marked increase in turbidity. At weekly intervals, over a period of 11 weeks, 20 cc from each flask were passed through a Seitz filter under a pressure of about 20 mm of mercury. Just immediately preceding filtration, preparations were made and stained to determine morphological features and acid-fastness of the bacilli. Two cc portions of the filtrates were transferred to glycerine-agar slants in tubes plugged with paraffined cotton and topped with tinfoil to prevent drying of the medium, and incubated at 37.5°C. At no time was there macroscopical or microscopical evidence of growth over the entire period of observation (3 months).

Summary. It would appear from the work of others that a filtrable form of *B. lepræ* exists *in vivo* for the respective bacilli of human and rat leprosy, since *in vitro* growth is said to have occurred from the cultured filtrates. Employing filtrates of cultures of *B. lepræ chrome* (Duval) we have been unable after repeated attempts to demonstrate a filtrable stage of this microörganism.

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Variations in Pulmonary Vital Capacity in Health: Daily, Seasonal, and at Moderate Altitudes.

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A. Daily and Seasonal Variations. Occasionally writers draw various deductions from small alterations in the vital capacity of their patients. Anyone, however, with any experience in spirometric work is aware of the daily variations in healthy people, which may amount to several hundred ccm. These are usually disregarded or explained as due to temperature and pressure. I am not aware, however, of any observations of such variations over prolonged periods.

When air is taken into the lungs it is not only heated to a temperature close to that of the body but it becomes charged with water vapor. When this air is expelled with all the forces of expiration into a spirometer at ordinary room temperature and pressure we would expect: (a) A contraction of volume with perhaps condensation of water vapor resulting in a spirometric reading less than the true vital capacity, an error which would increase with falling temperatures, and (b) the final volume of the mass of air forcibly expelled into the spirometer to vary inversely as the atmospheric pressure opposing its expulsion.

Observations of vital capacity (largest of 3 trials), laboratory temperature and barometric pressure were made several times each month for a year in 2 individuals, and for a part of the year in 6 others. Fig. 1, showing the results over a period of 7 months, is typical of all. (These observations were made in Melbourne, Australia, hence the inversion of the temperature record between February and August.)

Results. (1) The daily and the general directions of the vital capacity curve follow that of the temperature curve, swinging down in winter to rise again the following summer. This is not in agreement with the statement of Griffith, *et al.*,[5] that "neither the vital

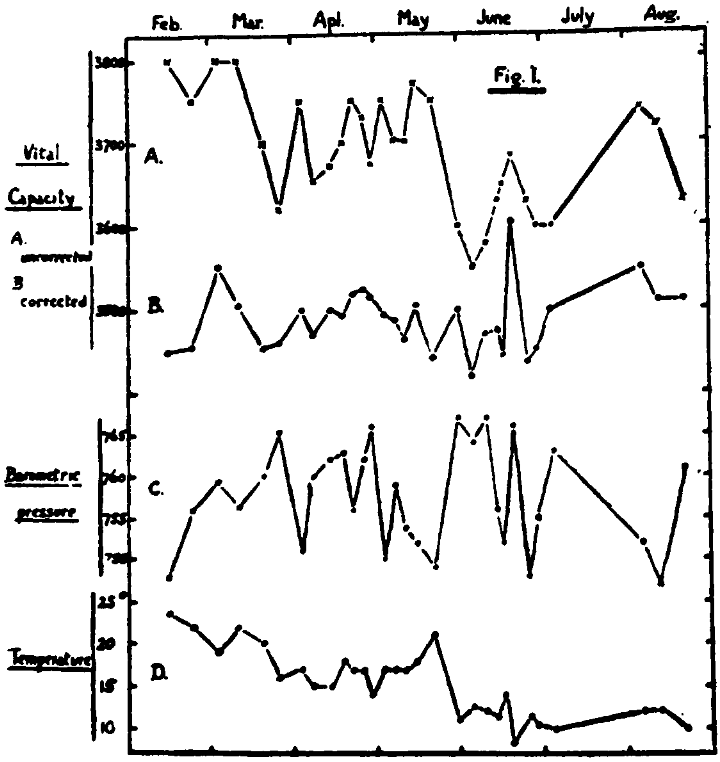

FIG. 1.

Variations in pulmonary vital capacity, barometric pressure and temperature over a period of 7 months in one subject. Curve B represents the results after correction of curve A for temperature and barometric pressure.

[5] Griffith, F. R., Pucher, G. W., Brownell, K. A., Kline, J. D., and Carmer, M. E., *Am. J. Physiol.*, 1929, **89**, 555.

capacity nor the respiratory rate . . . . has a seasonal variation." (2) The daily vital capacity volumes vary inversely as the barometric pressure, these two curves being almost the mirror image of each other. (3) When we correct for variations due to temperature and pressure, reducing all volumes to standard conditions, (0°C and 760 mm Hg) we find the volume curve levels off at about 3500±50 ccm. It is probable that these variations are due entirely to experimental error, since the shape of the curve is now entirely independent of temperature or pressure, *i. e.,* there cannot be any real changes in lung capacity due to either of these factors. This is in contrast to our results in section B, below.

B. Variation at Moderate Altitudes. A number of observations were also made on a group of 4 individuals before, during and following a one-day visit to an altitude of about 3000 feet, and on another group of 6 people during a 9-day sojourn at 6000 feet. The journeys were made by automobile, partly to avoid possible errors due to exercise and fatigue. The same spirometer was used on all occasions. Since the 2 groups were partly composed of different

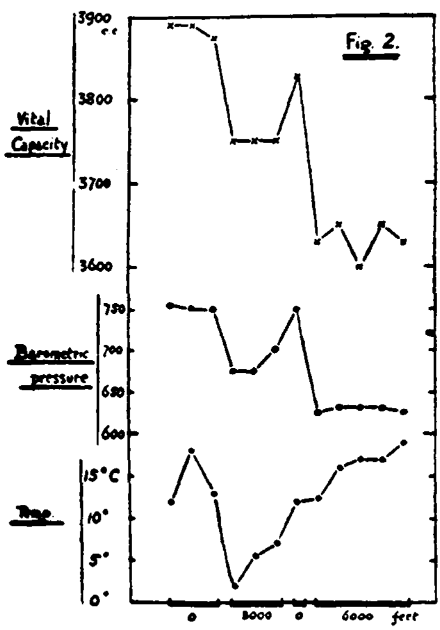

FIG. 2.
Variations in pulmonary vital capacity, barometric pressure and temperature at sea-level, 3000 feet and 6000 feet respectively, in one subject. (Note that the divisions of the barometric scale correspond to 50 mm Hg, *i. e.,* are 10 times those in Fig. 1.)

people it is not possible to compare their average figures. Fig. 2 shows the results in one individual who accompanied both groups, and his results are typical of all.

Results. Although, as shown in section A above, vital capacity volumes varied inversely as the small variations in barometric pressure at sea-level, we now find that vital capacity varies directly with the much larger changes in barometric pressure due to moderate altitudes. (Note that the barometric pressures are plotted in Fig. 2 on a scale 10 times greater than those in Fig. 1.) Furthermore, at 6000 feet there was a rising temperature, a condition which at sea-level we found to increase spirometric readings. At this level the vital capacity falls to 3600-3650 cc which, at standad conditions is reduced still further to 2800-2900 cc, a large reduction in oxygen supply.

The diminution of vital capacity at high altitudes has been noted by many others.[1, 6, 8] Zuntz[10] believed it to be due to expansion of intestinal gases, but Schneider[8] showed that there is no increase of abdominal girth. Durig[3] blamed fatigue of respiratory muscles and Fuchs[4] regarded it as the result of increased muscular tone due to cold. The last 2 possibilities were certainly absent in our experiments. Mosso's view,[7] that there is a dilatation of lung capillaries with some congestion and consequent diminution of air space, seems best to fit the facts. Thus a diminution of vital capacity can easily be brought about by lying down, or bandaging the limbs, and conversely vital capacity can be increased by subjecting men to increased atmospheric pressure (Schilling[9]) and by venesection (Budelmann[2]). Schneider,[8] experimenting with subjects in a low pressure chamber, believes that no fall in vital capacity occurs until the pressures fall to the equivalent of 10,000 to 15,000 feet, and further, that part of the effect is due to an anoxic loss of vascular tonus. Our observations, however, show a decreased vital capacity at 3000 and

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1 Bert, P., *La pression barométrique*, Paris, 1878.

2 Budelmann, G., *Klin. Wochschr.*, 1937, **16**, 704, 1711.

3 Durig, A., *Physiologische Ergebnisse der im Jahr 1906 durchgeführten Monte Rosa Expedition*, Vienna, 1909.

4 Fuchs, R. F., *Sitzungsb. Physick. Med. Soz., Erlangen*, 1908, **40**, 238.

6 Grollmann, A., *Am. J. Physiol.*, 1930, **93**, 19.

7 Mosso, A., *Life of Man on the High Alps*, London, 1889.

8 Schneider, E. C., *Am. J. Physiol.*, 1932, **100**, 426.

9 Shilling, C. W., Hansen, R. A., and Hawkins, J. A., *Am. J. Physiol.*, 1935, **110**, 616.

10 Zuntz, N., Loewy, F., Müller, F., and Caspari, W., *Höhenklima und Bergwanderungen*, Berlin, 1906.

6000 feet in all subjects, at which altitudes the question of anoxia can hardly arise. The simplest explanation is that the fall of atmospheric pressure, by removing some of the external support of the pulmonary capillaries, allows dilatation thereof, resulting in a larger volume of blood and a smaller volume of air in the lung. Such capillary dilatation, although increasing the surface of blood exposed to alveolar air, diminishes the surface: mass ratio for pulmonary blood as well as the vital capacity. Blood examinations, however, show that both of these are well compensated by increased ventilation.

It is possible that the increased mass of blood in the lungs may be one of the factors favorably influencing certain forms of pulmonary tuberculosis at moderate altitudes.

Summary and Conclusions. 1. Apparent variations in pulmonary vital capacity are found from day to day and from season to season. When corrections are made for temperature and atmospheric pressure, these variations disappear. 2. True vital capacity diminishes at moderate altitudes, changes appearing even at 3000 feet. The various factors to which these changes have been attributed by different observers were absent in our experiments. Diminished vital capacity at altitudes is believed to be due to dilatation of lung capillaries following the partial loss of external (atmospheric) support, with consequent diminution of air space. Such an increase of blood in the lungs may be a factor in the favorable effect of moderate altitudes on certain forms of pulmonary tuberculosis.

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Variability of Tubercle Bacillus. I. Mucoid Phase of the Human-Type Strain H-37.

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The characteristic attributes of both the rough and the smooth phases of the human tubercle bacillus have been amply recorded, but there has been no indication of the existence of a mucoid phase such as occurs in the dissociative pattern of many other microörganisms.[1] The isolation and subsequent growth of a mucoid phase in the human tubercle strain, H-37, is therefore briefly presented.

1 Hadley, P. B., *J. Inf. Dis.*, 1937, **60**, 129.