of dyspepsia suggested the diagnosis of a perforated peptic ulcer. This was confirmed by the presence of free gas under the right diaphragm shown on an x-ray film of the abdomen.

At laparotomy a perforated anterior duodenal ulcer was found with established generalized peritonitis. The perforation was closed with three chromic catgut sutures and an omental patch. After toilet of the peritoneal cavity, it was drained by tubes in the right subphrenic space and in the pelvis. After a further 14 hours on the respirator he was transferred to an oxygen tent. Tracheostomy was decided against, as he was by this time breathing well and was able to cough satisfactorily.

The Table indicates the blood gases during the immediate postoperative days. The patient showed steady improvement, but on the sixteenth postoperative day had a complete dehiscence of his abdominal wound. This was resutured under general anaesthesia without complications affecting the respiratory system. Ten days later he developed Friedlander's pneumonia and despite all attempts at resuscitation he died on 3 June. Unfortunately a post-mortem examination was refused.

**Table Showing Blood Changes**

<table>
<thead>
<tr>
<th>Date</th>
<th>Time</th>
<th>Treatment</th>
<th>pH</th>
<th>PO2 (mm. Hg)</th>
<th>Base Excess (mEq/L)</th>
<th>Standard bicarbonate (mEq/L)</th>
<th>PO2 (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>19/5/65</td>
<td>6.30 p.m.</td>
<td>E.R. respiration; Bicarb. 1 litre</td>
<td>7.33</td>
<td>70</td>
<td>65</td>
<td>+13</td>
<td>31</td>
</tr>
<tr>
<td>6/5/65</td>
<td></td>
<td></td>
<td>7.34</td>
<td>65</td>
<td>50</td>
<td>+15</td>
<td>30</td>
</tr>
<tr>
<td>2/5/65</td>
<td>11 a.m.</td>
<td>O2 tent</td>
<td>7.34</td>
<td>80</td>
<td>0</td>
<td>+10</td>
<td>31</td>
</tr>
<tr>
<td>2/5/65</td>
<td>6 a.m.</td>
<td>O2 tent</td>
<td>7.34</td>
<td>65</td>
<td>50</td>
<td>+15</td>
<td>31</td>
</tr>
<tr>
<td>2/5/65</td>
<td>4.00 p.m.</td>
<td>O2 tent</td>
<td>7.34</td>
<td>72</td>
<td>0</td>
<td>+8</td>
<td>31</td>
</tr>
</tbody>
</table>

We would like to think Mr. J. Moroney, under whose care this patient was admitted, for permission to treat and publish this case.

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**REFERENCES**


**Lupus-like Syndrome Induced by Procainamide**


Many drugs have been associated with the onset of a syndrome resembling systemic lupus erythematosus. Among the drugs implicated are hydralazine; sulphonamides; anticonvulsants of the phenytoin group, and trichotone; penicillin; phenylbutazone; tetracycline; streptomycin and isoniazid; griseofulvin; thiouracil derivatives; and procainamide.

Since the first description of a syndrome closely mimicking, if not identical with, systemic lupus erythematosus, and occurring after the administration of procainamide (Ladd, 1962), 13 cases have been reported—12 in patients receiving the drug for cardiac disease (Hahn, 1964; Colman and Sturgill, 1965; Kaplan et al., 1965; Paine, 1965; Carabia and Fortney, 1965) and one in a patient with dystrophy myotonica (Procopk, 1966).

All previous reports have appeared in the American literature.

**CASE REPORT**

A 50-year-old woman attended hospital in May 1965 with symptoms suggestive of angina of effort, and was subsequently found to be hypertensive, with a blood pressure of 260/150 mm. Hg. Investigation showed no cause for this, and a diagnosis of essential hypertension was made. She was initially treated with antihypertensive drugs, but these were withdrawn when she had a myocardial infarction. In September 1965 she developed paroxysmal ventricular tachycardia and was given quinidine sulphate, but because of a drug rash occurring within 24 hours this was replaced by procainamide 500 mg. q.i.d.

In January 1966 she was readmitted to hospital complaining of left-sided pleuritic pain of sudden onset. She had no cough, sputum, or haemoptysis. She had pleural rubs at the left costophrenic angle and signs of a left sided pleural effusion. Her pulse was in sinus rhythm and her blood pressure was 160/100 mm. Hg. E.S.R., blood picture, and blood urea were within normal limits. Serum aspartate aminotransferase and serum lactate dehydrogenase were also normal. X-ray examination showed a small pleural effusion at the left costophrenic angle and marked cardiac enlargement. Though there was no obvious source of emboli, an initial diagnosis of pulmonary embolism was made and anticoagulation was started. Procainamide was continued as prophylaxis against further cardiac arrhythmia.

Initially there was some improvement, but three weeks after admission the patient began to complain of severe pain in the small joints of both hands and in the muscles around the shoulder girdles. Subsequently she had a further episode of left-sided pleuritic pain, followed by pain and a pleural rub on the right side. X-ray examination showed an increase in the left-sided pleural effusion and the presence of fluid at the right base. The E.S.R. was 70 mm. in one hour and the serum gammaglobulin 1.5 g./100 ml. The most likely diagnosis was thought to be multiple pulmonary emboli, and the oral anticoagulants were supplemented by high doses of intravenous heparin. However, no clinical improvement was noted.

The possibility of a collagen disease—a procainamide-induced lupus syndrome—was then considered. Positive L.E.-cell preparations were found on several occasions, the L.E. latex fixation and Jones tests were positive, and the antinuclear factor was demonstrated.
Therefore the procainamide was withdrawn and replaced by propranolol 20 mg. q.i.d. The effect was dramatic. The myalgia and arthralgia subsided; the chest symptoms and signs abated, and X-ray examination showed a clear right lung field within one month, followed by a gradual resolution of the effusion on the left side; the E.S.R. fell to 10 mm. in the hour in two months; I.E.-cell preparations were negative after six weeks, and the antinuclear factor was negative five months later.

When seen in October 1966 the patient was well, and was able to do light housework and take mild exercise.

COMMENT

In the 14 cases reported previously, in which similar reactions occurred, procainamide was given in a dosage of 1-5 g. daily. Symptoms of the lupus syndrome appeared after three weeks to 22 months of therapy. The commonest manifestations were polyarthritis (14 cases), positive I.E.-cell preparations (13 cases), pleuropulmonary symptoms (nine cases), and the presence of antinuclear factor (in all five cases examined). Our patient took 2 g. of procainamide daily for five months before the appearance of chest symptoms.

Apart from two cases that were followed for only seven or eight weeks after procainamide was stopped, all have shown complete recovery within two years. This may represent a difference from the similar syndrome precipitated by hydralazine, in which hypergammaglobulinaemia or antinuclear factor antibodies were found five years after discontinuation of the drug and disappearance of symptoms (Alarcón-Segovia et al., 1965). Recrudescence has been found to follow re-exposure to the drug, but it was not attempted in this case.

There is still much controversy surrounding the mechanism giving rise to this drug-induced syndrome. Some authors Alarcón-Segovia et al., 1965; Lee et al., 1966) feel that the drug unmarks a predisposition to develop lupus erythematosus. Studies in patients with the "hydralazine syndrome" have shown that symptoms and signs possibly due to S.L.E. were present before the drug was given. Patients who at one time had developed clear-cut drug-induced S.L.E. have occasionally been observed to have apparently spontaneous recurrence of the disease years later. There is, however, as yet no case reported of spontaneous recurrence or chronic S.L.E. induced by procainamide.

There are two other possibilities. Firstly, the symptoms could be a manifestation of drug toxicity or hypersensitivity—that is, a lupus—drug reaction. In favour of this a case has been reported (Prockop, 1966) of a patient with established S.L.E. who received full doses of procainamide for 15 months for a myotonic illness with no exacerbations of S.L.E., though there were episodic symptoms long before and long after the period of drug administration. This may be evidence that the lupus-like drug reaction differs from the disease. Secondly, the appearance of S.L.E. during drug administration might be coincidental, but the increasing number of cases reported makes this unlikely.

A review of the cases would suggest that procainamide is used in circumstances—for example, atrial fibrillation, paroxysmal atrial tachycardia, ventricular ectopics—in America where other drugs would be the first choice in Britain. It was only because this patient, with recurrent ventricular tachycardia, was hypersensitive to quinidine that procainamide was used. It would seem that this drug should be prescribed on a long-term basis only when there is no less potentially harmful alternative.

We wish to express our thanks to Professor O. L. Wade for permission to publish this case, and for advice on the manuscript.

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REFERENCES


Hyopoparathyroidism and Malabsorption


The association of secondary hyperparathyroidism with osteomalacia caused by malabsorption has often been noted (Davies et al., 1956). There also seems to be a connexion between hypoparathyroidism and the malabsorption syndrome.

Bennett et al. (1932), in describing 15 cases of "idiopathic osteitis deformans," suggested that five may have had secondary hypoparathyroidism, in view of the finding of a raised serum inorganic phosphorus level. Several cases of idiopathic hypoparathyroidism associated with malabsorption and generalized moniliasis have been described (Collins-Williams, 1950; Salvesen and Bøe, 1953a; Clarkson et al., 1960). A few other cases of idiopathic hypoparathyroidism coexisting with malabsorption alone have been documented in recent years (Lowe et al., 1950; Case Records of Massachusetts General Hospital, 1954; MacGregor and Whitehead, 1954; Jackson et al., 1956; Williams and Wood, 1959; Cochrane et al., 1960; Taybi and Keele, 1962). In a case reported by Clarkson et al. (1960) a flat small-bowel mucosa was found on biopsy, and correction of the hypoparathyroid state converted to normal most of the demonstrated abnormalities of absorption.

CASE REPORT

An unmarried woman aged 58 was admitted to hospital for investigation of an iron-deficiency anaemia which had been present for nine months and had failed to respond to treatment. She gave a history of a "nervous breakdown" followed by depression at the age of 22. A year later she had noticed a progressive deterioration of vision, and was found to have bilateral cataract, one of which was successfully removed at operation.

Clinical examination revealed a pale, nervous woman, with finger-clipping. A postmature cataract was present in the left eye. Neurological examination showed a positive Trousselau's sign, but a negative Chvostek's sign.

Investigations showed a serum calcium level of 5.7 mg./100 ml. and a serum inorganic phosphorus of 6.1 mg./100 ml. Alkaline phosphatase was within the normal range. The serum albumin and serum iron were low, but serum vitamin B12, folate acid, and cholesterol were normal. Tests for faecal occult blood were consistently negative. Faecal fat collection showed a level of 15 g./day, but D-xylene excretion test gave an equivocal result. Radiology showed no bony change, and small-bowel meal and barium enema