4 g daily for 7 days or
- (2) 5 g daily for 5 days.

A few patients were given 4 g daily for ten days. The tablets were taken after meals with an alkaline mixture and abundant fluids.

Tests for Cure. After the completion of treatment the patient was examined at least once a week for three weeks. Smears of morning secretions were examined. After one month prostatic massage was carried out on three separate occasions at intervals of not less than a week and the secretions were examined. At the end of three months, full clinical examination was made, prostatic secretion was examined, urethroscopic examination, Wassermann reaction and complement fixation test for gonorrhea were carried out.

Results. Only 205 patients were assessed in the above way. Of these 14.1 per cent completed satisfactory tests after one course of sulphathiazole. The treatment failed in 176 cases. The evidence of failure was the persistence or recurrence of pyuria or the development of complications. 70 out of these were given a second course of sulphathiazole. Out of these 16 passed the test for cure. In 12 cases even 3-4 courses of sulfathiazole failed. The treatment by sulphathiazole failed in 85.8 per cent of cases. In 44 per cent of cases complications developed in spite of sulfonamides. Two developed severe complications due to sulfonamide therapy. One developed acute toxic edema, the other severe vomiting and prostration. A history of one or more attacks of gonorrhea was given by 17 per cent of those cured with sulphathiazole and 30 per cent of those in which treatment failed.

(Abstracted from the B. J. V. D. June 1949.)

B. C. G. VACCINATION

KRISHNA KUMAR L.S.M.F.

Tuberculosis Clinic, Civil Hospital, Ambala City.

B.C.G. Vaccine is a vaccine containing living avirulent strain of tubercle bacilli. The vaccine is potent up to 10 days only, after which the bacilli begin to die rapidly.

B.C.G. Vaccine has appreciably reduced the morbidity and mortality from tuberculosis. The tuberculosis lesions if they occur in the vaccinated are less extensive, of shorter duration and become calcified earlier. General mortality in the vaccinated is lowered by about 50 per cent as compared with the non-vaccinated.

Selection of candidates for B.C.G. The vaccine is given only to those who are strictly "negative" to tuberculin tests.

Any one of the following tests may be used :—

(1) Those who prove negative to Pirquet test twice at intervals of a week.

(2) Those who prove negative to Mantoux test with both 0.1 and 1 mg. of old tuberculin.

(3) Those who prove negative to 0.00002 and 0.005 mg. of P.P.D.

Whatever the method chosen, the same brand of tuberculin should be employed in all groups. Whenever possible the X-ray of the chest may be taken because some negative cases may show an active focus. The newborn can be given vaccine within the first 10 days, when the question of tuberculin testing does not arise.

Methods of Vaccination.—(1) Oral. This is specially suitable for the newborn within the first ten days of life. 1 cc of freshly emulsified cultures of the B.C.G. are given on empty stomach on alternate days thrice within the first ten days of life.

(2) Subcutaneous. 1-2 cc of the emulsion containing 1,000 mg per cc is given according to age. Abscess may form which heals up.

(3) Intradermic. 0.1 cc of the vaccine containing 0.1—0.15 mg of B.C.G. culture is injected slowly into the dermis through a tuberculin syringe and a 26-gauge platinum needle. After 48 hours a sharply defined nodule appears. Sometimes the centre may ulcerate after 3-4 weeks which heals up in 4-8 weeks.

(4) Multiple Puncture Method. This is the method used now. 95 per cent of the vaccinated persons become tuberculin positive. After one year the percentage falls to 93 per cent which remains negative for 3.5 years.

Highly exposed infants should be removed from source of infection for 6 weeks before B.C.G. vaccination. The vaccinated person also should be protected against exposure to infection by tubercle bacilli until they become tuberculin positive.

Duration of Immunity.—Immunity is conferred by the oral method by the end of the second month. It is much more earlier by the

subcutaneous or intradermic method. The immunity lasts for about 5 years. It is better to repeat the vaccination at the end of the III, VII, and 15th year. Those who have been able to withstand upto the 15th year may be considered to be able to resist tuberculosis in later life. In the case of infants reared in a tubercular environment, it is better to inoculate at the end of the 1st and 3rd year. (If the child is tuberculin negative).

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B.C.G. VACCINE—ITS EFFICACY AND DISADVANTAGES

B. B. MUKHERJEE, M.B.M.R.C.P.

Efficacy of the B. C. G. vaccination may be judged from the following reports :—

(1) Hyge in Copenhagen reported an epidemic of tuberculosis in a school for girls between 12—18 years of age. Before exposure 105 were tuberculin positive, 106 were vaccinated with B. C. G. and 94 were tuberculin negative. The source of infection was a temporary teacher. The duration of exposure was 2 months only. The results of follow up for a period of three years was :—Cases which developed pulmonary tuberculosis was 41 in tuberculin negative cases as against 2 in each of the other two cases. In the tuberculin negative cases one died of tuberculosis while none in the other two groups.

(2) Wallgren in Sweden vaccinated every infant that was considered to be in danger of infection from family contact. The infantile death rate from tuberculosis fell from 8.9 per 1000 to 0.3 per thousand.

(3) Heimbeck carried out an extensive investigation on nurses during the years 1924-1935. The morbidity rate in nurses not vaccinated was 23.2 as compared to that in those vaccinated and became tuberculin positive as 1.1 and 4.9 per thousand in those vaccinated but remained tuberculin negative.

Later on in 1947 he found morbidity and mortality rate in vaccinated persons as 24.1 and 2.1 per thousand as against 141.3 and 14.6 in those unvaccinated and tuberculin negative.

(4) Similarly Rosenthal et al found very high incidence of cases of tuberculosis among the controls as compared to those vaccinated with B.C.G.

(5) Holm reported from Denmark that all tuberculin negative children in a tuberculous environment as well as the new born were vaccinated. When they become tuberculin positive they were allowed to remain in the environment. Mortality from tuberculosis among the children in tuberculous environment had been reduced to almost zero though previously the mortality was very high. Those who got tuberculosis the attack was much milder.

(6) Aranson and Palmer investigated the results of B.C.G. vaccination among the North American Indians who are comparatively more

susceptible to tuberculosis. The rate of morbidity and mortality from tuberculosis among the vaccinated was 2 and 4 per thousand as compared to 9 and 28 among the non-vaccinated. In other places also similar results were confirmed.

Difficulties and Disadvantages.—(1) B.C.G. is a live vaccine and has to be used within a fortnight of its preparation though preferably in the first week.

(2) Local Lesions. Abscess or ulceration may develop at the site of inoculation in a small number of cases. These generally heal up in the course of a few months. These drawbacks are avoided by multiple puncture method but even in this method papules may develop which may last for weeks.

(3) Infants of tuberculous mothers are to be separated from their mothers at birth until B. C. G. has made them tuberculin positive. The already exposed child must be separated for at least six weeks before vaccination.

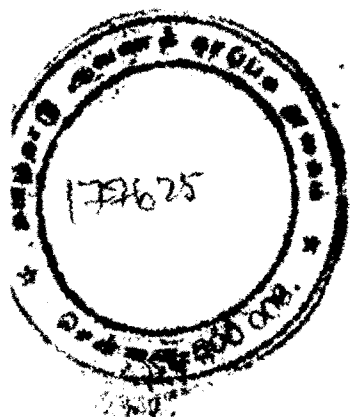
(4) Some 2-5% cases require revaccination.

(5) The immunity conferred by B. C. G. vaccination lasts from five to ten years or more. When they become again tuberculin negative vaccination must be done again. This is much more essential during the period of childhood and adolescence.

(6) Newborn. Newborn and very young infant requires large doses 0.1-0.2 mgm of B. C. G. Tuberculin testing is unnecessary as all of the newborn may be taken as tuberculin negative.

Local abscess and ulceration occur very seldom in infants. After vaccination of the newborn subsequent tuberculin testing can be done without risk by Mantoux method direct with 1 mgm.

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