the scope of the average general hospital in an agricultural area, but exclusion of airway obstruction and finding hyperventilation and possibly cyanosis on exertion may help to clarify a doubtful diagnosis.

After a severe attack recovery is still possible, but some patients are never the same again, and repeated recurrences may lead to the chronic stage, with increasing cellular infiltration of the lung and subsequent fibrosis. Respiratory function gradually fails, and the patient suffers increasing emphysema, pulmonary hypertension, and eventually cor pulmonale. The radiological picture becomes less typical and confusion with other chronic diseases of lung more likely. If the patient has left farming the mouldy-hay antigen test may become negative and the diagnosis, as in the acute stage, depend more on a careful occupational history than objective physical signs.

As in any disease with a natural tendency to remission the effects of treatment are hard to assess. Severe cases respond dramatically to corticosteroids. If recurring attacks are causing progressive disability the farmer must be advised to modify his farming methods (such as changing to open-pit silage) or leave the farm. The use of prophylactic corticosteroids in the winter months may be indicated in a minority of severe cases. Protective masks are of little value, and the Industrial Injuries Advisory Council[2] calls for a sustained programme of publicity to draw attention to the hazards of handling mouldy vegetable produce.

S.L.E. and the Lupus Diathesis

Until the introduction by M. M. Hargraves[1] and his colleagues of the L.E.-cell test systemic lupus erythematosus (S.L.E.) was regarded as a rare disease. But widespread use of the test soon showed that the original impression was wrong and that the disease is rather common.[2] Though it could be argued that there were many false-positive results, careful assessment of the patients in whom L.E. cells were found confirmed that in almost all the diagnosis of S.L.E. was correct. The most notable exception is rheumatoid arthritis, in which a small proportion, varying with the manner in which the test is performed,[3] may give consistently positive results.[4]

Though most people think that S.L.E. is a manifestation of some abnormality of the immunological apparatus—possibly a form of autoimmunity in which the normal immunological tolerance to the body's own antigens is disturbed—there is little evidence at present on how the disturbance arises. It is because of this ignorance that the discovery[5] that an essentially identical clinical picture can be induced by hydrallazine (a drug widely used for the treatment of hypertension) excited widespread interest.

Two diametrically opposite views have been advanced to account for this remarkable relationship between hydrallazine and S.L.E. The first[6] regards the symptoms as a manifestation of drug toxicity or hypersensitivity, and the resemblance to S.L.E. as purely coincidental. The second[7] regards the condition as a true example of S.L.E. in which the latent condition has been uncovered by the action of the drug.

The first is the more widely held view, because of the following arguments, which have been summarized by D. Alarcón-Segovia and his colleagues[8] at the Mayo Clinic. Firstly, the history of these patients does not usually support the presence of a lupus diathesis. Secondly, the syndrome rapidly remits when the drug is withdrawn. Thirdly, involvement of the kidney, a common and serious feature of the true disease, is rare in the induced variety. Fourthly, the same clinical picture can be produced in animals. Lastly, as judged by the incidence of the complication, about 10% of all hypertensive subjects would have the lupus diathesis. Alarcón-Segovia and his colleagues have re-examined these arguments in the light of their experience of 50 cases of S.L.E. induced by hydrallazine compared with 100 hypertensives without this complication. A suggestive personal history was almost six times as common and a suggestive family history more than four times as common in the test group as in the hypertensives. The authors therefore conclude that the previous arguments are invalid and that S.L.E. induced by hydrallazine is indeed a true example of the disease uncovered by the drug.

It is, however, difficult to accept their conclusion that a "lupus diathesis is present in a considerable proportion of patients with hypertension and perhaps in the general population." Patients with S.L.E. are notoriously susceptible to drug reactions, and any random group showing a high incidence of these reactions will contain a high proportion of such patients. Furthermore, renal disease, itself a common cause of hypertension, is also a common feature of S.L.E., which will therefore contribute an appreciable proportion to any group of hypertensives. A special clinic to which the more unusual cases are presumably referred may well see an unduly high proportion of these patients. Finally, the production of a lupoid syndrome in dogs[9 10] treated with hydrallazine is difficult to reconcile with any underlying lupoid diathesis.

[1] Hargraves, M. M., Richmond, H., and Morton, R., *Proc. Staff Meet. Mayo Clin.*, 1948, **23**, 25.
[2] Dubois, E. L., *Ann. intern. Med.*, 1953, **38**, 1265.
[3] *Brit. med. J.*, 1965, **2**, 1347.
[4] Kievits, J. H., Goslings, J., Schuit, H. R. E., and Himans, W., *Ann. rheum. Dis.*, 1956, **15**, 211.
[5] Dustan, H. P., Taylor, R. D., Corcoran, A. C., and Page, I. H., *J. Amer. med. Ass.*, 1954, **154**, 23.
[6] Comens, P., in *Inflammation and Diseases of Connective Tissue*, edited by L. C. Mills and J. H. Moyer, p. 190, 1961. Saunders, Philadelphia.
[7] Dubois, E. L., Katz, Y. J., Freeman, V., and Garbak, F., *J. Lab. clin. Med.*, 1957, **50**, 119.
[8] Alarcón-Segovia, D., Worthington, J. W., Ward, L. E., and Wakim, K. G., *New Engl. J. Med.*, 1965, **272**, 462.
[9] Comens, P., *J. Lab. clin. Med.*, 1956, **47**, 444.
[10] Gardner, D. L., *Brit. J. exp. Path.*, 1957, **38**, 227.

Acute Non-specific Pericarditis

The report of a fatal case of acute non-specific pericarditis[1] brings the recorded number of deaths from this disease to eleven, so we should certainly abandon the epithet "benign" and confess our ignorance of the cause in most cases. The condition is not particularly uncommon. Several large collections of cases have been published,[2-4] mostly in the United States. The largest British series consists of fourteen cases, with one death.[5]

The clinical picture of the ordinary uncomplicated case of acute non-specific pericarditis is usually that of a young man who complains of severe retrosternal pain which is made