Leprosy in Iceland.

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LEPROSY is said to have been prevalent in Iceland in the Middle Ages, but the exact date of its introduction or from whence it came is not known. One theory is that the Norwegians brought the disease into the country when colonising Iceland, another is that during the Viking invasion of Great Britain and Ireland, the Vikings captured a number of Irishmen and took them back to Iceland, and it is thought that these Irishmen caused the introduction of the disease. Leprosy is mentioned in the Icelandic writings dated about the 12-14th Century, but always in connection with the lives of saints. It is related that at the beginning of the 15th Century, one of the two Icelandic Bishops was ordered by the Pope to resign his position on account of his having developed the disease. The description of leprosy in the Pope's letter was probably the first to appear in Norway. This Bishop came from Bergen, so whether he brought it from there or developed it in Iceland cannot be said.

About the middle of the 16th Century, the disease had spread considerably and during the years 1652-54 four small hospitals were built. These hospitals were far from

perfect, and in all had room for only 30-40 patients. These hospitals were shut up in the year 1848 after having been used for 200 years, as the disease had diminished so much that they were often empty, or only half full. The theory that leprosy was a hereditary disease had now been superseded by the theory of infection. These hospitals were evidently closed at the wrong time, as leprosy commenced to increase rapidly in the last half of the 19th Century. This was further substantiated when Armour Hansen discovered the leper bacillus about 1870.

At the end of the year 1896, 250 patients were known but probably there were a few more. In 1898, laws relating to compulsory segregation of lepers were passed and a hospital was built near Reykjavik with 60 beds. A well known Danish doctor, Elhers of Copenhagen, induced the Danish Oddfellows to build this, and it was opened for patients on October 1st, 1898. The Icelandic State has run this hospital ever since. As the following statistics show, the disease has diminished considerably. The statistics from 1897-1900 are very imperfect.

Dec. 31st.	1896	..	250	lepers	Dec. 31st.	1915	..	78	lepers
,,	1901	..	169	,,	,,	1916	..	77	,,
,,	1902	..	163	,,	,,	1917	..	77	,,
,,	1903	..	158	,,	,,	1918	..	73	,,
,,	1904	..	145	,,	,,	1919	..	70	,,
,,	1905	..	130	,,	,,	1920	..	67	,,
,,	1906	..	123	,,	,,	1921	..	61	,,
,,	1907	..	110	,,	,,	1922	..	60	,,
,,	1908	..	104	,,	,,	1923	..	59	,,
,,	1909	..	104	,,	,,	1924	..	56	,,
,,	1910	..	96	,,	,,	1925	..	53	,,
,,	1911	..	90	,,	,,	1926	..	51	,,
,,	1912	..	93	,,	,,	1927	..	46	,,
,,	1913	..	90	,,	,,	1928	..	40	,,
,,	1914	..	85	,,	,,	1929	..	37	,,

As a result of the war being waged against the disease, the number of patients was reduced from 250 in 1896 to 37 in 1929, and during this time the population increased from 75,680 to 106,000.

During the first five years after the hospital was opened (1896-1901), the decline in the number of patients was only 81 (from 250 to 169), but it must be remembered that up to the date when the leper hospital was opened the lepers had received little or no treatment, and at first not nearly all the patients in need of nursing could be taken in. The worst cases were given precedence and especially those in the nodular stage of the disease. Many of the lepers were in a terrible state on entering the hospital. The segregation

of these patients was probably a great factor in hindering the spread of the disease.

During the years 1898-1904 the same experiments in treatment, as were being carried out elsewhere, were used at the hospital, namely the use of salicylates, mercury, arsenic, but the results were not good. The patient became worse and generally died as a result of some complication, *e.g.*, sepsis or amyloid degeneration, tuberculosis, pneumonia, etc.

Some of the chief leprologists about the year 1902 spoke favourably of the old eastern remedy for leprosy, chaulmoogra oil, and I decided to try it, and have used some derivative or other of the oil since 1907, *e.g.*, oil by mouth, ethyl esters, hydnocarpates, etc. The improvement in the condition of the patients was enormous, and their whole aspect towards treatment changed and this resulted in a checking of the virulence of the disease, fresh eruptions and reactions were less frequent, leprous nodules cleared up, and diffuse infiltration diminished, and often disappeared totally. The torpid ulcers of long duration became clean and healed, though the complete healing took a considerably longer time, and changes could be detected after a few months of treatment. These changes were especially noticeable in nodular lepers and mixed cases, but I never noticed any improvement in the pure anæsthetic form of the disease.

It should be noted, however, that most of the pure nerve cases had suffered from the disease for years, and that it had become naturally arrested. The patients were already deformed before treatment commenced.

In Iceland we have little experience of the efficacy of treatment on the early case of nerve leprosy. Chaulmoogra preparations appear to have little, if any, effect on trophic ulcers. The obstinate perforating ulcer can be healed with usual treatments, especially if the patient is confined to bed for weeks. This is a difficulty for patients object to staying in bed, and when they get up the ulcers recur. Ulcerations are nearly always found where pressure is greatest, *e.g.*, the metatarso-phalangeal articulations. The most efficient method of treatment is amputation of the toes in question with the head of the metatarsal bone. If ulceration recurs it is sometimes due to the fact that the head of the metatarsal bone has been left. A perforating ulcer of the heel is most persistent and difficult to heal because little radical surgery apart from an extensive operation can be attempted.