is indicated despite the presence of toxaemia of mild or even medium degree. It is at this stage in these cases when air-bathing and/or ultra-violet light from an artificial source will be found of value, provided that the patient has the necessary powers of response. The publications of Lawrason Brown and Sampson on the treatment of intestinal tuberculosis—apparently an earlier complication in some cases of pulmonary tuberculosis than was formerly realized—would seem to confirm this point of view.

In conclusion, I believe that sunlight treatment (using the term in the broad sense indicated above) has a definite and wide sphere of usefulness in the treatment of pulmonary tuberculosis. For its safe application, however, expert supervision, careful selection of cases, and scrupulous attention to dosage are essential.—I am, etc.,

Montana, Switzerland, Nov. 19th. HILARY ROCHE.

DIET AND PUERPERAL SEPSIS

SIR,—Dr. H. P. Dawson, in your issue of November 21st (p. 965), reciting a farmer's experience with his lambing ewes, opens up a large field of speculative interest to students of human and comparative pathology.

Puerperal fever, whether in the higher or the lower animal, is caused by specific pathogenic organisms; eliminate them, and no such condition could exist. But there are contributory factors which go a long way to assist those aggressive bacteria in the production of disease, and, as this practical farmer suggests, diet is one of them. To attribute it wholly to diet, however, is erroneous. One must remember that the present-day ovine is a phlegmatic animal. It obtains all the food it wants with little or no effort; consequently a plethora of blood and adipose tissue is the order of things. Further, in the case of the pregnant ewe exercise is an effort. When, therefore, parturition takes place, the subject is soft and flabby generally, and the mucosae and underlying structures of the generative system do not escape this fatty infiltration. The passage of a foetus through the vaginal canal, made narrower by an excess of submucosal fat, is bound to cause pressure and consequent cellular shock and depression, giving thereby an opportunity for the normal *saprophytic* organisms of those parts to become *parasitic*. Streptococci (haemolytic and non-haemolytic), *B. coli*, etc., become very virulent as they pass from animal to animal. A septic intoxication is soon followed by a septic infection once the first line of defence has given way, and death soon follows from a generalized septicaemia.

The practical conclusion to be drawn from this line of thought is: let the pregnant animal hunt for her rations, for by so doing she will become "fit," and, being so, more resistant to the bacterial complement she unconsciously harbours. The ewe is not the only animal whose plethora is a drawback to normal delivery, for we find the same obtains in others, the mare and cow in particular. In practice one always finds that those animals most likely to develop septic conditions in difficult deliveries are the full-blooded, high-conditioned ones, even when the most rigorous aseptic measures are adopted; while, *per contra*, if the animal is in hard, firm condition, severe bruising and even laceration of the mucosa itself will be followed by no untoward sequelae.

There is another condition in animals which is greatly dependent upon plethora as an exciting cause. I mean quarter ill (gangrena emphysematosa) in cattle. Speaking generally, this disease manifests itself most prominently in herds which are improving in condition rapidly. The causal organism is an anaerobe (sporulating). In this and in the case of the gravid ewe, why should plethoric subjects be less resistant to bacterial invasion? Is their blood less bacteriotropic? The cellular and humoral elements appear to put up a very poor fight—a negative chemotaxis from the outset. Is there a deficient calcium content or the opposite? If the latter, local injury would bring about early coagulation, and so provide a safe "housing" for the bacterial elements. A speculative hypothesis no doubt, but the primary object I had in view in making these observations was to draw attention to the fruitful scope there is here for scientific team work.—I am, etc.,

Bridgwater, Nov. 27th. W. M. SCOTT, F.R.C.V.S.

MIGRATION AND HEALTH

SIR,—In the *British Medical Journal* of January 3rd, 1931, I asked that the word "sixth" be substituted for "fourth" in the statement "There is no one living in Australia (other than Aborigines) who is a descendant of the fourth generation of Australians" in my paper published in your issue of September 27th, 1930. Dr. A. C. F. Halford of Brisbane now writes to me as follows:

"From what I read in the *British Medical Journal*, I understand you to say that you had not succeeded in finding a sixth generation of Australian-born in our midst. I have much pleasure in sending you particulars of a sixth generation of a vigorous and well-born type, which is not likely to be displaced as the first in the category."

Mr. E. M. Tooth of Brisbane, writing to Dr. Halford, states:

"I happen to be a member of a family in which six generations have been born in Australia, being the great-great-grandson of the Rev. Samuel Marsden, who arrived in Sydney in the year 1793.

"*The First Generation Born in Australia.*—Ann Marsden, born March 2nd, 1793, birth registered Parramatta. Daughter of the Rev. Samuel Marsden. Ann Marsden married the Rev. Thomas Hassall.

"*The Second Generation Born in Australia.*—Rev. J. S. Hassall, born October 12th, 1823, who married Miss Oxley, niece of Surveyor General Oxley.

"*The Third Generation Born in Australia.*—Robert Hassall, born April 12th, 1855.

"*The Fourth Generation.*—Emily Hassall, born July 17th, 1880. Now resident in Sydney.

"*The Fifth Generation.*—Everil Bloomfield, born May 24th, 1903. Resident Bundaberg, Queensland.

"*The Sixth Generation.*—Richard Bloomfield, born March 15th, 1929."

The amended statement, "There is no one living in Australia (other than Aborigines) who is a descendant of the sixth generation of Australians," is still correct, as Richard Bloomfield, who is the sixth generation, at the time of writing is only 2½ years of age. It is unlikely that there will be a seventh generation before 1950.—I am, etc.,

October 7th. J. S. PURDY, M.D.
Metropolitan Medical Officer of Health, Sydney, N.S.W.

LONDON CLINIC AND NURSING HOME

SIR,—The London Clinic and Nursing Home is now nearing completion, and it is hoped that it will be open for the reception of patients early in January. In view of the dimensions and novelty of the undertaking, we regard it as of the greatest importance that its aims should be clearly understood, and the means by which it is hoped to achieve them.

The nursing home will contain 200 separate rooms, and these will be available for the patients of any registered medical practitioner, who may look after his patients and call in the services of any consultant with precisely the same freedom as he would enjoy in any ordinary nursing home. He will, in addition, have the advantages of all the resources of a modern hospital. It is, of course, obvious that the use of the operating theatres for major surgical procedures must be limited to surgeons on the staff of recognized hospitals. The charges for the rooms will range from seven guineas a week, according to the