branching remained undecided. In young cultures on Loeffler's serum spindle-shaped rods and filaments predominated, the latter being shorter than in fluid media. The filaments also showed central, terminal, or subterminal swellings (moniliform bodies) similar to those seen by one of us (I.R.W.L.) in Levaditi's laboratory in 1936. It was noted that the moniliform bodies tended to disappear in older cultures.

Staining Reactions.—Poor results were obtained with Gram's stain, but on the whole in very young cultures the organism tended to be Gram-positive and in older cultures Gram-negative. With the other stains previously mentioned the staining was irregular, the moniliform bodies being coloured more intensely than the filaments. When stained with Ziehl-Neelsen's carbol-fuchsin the organism was not acid-fast.

Characteristics in Culture.—On solid media no growth occurred on ordinary agar, very scanty growth on 5% blood agar, good growth on 25% horse-serum agar, and abundant growth on glucose-serum agar and Loeffler's serum. The colonies were at first small and translucent, but became granular and rough after several days. Even after 24 hours a number of the colonies were of R type. In deep glucose-serum agar shake cultures mulberry-shaped colonies appeared which were not limited to the aerobic zone. The organism is thus aerobic but a facultative anaerobe. Growth did not appear to be stimulated by increased carbon dioxide pressure. In fluid media abundant growth occurred, as already indicated, when serum and fermentable carbohydrates were present. The sugar reactions were as follows: acid but no gas from glucose and starch, slight acid from lactose and maltose; no indole formation; gelatin not liquefied. In fluid media containing carbohydrates subcultures were found to be essential every 48 hours. In carbohydrate-free media the organism can remain alive for five days but is dead in seven days. It is killed by exposure to 60° C. for 20 minutes. The organism isolated was found to be very sensitive to penicillin, all growth being inhibited by 0.01 unit of the drug per 1 ml. of medium.

Animal Inoculation.—The organism is pathogenic for mice, and both animals inoculated intraperitoneally with 1 ml. of the patient's blood died, in 9 and 11 days. Similar inoculations from 24-hour cultures (0.5 to 1 ml.) caused death in four to six days, but no characteristic lesions were observed at necropsy, although heart-blood cultures were positive. Both guinea-pigs and rabbits seem to be insusceptible.

Two additional points of interest may be noted. Attempts to demonstrate agglutinins, both in the patient's blood and in the susceptible animals, failed, for the organism was auto-agglutinable even in media free from carbohydrates. The whole stock of hooded rats from the cage containing the original culprit was examined, and *Streptobacillus moniliformis* was obtained by swabbing the nasopharynx in seven out of the 10 rats examined.

Discussion

There are records of at least five patients previously treated with penicillin, but their illnesses occurred in the time of penicillin scarcity and a much smaller dosage was employed. Kane (1944a, 1944b) reported the first case from Belfast, in which a positive blood culture was obtained only on the 25th day of illness. Gold therapy was tried without success, and penicillin was first used on the 62nd day, just after the beginning of a bout of fever. In all 200,000 units were injected over 48 hours; but within 12 hours, after only 60,000 units, the fever vanished and convalescence was uninterrupted. Weber and Favour (1945) began penicillin treatment of their patient on the 13th day, using at first 100,000 units a day, reduced to 80,000 units on the third day and continued for 10 days. Fever was abolished only on the seventh day. Altemeier, Snyder, and Howe (1945) gave 302,500 units over 8½ days to a child aged 10 months, 212,500 units to a child of 2 years, and 132,000 units over five days (all the drug available) to a child of 4½ years. Cure was immediate and complete in the first and second cases, but the third child relapsed four days after penicillin was discontinued and blood culture again became positive.

Summary

A case of rat-bite fever due to *Streptobacillus moniliformis* (Levaditi) is reported, developing 48 hours after the bite of a hooded rat.

Treatment with penicillin was completely successful, the disease being cured at once. Much larger dosage, probably excessive, was employed than in the few cases previously described, but the illness was severe and relapse had occurred in the days of penicillin scarcity.

The methods of diagnosis and the characteristics of the organism isolated are briefly described.

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A CASE OF ACUTE DERMATOMYOSITIS

BY

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Acute non-suppurative myositis involving several muscle groups is a rare condition and deserves special notice when it occurs. It was first described in 1887 almost simultaneously by Wagner, Unverricht, and Hepp, but little has been added to our knowledge of the pathology of the condition since that time. The case recorded here is one of the very few in which an organism that might be regarded as causative has been isolated.

The chief clinical features of the disease are widespread oedema of muscle groups associated with intense pain and tenderness, fever, and often inflammatory lesions of the overlying skin. Unverricht coined the term "dermatomyositis" to describe the disease with cutaneous lesions, but this has since been applied to a large and ill-defined group of skin affections associated with muscular swelling or atrophy and with wide variation in the severity and duration of symptoms. Probably some of these are aetiologically different from the acute polymyositis under consideration, but it is possible that allergic sensitivity may be a common factor.

Pathology

The clinical and histological features of dermatomyositis of the acute type suggest that it is infective in origin, but organisms have very rarely been isolated and the standard textbook descriptions of the disease state that the aetiology is unknown. Deák (1931) isolated a haemolytic streptococcus in five cases of "acute haemorrhagic myositis," and Karelitz and Welt (1932) found a haemolytic streptococcus in blood culture shortly before death in one case. Other attempts to demonstrate a causative organism have been unsuccessful. Therapeutic evidence of an infective cause has recently been furnished by the successful treatment of the condition with sulphonamides and penicillin. Clark (1946) reports two cases—one treated with sulphonamides and the other with penicillin. Friedmann and Por (1947) also describe a case successfully treated with penicillin. In none of these, however, was an organism isolated from blood cultures. My case appears to be the first in which an

organism has been grown from a biopsy specimen of muscle.

The affected muscles are usually soft and friable and may be adherent to the surrounding skin and subcutaneous tissues. Later they may become indurated and scarred. The muscles are severely oedematous in the acute stages and areas of haemorrhage may be seen. The skin usually shows macular or petechial haemorrhages and a variable degree of inflammatory infiltration and oedema.

Microscopically there is extensive degeneration of muscle fibres and infiltration with lymphocytic, mononuclear, or polymorphonuclear cells. Occasionally eosinophilic infiltration is seen. The infiltration is usually perivascular in distribution and oedema is intense. The degree of inflammatory change varies with the severity and stage of the disease. In the late stages the muscle fibres may be entirely replaced by fibrous tissue, with perhaps a few areas of cellular infiltration—the picture of “myositis fibrosa.” Occasionally the inflammatory changes involve the walls of blood vessels. In some cases the myocardium and muscles of deglutition are affected.

The blood picture is variable. In many cases there is no leucocytosis even in the acute phases of the disease, but in others a moderate or even high polymorph leucocytosis occurs. There is often a moderate degree of splenomegaly.

Differential Diagnosis

The clinical picture, once seen, should be unmistakable. The only condition which at first sight may give difficulty is trichiniasis. The disease was called “pseudo-trichinosis” by Hepp in 1887, but a high degree of eosinophilia is an almost invariable accompaniment of trichiniasis, and microscopical examination of a piece of excised muscle will usually reveal encysted forms of *Trichinella spiralis*. The skin lesions may suggest a diagnosis of the so-called Stevens-Johnson syndrome or of acute lupus erythematosus (Libman-Sachs syndrome), but myositis is not associated with these conditions. Polyarteritis nodosa may have to be considered in a few cases, particularly as some degree of myositis may occur in this disease as well as skin lesions. There should be no difficulty in distinguishing the disease from acute suppurative myositis.

Prognosis

The disease has had a high mortality in the past, but few cases have been recorded since sulphonamides and penicillin have been available, and it seems probable that chemotherapy may change the outlook considerably. Recurrences after apparent recovery are common. Many of the patients who survive are left with muscular atrophy or contractures and scleroderma. It is possible that myositis fibrosa is a terminal stage of the disease. Calcinosis is said to be a sequel in some cases. When death occurs it is usually due to involvement of the muscles of respiration or deglutition and sometimes of the myocardium.

Case Report

A boy, aged 15, was admitted to Crewe and District Memorial Hospital on Feb. 16, 1947. The illness had started seven days before with sore throat followed by pain in the right ear. A few days later a painful swelling appeared in the right arm above the elbow, with oedema of the right pinna and bulging of the right eardrum, red tonsils and fauces, and slight cervical adenitis. The right arm was grossly oedematous from wrist to shoulder. Some swelling was also present in the left arm above the elbow, with extreme tenderness. Temperature 101° F. (38.3° C.), pulse 120, respiration 24. No cardiac murmurs; blood pressure 125/75; urine normal. Leucocytes 30,000 per c.mm.—polymorphs 80%. Penicillin, 20,000 units three-hourly, was begun. The muscular swelling spread rapidly to both legs and the face,

and the limbs and trunk became covered with a macular erythematous rash and scattered purpuric patches. Many of the purpuric areas became confluent, and as they faded more appeared. The play of the colours known as erythema iris was seen in the skin lesions. Bullae containing serous fluid appeared on the legs from time to time. The spleen became palpable and tender during the second week of the illness. The patient was immobilized by intense pain, which was only partly relieved by morphine. No improvement occurred until March 3. After this the skin lesions faded, leaving a brownish pigmentation. Pyrexia of 101°–102° F. (38.3°–38.9° C.) continued and gradually returned to normal on March 28. Penicillin was discontinued on March 16, a total of 3,300,000 units having been given. On March 25 the leucocytes were 5,000 per c.mm.—polymorphs 65%. Blood cultures were sterile on three occasions—once before treatment with penicillin and twice during treatment. Culture of fluid from a bullous skin lesion was also sterile. The patient was discharged home on April 16. The muscles of his arms and legs were very wasted and some contracture at the knees and elbows was present. Massage and exercises corrected these deformities, and by July 1 he was able to work as a packer. The muscular volume had returned to normal and there was no loss of motor power.

The patient was again admitted to Crewe Hospital on Sept. 20, 1947, with a severe recurrence of the polymyositis, similar to the first attack, but more intense. The spleen was again enlarged and tender, and the polymorphic skin lesions more widespread. Temperature 103° F. (39.4° C.), pulse 120, respiration 24. Blood culture was again sterile and remained so after incubation for 18 days. Leucocytes amounted to 25,600 per c.mm.—polymorphs 83%, eosinophils 3%. Penicillin, 30,000 units three-hourly, was begun. No improvement occurred during the next week, and the patient was transferred to the Staffordshire General Infirmary on Sept. 26. A diagnosis of dermatomyositis still seemed the most probable one, but the outlook appeared hopeless. A piece of muscle was excised from a tender oedematous site in the right adductor longus on Sept. 29 to exclude the possibility of trichiniasis or polyarteritis nodosa. Half of this piece was kept for histology and half was placed into broth and incubated. By Oct. 2 this culture yielded a pure and heavy growth of *Streptococcus viridans*. Two further blood cultures and culture of oedema fluid from the leg were sterile. Leucocyte counts were as follows during the illness.

Date	Total Leucocytes per c.mm.	Polymorphs %	Lympho- cytes %	Monocytes %	Eosinophils %
1/10/47	63,600	89	8	3	
3/10/47	59,800	86	11	3	
7/10/47	43,600	83	10	6	
13/10/47	15,600	78	17	3	
27/10/47	15,600	46	51	3	

In spite of this intense leucocytosis there was no evidence of suppuration at any time. Through an unfortunate laboratory error the sensitivity of the *Str. viridans* to penicillin was not determined, but the penicillin dosage was increased to 80,000 units three-hourly on Oct. 3, and sulphadiazine, 1 g. four-hourly, was also begun. The sulphadiazine was stopped on Oct. 14 after a total of 70 g. had been given, and the penicillin was stopped on Oct. 18 after a total of 8,120,000 units. “Benadryl” in doses of 200 mg. daily had been given for four days from Oct. 2, but without visible effect on the skin lesions or oedema. The skin lesions and oedema began to diminish on Oct. 13 and the temperature had become normal by Oct. 23. Electrocardiograms on Oct. 3 and Nov. 4 showed no abnormality apart from low voltage. The urine showed no abnormality throughout the illness apart from a trace of albumin. The patient returned home on Nov. 16. The muscle-wasting was more pronounced than in the previous attack, but there has since been a rapid recovery.

Dr. A. J. McCall has kindly given the following report on the sections of excised muscle. “The muscle shows numerous inflammatory lesions in which the muscle fibres are separated by collections of histiocytes and lymphocytes, with only occasional eosinophils and polymorphs (Figs. 1 and 2). In some of these areas there is albuminous coagulum, but frequently

there is none. The lesions are often perivascular, and they then tend to be spindle-shaped. One or two small arteries show what is probably oedema of the vessel wall, with occasional inflammatory cells in the outer media, but there is no necrosis or thrombosis. Some muscle fibres appear normal, but others show damage varying from oedema to complete destruction and replacement by granular eosinophil material. Proliferation of sarcolemma nuclei is sometimes seen. The frequency of the lesions varied considerably in sections taken from different levels in the block, and it is evident that they are patchy in distribution. No parasites or organisms could be found in any of the sections."

FIG. 1.—Section of muscle showing cellular infiltration and damage to muscle fibres. ($\times 80$.)

FIG. 2.—Showing perivascular infiltration and slight inflammatory reaction in outer coats of small artery. ($\times 250$.)

Discussion

The clinical manifestations of acute dermatomyositis have been well recognized for more than sixty years, but although these are suggestive of a fulminating bacterial infection it is remarkable that an organism has been isolated on so few occasions. The isolation of a possibly causative organism in this case is so unusual that it must be examined with some scepticism.

It seems improbable that the *Str. viridans* was an accidental contaminant of the culture, but lesions of the severity described are by no means a characteristic of *viridans* infections. They are never seen, for instance, in bacterial endocarditis due to this organism, even when a considerable degree of septicaemia is present. There was no evidence of cerebral, renal, or pulmonary lesions, which would be expected in a fulminating bacterial infection with such an intense leucocytosis. The localization of the lesions to the muscles and skin is the most remarkable feature of the case.

The similarity of the clinical picture to that of acute disseminated lupus erythematosus (Libman-Sachs syndrome), acute erythema multiforme of the Stevens-Johnson type, and polyarteritis nodosa has been mentioned, and it is possible that they also have a similar aetiology. In the past these diseases have been thought to be due to bacterial infection, but most authorities now regard them as manifestations of allergic hypersensitivity, in some cases to bacterial antigens. Ophüls (1923) first suggested that poly-

arteritis nodosa might be due to allergic sensitivity to streptococci, and Rich (1946) now emphasizes that substances of widely differing chemical nature must be added to the list of sensitizing antigens capable of producing vascular lesions. This conception of allergic hypersensitivity has also been applied to the Stevens-Johnson syndrome (Sutton and Sutton, 1939, quoted by Nellen, 1947) and to the Libman-Sachs syndrome (Fox, 1943).

Banks (1941) first discussed the question of whether there is a common denominator between scleroderma, dermatomyositis, acute lupus erythematosus, and poly-

arteritis nodosa. He draws attention to the overlapping of the morbid anatomical features in all these conditions, particularly of the arteriolar changes. The very slight histological evidence of vascular damage in this case is an inconclusive finding, but the severe recurrent symptoms localized to muscles and skin and the slow response to penicillin and sulphadiazine could be explained by the assumption that the disease was due to an allergic response to the *Str. viridans*.

Summary

Two severe attacks of dermatomyositis in a boy aged 15 are described. After many unsuccessful attempts to grow an organism from the blood and oedema fluid *Str. viridans* was obtained in pure culture from a muscle biopsy during the second attack.

A piece of excised muscle showed patchy inflammatory changes with perivascular cellular infiltration and necrosis of muscle fibres in some places. Only slight infiltration of arterial walls was seen in the sections examined.

Complete recovery from both attacks occurred. In the first there was a slow response to penicillin alone, and in the second a slower response to larger doses of penicillin and sulphadiazine.

It is suggested that the severe symptoms were due to an allergic reaction in muscles and skin to the *Str. viridans* infection and that a clinical and possibly an aetiological similarity exists between dermatomyositis, polyarteritis nodosa, acute disseminated lupus erythematosus, and erythema multiforme of the "Stevens-Johnson" type.

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POST-PRANDIAL SYMPTOMS FOLLOWING PARTIAL GASTRECTOMY

BY

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While the results following subtotal gastrectomy for ulcer are generally excellent, in a small proportion of cases there develop post-prandial symptoms which may persist for many months. Such post-prandial upsets after gastrectomy have been described in British, German, and American literature, and frequent attempts have been made to relate these symptoms to changes in sugar levels in the blood. Lawrence (1936) noticed that after such surgical procedures patients developed hyperglycaemic then hypoglycaemic sugar levels following ingestion of glucose. Evensen (1942) found that 34 out of 95 cases treated by partial gastrectomy developed unusually low blood-sugar levels after meals. Adlersberg and Hammerschlag (1947) divided such upsets into two groups—early and late. The early symptoms (sense of pressure and fullness in the epigastrium) they attributed to the rapid emptying from the stomach remnant. Later symptoms (palpitations, headache, dizziness, and perspiration) they believed to be due to chemical factors, especially hypoglycaemia. Gilbert and Dunlop (1947) believed the symptoms were due to hypoglycaemia, and they found that ephedrine administration lessened the upset. They suggested that the development of insulin sensitivity was a causal factor.

Present Investigation

Twenty-four cases were studied—23 men and one woman. All had had subtotal gastrectomy for duodenal or simple gastric ulcer. The type of operation performed in every case was a gastrectomy, removing three-quarters of the stomach and the first inch of the duodenum, with an antecolic anastomosis. No valve mechanism was formed.

The Nature of the Symptoms.—In mild cases (the majority) the patients complain of a heaviness and fullness in the epigastrium, accompanied by a curious sense of muscular or body fatigue, and want to sit down; in more severe cases they may have to lie down and may fall asleep. A flushing of the skin may occur and the patient often "feels hot" and may actively perspire. Giddiness and palpitations often occur. Such symptoms were present in all 24 cases. To this list of symptoms must be added bilious vomiting, which occurred in two of the cases. These two patients had all the other symptoms, but in addition after meals they felt very upset and wanted to vomit. They usually vomited about 30 to 45 minutes after meals, and thereafter felt very well and were able to go about their business. The vomited material consisted either of food heavily bile-stained or, as often, of 1½ pints (0.85 litre) of unmixed pure bile.

Time Interval between Operation and Symptoms.—In every case the symptoms started either while the patient was in hospital or as soon as he went home and began to

live a normal life. In no case was there a time interval of some weeks, such as has been described by those who believe the condition to be due to the development of insulin sensitivity.

Time of Onset of Symptoms in Relation to Meals.—In each case the symptoms started almost immediately after meals or, indeed, while the patient was still eating. The upset usually lasted 20 to 30 minutes. The rapidity of onset suggested a mechanical causation, and in nearly every case the symptoms could be brought on rapidly by eating more quickly, and by giving a larger meal the symptoms usually increased in severity. In no case did a group of late symptoms appear 1 to 1½ hours after meals.

Type of Meal which Caused the Symptoms

In all 24 cases a careful study was made of the type of food and the amount taken at each meal in the day, and a rough measure of the total bulk of each meal and its total carbohydrate content was made. In 23 cases the symptoms appeared only after "the big meal"—i.e., the most bulky meal of the day. In almost all of these cases the carbohydrate content of "the big meal" was considerably less than the carbohydrate content of another meal in the same day which produced no symptoms. Indeed, the patients themselves in most cases clearly realized that it was the bulkiness of the meal which troubled them, and many noted that by missing the soup one day, and doing without pudding the next, they could avoid such symptoms altogether. In other words, the incidence of symptoms depended on the total amount of food eaten and not on its carbohydrate content.

In order to clarify still further the question whether the symptoms were due to bulk or to carbohydrate content each patient was given 50 g. of glucose in water and the severity of the symptoms and their time of onset, etc., were recorded; the blood-sugar levels were estimated by MacLean's method. On the following day the patient was given a bulk meal containing scarcely any carbohydrate (about 6 g.) and the severity and time of symptoms were again recorded. The blood-sugar levels were again estimated to check the fact that there was no rise in the blood sugar. In 16 of the 24 cases the symptoms were more pronounced after this bulk meal than they were after the ingestion of 50 g. of glucose in water. In four cases the severity of the upset was equal, and in four cases the 50 g. of glucose caused more upset than the bulk meal. In short, it was again shown that it was the bulk and not the carbohydrate content of the meal which mattered. It is, however, interesting to note that after these bulk meals the blood-sugar level rose to a moderate extent in most cases. Indeed, blood-sugar levels were recorded which were higher than one would expect after a meal of such small carbohydrate content. It will be suggested that this may be due to stimulation of the sympathetic system caused by distension of the gut, bringing about an adrenaline effect.

Relation of Symptoms to Blood-sugar Level

It has already been stated that each patient had blood-sugar estimations done after the ingestion of 50 g. of glucose in water, and the time during which the symptoms were present was carefully noted. The results are shown in the accompanying Table. By graphing the blood-sugar levels and marking on the graph the period of upset the following points were noted.

The symptoms appeared long before the sugar level in the blood had reached its peak. The symptoms were usually over before the blood-sugar level started to fall or before it fell below normal limits. In short, the symptoms appeared during the period of hyperglycaemia and the symptoms had