concentration, both by condensation and by elimination of diluents should be therapeutic endeavours. It has been determined that one of the most important factors of vitiated air of overcrowded interiors is the humidity resulting. Here, however, we also have an excess of carbonic acid gas and a corresponding deficiency of oxygen. The sum of it all, however, is suboxygenation susceptibility.

The role of rarefied and moisture impregnated atmospheres is not far to seek. The oxygen volume conveyed to the pulmonary areoles per unit of respiratory activity, is diminished correspondingly to the atmospheric rarefaction and to the absolute humidity. Taken separately, the rarefaction of air, independent of moisture, as encountered at high altitudes by aeronauts and mountain climbers, is a pathogenic factor in ratio to the rarefaction obtaining. For example, 100 per cent. increase of rarefaction is equal to 50 per cent. reduction in the aeration of the lungs per unit time and respiratory effort.

The role of humidity, however, can never be reckoned with independently of the degree of atmospheric heat. We have seen wherein atmospheric rise of temperature involves a rise of humidity in increasing ratio, whereas the fall does not occur in any fixed relation, but depends upon extrinsic conditions, such as winds, season of the year, etc.

Moreover, humidity exerts less pathogenic (suboxygenative) influence, apart from that of atmospheric rarefaction, for the obvious reason that the oxygen molecules of the air owe their separation to temperature and not to the aqueous vapour that interposes them. However, when condensation by cold is produced, and the absolute humidity is highest, the humidity, *per se*, is a potent factor of the essential cause of the dilution of the air by vapour.

A comprehension of the above tedious effort at definition of the atmospheric factors, gives us a basis of understanding the mode of operation of these factors in pathogenesis.

Hot weather and hot climates exhibit a two-fold effect upon susceptibility to infection. Firstly, the *direct* effect of the heat upon the body tissues, in rarefying them and activating considerable fall of tissue density; secondly, in the *indirect* effect exerted to the same end, by virtue of suboxygenation through respiratory insufficiency.

The Heredity Factor.

The direct transmission of tuberculosis by heredity does not occur. Predisposition to infectious disease, but not to tuberculosis in particular, is involved in all congenital cardiac disabilities, narrow and flat chests, narrow arteries, etc. Suboxygenation from the above and all other congenital malformation factors predispose to infection in general. Low tissue density, both general and local, is inheritable, and as such plays its role in susceptibility, but is subject to treatment (hardening processes).

The Bovine Origin of Human Tuberculosis.

The writer points out that in the nation-wide organised crusade against tuberculosis, the milk source of the disease, of bovine origin, has received attention. The cow has been recognised as an extremely highly infected animal, as one of the most prolific sources of disease in milk consuming man, yet little or nothing has been done or agitated to safeguard the cow against the disease.

An example of the "penny wise and pound foolish" procedure has been the wholesale slaughter of animals throughout the country, thus creating almost a beef and milk famine, and certainly exorbitant prices of flesh and milk as foods, by virtue of the scarcity artificially produced, and yet nothing of any perceptible moment has been done to prevent the contraction of tuberculosis by the cow. The writer shows that the conditions of cows while in winter quarters in relation to food, air, light, and sanitation, are universally such as to invite debility, glandular disease, susceptibility to infection, and to the facilitation of the infection *per se*. Yet no notice has been taken of it by the medical men who have the crusade against tuberculosis in charge, by departments of health or milk commissions.

The *clean* milk propagandist also, we are told, overlooks the fact that the cow is the captive and slave of man. During the summer months when she shifts for herself her life is generally healthful; conversely from October to May she is kept in a shed in close confinement, with vitiated and sub-oxygenated air, deficient sunlight, practically no exercise, and on food deficient in the nitrogenous elements. Such food and hygiene is recognised as productive of scrofulosis, glandular disease and tuberculosis in man, yet the cow with continuous overlapping pregnancies and lactations, is ignored, and though perhaps now daily curried and washed, and milked by white garbed milkers, she does not fail to transmit her disease to the consumers of her milk, in toxins, if not in tubercle bacilli.

Just so long as this essential factor is ignored, infants will continue to die in prodigious numbers during the milk feeding period, and scarlet fever, scrofulosis and other glandular diseases and tuberculosis will be

previous page next page