Estimations of renal and muscle K^+ 24 hours after injection of 5-aminosalicylic acid were not significantly different from those in control animals. The incidence of renal damage in these animals was similar to that recorded in the first experiment.

Discussion

These findings show that a single compound with a structural resemblance to both salicylic acid and phenacetin can produce damage to segments of the nephron in both the cortex and the medulla and seem to be relevant to the problem of renal damage associated with analgesic abuse (Spühler and Zollinger, 1953; Kincaid-Smith, 1967).

The sporadic and incomplete incidence of renal lesions in this series of experimental animals, evident at all dose levels, recalls the sporadic incidence of renal damage in patients taking analgesics over long periods. The factors responsible for this variable experimental effect may be of importance in indicating the nature of the processes responsible for the variable incidence of the clinical syndrome. It has been shown that aspirin and phenacetin derivatives, if acutely nephrotoxic to rats, produce necrosis of proximal convoluted tubules rather than renal papillary necrosis (Calder *et al.*, 1971). It is therefore of interest that 5-aminosalicylic acid, which has the structural characteristics of both analgesic groups, should produce a combined lesion of the renal cortex and medulla. Such a pattern suggests that synergism between components or metabolites of combined analgesic preparations may be responsible for the cortical and medullary components of the renal damage associated with analgesic abuse and reinforces previous suggestions (Green, Ham, and Tange, 1969; Calder *et al.*, 1971) that analgesic compounds may have a toxic effect on the renal cortex rather than on the medulla alone.

Droplet formation in the collecting duct epithelium of the tip of the papilla is the only lesion found six hours after injection of 5-aminosalicylic acid. Similar droplets have been found after temporary renal ischaemia (Sheehan and Davis, 1960) and in papillary necrosis produced by ethyleneimine (Oka, 1913), especially when necrosis is confined to a small portion of the papilla (Funder, Ham, and Tange, unpublished). The combination of droplets in collecting duct epithelium, mitoses in collecting duct epithelium in adjacent regions of the medulla, and damage to epithelium in proximal convoluted tubules resembles the lesion produced in rats by experimental K^+ deficiency (Milne, Muehrcke, and Heard, 1957). No evidence for K^+ deficiency was obtained in these animals and it is improbable that such deficiency could have developed so quickly. Thus it is apparent that the experimental renal lesions produced by 5-aminosalicylic acid are not mediated by K^+ deficiency, but it is conceivable that similar disorders of intracellular structure and function may be involved, and this may also prove to be an indication of the processes preceding renal papillary necrosis however produced.

It should not be inferred that 5-aminosalicylic acid is directly concerned in the genesis of renal damage incident to analgesic abuse or that it is necessarily nephrotoxic in man. 5-Aminosalicylic acid is known to be a metabolite of salicyl-azosulphapyridine (Hanngren, Hansson, Svartz, and Ullberg, 1963), and renal lesions have not been noted in a series of patients treated intermittently with this drug (Cuthbertson, 1971) over a 10-year period.

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MEDICAL MEMORANDA

Spontaneous Rupture of Testicular Teratoma

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British Medical Journal, 1972, 1, 154-155

A case of teratoma of a testis presenting as sudden spontaneous rupture of the testis without previous injury is described.

Case Report

An 18-year-old youth was out walking when he experienced a sudden severe, sickening pain in the right scrotum, which started to swell up rapidly. He was taken to hospital and admitted to the general surgical ward by the casualty officer with a diagnosis of ? strangulated right inguinal hernia.

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When seen in the ward the patient was obviously in pain. He was emphatic about the sudden onset of his symptoms and was not aware of any recent injury to the scrotum, though during the previous month he had noticed that his right testicle was larger than the left. The only relevant point in the past history was the information subsequently offered by his parents—namely, that his left testicle was undescended at birth. As they were not concerned about this they had never again checked the position of the testicle.

On examination the left testicle was in the scrotum but was about half the normal size. The right scrotum was swollen, tense, bluish, and very tender but did not look inflamed. The swelling extended to the groin, with pronounced thickening of the cord. The right testicle could not be felt as the patient resented any handling of the scrotum. The most likely diagnosis was thought to be acute torsion of the testicle, and preparations were made to explore the scrotum.

With the patient under general anaesthesia an incision was made in the oedematous right scrotum. A bulging, bluish swelling was exposed. A nick was made with care in the sac and a gush of fresh blood was obtained. It soon became obvious that this was a haematocele. After the blood was evacuated by suction the contents of the scrotum were inspected. There was no evidence of torsion of the testicle, which was of normal colour and sheen though it was enlarged to twice the normal size. The cord was oedematous, but not twisted, with good pulsation throughout. There was a stellate tear in the lower pole of the testis through which fresh, bright red blood was spurting. The testicular core was digitally explored through the rent in the tunica vaginalis and was felt to be cystic and necrotic. In view of the possibility of malignancy it was

decided to proceed to orchidectomy, which was performed through a right inguinal incision, the cord being clamped before division.

The postoperative course was uneventful.

Histologically the "picture of this tumour is in keeping with the Malignant Teratoma Intermediate (Sub Group A) of the Testicular Tumour Panel and Registry of the Pathological Society of Great Britain and Ireland."

Subsequently the patient received a course of radiotherapy to the right scrotum, the iliac and inguinal lymph nodes, and the para-aortic nodes up to the xiphisternum.

He was alive and well with no evidence of metastases 18 months after operation.

Comment

This case illustrates a distinctly rare presentation of an uncommon tumour. Only one other similar case has been recorded in the literature (Cassie, 1956). There are some dissimilarities between the two cases. Cassie's patient presented in the outpatient department with a haematocele two weeks after a trivial injury to the scrotum resulting in a painless rupture of a testis harbouring a seminoma, which had already metastasized to the lungs. The present case presented as an acute emergency, the patient complaining of severe pain on the side of the ruptured testis. The tumour was a teratoma without evidence of metastases. He would not admit any history of injury to the scrotum, not even in retrospect, though tumour involvement may have rendered the testis so insensitive that minor trauma to it may have been ignored.

I would like to thank Mr. D. G. Jenkins for permission to report this case. The histological examination was done by Dr N. J. H. Davidson. Radiotherapy was given by Dr. Ian Bell.

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Dermatomyositis with Parenchymal Lung Involvement

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Primary lung involvement in dermatomyositis is not a well recognized clinical entity, in contrast to its occurrence in other connective tissue disorders such as progressive systemic sclerosis, systemic lupus erythematosus, and rheumatoid arthritis (Beerman and Kirshbaum, 1961). We report here a case of dermatomyositis in which pulmonary manifestations were the presenting feature. A good remission of all symptoms was obtained for nine months with corticosteroid therapy.

Case Report

The patient, a 39-year-old housewife, was first seen in September 1970. She gave a four-month history of cough, sputum, and increasing exertional dyspnoea. For two months she had had generalized muscle tenderness, proximal muscle weakness, and arthralgia affecting the metacarpophalangeal and proximal interphalangeal joints of both hands. During the month before admission she had noticed a transient papular rash on the dorsum of both hands and feet and had Raynaud's phenomenon in the hands.

On examination she was pale and orthopnoeic but not cyanosed. She was unable to sit or stand unaided and there was severe proximal muscle weakness and tenderness but no wasting. Heliotrope streaking and scaling of the skin was seen over the dorsum of the metacarpophalangeal joints of the hands. Her respiratory rate was 28/min, chest expansion ½ in (1·3 cm), and there were inspiratory crepitations with diminished air entry at both lung bases. There was no evidence of joint disease.

Haemoglobin was 12·0 g/100 ml, E.S.R. (Westergren) 60 mm/hr, and latex test for rheumatoid factor, antinuclear factor test, and L.E. cell test were all negative; results of other routine tests were normal. A chest *x*-ray picture showed bilateral widespread interstitial infiltrations. The results of lung function studies are shown in the Table. The serum creatinine phosphokinase was 1,250 (normal <70) mIU/ml, and serum aldolase 37·9 (normal <7) mIU/ml. Muscle biopsy showed necrosis and regeneration of muscle fibres with an associated inflammatory infiltrate of histiocytes, lymphocytes, plasma cells, and a few eosinophils. The features were consistent with myositis.

Treatment with prednisolone 15 mg daily was started on 1 October 1970. Within a few hours of the first tablet of 5 mg she was able to sit up unaided. By the next day her dyspnoea and the abnormal physical signs in the respiratory system had disappeared. This improvement was maintained and she was discharged home after three weeks. The changes in the serum levels of the muscle enzymes and in the sedimentation rate in response to corticosteroid therapy are shown in the Chart. A cut in prednisolone dosage from 15 mg a day to 10 mg a day in January 1971 was associated with the return of slight muscle weakness and dyspnoea. She was maintained symptom-free on prednisolone 15 mg daily. No further respiratory symptoms or signs developed, and though the chest *x*-ray picture remained unchanged the lung function tests continued to improve (see Table). No side effects from corticosteroid therapy were recorded.

Dates and Results of Pulmonary Function Studies

	24/9/70	9/10/70	23/3/71
FVC (l.)	1·33	1·59	1·93
FVC (% predicted value)	43	51	62
FEV_1 (l.)	1·14	1·35	1·64
FEV_1/FVC (%)	85	85	85
Pao_2 (mm Hg)	82·2	94·2	94·1
$Paco_2$ (mm Hg)	33·6	32·4	33·6
pH	7·470	7·444	7·391
$A\text{-}aDo_2$ (mm Hg)	33·9	24	6

Comment

The diagnosis of dermatomyositis was confirmed by the raised serum levels of muscle enzymes and the histological evidence of myositis. The clinical features accorded with type 2 described by Walton and Adams (1958). The lung function studies showed hypoxaemia associated with restriction of the vital capacity but without airways obstruction. The increase in alveolar-arterial oxygen gradient ($A\text{-}aDo_2$) indicated ventilation-perfusion imbalance or diffusion block. Transfer factor for carbon monoxide was reduced to 15·2 ml/min/mm Hg (predicted normal value 19-29) on 9 February 1971, the one occasion when it was measured. The abnormal $A\text{-}aDo_2$ resolved on treatment but the vital capacity remained restricted to 62% of the predicted normal value, possibly signifying some restriction of chest wall movement.

Three types of pulmonary involvement have been recorded in dermatomyositis (Hepper *et al.*, 1964). Primary pulmonary

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