No. 18 or a No. 20 evacuating cannula. Most surgeons who have done much stone work in India will, I think, bear me out when I state that it is but comparatively seldom that a No. 20 evacuating cannula can be readily passed into the bladder of a Hindu. Then how can we account for the great measure of success achieved by the skilled litholapaxist in India, if it be not due to the greater tolerance of operative interference by the native of India as compared to that of the native of Europe? The explanation is very simple. The main reason why litholapaxy is more successful in India than in Europe is that, as a general rule, it is done more thoroughly and more skilfully in the former than in the latter. And the reason why it is done more skilfully in India than in Europe is explained by the frequently repeated opportunities which surgeons in India enjoy of practising this operation. But we surgeons who have passed many years of our lives in the calculous districts of India, must not plume ourselves because we happen to be very expert at working with the lithotrite, and we need not be very proud because we are able to crush a stone in the bladder skilfully and with little danger to our patients. For we have done no more than take advantage of the unrivalled opportunities afforded us in this special department of operative surgery.

Now, if the great success obtained by surgeons in India in litholapaxy depended solely on the peculiarly favourable conditions of their patients, then the novice in litholapaxy in India should obtain as good results as a master of the craft. But this is notoriously not the case. I may cite three examples out of many to illustrate this point. In the July number of the *Indian Medical Gazette* for 1892 I gave an analysis of the first 500 litholapaxies performed at the Indore Hospital, Central India, on patients at all ages. Deducting the litholapaxies in boys there were 286 operations in males between fifteen and ninety years of age, with 11 deaths, or with a death-rate of 3·84 per cent. But when I come to examine this mortality more closely I find that 7 out of the 11 deaths occurred among our first 52 litholapaxies, leaving a balance of 234 litholapaxies with only 4 deaths. The death-rate among the first 52 litholapaxies was 13·4 per cent., while it was only 1·7 per cent. among the last 234. There was no marked difference in the ages, caste, and general health conditions of the patients who constituted the first division of 52 and the second division of 234, and the average weight and composition of calculi removed from both sections of patients did not differ materially. A death-rate of 13·4 per cent. in 52 litholapaxies was certainly not a very brilliant result; but a death-rate of 1·7 per cent. in 234 litholapaxies left little to be desired. The second instance refers to the work done at Hyderabad, Sindh. Brigade-Surgeon-Lieutenant-Colonel Forbes Keith has placed at my disposal a tabular statement of all operations performed by him for stone in the bladder at Hyderabad during five and a half years ending May, 1894. The number of operations was 1933. Eliminating all the lateral lithotomies in boys and men and 57 litholapaxies in females, I find that he performed 676 litholapaxies in adult males with 22 deaths, a rate of mortality of 3·2 per cent.—a result highly satisfactory. He, however, lost 16 patients in his first 111 cases, a death-rate of 14·4 per cent. In his last 565 operations he lost but 6 patients, a death-rate of only 1·06 per cent. As in the case at Indore, there was no marked difference in the ages, caste, and general health conditions of the patients, or in the average weight and composition of the calculi removed in his first 111 and his last 565 litholapaxies. The third instance refers to Surgeon-Lieutenant-Colonel Freyer's experience. In reply to my questions on this subject he writes: "I find that amongst my first 50 litholapaxies there were 3 deaths, or 6 per cent.; but it must be remembered that my earlier cases were selected, the worst cases being subjected to lithotomy. My results have gradually improved as I have gained experience of the operation, so that amongst my last 300 litholapaxies there were only 3 deaths, or 1 per cent., though I rarely use the knife now. As I gain experience of Bigelow's operation amongst Europeans I am gradually coming to the conclusion that the European stands the operation better than the native of India." Then how can we account for the vast difference in the mortality which followed our first and last series of litholapaxies? The answer is easy. We were learning our work at the beginning, and as soon as we had gained experience and confidence in performing the operation, and had learned how to use the lithotrite with skill and precision our success increased most rapidly. And

so, after all, it comes to this, that the bladder and urinary organs of the natives of India, like those of the natives of Europe, become tolerant of the use of the lithotrite when the surgeon has learned the art of manipulating it with skill and precision.

It is, perhaps, a somewhat ungrateful task to dispel a fondly cherished and consoling illusion of the unskilled litholapaxist in Europe, when he ascribes the great success achieved by his Indian *confrère* in crushing vesical calculi to conditions in the latter's patients which have only a mythical existence. The truth, however, must be told, disagreeable as it often is to hear it; and now, I trust, we shall read and hear no more about the tolerance of the Indian bladder, and of those mythical Indian patients who, though suffering during a long series of years from stone in the bladder, are lucky enough to retain their kidney power unimpaired. In few operations in the whole range of surgery are constant practice and the personal factor of the operator of greater importance than in litholapaxy. No surgeon need hope to acquire a practical familiarity with the lithotrite until he has performed between fifty and a hundred litholapaxies, and even then, he has still much to learn. In the beginning he may be so fortunate as to get a run of a dozen simple and straightforward cases which may not tax his powers of manipulation too severely, but the difficult cases are sure to turn up sooner or later. Then how, it will be asked, is the general surgeon in Europe, who only meets with cases of stone in the bladder at fitful intervals in his professional career, ever to acquire a practical familiarity with the lithotrite? Again the answer to this question is simple. The general surgeon in England, and throughout Europe, as a matter of fact, seldom, if ever, acquires this familiarity, and he passes away or retires from practice without ever having acquired it. The span of active professional life is too short to learn the art of performing litholapaxy with dexterity and safety to one's patients unless a surgeon has been so favourably placed as to be afforded frequently repeated opportunities of working with the lithotrite. The fact of the matter is that in England and throughout Europe, there are not enough cases of stone in the bladder to go round among the general surgeons, and so if any young surgeon cherishes the ambition of developing into a skilled litholapaxist before middle-age shall overtake him he should go to Egypt or India to learn this special operation. With this object in view, a year spent in the hospital at Hyderabad, Sindh, would be worth a lifetime of practice in England.

Cresswell-gardens.

A CASE OF MUMPS, WITH CEREBRAL SYMPTOMS AND HIGH TEMPERATURE;

WITH A REVIEW OF SOME OTHER CASES PREVIOUSLY RECORDED.

By F. LUCAS BENHAM, M.D., M.R.C.P. LOND.

THE following are the notes of a case of some interest which I lately attended in conjunction with Mr. H. G. Brigham.

The patient was a lad, barely sixteen years of age, who was at school a little way out of London, where there had recently been a few cases of mumps. On Nov. 1st, 1896, an attack of mumps set in, with ordinary symptoms. The left parotid gland swelled first, then the other became swollen before the first had subsided. He was kept in bed. On the 2nd the parotid glands were still swollen. He was kept in bed part of the day. Some pain was felt in the left testis. On the 3rd he seemed much better and was allowed to go home to London, but had to walk more than a mile to the station. On the 4th he was not so well. The testis was distinctly swollen. He was feverish, but had no rigor or vomiting. The temperature was 104·7° F. in the evening. On the 5th he was still kept in bed and was very feverish. The temperature was 103°. There was no swelling over the parotid glands. The left testis was somewhat swollen and tender and the scrotum was red. There was a fine papular rash, something like that of scarlet fever, on the outer aspect of the left forearm. The buttocks were slightly erythematous,

but there was no distinct rash elsewhere. The tongue was moist and coated with a thick yellowish fur. There was no swelling or redness of the tonsils or throat. There was some headache, but the patient was cheerful and wished to get up. The pulse was not very frequent. He was still kept in bed. He was ordered three grains of calomel and a diaphoretic mixture. The temperature was 104.8° in the evening. On the 6th the temperature was 105° and the pulse 120. The tongue was moist and thickly coated; the velum palati looked raw and dry and was partially covered with tenacious mucus, but the throat was not red or sore, and the tonsils were not swollen. The rash had quite disappeared. Tache cérébrale was visible on the abdomen. He was thirsty. He had been sick on the previous day after taking the powder. The bowels had been opened two or three times. The eyes were natural and the pupils normal. He had been delirious, wandering, and talkative, asking for beer, &c., at night. The following mixture was ordered three times a day; half a grain of potassio-tartrate of antimony, fifteen grains of carbonate of magnesia, half a drachm of sulphate of magnesia, and peppermint water to one ounce. He was also to be sponged, when hot, with vinegar and water; and he was to have a milk diet. At 9.30 P.M. he had been dull, drowsy, and heavy, almost in a state of stupor, from which he could be momentarily roused without much trouble, all day. The temperature was 103.4° and the pulse 108 and regular. He had vomited two or three times curds and bile. The bowels were opened twice. The testis was more swollen; the scrotum was red and cedematous. There was frontal headache, but not very intense; the patient lay on his side and slept, but sleep was not continuous and was broken by horrible dreams of accidents, &c. The tongue was moist and cleaner; there was no pain in the back or limbs, but he disliked being disturbed. He had perspired freely. On the 7th he had had a restless night, but was brighter and more cheerful in the morning. His head was clearer, the headache was gone, though it was still brought on by movement. The temperature was 102.6° and the pulse 100, dicrotous. The tongue was moist with yellowish fur. There was no more vomiting. The bowels were opened three times. He took milk badly, and was ordered beef-tea, toast, cooked apples, &c., and to continue the medicine. The left testis was more swollen, and tender on pressure. He passed urine involuntarily, as he had done the day before. The temperature was 103.8° in the evening, and the pulse 100. On the 8th he had slept better, though he was restless; he had taken more liquid food. There was no vomiting. The bowels had been opened eight times. In the morning he was much better in all ways. His temperature was 99.2° and the pulse 80 and regular. The headache was almost gone. His mind was clear and he was more lively and natural. The tongue was still coated but was clean at the tip. There was copious epistaxis in the morning. He complained of pain over the lower part of the sternum; on auscultation the sounds of the heart were found to be quite normal. The testis was still very large and tender and the scrotum cedematous. The temperature was 101° in the evening. On the 9th he had slept better, but was still restless. The temperature was 100.2° and the pulse 96. The tongue was slowly cleaning from the tip. The epistaxis was renewed in the morning. The headache was gone. The left testis was smaller, but painful and tender, and there was much edema of the scrotum. The right testis was somewhat tender also, but was not swollen. The temperature was 102.2° in the evening. The medicine was discontinued. On the 10th the temperature was 97.8° . The tongue was cleaner. The left testis was smaller and the right testis tender; hot fomentations were applied. At 9 P.M. the patient looked well. The temperature was 97.6° and the pulse 72. The tongue was clean. The appetite was becoming ravenous. On the 11th he was still in bed, but convalescent. On the 13th he sat up for the first time. He slept while sitting in a chair; both arms and legs twitched during sleep. The bladder had been irritable during the illness, but this was now all right. On the 15th he left the room for the first time, but was very shaky; he was weak on the legs and had to be assisted on returning upstairs.

The testis subsequently underwent considerable atrophy, as is usual, I believe, in such cases as this.

Remarks.—The nervous phenomena which may complicate mumps are not novelties; they have been observed and remarked upon from time to time for a long while. But all the same they are infrequent, even very exceptional,

considering what a common disease mumps is. They are said to be more common in some epidemics than others. The subject is mentioned in standard text-books of medicine, but generally in a vague and inaccurate way. It is, perhaps, because mumps is generally such a trivial complaint that so little attention is devoted to its curious occasional phenomena. But surely it is an error to style the disease, as is done in more than one text-book, "Idiopathic Parotitis," which is quite a different thing. I should like also to raise the question whether meningitis is a possible complication as it is sometimes stated to be. Some writers, by-the-way, think proper to place mumps among diseases of the mouth, &c., instead of with general and infectious diseases. A good many cases complicated with brain symptoms have now been put on record, but they are only to be found here and there in various periodicals, British and foreign; it seems, moreover, that the literature of the subject is more abundant in other countries, especially France and America, than in this. These cases are remarkable, both on account of the striking nature and sudden appearance of the symptoms, and on account of the obscurity of their pathology, which is still a mystery. Fortunately, even although the symptoms be alarming, death very rarely takes place. Moreover, when death does occur in mumps, it seems often to be due to some indirect complication which may almost be called accidental. I will refer briefly to the fatal cases that I have been able to discover, either described or mentioned. Three fatal cases are cited by Haldeman.¹ The first of these, complicated with orchitis, died from acute pneumonia, which set in on the fifth, and was fatal on the eleventh, day of the illness. The author ascribes the pneumonia to embolism. The second case, in which both parotids and both testes were involved, succumbed to the bursting of an abscess of the liver into the lungs and air-passages, causing asphyxia on the thirteenth day. His third case is that of a woman, seven months pregnant, who aborted on the fifth day and died from septicæmia two days later.

A remarkable case of sudden death in mumps is related by Gillet.² The patient was a young conscript, in whom swelling of the right parotid gland set in, which spread to the sub-maxillary gland also; he did not take to bed or rest at once. On the fourth night he suddenly awoke, rose up and tried to walk, but fell in a state of complete syncope; he rallied a little, but soon became collapsed again and died. It is stated that he could breathe and swallow during the attack. Only the parotid gland was examined post mortem. Death was ascribed to embolism of one of the arteries at the base of the brain due to cardiac thrombosis consequent on endocarditis. In a case reported by Hamilton³ and in another by Shreve⁴ furious delirium came on suddenly, with great excitement, and attended in the former with opisthotonos and muscular rigidity, and death ensued in each in two or three days. Other fatal cases have been recorded, two by Lindsley,⁵ another by Pollard,⁶ a few others (mentioned by Lannois and Lemoine) by various French observers, but no details are furnished when quoted, and I have not obtained access to the sources of information. Niemeyer⁷ says: "Cases have also been recorded where, in the course of idiopathic parotitis, fatal meningitis has been developed." Eberle⁸ also speaks of a fatal case with convulsions. I am not aware, however, that a post-mortem examination was made in any of the fatal cases referred to or that there was positive evidence of meningitis in any of them. From a survey of all the cases to which I could find a reference and have been able to peruse the details it seems that the cases of mumps with cerebral complications fall into three different classes according as the nervous symptoms are marked by: (a) high fever and delirium; (b) insanity; and (c) paralysis.

A.—The case above related is a characteristic example of the first group. Cases of this sort are met with, I believe, only in males, never in children, and chiefly in lads about the age of puberty or in young men, and is almost always, if not invariably, associated with orchitis, though of course orchitis often complicates mumps without there being any serious constitutional disturbance. Lynch⁹ states that high fever and delirium may occur in mumps when the disease is

¹ Journal of the American Medical Association, Chicago, 1887, p. 543.

² Gazette des Hôpitaux de Paris, 1873, p. 1156.

³ Lond. Med. Journal, vol. ix. (quoted by Lannois and Lemoine).

⁴ Boston Medical and Surgical Journal, 1875, p. 739.

⁵ Stethoscope and Virginia Medical Gazette, 1851.

⁶ Bellevue Hospital, Virginia.

⁷ Text-book of Practical Medicine, vol. i., p. 439.

⁸ Treatise on Practical Medicine, vol. ii., p. 333.

⁹ Dublin Quarterly Journal of Medical Science, 1856, xxi., p. 290.

confined to the parotid glands, but I have not been able to find the account of any such case where the presence of orchitis is not mentioned. It may be that the brain and testes are affected in common simply because of the intensity of the disease, but the constant association of the brain symptoms with severe orchitis suggests the possibility of some causal relation between the two. In some cases—e.g., those related by Trousseau,¹⁰ Hall,¹¹ Walmsley,¹² Davis,¹³ and Lees,¹⁴ the extreme febrile symptoms and delirium supervened on the subsidence of the swelling of the parotids and disappeared on the manifestation of orchitis. It is to this particular feature that Trousseau calls attention, and many readers will most likely recollect his vivid account of the two cases which came under his own notice with an interval of twenty years. But in other cases (and they form the majority, as far as my inquiries go), as in the case related in this paper, also those by Hamilton,¹⁵ Appleyard,¹⁶ Beale,¹⁷ Monro,¹⁸ Parrott,¹⁹ Doudney,²⁰ Minot,²¹ Shreve,²² Sorel,²³ Janson Zuède,²⁴ and Lynch,²⁵ orchitis appeared first, and was attended either immediately or after a short interval, by high temperature (sometimes reaching 106° or 107° F.) and severe febrile symptoms, such as delirium and other nervous phenomena, and the local inflammation and general symptoms subsided *pari passu*. The nature and pathology of these cases is uncertain and obscure. By some observers and writers the nervous symptoms have been assigned, rather hastily I think, to meningitis; but in almost all the cases referred to above, this cause is, to say the least, very doubtful. The symptoms are hardly those of meningitis, and the cases certainly bear very little resemblance to the description, say, of epidemic cerebro-spinal meningitis. Moreover the difference in mortality is very great. Even in Hamilton's and Shreve's cases (*vide supra*) the symptoms do not point conclusively to meningitis; the inference is rather that death took place from exhaustion consequent on violent excitement, but no necropsy seems to have been held either in these or in any of the reported fatal cases of mumps, and in the absence of such unequivocal evidence it is impossible to affirm the existence of meningitis. In Munro's case—which was a very severe one and accompanied by a very high temperature (107.2°) and great delirious excitement with much prostration—although recovery took place it is stated that for a considerable time after the acute symptoms subsided the patient was excitable and also suffered from difficulty in walking; the gait was unsteady and incoördinate, the speech hesitating and impulsive, and agraphia was marked. This condition is certainly suggestive of cerebral and spinal meningitis, but cannot, I think, be pronounced conclusive. These cases seem to me to be much more closely related to those of acute rheumatism with hyperpyrexia and cerebral symptoms, and the affinity is strengthened by the fact that in some of these cases of mumps certain symptoms of acute rheumatism have been superadded; for example, in a case recorded by Charles Aitken,²⁶ an attack of mumps with orchitis was immediately followed by "a rather severe attack of rheumatic fever, with pains in the joints, but no cardiac mischief; this was almost immediately succeeded by a short attack of chorea." In Davis's case subsidence of orchitis was attended by general articular rheumatism, with much pain, but without fever. Endocardial murmurs developed in the course of Appleyard's case—a severe one with very high temperature though in this case there may have been pre-existing valvular disease. Metastatic endocarditis was also the chief feature of one of the set of cases reported by Haldeman,²⁷ which recovered, however, and there was thought to be endocarditis in Gillet's case also, which, as has been already mentioned, ended in sudden death.

Thus instead of ascribing any cases of this group to meningitis I think it far more probable—almost certain, indeed—that the grave symptoms are really those of cerebral irritation due to some kind of blood poison which is developed in the course of the disease, especially about the period at which inflammation settles on the testes.

B.—A similar sort of explanation probably applies to this class also. Resembling the former group in the presence of delirium, it differs in the fact that the temperature and febrile symptoms are generally less high; the delirium begins about the same period as in the former class or when the orchitis is disappearing, but does not subside with the natural subsidence of the disease being prolonged, often for a month or two, in the form of insanity either mania or melancholia. Cases of this sort have been recorded—one by Radcliffe²⁸ and three (one of whom was a woman, but it is not stated whether her sexual organs were affected) by Van Bibber.²⁹ All these cases recovered.

This occasional sequence of insanity constitutes another point of affinity to *acute* rheumatism.

C.—On the other hand, this explanation will scarcely cover all cases of the cerebral symptoms of mumps. In an able paper by Lannois and Lemoine (*Des Manifestations Méningitiques et Cérébrales des Oreillons*),³⁰ three cases are described (one observed by themselves, the other two quoted from Sorel and Janson-Zuède respectively) which presented definite and persistent paralyses, which cannot well be ascribed to nervous debility following the disease. In the first case—in which, by the way, there is no mention of orchitis—there suddenly supervened after the parotid swellings had subsided in the usual way a condition of coma, with right hemiplegia and hemianæsthesia, as well as conjugate deviation of the eyes, unilateral paralysis of the face and tongue, and aphasia. The hemiplegia and aphasia lasted for some time, but ultimately recovery took place. A closely similar case is reported by Hitchcock.³¹ The patient was a woman who, after the subsidence of a severe febrile attack of mumps, was suddenly seized on the eleventh day with cerebral disturbance and vomiting, soon succeeded by coma, paralysis of the right arm, and convulsions. In a month's time the paralysis disappeared and the patient recovered. In Sorel's case (*Orchite ourlienne atrophiant; complications cérébrales; aphasie légère mais prolongée*)³² the temperature reached 107° F. while the orchitis lasted, and the attack was severe. Convalescence was gradual, and attended by a variety of aphasia. In Janson-Zuède's case³³ furious delirium supervened during the orchitis and was accompanied by aphasia and paralysis of the right arm as well as anæsthesia and a slow pulse. The patient partially recovered, however. Partial paralysis of the arms is mentioned also in one of Haldeman's fatal cases. The explanation offered by Lannois and Lemoine of the whole class of cases of cerebral complications of mumps is that the cause is probably active hyperæmia of the meninges in the slighter cases, and in the more severe ones, in which some degree of actual paralysis occurs, the hyperæmia is more intense, extending more deeply and damaging the surface of the brain itself. This hypothesis may possibly be the true one, yet it is very doubtful. It seems much more probable from the histories that the paralysis is due to some accident, such as hæmorrhage or embolism, rather than to an inflammatory process. I think it is most likely that the real condition of these cases is embolism, for there is corroborative evidence in certain cases which show a tendency to thrombosis in mumps. Gillet's case, already quoted, was probably fatal by embolism; but instead of its fatal end being brought about by cardiac thrombosis and embolism of a cerebral artery, I would suggest that it might be due to venous thrombosis and embolism of the pulmonary artery. I have seen sudden death happen in an exactly similar way from pulmonary embolism in a case of cancer of the tongue; the cases of endocarditis related by Appleyard and Haldeman also mark a liability to embolism. There is also a remarkable case narrated by Logan (*Metastasis in parotitis*)³⁴ in which, about three weeks after the onset of an attack of mumps affecting the parotids and testes, phlebitis set in, first in the left lower limb, involving the

¹⁰ Clinical Lectures, Sydenham Society's Translation, vol. ii., p. 77.

¹¹ Brit. Med. Jour., 1881, vol. i., p. 966.

¹² THE LANCET, Aug. 31st, 1878, p. 312.

¹³ Brit. Med. Jour., 1889, vol. i., p. 648.

¹⁴ Dublin Quarterly Journal of Medical Science, 1854, p. 457.

¹⁵ Loc. cit.

¹⁶ THE LANCET, Jan. 21st, 1882, p. 101.

¹⁷ Medical Times and Gazette, 1875, vol. i., p. 416.

¹⁸ THE LANCET, Aug. 18th, 1883, vol. ii., p. 280.

¹⁹ Brit. Med. Jour., 1889, vol. ii., p. 819.

²⁰ THE LANCET, Nov. 29th, 1890, p. 1156.

²¹ American Journal of Medical Sciences, Philadelphia, 1850, p. 377.

²² Loc. cit.

²³ Archives de Médecine Militaire, December, 1883.

²⁴ Annales de la Société Médico-Chirurgicale de Liège, May, 1884.

²⁵ Loc. cit.

²⁶ Brit. Med. Jour., vol. i., p. 158.

²⁷ Loc. cit.

²⁸ Philadelphia Medical Times, 1873-74, p. 721.

²⁹ Medical News, Philadelphia, 1885, p. 678.

³⁰ Archiv de Neurologie, Paris, 1885, vol. xi., p. 1.

³¹ Boston Medical and Surgical Journal, 1842, p. 351.

³² Loc. cit.

³³ Loc. cit.

³⁴ Medical News, Philadelphia, 1832, p. 569.

femoral, popliteal, and one-third of the anterior and posterior tibial veins, then the left upper limb, involving the jugular, subclavian, brachial, and part of the ulnar veins; after this, the right lower limb was involved as the left had been. Thus the main veins of three limbs were successively plugged. In each of these attacks of phlebitis the inflammation seemed to start from one of the glands which had been involved. In such a condition it is easy to see what a liability to embolism there must be. This patient recovered. However, in the absence of necropsies on fatal cases it is impossible to do more than conjecture. It is a matter that requires more light to be thrown upon it, and it is with a view to calling attention to the subject that these suggestions are offered.

A last word may be said about treatment. As has been already said, nearly all cases recover, and therefore the prognosis is good. But in the way of giving relief and promoting cure in the feverish, delirious patients, I think no measures have proved so effectual as the administration of cooling, diaphoretic, and aperient medicines, such as were exhibited in the case I have narrated. Narcotics should, I think, be avoided. It was also worthy of note that in a large proportion of the cases here collected the grave symptoms followed and were apparently induced by carelessness and exposure to cold, &c., while recovering from a simple attack of mumps. It may, therefore, be of importance to exercise prudence and to take necessary precautions during convalescence from the disease, especially in the case of adults.

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ON THE PATHOLOGY AND TREATMENT OF CHILBLAINS.

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I HAVE in a recent issue of THE LANCET¹ dealt with the general question of "serous hæmorrhage" (i.e., of increased transudation of the blood fluids into the tissues), and I have pointed out (a) that this condition is very often dependent upon a defect of blood-coagulability, and (b) that serous hæmorrhage can to a large extent be controlled by increasing the coagulability of the blood. I had in previous papers dealt with this question of serous hæmorrhage in its special relation to urticaria, and had demonstrated not only that urticaria is very often dependent upon a condition of defective blood-coagulability, but also that this affection can often be relieved in a very striking manner by increasing the patient's blood-coagulability. I propose in this paper to adduce evidence to show that what holds true of urticaria holds true also of that very familiar form of serous hæmatoma which we denote by the name "chilblain."

Association of chilblains with a condition of defective blood-coagulability.—I have investigated the condition of blood-coagulability in ten cases of chilblains. Two of these cases were cases of aggravated chilblains occurring in adult males. The blood-coagulation times of these patients were respectively *nine minutes*, and *nine and a quarter minutes*. Four of these cases were cases of aggravated chilblains occurring in adult females. The respective blood-coagulation times of these cases were respectively *thirteen minutes*, *eleven minutes*, *eight and three-quarter minutes*, and *seven and a half minutes*. Lastly, four of these ten cases were mild cases of chilblains occurring in schoolboys. The coagulation times of these cases were respectively *eleven minutes*, *nine and a quarter minutes*, *seven and three-quarter minutes*, and *four and a half minutes*.² It is obvious, therefore, when we consider that the normal blood-coagulation time varies between *three and four minutes*, that in all these cases of chilblains, with the exception of the last case, were associated with a very notable defect of blood-coagulability.

¹ THE LANCET, Sept. 19th, 1896.

² All these coagulation estimations were made with coagulation-tubes of 0.25 mm. internal diameter, obtained from Mr. A. E. Dean, jun., 73, Hatton-garden, London, E.C. In order to bring the tubes to the standard temperature of 18.5° C. (half blood heat), their butt-ends were first fitted with indiarubber caps, and they were then immersed (point upwards) into a tumbler two-thirds full of water at that temperature. After filling in with blood the tubes were again immersed (this time point downwards and without caps) in the tumbler of water.

This fact stands in relation with certain other facts which obtrude themselves more directly upon the clinician's attention. These facts are—(a) the superior liability of children to chilblains; (b) the fact that chilblains are prone to occur in persons who give a history either of nose-bleeding or of urticaria; (c) the occurrence of chilblains in persons who are characterised by a lymphatic habit of body; (d) the not unfrequent occurrence of chilblains in persons who are the subjects of malarial cachexia; and (e) the not unfrequent occurrence of chilblains in hæmophilic families. We may briefly consider each of these predisposing causes.

Childhood.—The notorious liability of children to chilblains is no doubt in part referable to the fact that the influence of cold makes itself felt more upon the relatively small extremities of the child than upon the relatively large extremities of the adult. Another probable factor in the etiology is the fact that the lime salts upon which the coagulability of the blood depends are in the growing child being continually removed from the blood in order that they may be deposited in the bones.

Predisposition to urticaria and epistaxis.—There is an obvious relation between the predisposition to epistaxis, the predisposition to urticaria, and the predisposition to chilblains, inasmuch as these predispositions have been shown to depend upon a defect of blood-coagulability. In two cases which have recently come under my observation I have seen urticaria alternate with chilblains. Both these forms of serous hæmorrhage were apparently brought on in susceptible patients by exposure to cold.

Lymphatic habit of body.—We shall understand the relation between the lymphatic constitution and a predisposition to chilblains if we consider, first, that the essence of the lymphatic constitution is to be found in a water-logging of the tissues which is dependent upon an excessive transudation of lymph; secondly, that it will require only a very slight increase of transudation to convert such a water-logged condition of the tissues into perfectly definite hæmatomata such as we see in chilblains; and thirdly, that in all probability both chilblains and the water-logged condition of the tissues which we meet with in the lymphatic patient are ultimately referable to a defect of blood-coagulability.³

Malarial cachexia.—The subjects of malarial cachexia are not unfrequently also the subjects of chilblains. It is even possible, as I am assured by a medical officer who has experience of the truth of this fact in his own person, to suffer from chilblains on the West Coast of Africa after a severe attack of malarial fever. This liability of the malarious subject to chilblains is in absolute conformity with the fact that the blood of patients, who are the subjects of malarial cachexia, is characterised by a defect of blood-coagulability which is dependent upon a great paucity of white blood corpuscles,⁴ especially of polynuclear white blood corpuscles.

Hæmophilic constitution.—I have pointed out in previous papers that the chilblains are of very frequent occurrence in hæmophilic families. This stands in connexion with the fact that hæmophilic blood is, as I have previously shown, characterised by an extreme defect of blood-coagulability which is dependent upon an extreme and hereditary paucity⁵ of white blood corpuscles and, in particular, upon a paucity of polynuclear white blood corpuscles.

Treatment of chilblains.—In view of the etiological facts which are thus disclosed, the obvious indication for treatment in a case of chilblains is to take steps to augment the patient's blood-coagulability. In conformity with these indications I placed my patients upon a regimen of calcium chloride (after duly cautioning them against lowering their blood-coagulability by the ingestion of sour fruits, alcohol, or excessive quantities of fluid⁶). Of the 8 cases which are particularised below, six cases (Cases 1, 4, 5, 6, 7, and 8)

³ It is customary to impute the liability of the lymphatic patient to chilblains to the patient's bad circulation. But in many cases it is, in all probability, not the bad circulation which produces the œdema. It is rather the œdema which impedes the circulation of the blood in the tissues, especially in the tissues of the extremities.

⁴ There may in malarial cachexia be as few as 400 white blood corpuscles in the c.mm. of blood.

⁵ It is unusual to find more than from 4000 to 5000 white blood corpuscles in the c.mm. of hæmophilic blood, and as few as 35 per cent. of these may be polynuclear white blood corpuscles.

⁶ I find that it is quite unnecessary to caution a lymphatic patient against ingesting excessive quantities of fluid, for the typical lymphatic patient, owing probably to that water-logging of the tissues which has been referred to above, is apparently never thirsty. The lymphatic young woman contrasts markedly in this respect with the wizened elderly woman, whose dry tissues seem to require to be irrigated with perpetual draughts of tea.