APPENDIX

Morphological and Other Characteristics of the Three Strains of Organism Isolated.—PS: From pustule at site of injury on June 23. BC: From blood culture on June 23. D: From dog's mouth, July 1.

Morphology.—A short, oval coccobacillus with rounded ends, about 1.5 by 0.5–0.8 μ, occurring singly, in pairs, and in short chains. Pleomorphism noted in subculture and increased with repeated subculture. It stained Gram-negative; bipolar staining was not a feature and no capsule was seen. (The organism was not observed in tissue-smears.)

Cultural Characters.—Aerobic and anaerobic at room temperature and at 37° C. on ordinary media but not on media containing bile salts. Colonies on agar and blood-agar after 24 hours were small (1–2 mm. diameter) translucent greyish-white disks. No haemolysis seen. No motility in liquid media.

Biochemical reactions to the three strains (PS, BC, and D) were: Glucose +, lactose +, dulcitol −, sucrose +, mannitol +, maltose +, indole +, urease −, gelatin liquefaction −.

Sensitivity to Antimicrobial Agents (Disk Diffusion Method).—

	Concentration per Disk	Zone of Inhibition (Diameter)	
Penicillin	2·5 units	21 mm.	Highly sensitive
Erythromycin	10 μg.	21 ,,	
Chloramphenicol	40 ,,	23 ,,	
Tetracycline	10 ,,	17 ,,	
Sulphafurazole	1 mg.	25 ,,	
Nitrofurantoin	10 ,,	17 ,,	
Novobiocin	10 μg.	13 ,,	Moderately sensitive
Streptomycin	80 ,,	0 ,,	Resistant
Neomycin	100 ,,	0 ,,	
Bacitracin	5·5 units (100 μg.)	0 ,,	

Serum Agglutination Reactions.—These were as follows:

	PS	D
Patient's serum July 29	1/160	1/160
,, ,, October 29	1/80	1/40
Control serum	1/20	1/20
Patient's serum, December 22	1/1,280	1/20
Control serum	1/10	Less than 1/10

Pathogenicity to Laboratory Animals.—PS: Killed a mouse in 24 to 36 hours and caused an abscess at site of inoculation in a guinea-pig in seven days. D: Killed a mouse in seven days. Non-pathogenic to guinea-pigs. BC: Not done.

Reaction with Specific Antisera.—PS and D: No agglutination titre obtained higher than 1:20.

It will be seen that the organism, though closely resembling *P. septica,* differed from it in fermenting lactose and in failing to agglutinate with a specific pasteurella antiserum except in very low dilution. Neither of these differences can be taken too seriously. In the past, several strains fermenting lactose have been isolated from animals, and more recently a group of lactose-fermenting strains possessing haemolytic activity have been defined. Agglutination reactions with members of the *Pasteurella* group are often very irregular, varying sometimes even from day to day with subcultures of the same organism. The general properties of the strain described here, taken in conjunction with its pathogenicity for mice and guinea-pigs, render it justifiable, in the absence of more detailed investigation, to include it tentatively in the *Pasteurella* group.

REFERENCES

Allott, E. N., Cruickshank, R., Cyrlas-Williams, R., Glass, V., Meyer, I. H., Straker, E. A., and Tee, G. (1944). *J. Path. Bact.*, **56**, 411.
Bartley, E. O., and Hunter, K. (1947). *Lancet*, **1**, 908.
Bearn, A. G., Jacobs, K., and McCarty, M. (1955). *Amer. J. Med.*, **18**, 167.
—— Kunkel, H. G., and Slater, R. J. (1956). Ibid., **21**, 3.
Braunstein, H. (1956). *A.M.A. Arch. Path.*, **62**, 87.
Brunsdon, D. F. V., and Mallett, B. L. (1953). *Brit. med. J.*, **2**, 607.
Byrne, J. J., Boyd, T. F., and Daly, A. K. (1956). *Surg. Gynec. Obstet.*, **103**, 57.
Cawson, R. A., and Talbot, J. M. (1955). *J. clin. Path.*, **8**, 49.
Emson, H. E. (1957). Ibid., **10** (May), p. 187.
Gorzynski, E. A., and Neter, E. (1953). *Antibiot. and Chemother.*, **3**, 1211.
Horne, W. I., and Berlyne, G. M. (1958). *Brit. med. J.*, **2**, 896.
Lee, M. L. H., and Buhr, A. J. (1960). Ibid., **1**, 169.
Ludlam, G. B. (1944). *J. Path. Bact.*, **56**, 307.
Mautner, L. S., and McIntyre, J. A. (1956). *Canad. med. Ass. J.*, **75**, 218.
Needham, G. M. (1948). *Proc. Mayo Clin.*, **23**, 361.
Peltier, M., Arquie, E., Jonchere, H., and Le Blough, G. (1938). *Bull. Soc. Path. exot.*, **31**, 475.
Pizey, N. C. D. (1953). *Lancet*, **2**, 324.
Regamey, R. (1938). *Schweiz. med. Wschr.*, **68**, 666.
Rivoalen, A. (1936). *Bull. Soc. Path. exot.*, **29**, 709.
Robinson, R. (1944). *Brit. med. J.*, **2**, 725.
Schipper, G. J. (1947). *Bull. Johns Hopk. Hosp.*, **81**, 333.
Schlecht, H. (1931). *Med. Klinik.*, **27**ii, 1100.
Sherlock, S. (1948). *Lancet*, **1**, 817.
Smith, J. E. (1955). *J. comp. Path.*, **65**, 239.
Svendsen, M. (1947). *Acta path. microbiol. scand.*, **24**, 150.
Whipple, R. L., and Harris, J. F. (1950). *Ann. intern. Med.*, **33**, 462.
Wilson, G. S., and Miles, A. A. (1955). *Topley and Wilson's Principles of Bacteriology and Immunity*, 4th ed. Arnold, London.

ANABOLIC HORMONES IN DERMATOMYOSITIS

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Spontaneous remission and relapse are not uncommon in dermatomyositis, making assessment of the results of treatment difficult. It is apparent from the many remedies listed that no single treatment has been successful. Since their introduction the corticosteroids have been most frequently prescribed, but, while some authors have claimed that these have revolutionized treatment in this group of disorders (Walton and Adams, 1958), others have found their effects to be slight or frankly disappointing (Ragan, 1950; Anderson, 1952; Gross, 1952; Kierland *et al.*, 1952; Wedgwood *et al.*, 1953; Wilson, 1954; Smith, 1955).

We report here two cases of dermatomyositis in which remission occurred after the use of anabolic hormones.

Case 1

Mrs. A, a schoolteacher aged 36, was admitted to hospital in May, 1957, with an acute febrile illness associated with nausea, diarrhoea, and myalgia. On examination she was seen to be of average build, with a

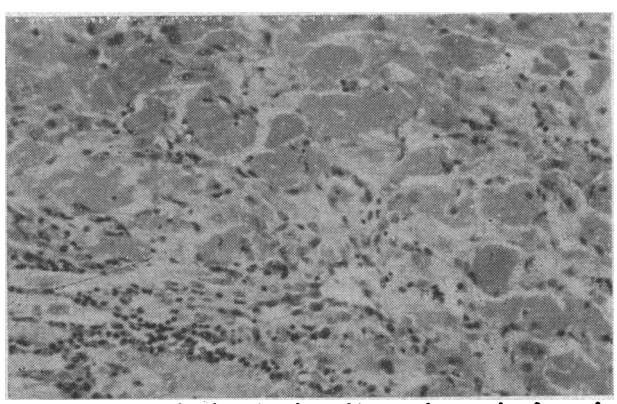

FIG. 1.—Case 1. Section showing widespread necrosis of muscle fibres with aggregations of inflammatory cells. (×400.)

widespread erythematous rash. There was profound generalized muscle-weakness without loss of deep reflexes. A partial thyroidectomy had been performed in 1944 for thyrotoxicosis, and, although clinically she was euthyroid, slight residual exophthalmos was present. A coarse pleural friction rub was audible at the base of the left lung. The relevant findings at this stage were as follows. Urine: albumin, 2 parts, Esbach; granular casts present. Blood urea, 30 mg./100 ml.; E.S.R., 54 mm. per hour (Westergren); W.B.C., 14,000/c.mm. No L.E. cells were present in a suitable preparation. Biopsy showed widespread necrosis of muscle fibres, with diffuse round-cell infiltration and aggregations of inflammatory cells, mainly plasma cells. The arteries were unaffected (see Fig. 1).

Prednisolone 60 mg. a day and A.C.T.H. 40 units a day were given with some improvement, but over the next few months relapse occurred on each occasion that the dose was reduced. After adding norethandrolone ("nilevar") to this regimen there was a more sustained improvement in muscle power, and she became able to walk. On discharge home she left the district but remained well for four months, at the end of which period norethandrolone was stopped. Although oral corticosteroids were continued, within a week muscle-weakness again became apparent and progressive. Four months later, when completely bedridden, norethandrolone was restarted, but the response was slight and unsustained.

She was readmitted to our wards at this stage. There had been considerable deterioration. Muscle-weakness was so pronounced that she was unable to lift herself from the bed, and had difficulty in feeding herself. Laboratory findings were: no albuminuria; creatinuria, 1.8 g. a day; E.S.R., 34 mm. per hour (Westergren); total serum proteins, 6.9 g./100 ml. The electrophoretic pattern showed a hyperglobulinaemia due to an increase in the α_2 and γ fractions. The E.C.G. was normal. Radioiodine assay of thyroid function gave normal results.

While corticosteroids were continued in the same dosage, norethandrolone was replaced by another anabolic hormone, methandienone ("dianabol") 30 mg. daily. Over the next three months progress continued, so that at the time of dismissal she was able to rise from a chair and walk unaided. Six months later she was on maintenance doses and was managing light housework.

Case 2

Miss B, a schoolteacher aged 45, had been unwell for six weeks before her admission to hospital in April, 1959, with heaviness and aching in her limbs. Swelling of the face, arms, and legs had been present for two weeks, and shortly after admission an erythematous rash developed over the trunk. Apart from marked muscle-weakness, which prevented her from raising her arms or sitting up unsupported in bed, examination was negative. Laboratory findings were: urine, a trace of albumin; a catheter specimen was sterile but showed granular casts; creatinuria, 1.7 g. a day. Haemoglobin, 10.7 g./100 ml; W.B.C., 16,000/c.mm.; E.S.R., 26 mm. per hour (Westergren); blood urea, 36 mg./100 ml. L.E. cells were not found after several examinations. Chest *x*-ray films and E.C.G. were normal. Total serum proteins, 7.4 g./100 ml. The electrophoretic pattern showed a hyperglobulinaemia due to an increase in the α_2 and γ components. The first muscle biopsy was normal, but the second showed extensive necrosis of muscle fibres (see Fig. 2).

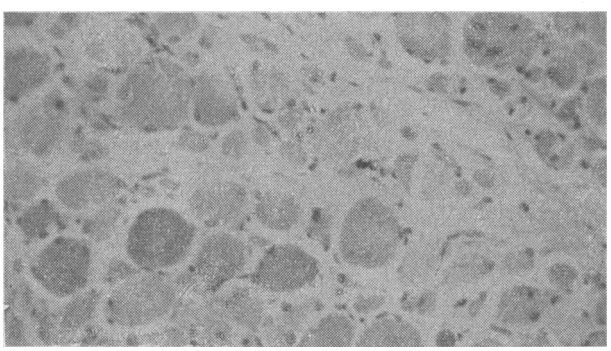

FIG. 2.—Case 2. Section showing extensive necrosis of muscle fibres. (×400.)

Despite treatment with 60 mg. of prednisolone daily and 40 units of A.C.T.H. gel weekly, her condition deteriorated. Dysphagia and weakness of the respiratory muscles developed, complicated by a respiratory infection not controlled by antibiotics. Three weeks after admission a tracheotomy was performed as an emergency measure, and the upper respiratory passages were cleared of much secretion and mucus. The extreme muscle-weakness persisted.

Methandienone was started, 30 mg. daily, two months after admission. After 10 days' treatment a definite and sustained improvement in muscle power became apparent, allowing the removal of the tracheotomy tube. At the time of discharge she was ambulant, and her progress as an outpatient has been maintained. She has returned to full-time teaching and, six months after leaving hospital, remained well. Maintenance doses of prednisolone and methandienone have been continued.

Discussion

Corticosteroids and A.C.T.H. have been the mainstay of treatment of the polymyositis group of diseases, but the results of therapy have been variable and unpredictable, particularly in the subacute and chronic forms (Ragan, 1950; Anderson, 1952; Gross, 1952; Kierland *et al.*, 1952; Wedgwood *et al.*, 1953). As they were the accepted form of treatment, A.C.T.H. and prednisolone were given in adequate doses in both cases throughout. In the first case there was evidence of some remission, but this was not sustained, and attempts to reduce the high dosage of corticosteroids resulted in an acute exacerbation of muscle-weakness. There was steady deterioration in the second case despite corticosteroid therapy, so that emergency tracheotomy became necessary.

We thought it rational to use an anabolic hormone in dermatomyositis because, although the histological picture is largely one of muscle-fibre degeneration and inflammatory cell infiltration, there is also characteristically present regeneration of muscle (Walton and Adams, 1958), and it seemed that the anabolic hormones would aid this process. In addition, their anabolic property would counteract the katabolic effect of long-term corticosteroid therapy.

Testosterone has been tried in dermatomyositis with variable results. Wedgwood *et al.* (1953) stated that testosterone was "possibly helpful" in 10 out of 12 cases that received it, but thought that orthopaedic treatment and physiotherapy were more effective. Where there was rapid destruction of muscle mass, Black and Bunim (1958) employed testosterone in large doses, and found that in some cases there was an increase in weight and muscle strength. On the other hand, Garcin *et al.* (1955), in their review of polymyositis, thought that testosterone was of doubtful value. In addition, the virilizing property of testosterone precludes its long-term use, particularly in females and pre-pubertal males.

In the past three years a number of steroid compounds have been produced which are predominantly anabolic and have little virilizing effect. One of the first of these agents was norethandrolone, but recently methandienone has been claimed to be the most effective anabolic agent orally, and the least androgenic (Foss, 1959).

While corticosteroid therapy was maintained in both our cases, methandienone was introduced, and improvement occurred. In the first patient muscle-weakness regressed in a slow but sustained fashion over a period of months, whereas in the second patient the improvement was more dramatic, and was evident in the first 10 days of treatment. While we may have observed the natural remission of the disease in these patients, its direct relationship to the start of anabolic hormone suggests that it was drug-induced. We consider that anabolic hormones were of value in the cases described and are worthy of further trial in dermatomyositis.

Summary

Two cases of dermatomyositis are described. Although the results of corticosteroid therapy were disappointing, improvement occurred in both cases after the use of methandienone, an anabolic hormone.

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REFERENCES

Anderson, T. E. (1952). *Brit. J. Derm.*, **64**, 73.
Black, R. L., and Bunim, J. J. (1958). *Med. Clin. N. Amer.*, **42**, 1653.
Foss, G. L. (1959). *Brit. J. clin. Pract.*, **13**, 541.
Garcin, R., Lapresle, J., Gruner, J., and Scherrer, J. (1955). *Rev. neurol.*, **92**, 465.
Gross, P. (1952). *Arch. Derm. Syph. (Chicago)*, **66**, 759.
Kierland, R. R., O'Leary, P. A., Brunsting, L. A., and Didcoct, J. W. (1952). *J. Amer. med. Ass.*, **148**, 23.
Ragan, C. (1950). *Proceedings of the First Clinical ACTH Conference, 1949*, edited by J. R. Mote, p. 423. Blakiston, New York.
Smith, H. G. (1955). *Brit. med. J.*, **1**, 770.
Walton, J. N., and Adams, R. D. (1958). *Polymyositis*. Livingstone, Edinburgh.
Wedgwood, R. J. P., Cooke, C. D., and Cohen, J. (1953). *Pediatrics*, **12**, 447.
Wilson, T. G. (1954). *Arch. Middlx Hosp.*, **4**, 151.

SCHISTOSOMIASIS OF THE SPINAL CORD

REPORT OF A CASE

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Schistosomiasis of the cord has been infrequently reported. It is therefore considered worth while reporting a further case.

Kane and Most (1948) reviewed the literature up to 1944. They recorded 24 cases of cerebral complications of schistosomiasis, of which 14 were due to *S. japonicum*. Two authenticated cases of myelitis due to *S. haematobium* and two due to *S. mansoni* had been described during this period. They comment on the tendency of *S. japonicum* to affect the brain and of *S. mansoni* and *S. haematobium* to affect the cord. Since their paper further cases of spinal-cord involvement have been reported sporadically.

Gama and de Sá (1946) record a case operated on for suspected spinal tumour. A cystic swelling at the second lumbar segment was removed from which *S. mansoni* eggs were isolated. The neurological signs were not affected. Raper (1948) records the finding, at necropsy on an African soldier, of a schistosome in a dense scar compressing the thoracic cord. Two years previously the patient had been offered but had refused surgery for a suspected spinal-cord tumour. Gelfand (1950) reported a personal case in which *S. haematobium* eggs were found in the cord. He comments on the frequency in which schistosome ova of both species are found on digestion of the brains of cases of schistosomiasis, but found ova only in one of the 25 cords examined. Ross *et al.* (1952) report a case of *S. mansoni* of the cord discovered at operation for suspected tumour. The patient improved after operation and antimonial therapy. Horrax *et al.* (1958) describe a case of *S. mansoni* granuloma of the cord which was operated on for suspected spinal tumour.

There are at least four further cases of cord involvement with schistosomiasis in the South American literature not available to us.

Case Report

A European man aged 73 had been a pilot in the first world war. After a flying accident in 1918 he had had an arthrodesis of his right hip and an ankylosis of the right knee-joint. Despite this he had been very active and had actually been playing tennis not very long before the present illness. In July, 1958, he fell and sustained severe bruising of his back and left hip. After this his wife thought he did not walk so well and that his left foot turned in more. Before his arrival in Uganda he had spent some time in Southern Rhodesia and then some months in Tanganyika.

Ten days before admission he had motored up 1,400 miles (2,250 km.) from Tanganyika, driving himself all the way. He complained of stiffness and cramps in his legs. He attended the out-patient department. No abnormalities were noted. A white count at this time was normal. Five days before admission he had another fall; he was unable to get up and was helped into bed. He had some difficulty in moving his legs and had pain in his legs and lower abdomen, but attributed this to the fall.

After this he found difficulty in passing urine and had to strain. The same day he had an involuntary movement of the bowels. Two days before admission he developed retention with overflow. The day before admission he became completely unable to move his legs.

On admission he was an elderly alert man. He had a blood-pressure of 180/100. C.N.S.: The cranial nerves and upper limbs were normal. There was a band of hyperaesthesia to pin-prick from D 9 to D 12. He stated that he felt as though he had an iron band round his abdomen. There was a motor paresis of the lower extremities, more marked on the right than on the left, although fixation of the right knee- and hip-joints made testing difficult. There was a loss of all forms of sensation below D 12, except for some sparing on the inner side of the right thigh and left calf, and some diminution of sensation to light touch in D 11 and D 12.

The abdominal reflexes were brisk. The knee- and ankle-jerks were not elicited, nor were the plantar responses. The bladder was distended, with a dribbling overflow, and the rectal sphincter was toneless. The prostate was normal. There was a suggestion of a kyphus at L 3 to 4, and there was tenderness over the lumbar spines. A white count showed 10,000 cells, of which 78% were neutrophils. There were no eosinophils at this time. The C.S.F. showed a normal pressure with a free rise and fall on jugular compression. The fluid was clear, contained 90 mg./100 ml. of protein, and 55 lymphocytes per c.mm.; the sugar was 46 mg./100 ml., and the Khan reaction was negative.

A further C.S.F. three weeks later showed a protein of 10 mg./100 ml., 15 lymphocytes per c.mm., and a sugar of 14 mg./100 ml. This low figure, which was checked, was not due to delay in examining the fluid.

Urine showed no protein, sugar, or abnormal cellular deposit. The test for Bence Jones proteose was negative.