other fatal case of purpura was mentioned at a discussion on quinidine therapy (Gold, 1954), but details are not available. Although spontaneous recovery is the rule, it is by no means certain or predictable. In view of the action of cortisone and corticotrophin in suppressing antigen-antibody reactions, there are good grounds for the early use of these drugs in all cases of drug purpura.

CONCLUSIONS

A test dose of quinidine, however small, is contraindicated in patients who have had purpura due to quinidine. Drug purpura should be treated with cortisone or corticotrophin, since the condition is potentially fatal and antigen-antibody reactions appear to be involved.

We are grateful to Dr. R. L. Mackay for permission to report this case and to Dr. A. G. Marshall for the necropsy findings.

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Rheumatoid Arthritis of the Larynx

A male clerk aged 61 had developed rheumatoid arthritis 30 years previously and had received various treatments during this period, leaving him with marked deformity of hands, wrists, and elbows, and rheumatoid nodules over the latter. For some years he had had a productive cough, and chest *x*-ray changes were known to be compatible with some bilateral basal bronchiectasis. For three months he had noticed increasing dyspnoea on exertion and huskiness of his voice; severe dyspnoea and wheezing had supervened for two days before I was asked to see him at his home as a case of "? asthma." He had marked inspiratory and moderate expiratory stridor; laryngeal obstruction was diagnosed and he was admitted to hospital at once.

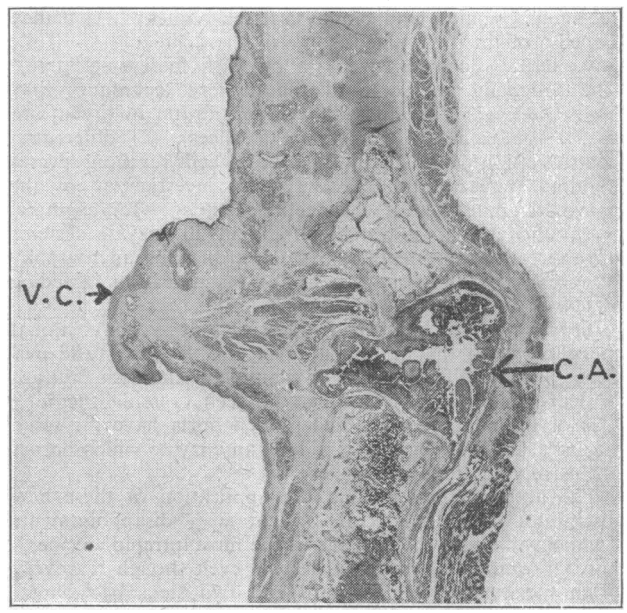
Laryngoscopy and bronchoscopy were performed under local analgesia by Mr. Mearns Milne. The vocal cords showed some whitish nodules on both vocal processes and a very narrow glottic aperture. The bronchial tree contained a large quantity of infected sputum but was otherwise normal. Tracheostomy was performed with great relief to the patient's condition. Biopsy of one of the vocal nodules showed a vascular fibrous core covered by somewhat hyperplastic squamous epithelium; no significant inflammatory reaction was present. Indirect laryngoscopy was later performed by Mr. Douglas Fairman, who reported that the vocal cords showed slight abduction on inspiration so as to produce a very small glottic chink, and they adducted on phonation. He confirmed the presence of nodules on the vocal processes of the arytenoids and the upper surface of the left vocal cord, and he suggested that the laryngeal condition was produced by an ankylosing arthritis of the crico-arytenoid joints.

The patient was thereafter treated by physiotherapy for his bronchial infection, together with catheter suction through his tracheostomy tube, which was later changed to a permanent type. Constant difficulty in expectoration persisted, however, and four months after admission he developed an acute respiratory infection with extensive consolidation in both lower lobes. This failed to respond to treatment, including antibiotics, and he died.

Necropsy by Dr. R. J. Sandry confirmed the cause of death as bilateral basal bronchopneumonia; some bronchiolectasis was present in both lower lobes, but no "rheumatoid" changes were found in the lungs. The laryngeal appearances previously seen were confirmed, and histological section showed, as suspected, changes in the crico-arytenoid joints characteristic of rheumatoid arthritis (see illustration).

COMMENT

This same condition seemed very probable in a patient who recently died at home, with no necropsy. When seen at the age of 83 she gave a 35-year history of rheumatoid arthritis, with which she was completely crippled. Some degree of laryngeal stridor and dyspnoea at rest had been noted progressively for over three years, and her voice had become more husky and deep in tone, rather similar to that in myxoedema, of which in her case there was no evidence. Her general condition and neck posture made even indirect laryngoscopy impossible, so that the laryngeal diagnosis remained unproved.



Longitudinal section of larynx (×4). V.C.=vocal cord. C.A.=crico-arytenoid joint showing villous synovial proliferation.

While crico-arytenoid arthritis has been recorded by laryngologists (Ellis, 1951; de Vido and Ancetti, 1952) it is a rare and scarcely recognized condition in general medical practice. Hart and Mackenzie (1955) encountered laryngeal stridor requiring tracheostomy in a man with chronic rheumatoid arthritis. They considered that abductor palsy was the cause but could not account for this except possibly on a basis of previous deep *x*-ray therapy for thyrotoxicosis. Histologically they reported diffuse fibrosis in the region of the vocal cords, but did not specifically refer to the crico-arytenoid joints. It seems likely that, in their case also, arthritis of these joints was present as an explanation of the findings.

If laryngeal stridor occurs in a case of rheumatoid arthritis, it is suggested that the most likely cause is involvement of the crico-arytenoid joints by the disease process.

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