to be more likely, in these two cases, that the delayed reaction would have set in whether the operation had taken half an hour or one hour, and that the death from heart failure was a direct result of the strain of the reaction on a weakened myocardium.

As after a blood transfusion of one pint the haemoglobin percentage only rises by 8 to 12, it seems unlikely that "a further factor complicating transfusion reactions is the sudden raising of the haemoglobin percentage of the blood," according to Dr. Leigh Silver's theory.

Professor Baker and Professor Dodds, in connexion with a statement of mine on "our ignorance in regard to the renal changes" in cases of uraemia following blood transfusion, refer me to their experimental and chemical study of the question in the *British Journal of Experimental Pathology* (1925, **6,** 247). I had previously studied this excellent work with great interest, and I regret that lack of space did not allow amplification of my statement with reference to their article.

It seems to me, however, that the exact nature of the substance in the renal tubules still has to be determined, for although by experimental work in rabbits they produced a similar appearance in the kidney their exact analyses were concerned with a substance in the urine. Moreover, as these authors themselves say, a similar condition should often occur in paroxysmal haemoglobinuria—an objection which they explain in the following words: "It is reasonable to suppose that in paroxysmal haemoglobinuria and blackwater fever the amount of haemoglobin set free during any one attack is, as a rule, small compared with that liberated in a patient transfused with incompatible blood." To my mind this is an unreasonable assumption. Further, in some cases of death from uraemia following transfusion, showing the typical appearance of pigmented debris in the tubules, there is no evidence of haemolysis as shown by jaundice, or, in one case, even by van den Bergh tests (Witts, L. J.: *Lancet,* 1929, **1,** 1297; De Gowin, E. L., and Baldridge, C. W.: *Amer. J. med. Sci.,* 1934, **188,** 555; Bordley, J.: *Arch. intern. Med.,* 1931, **47,** 288).

—I am, etc.,

London, W.1, Jan. 4. N. S. PLUMMER.

SIR,—May I correct any misunderstanding that may exist regarding a side issue in my letter on blood transfusion fatalities (*Journal,* December 26, 1936, p. 1333). Transfusion of saline solutions by various methods has, of course, been used for some time; I believe, however, the particular method of blood transfusion described by Drs. Marriott and Kekwick, using more than one donor, to be the first of its kind, in this country or elsewhere.

—I am, etc.,

A. L. LEIGH SILVER.

Medical Department, Royal Arsenal, Woolwich, Jan. 3.

## Prevention of Silicosis

SIR,—If a national industrial health service is ever established silicosis should be one of the first complaints to engage its attention. In the mortality tables for 1935 the number of deaths from "chronic interstitial pneumonia, including occupational diseases of the lung" is given as 367, but that modest figure should not be regarded as an index of the harm done by breathing particles of silica.

The coal miner is usually exempt from this disease, and yet the post-mortem appearance of a collier's lungs indicates that millions of particles of coal dust have become embedded in the lung tissue without any obvious disadvantage. In spite of popular opinion to the contrary, if we exclude deaths from accidents, it cannot be said that a miner's occupation is unhealthy. I write from experience, for I practised among them for twenty-one years. It is true they are shut off from sunlight; but they have many compensating advantages which need not be discussed here. In the mortality figures for the various trades and professions the miner occupies a good position. The statistics show that he lives as long as builders and commercial travellers. The question naturally arises, Why can a coal miner have his air cells bombarded with coal dust and take no harm, while a stone grinder ultimately dies from breathing silica dust? The usual answer is that the sharp particles of silica inflict minute wounds in the lung tissue and germ infection follows. That explanation is no doubt partly true, but it has never wholly satisfied me. I believe the immunity enjoyed from coal-dust penetration is largely due to its antiseptic properties. The bane and the antidote are administered together. When wounds are inflicted by silica there is no accompanying germicide.

If there is any truth in the suggestion just made obviously it might be possible to prevent the more serious consequences which follow the inhalation of silica particles by the simultaneous administration of a germicide. This, I believe, could be accomplished in a natural and unobtrusive way by iodizing the atmosphere breathed by workers who, by the nature of their occupation, are compelled to inhale dangerous dust. I think the plan is worthy of an extended trial in this country, and I would also like to see it tried out in one or two of the South African gold mines, where miners' phthisis takes such a heavy toll of the natives.—I am, etc.,

J. A. GOODFELLOW.

Kenwood, Chesterfield, Dec. 30, 1936.

## Metabolic Factor in Rheumatism

SIR,—Dr. R. G. Gordon in his interesting article on the metabolic factor in chronic rheumatism (*Journal,* December 19, 1936, p. 1243) states his belief that the majority of cases of fibrositis are metabolic rather than infective in origin, and are due to minor degrees of thyroid inadequacy and to sympathetic dystonia, both of which conditions favour the deposition of "irritating metabolites" in certain tissues. That he does not indicate the nature of such disturbing bodies seems to imply that he has not isolated them, and surely they need to be isolated before they can be considered to hold a causative relation to the commonest of all "rheumatic" conditions. For fibrositis, if not universal in its incidence, is at any rate so extremely common that its cause is likely to be found among conditions which operate very widely. Infection is such a cause, and should not be neglected simply because the focal theory of infection often produces disappointing results.

I have put forward evidence to explain why the focal theory is of little practical value in the treatment of fibrositis, not because infection is ever absent but because the theory is inadequate to account for what is really happening. In a short paper on "A New Treatment of Fibrositis" in the *Journal* of February 15, 1936, I suggested that the primary focus is no more than the first stage of the disease, the portal whereby organisms gain entry to the circulation, and that the appearance of fibrositis denotes the further stage in which they have established outposts in the tissues. Such local collections of living organisms are self-sufficient and need no reinforcement for their continued existence or eventual spread to other parts. I further tried to show that the resolution of such "secondary foci" rarely takes place without adequate local treatment, by means of massage in slighter cases, and by means of oil-soluble chemicals and oil-

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