But much more important to the seeker after health than formal resorts are the devices which Canadians themselves employ. In the vicinity of Montreal are numberless small camps, owned by groups of men, where they are in the habit of going to recuperate. Life in the woods is extremely simple, and it is a sovereign remedy for disturbances of the digestive and nervous systems. Canadians are a kindly people, and any visitor with some slight introduction can easily gain admission to these silent places.

LABB AND SALOMON.. "Les Anémies Pernicieuses." *Revue de Médicine,* April and May, '08.

This paper, of sixty-four pages, which has recently appeared in the *Revue de Médicine,* is an exhaustive study on Pernicious Anæmia, and one worthy of careful perusal by all interested in clinical medicine. After a short historical sketch from Biermer's first description (1868) to the recent publications of Hayden and others of the French school, the authors conclude that pernicious anæmia is not a specific entity but a clinical syndrome of varying etiology.

Etiologically, the disease can be considered as "Phenerogenetic," or of evident origin and cryptogenetic or of concealed origin. Under the former he groups, (1) repeated hæmorrhage (gastric, uterine, nasal and vesical), (2) intestinal parasitis (Botheriocephalus and anchylostoma), (3) malaria, (4) bacterial infections, (5) tuberculosis, (6) syphilis, (7) cancer, especially gastric, (8) gastro-intestinal disorders and auto-intoxications, which are said to be the cause of the so-called idiopathic cases, (9) nephritis, (10) pregnancy, (11) lead, (12) carbon monoxide, arsenic and opium. The factors necessary for any of the above conditions to result in this syndrome are, (i) an excessive intensity of the morbid cause, (ii) the localization of the infection, (iii) the duration or repetition of the cause, (iv) an accumulation of the morbid condition, (v) predisposition.

To sum up, they conclude that "Progressive Pernicious Anæmia can be the final stage of secondary anæmias..... if there be an insufficiency of blood repair."

The anæmic symptoms—pallor, dyspnœa, œdema, etc., predominate. As pointed out by Kraus, the cardiac area may be increased on percussion and in the radiograph, due to a dilatation of one or both ventricles, or to a relative mitral or tricaspid insufficiency. Attention is called to the "Pseudo-tabetic type," described by Lichtheim, Déjerine and others, in which there are paræsthesias, less commonly hypo-or hyperæsthesias, paralysis or ataxia, a gait resembling that of a convalescent from a severe illness, diminished or absent tendon reflexes and retinal hæmorrhages; post-mortem one finds capillary hæmorrhages or sclerosis