far (as shown in Fig. 2) was entirely due to the capillary action of the silk, aided by gravity :—

	Oct. 14th.	Oct. 27th.
	Inches.	Inches.
Circumference at mid-tarsal joint ...	14½	11½
„ above ankle	21¾	13½
„ of leg below knee ...	25¾	17
„ at knee-joint... ..	20	16¾
„ of thigh 9 inches } above knee	24¾	21¼

(Second operation of lymphangioplasty on Oct. 17th, 1908.)

Since about Nov. 12th the patient has been allowed to get up and to walk about for several hours daily, and an elastic bandage has been applied to the limb. In this way a still further diminution in the size of the limb has been obtained. The tissues are now abnormally softened and lax, hanging in folds about the bones, and the thickened skin has become thin and supple.

In conclusion, my best thanks are owing to Mr. Foulerton and to his assistants Mr. W. T. Hillier and Miss H. K. Whittingham for undertaking the bacterial investigations without which the case could not have been brought to a successful issue, to Dr. H. P. Noble who gave the anæsthetic, to Mr. P. J. Chissell and Dr. D. Ligat who as house surgeons successively had charge of the case, and to the nursing staff of Bischoffsheim Ward who were responsible for the herculean task of cleansing the thickened skin.

I believe that Mr. Foulerton proposes to publish later a detailed account of the case in its bacteriological aspects.

New Cavendish-street.

Clinical Notes :

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

FAMILY TENDENCY TO RELAPSE IN SCARLET FEVER, WITH NOTES ON FOUR CASES.

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THE occurrence of relapse in cases of scarlet fever is an event which receives but scant attention in the ordinary text-books. In view of this fact a note on four cases of relapse recently observed in the London Fever Hospital may not be without interest, and more particularly as all four patients were members of one and the same family—two brothers and two sisters.

CASE 1.—The patient, a boy, aged 11 years, was admitted to hospital suffering from a typical attack of scarlet fever on Oct. 10th, 1907, the fourth day of his illness, with a history of sore throat on the 7th and headache and rash on the 8th. Examination on admission showed the presence of a widely spread fading punctate erythema, injected and swollen tonsils and fauces, enlarged and tender cervical glands, and a peeling tongue. The temperature, which was 99·5° F. on admission, became normal on the 15th, on which day typical “pin-point” desquamation appeared on the neck. From this time onwards until Nov. 10th the patient had a normal convalescence; peeling was noted on the hands on Oct. 26th and on the feet on the 29th. On Nov. 10th the boy did not feel well, he vomited two or three times, and the temperature rose to 100°. On the next morning the temperature was 101°, the boy complained of sore throat, and a few hours afterwards a punctate erythema developed on the chest and slowly spread over the whole body and limbs. The fauces and tonsils were injected and much swollen, but there was no adenitis. Two days later a second desquamation began and followed the usual course. The second attack was more severe than the first, for it was attended with well-marked secondary adenitis and rheumatism. The patient was discharged well on Dec. 14th.

CASE 2.—The patient, a boy, aged six years, was sent to

hospital on Oct. 10th, 1907, suffering from a severe attack of scarlet fever, with a history of vomiting, sore throat, and rash on the day of admission. The history of contact with his brother, the patient in Case 1, who was admitted on the same day with symptoms commencing three days previously, sufficiently indicated the source of infection. The patient exhibited a universal punctate erythema, circum-oral pallor, severe adenitis and angina, and a typical so-called “straw-berry” tongue. Desquamation began on the face and neck on the 15th and was noted on the hands on the 30th. Three days later the patient complained of sore throat, the pulse went up to 130, but the temperature was only raised to 99° F. Examination showed the presence of a punctate erythematous rash and a moderate degree of swelling and injection of the throat. A second desquamation did not take place, the second attack was less severe than the first, and the boy was discharged well on Dec. 14th.

CASE 3.—A sister of the two patients whose cases have been related, aged five years, was admitted to hospital on Jan. 10th, 1908, suffering from a well-defined attack of scarlet fever. There was a history of sore throat and vomiting on the 7th, with rash on the 10th. On admission the rash was definite; the fauces, tonsils, and cervical glands were moderately affected. A few days after admission peeling began and the patient remained quite well until Feb. 3rd. On that day vomiting took place and the girl was found to have a well-marked punctate erythema on the body and limbs and a moderately inflamed throat. The temperature was 99·5° F., but it reached 100·5° two days later, and then fell to normal on the 10th. A second desquamation began on the 6th and the second attack proved to be mild in character. The patient was discharged well on March 5th, 1908.

CASE 4.—The patient was another sister, aged ten years, who had been in contact with the subject of Case 3. She complained of sore throat and headache on Jan. 14th, 1908, and the rash was first noticed on the 17th. She was admitted to hospital on the same day with a moderate but well-defined attack of scarlet fever, showing in fact a peeling tongue at the time of admission. Desquamation appeared and proceeded in the usual way, and the child was quite well until the morning of Feb. 4th, when she complained of sore throat and was sick. The temperature was 101·5° F. In the evening a characteristic rash appeared, but this was not followed by a second desquamation. The secondary attack was more severe than the primary, being attended with troublesome adenitis. The patient was discharged well on March 5th.

It may be pointed out that three of these cases (Cases 1, 2, and 4) illustrate the complementary nature of the primary and secondary attacks. In many cases of scarlatinal relapse the secondary attack is severe if the primary has been mild and *vice versa*. An inquiry failed to elicit a history of similar relapses in the case of the parents or members of collateral branches of the family.

I have to thank Dr. Sidney P. Phillips and Dr. William Hunter, under whose care the patients were treated, for permission to report the cases.

London Fever Hospital.

A NOTE ON A CASE OF CEREBRAL VENOUS THROMBOSIS.

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THE following note may be of interest in connexion with Dr. C. O. Hawthorne’s article upon thrombosis and its relation to the cerebral and ocular complications of anæmia which appeared in THE LANCET recently.¹

About ten years ago I was called to treat a mill girl of about 20 years of age. Her chief complaint was vertical headache which appeared to be of the clavus variety. She had mistiness and dimness of vision at times. She appeared to be slightly anæmic, but no accurate determination was made of this. However, she looked anæmic enough to cause me to treat her for chlorosis. She had dysmenorrhœa and dreaded her periods. She remained at her work of weaving, and one day she lost consciousness at the loom and fell backwards and struck the back of her

¹ THE LANCET, Sept. 19th, 1908, p. 857.

head on the floor. She was taken home and on the next day she returned to work. I found no objective signs of disease with the exception of the anæmia, and there were many girls with apparently as much who did not complain. The fundus oculi was normal, as was the pupillary light reflex; the pupils were somewhat larger than usual; there was no reason to suspect syphilis. The headaches persisted, and one day a week after her fall, whilst she was sitting at a table, she gave a loud and fearful scream and fell from her chair to the floor unconscious. I arrived within half an hour and found the girl in convulsions on the floor where she fell. She was unconscious, with fixed and staring eyes. She had passed fæces into her clothes. The conjunctival reflex was almost gone and soon disappeared. Gradually as I watched her the pupils became more and more widely dilated, the breathing more and more stertorous, the convulsions at longer intervals, and she died about two hours from the commencement of her attack. My colleague and myself were at a loss to account for this sudden death of a young girl who, with the exception of the before-mentioned symptoms, had been healthy and who had been at work on the day of her death.

After much trouble I obtained consent to carry out a post-mortem examination limited to the brain alone. On removing the skull-cap the brain was found to be adherent to the dura mater over an area of about one inch square, situated at the junction of the posterior and middle thirds of the superior longitudinal sinus. Otherwise the meninges were normal. On removing the entire brain there was nothing else abnormal to be seen on the surface. I then sliced the brain horizontally from above downwards. No abnormality was met with until the lateral ventricles were opened. These were found to be distended with about two ounces of dark clotted venous blood; the iter and the fourth ventricle were full of the same kind of blood. On examining the velum interpositum it was found that the veins of Galen were full of soft blood clot. The torcular Herophili and the longitudinal and lateral sinuses were not filled with blood clot, but were fairly empty. No other abnormalities were found in the skull or brain, but the post-mortem examination had to be somewhat hurriedly concluded on account of the clamouring of the friends who wished to bury the body. There was no history of ear or nose suppuration.

This case was unique in my experience and in that of my colleague, nor have I met with a similar case since, although I have been on the look out for one. I think that such cases must be rare, and I publish it in the hope that others who have met with similar cases may be induced to publish them. But whilst such cases are infrequent their occurrence makes it practically certain that there must be others of less severity: cases where death does not take place; cases which may very likely be protean in their symptomatology and which may at present pass without recognition.

I have no theory to account for the sudden decease of a fairly vigorous young girl from profuse venous hæmorrhage into the lateral ventricles, due, apparently, to thrombosis of the veins of Galen. The subject of thrombosis is not understood by physiologists and pathologists. When it is such cases will probably be explained.

Jullundur, Punjab.

NOTE ON A CURIOUS CASE OF LIGHTNING STROKE.

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A CAPE "boy," aged 28 years, was taking a mule out of a cart in an open yard when, after a frightful flash of lightning, both "boy" and mule were struck to the ground. The Cape "boy" was unconscious and remained so for three days and then died. The mule was killed outright, sustaining a fractured neck. On examination of the native there was a burn on the right arm, with fracture of the humerus; also another burn over the dorsal spine, with fracture of the vertebra. A post-mortem examination held eight hours after death revealed the following: Fracture of the humerus and spine, lungs congested posteriorly, heart empty, no clots, blood thin, abdominal organs and intestines opposite seat of fractured spine deeply congested and purple as regards the bowel, rigor mortis absent, and no bruises or marks on the body, with the exception of the two burns. When it is taken into consideration that the accident occurred in an

open space, where no heavy body was likely to strike either the "boy" or the mule, the question arises, Is it possible for lightning stroke to cause such violent contortions or contractions of the muscles as to break the said bones?¹

Krugersdorp.

Medical Societies.

ROYAL SOCIETY OF MEDICINE.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Discussion upon Whooping-cough.

At a meeting of this section held on Dec. 18th, 1908,

Dr. J. PORTER PARKINSON, in opening a discussion upon Whooping-cough, pointed out that in England the death-rate from this disease had diminished of late years, from 527 in the years 1861-70 to 450 in the years 1881-90, and in 1895 to 316. It was more fatal in Scotland than in England and less so in Ireland. The contagion must be a specific organism, several of which had been described, but the real one appeared not yet to have been isolated with any certainty. The infection was conveyed by the sputum, which was said to be most virulent during the catarrhal and early paroxysmal stages. The virus remained active after drying for at least several weeks, as had been proved by epidemics arising on board ship where no other source was probable. One-half of the cases occurred during the first two years of life, and it was not infrequent during the first six months of life, showing a stronger tendency to attack the young than any other infectious disease. Dr. Parkinson next described the case of a child, 12 days old, whose mother was suffering from the disease at the time when the child was born. There was, he said, a leucocytosis, especially in the paroxysmal stage. It was easy to overlook the primary disease when bronchitis or pneumonia had supervened, as the whoop, under these circumstances, frequently ceased. The complications, including vesicular and interstitial emphysema, acute bronchiectasis, glottic spasm, polyneuritis, papillitis, albuminuria, and post-nasal catarrh, in addition to those commonly observed, were next referred to. Two-thirds of the deaths from whooping-cough occurred during the first year of life, chiefly from lung complications. Paroxysmal coughs, which might be mistaken for whooping-cough, were mentioned. A short survey was then given of the various methods of treatment, including the diet and various drugs, and bromoform was recommended; it should, however, be dispensed in solution one drop to an ounce of water with a few drops of alcohol, from one to six drachms being given in a dose.

Dr. L. G. GUTHRIE, in referring to the history of whooping-cough, mentioned the term "chin-cough," still used in some parts of the country, as being derived from the name "la quintain" or "tussis quintana," by which the disease was known in Paris in 1578, the paroxysms being believed to occur every five hours. He referred to the diagnosis from the paroxysmal cough of influenza and of enlarged bronchial glands. He believed the cough, depending upon enlarged glands, commonly occurred in children who had already had whooping-cough. He had witnessed one death after a paroxysm in a boy, aged three years, who died in the waiting-room of the out-patient department. He thought cerebral hæmorrhage was very rare. Bromide of potassium and belladonna were his favourite drugs for relieving the symptoms.

Dr. J. WALTER CARR said that the diagnosis in cases of doubt lay between enlarged bronchial glands, influenza, and bronchiectasis. The cough with dilated tubes closely simulated that of whooping-cough, though the history and physical examination usually made the diagnosis clear. In bronchitis the cough just stopped short of the whoop. As regarded prognosis, early manifestations were no guide to the subsequent severity of the disease. In treatment he regarded pure fresh air at a temperature of about 60° F. as very important. If the child's temperature was 100° or over the

¹ In a leading article on p. 46, we discuss this question.