CASE 4.—M.G., a man 34 years of age, was seen in November 1953 because of a slowly growing mass on the left side of his neck. The mass was first noted by the patient two months previously. There was no pain or pressure symptoms. On examination a large 4 x 4 cm. mass was felt on the left side of the neck below the angle of the mandible and partly covered by the sternomastoid muscle. Operation on November 14 revealed a large cystic mass lying over the thyrolingual trunk. The postoperative course was normal.

Pathology.—The cyst contained a thick greyish discharge. Its thin wall was lined with squamous cell epithelium in which numerous lymphocytes were scattered. Beneath the epithelium lay a lymphoid tissue chorium with germinal centres. The lympho-epithelial symbiosis characteristic of these congenital cysts and fistulæ was well demonstrated.

CASE 5.—J.P.A., a 3½-year-old boy, was referred in October 1952 because of discharging cervical sinus since birth, repeated sore throat and nasal obstruction. On examination bilateral preauricular fistulæ were noted, the right one being scarred after repeated incisions and drainage. The left fistula was a mere dimple and had never discharged. Adenoids and large infected tonsils were present. Small discharging sinuses were noted on each side of the neck, in front of the sternomastoid muscle at the level of the thyroid cartilage. The surrounding skin was excoriated.

On October 27 the tonsils and adenoids were removed and the right preauricular fistula was excised. The child was then allowed home with instructions to return in one month for excision of the cervical sinuses. The sinuses were excised at two sittings on November 25 and 27 using the stepladder incision. Both sinuses were found to extend to the pharynx without opening into it, and passed between the two carotids. The postoperative course was uneventful.

Pathology.—Sections of the fistula revealed a columnar epithelium with lymphocytic infiltration, overlying a lymphoid tissue base.

CASE 6.—T.M., a 21-year-old man, gave a history of discharging cervical sinuses since birth. His mother also presented the same anomaly. On examination, bilateral preauricular and cervical fistulæ were noted. The cervical fistulæ were somewhat lower than the level of the thyroid cartilage. A considerable skin reaction due to the continuous discharge surrounded the sinus opening. At operation on June 13, the findings were similar to those in Case 5. This patient later died from nephritis.

SUMMARY

A brief review of congenital cysts and fistulæ of the neck is made without any attempt to explain their pathogenesis. The importance of correct diagnosis and proper management, which is by total excision, is stressed.

Six cases are presented particularly to demonstrate certain histological features.

It is believed that these conditions belong in the field of otolaryngology and should be treated by the otolaryngologist. This is logical for he is better acquainted with the embryology and anatomy of this region. Furthermore, only he is capable of properly examining the different cavities of the head and neck, an examination which is so important to eliminate a possible focus or primary tumour whenever dealing with lumps in the neck.

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Case Reports

LEPTOSPIROSIS SEJROE*
WITH REPORT OF A CASE

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IN RECENT YEARS increased attention has been drawn to human leptospiral infection in North America. Before 1938 only infection with *L. icterohæmorrhagiæ* had been reported. In that year, Meyer, Eddie and Anderson-Stewart described the first case of canicola fever[1] and other reports followed.[2 etc.] More recently there have been reports of *L. pomona*,[3, 4] *L. grippotyphosa*[5] and *L. bataviæ*[6] infections. Then the organism of Fort Bragg fever, after 10 years of passage through laboratory animal hosts, was identified as *L. autumnalis*.[7] *L. ballum* and serotypes of the *pyrogenes* and *hebdomadis* groups have been reported in man from Puerto Rico.[8, 9, 10]

In Canada, several cases of Weil's disease due to *L. icterohæmorrhagiæ* have been described,[11, 12, 13] but no other leptospiral infections

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were reported until 1955 when Saint-Martin reported a case of canicola fever with meningitis.[14]

Although agglutinations against the organism have been observed in livestock herds in the U.S.A., particularly in Florida,[10] the following is thought to be the first instance of human infection with *Leptospira sejroe* reported in North America.

The patient was a 39-year-old bank manager residing in a lakeshore suburb near Montreal. He was quite well until September 27, 1954, when he developed a headache, anorexia and pains in his joints. That night he suffered from bad dreams and continued to do so on succeeding nights.

On September 28 he suffered from pains in both knees and called his physician, who found fever (102° F.) and throat redness. The patient also pointed out a skin lesion remaining from a dog bite suffered two weeks before on the right index finger. He complained of a slightly tender lump in the right axilla. The physician noted red streaks up the right arm. Neither local lesion nor streaks were very tender. The patient was given 900,000 units of penicillin intramuscularly.

On September 29, the patient seemed somewhat better, and was given 600,000 units of penicillin. That day he developed severe headache and photophobia and was quite confused during the night. On September 30, he was admitted to Queen Mary Veterans' Hospital at 10.45 a.m.

On September 12, the patient had been bitten by his 1½-year-old cocker spaniel. There was merely a superficial abrasion on the right index finger, which healed at first but then developed a vesicle just before the generalized illness began. In hospital the lesion presented as an indolent ulcer with slight remaining vesiculation.

The dog had been ill repeatedly since acquired by the patient 1½ years previously. About every four months he suffered from diarrhœa, with blood and mucus in his stools. He had been seen by several veterinarians who had diagnosed various conditions, including hepatitis. He had been treated shortly before the bite with an injection for an illness similar to the others.

When the patient's illness began, he decided that the dog should be destroyed, and this was arranged on Oct. 2. Unfortunately this was done before samples of the dog's serum or urine had been obtained. Furthermore, the dog had been dead for some time before the patient's physicians were notified and it was then thought too late for necropsy to be of any value.

The patient kept a garden, but it was not damp or swampy and not many mice had been observed about it. The dog had not been seen catching mice.

Condition on admission (Sept. 30, fourth day of illness).—Temperature was 98.2° F., rising in the afternoon to 99.8°, but subsiding then to 99° or lower for two days. Pulse rate was 95; blood pressure was 110/70 mm. Hg. Physical examination was negative except for the right hand and arm; there was a small partly healed but slightly moist lesion on the dorsum of the right index finger, with a small vesicle at its proximal end. Streaks of lymphangitis were seen running up the forearm and arm and over the lateral aspect of the right chest. A hard gland the size of a plum was felt in the right axilla. There was tenderness but not the marked tenderness of pyogenic adenitis. He was complaining of constant headache and had periods of mental confusion. There was moderate photophobia. No retinal or conjunctival changes were noted.

Course: He was started on rather small doses of antibiotics, 300,000 units of penicillin and 0.5 g. streptomycin, each twice daily. On this medication his temperature seemed well controlled, but he still suffered from mental confusion at night. On Oct. 3, he complained of severe headache and in the afternoon the temperature rose to 102° F. There was no definite neck rigidity, but Kernig's sign was moderately positive.

Lumbar puncture was done on Oct. 4. Pressure was 280 mm. H_2O; fluid was clear and flowed freely. There were 234 neutrophils and 416 monocytes per cu. mm. The level of total proteins was 94 mg. %, sugar 76 mg. % and chlorides 679 mg. % (as NaCl). Dark-field examination of the spinal fluid revealed no spirochætes.

Antibiotic dosage was increased on Oct. 4 to crystalline penicillin, one million units every three hours, and streptomycin, 0.5 g. twice a day. At the same time triple sulphonamides were started. From this time the temperature fell, the symptoms abated and mental confusion ceased. The penicillin was continued till Oct. 10 (total 45,400,000 units in hospital), the streptomycin and sulphonamides till Oct. 8. He was discharged quite well on Oct. 10, and returned to work on Nov. 1. From Dec. 5 to 17 and occasionally subsequently he suffered from vertigo. He was seen for this in the neurology clinic on Dec. 21. Neurological examination was negative and the condition was thought to be a postural vertigo. Otherwise, he has had no significant illness to date (May 1955).

Other Laboratory Data: Cultures of blood, urine, C.S.F. and a swab of the lesion on the right index finger all yielded no growth of bacteria, leptospira or other organisms. The cultures were put up on routine media; the C.S.F. was also cultured on Fildes' medium and cysteine agar.

Dark-field examinations of blood, C.S.F., urine and material from finger lesion and also stained smears were all negative.

Animal inoculation (guinea pig, rabbit and mouse): blood smears, tissue cultures and animal autopsy material were all negative.

Urinalysis: Negative on admission. Developed 17 mg. % of albumin on Oct. 3, with 15 to 20 white cells, occasional red cells and rare small granular casts. Albumin level rose to 37 mg. % on Oct. 4, but no albumin or casts were found on Oct. 6 or thereafter.

Hæmogram: Sept. 30: white cells 7,700; differential: neutrophils 88% (mature cells 78%, young cells 10%, immature cells 0%), lymphocytes 9%, monocytes 3%; hæmoglobin 14.7 g. %.

Oct. 4: white cells 11,100; differential: neutrophils 77% (mature cells 75%, young cells 2%, immature cells 0%), lymphocytes 17%, monocytes 6%; hæmoglobin 12.9 g. %; erythrocyte sedimentation rate 92 (Westergren).

Oct. 12: white cells 6,800 (N. 64%, L. 35%, E. 1%); Hb. 14.2 g. %; E.S.R. 25.

Serology: VDRL exclusion test negative.

C.S.F. Wassermann: anti-complementary.

Blood Chemistry: Oct. 4: blood urea 18.1 mg. %, bilirubin 0.5 mg. %; cephalin cholesterol flocculation negative. Oct. 5: glucose (AC) 86 mg. %, (PC) 176 mg. %. Oct. 12: blood urea 13.3 mg. %; bilirubin 0.5 mg. %, bromsulphalein test: 2.4% of dye retention in ½ hour.

Agglutinations.—Serum samples were submitted to Dr. R. D. Stuart, Professor of Bacteriology at the University of Alberta, who kindly performed the leptospiral agglutinations. Each serum was tested against the following leptospiral sero-types: *autumnalis, ballum, bataviæ, bovis, canicola, grippo-typhosa, hebdomadis, hyos, icterohæmorrhagiæ, pomona, pyrogenes and saxkœbing.*

On Oct. 4, 1954, the serum titre to *L. sejroe* was 1/300 (weak 1/1,000), on Oct. 12, 1/1,000, on Oct. 19, 1/300, and on May 19, 1955, 1/10 (trace 1/30). Low titre cross-agglutinations (1/10 to 1/100) to *L. grippo-typhosa, L. hebdomadis* and *L. bovis* were demonstrated in certain of these sera, but in each case *L. sejroe* seemed to be the only significant agglutination.

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DISCUSSION

Leptospira sejroe was recognized as a separate type by Borg-Petersen and Christensen in Denmark.[15-17] In 1936-37 some indications were given that there might be a new type from serological reactions on routine specimens.[18] Finally, in November 1937, a new type of leptospira was isolated from the blood of a young fisherman, on the little island of Sejroe near Seeland, who was suffering from an acute illness characterized by sudden onset of fever, headache, vomiting, muscular pains, herpes labialis, mental disturbances and slightly increased C.S.F. pressure, but no increase in cells. The spirochætes showed cross-agglutinations with other strains, notably *L. hebdomadis*, but differed to a certain extent from all of them, so that it was considered a distinct strain and was named after the island on which the first patient resided.

Borg-Petersen and Christensen described 10 cases in 1939, in one of which there were jaundice and severe neurological effects; this case was also reported separately by Mortensen.[19] In 1949 Borg-Petersen reported 414 cases diagnosed at the State Serum Institute in Copenhagen.[18] Nielsen and Hertel described 29 cases seen in Denmark from 1937 to 1943 with particular attention to clinical detail.[20]

These reports tend to group cases as "leptospirosis sejroe" although some appeared to be due to *L. saxkœbing*, a closely allied type described by Borg-Petersen in 1944; still others appeared to be due to infection with a third strain whose status has not been clearly established.

Mino described the disease in Italy[21] and Babudieri has written at length about leptospirosis in the Italian rice fields, some of it due to *L. sejroe*.[22] Other reports have been received from Germany, Czechoslovakia, Switzerland[23, 24] and Yugoslavia.[25] Buckland and Stuart reported a case in a British soldier in N.W. Europe.[26] As noted previously, the presence of animal infection in the U.S.A. has been suggested by agglutination reactions.[10]

The disease occurs about equally among males and females. It is rare below the age of 14. The onset is sudden with high fever (104° to 104.8° F.) lasting 9 to 10 days. Headache occurs in all cases and is typically aggravated by sitting up. Vomiting, shivering fits, myalgia and injection of episcleral vessels occur in 50% of cases or more. Mental slowing and amnesia are common. Meningeal symptoms were seen in 25% of Nielsen and Hertel's cases. Five of their cases showed pathological changes in the cerebrospinal fluid with a rise in albumin, globulin and cells, chiefly lymphocytes. They describe two cases with disturbances of cardiac rhythm doubtfully due to specific effects of the disease. Redness of the fauces and exanthems of varying type have been described. Jaundice was observed in 56 (13.5%) of 414 cases of *L. sejroe* and similar infections recorded in Denmark up to 1948.[18] Peripheral nerve lesions occurred in 25% of Nielsen and Hertel's cases. Two of these patients, one being also reported by Mortensen, suffered from paralysis of the lower limbs.[19, 20]

There is no typical white cell count. The sedimentation rate is usually elevated. Albuminuria is common, and in some cases red blood cells and granular casts are found.

Leptospira have been isolated from the blood in a small proportion of cases from the second to seventh days of illness. In a few cases the organisms have been found in the urine about the 21st day of disease. Serological reactions develop about the beginning of the second week. Agglutination of leptospira by the patient's serum usually occurs to a titre of 1/300, but in some cases to 1/1,000.

The disease usually runs a benign course but deaths have been reported. Borg-Petersen reported three deaths (0.7%) in the 414 cases of the *sejroe* group up to 1948.[18] Babudieri implies that the disease is more severe in Italy than in Denmark and that fatal cases sometimes occur.[21]

The main reservoir of the infection in Denmark is the harvest mouse *(Mus musculus spicilegus)*. It is also found in the woodmouse *(Apodemus sylvaticus)*. Petersen found the organism in the kidneys of a mouse from a farm where a human case of *L. sejroe* had occurred. Case incidence in humans shows a rise in August for males and in September for females. This is presumably because the males become infected in the fields at harvest time; the mice then follow the crops to the barns, and hence the later occurrence of cases among females.[18] Practically all cases occur among the rural population.

Besides occurring in mice, the disease has been found in swine, horses and cattle.[28] Steigner[27] reports the case of a sick dog with a positive agglutination for *L. sejroe*. The dog de-

veloped nephritis and died. Necropsy was refused.

Infection usually occurs indirectly. The animal carrier excretes leptospira in the urine, this contaminating mud or water. The organisms then enter the human body through the skin or mucous membranes. Usually infection occurs through abrasions, but entry can take place through intact skin if it is softened by prolonged soaking. Infection may also occur from the saliva of an infected dog.[22]

In the present case, no proof of etiology was obtained by smear or culture of leptospira from either the patient or an animal source of infection. It is possible that no growth was obtained in the cultures from the patient because of a suppressive effect of antibiotics. However, the typical clinical course and the agglutination reactions give reasonable grounds for the diagnosis. Circumstantial evidence tends to incriminate the dog as the source of infection. The dog, in turn, probably acquired the infection in some way from a murine source. This leads to the conclusion that *L. sejroe* and other lesser known leptospira infect their usual hosts in Canada as in other countries. It is also possible that some cases of undiagnosed fever and "aseptic" meningitis in human subjects may be due to leptospiral infections. Consideration of this diagnosis by attending physicians may lead to confirmation by culture or agglutination.

TREATMENT

Most of the studies on treatment of the leptospiroses have been in reference to *L. icterohæmorrhagiæ*. In the earlier years arsenic, bismuth and sulphonamides were used with little effect. Abroad, antisera were used but there were great practical difficulties caused by delay in type diagnosis. Patterson in Hawaii[28] found that transfusions of blood from recovered cases had a prompt effect.

Several scattered reports have appeared to indicate efficiency of penicillin and other antibiotics. Bulmer,[29] Patterson,[28] and Suchett-Kaye[30] all reported the efficacy of penicillin if given early in the disease. In Reid and Reed's case[13] improvement seemed to follow institution of penicillin therapy; streptomycin was used in the latter part of the disease. There have been reports also of an apparent effect from chlortetracycline[31, 32] and oxytetracycline.[33]

Meyer and Brunner[34] discuss effects of antibiotics in dogs. They conclude that penicillin is effective in the first week of disease, but fails to eradicate the organisms after they have become established in the renal tubules. Streptomycin is also effective in the early stages. They investigated treatment of the carrier state with streptomycin and considered it effective.

On the other hand, in a survey based on 67 cases of infection with various leptospira in Puerto Rico, Hall *et al.*[35] studied the effect of five antibiotics and concluded that none was particularly effective. However, their daily dose of penicillin was 550,000 units and the mean total dosage was 6.5 million units. In the present case of *L. sejroe*, at such dosage levels the disease was not controlled; but improvement seemed immediate when the dose was increased to eight million units daily. The interpretation of this response is complicated by the coincidental administration of streptomycin and sulphonamides, either of which may have had some effect in controlling the infection.

SUMMARY

1. A case is reported of fever, meningitis, mental disturbances and mild transient nephritis.

2. Immunological reactions suggest that the condition was due to infection with *Leptospira sejroe*.

3. Circumstantial evidence suggests that the infection was contracted through a dog bite.

4. The history, clinical nature and geographical incidence of the disease are reviewed. This is thought to be the first human case in North America.

5. The disease appeared uncontrolled by lower dosages of penicillin but improvement seemed to begin when the dose was increased to eight million units daily.

6. It is suggested that some cases in Canada of fever or meningitis of doubtful etiology may be caused by the lesser known leptospira.

Thanks are expressed to Dr. Hugh Starkey, Chief of Laboratory Services, Queen Mary Veterans' Hospital, and to Professor R. D. Stuart of the University of Alberta, both of whom reviewed the early draft of this report and offered valuable suggestions. Dr. Starkey was the first to suggest the possibility of a spirochætal infection causing the patient's fever.

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MASSIVE NECROSIS OF THE LIVER DUE TO TRICHLORETHYLENE*

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TRICHLORETHYLENE is widely used in industry as a degreasing fluid, dry-cleaning agent and solvent. It has largely replaced other chlorinated hydrocarbons previously used for these purposes, such as carbon tetrachloride and tetrachlorethane, because it is much less toxic.

The most marked effects of trichlorethylene are on the nervous system, and indeed it is widely used as an anæsthetic. Industrial poisoning has resulted in giddiness, ataxia, cranial nerve palsies, polyneuritis, loss of consciousness and death due to respiratory failure, cardiac arrest or ventricular fibrillation. Several standard textbooks and monographs state that trichlorethylene is not toxic to the liver.[1-5] However, a search of the literature reveals that death due to massive hepatic necrosis has been produced by this agent.[6-13] The purpose of this report is to emphasize the danger of liver damage from trichlorethylene by presenting the case of a man who died of massive hepatic necrosis following exposure to this solvent.

E.F., male, age 37, consulted one of us (G.W.H.) on August 7, 1954. He had felt tired for several months but since Aug. 1 his fatigue had increased considerably and in addition he had nausea and loss of appetite. The patient was a chemist in a plant where trichlorethylene was used as a solvent. On July 31, he had supervised the preparation of the solvent mixture and had been exposed to trichlorethylene vapour for a period of 2½ hours. He stated further that he had always previously worn a mask for this work, but on July 31 he could not find a filter for his mask and so carried on without it.

He was a thin man with a slight icteric tint to his scleræ. The blood pressure was 90/70 mm. Hg. The liver was not enlarged or tender. There were no abnormal neurological signs and the rest of the physical examination was negative. Admission to hospital was recommended.

When he was admitted on August 10, the jaundice had increased but the physical findings were unchanged. The fasting blood sugar (87 mg. per 100 ml.), blood urea nitrogen (14 mg. per 100 ml.) and serum alkaline phosphatase (7 units) were normal. Serum bilirubin was 2.4 mg. per 100 ml. and the cephalin flocculation reaction was strongly positive. The urine specific gravity was 1.019, there was no albumin or glucose, and microscopic examination did not reveal any cells or casts. A diagnosis of toxic hepatitis due to trichlorethylene was made and he was given a high carbohydrate, high protein diet, supplemented by intravenous glucose.

During the next four weeks the jaundice increased and his general condition became worse. On August 28, spider nævi and ascites were noted. On September 2, he became confused and lapsed into coma. He was now deeply jaundiced and died three days later. His temperature had been normal throughout but rose to 100.2° F on the last day.

The prominent post-mortem finding was acute massive necrosis of the liver. The liver weighed only 1,070 grams. The left lobe was almost totally necrotic but the right lobe was much less involved. There was no productive fibrosis. Additional findings were ascites, bilateral pleural effusion, acute pulmonary congestion and biliary nephrosis.

There is no doubt that this patient was exposed to trichlorethylene. He spent most of his working time performing analytical assays of pulp and paper. This did not involve the use of chlorinated hydrocarbons or other liver poisons. Contact with trichlorethylene occurred during the preparation of a solution used for processing paper, 1000 lb. of trichlorethylene being piped

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