patients treated for hyperthyroidism with radioiodine will prove to be significantly higher than the occurrence of leukemia in the general population.

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References and Notes

1. W. C. Moloney and M. A. Kastenbaum, *Science* 121, 308 (1955).
2. H. S. Kaplan, *Cancer Research* 14, 535 (1954).
3. Much of the material contained in this communication was presented at the Clinical Research Session of the New York Academy of Medicine on 26 Jan. 1955.
4. S. M. Seidlin, *et al.*, *J. Clin. Endocrinol.* 9, 1122 (1949); S. M. Seidlin, *Med. Clin. N. Amer.* 36, 663 (1952).
5. This investigation was supported in part by a research grant from the National Cancer Institute, National Institutes of Health, U.S. Public Health Service. The I^{131} used since 1947 was supplied on allocation from the Isotopes Division, U.S. Atomic Energy Commission, by Oak Ridge National Laboratory. We are grateful to D. Laszlo, chief of the Neoplastic Division, Montefiore Hospital, for his advice and suggestions relating to the preparation of this report. We wish to acknowledge the excellent technical assistance rendered by Morris Hodara.
6. S. M. Seidlin, A. A. Yalow, E. Siegel, *Radiology* 63, 797 (1954).
7. J. Furth and O. B. Furth, *Am. J. Cancer* 28, 54 (1936).
8. H. C. March, *Am. J. Med. Sci.* 220, 282 (1950).
9. J. Delarue, M. Tubiana, J. Dutreix, *Bull. Cancer* 40, 263 (1953); P. S. Blom, A. Querido, C. H. W. Leeksma, *Brit. J. Radiol.* 28, 165 (1955).
10. W. C. Moloney, *N. Engl. J. Med.* 253, 88 (1955).

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24 October 1955

Palynological Study of Pleistocene Deposits on Banks Island, Northwest Territories, Canada

In connection with palynological studies made on a variety of Pleistocene deposits in Canada, I have examined several samples from Banks Island, District of Franklin, for plant microfossils.

The locality from which the samples were collected is in the general vicinity of Cape Kellett along the western shore of the Banks Island, approximate lat. 72°N, long. 120°W. According to Pleistocene geologists, this area was not glaciated during the Pleistocene time. However, deposits associated with glaciations cover this part of the island and form a shore cliff more than 100 ft high at the sampling locality. The fine plant debris occurs as lenses and streaks in the stratified gravels and silts.

The samples were collected from these beds of plant debris. The results obtained from palynological studies of two of the samples (No. 4 and No. 17) are given in Tables 1 and 2. In addition to the pollens listed in Table 1, fossil remains of fungus, fragments of bark and woody tissue, stomata of coniferous trees, and spores of mosses were also identified in sample No. 4. In sample No. 17 (Table 2) were further identified the spores of *Sphagnum* and stomata, fungus remains, and fragments of brown mosses. Among the nonarboreal pollens were identified pollen grains of Ericaceae, Caryophyllaceae, Cyperaceae, Gramineae, Polemoniaceae, and two pollen grains of *Ephedra* sp.

The palynological study suggests that, at the time when the beds from which the samples were collected were deposited, considerably more favorable climatic conditions than those now prevailing must have been present on Banks Island to account for the assemblage of pollen grains, spores, and other plant fossils present in these deposits. The total assemblage and relative numbers of pollen grains further suggest local forest coverage. The present timber line lies about 200 mi southwest of Banks Island.

The presence of pollen grains of *Ulmus*, *Tilia*, and *Carya* as well as *Tsuga heterophylla* made me think of the possibility that some of these pollen grains may have been transported by wind from a distant locality several hundred miles away. Even if that is true, these trees must have had a much wider distribution in earlier Pleistocene time than they do now. The lithological character of the material (gravel and silty sand) suggests rather rapid sedimentation, in which case the very low number of grains of tree pollen that would be transported by wind from a distant locality would not enter strongly into the assemblage of pollen grains and spores. This is also shown by the fact that the number of pollen grains and spores per unit volume of material is high. The high frequency cannot be described as primarily the result of a slow accumulation of pollen grains and spores transported from a distant locality by wind. The possibility of contamination seems unlikely, for several other samples that were analyzed at the same time did not yield any pollen grains at all. In addition, particular care was exercised to avoid contamination during analysis.

Of special interest is the discovery of pollen grains of *Ephedra* sp. in this material (Fig. 1). For identification, the fossil pollen grains were compared with modern reference material and descriptions of *Ephedra* pollen in palynological texts and with photographs and descriptions of modern and fossil *Ephedra* pollen given by Andersen (*1*).

The present distribution of *Ephedra* is limited to the southern parts of the Rocky Mountains (*2*). However, pollen grains of *Ephedra* have recently been found in the early postglacial sediments in the Great Lakes region by me and by Andersen (*1*). As also pointed out by Andersen (*2*, p. 19) *Ephedra* is not specific in its thermal requirements and is able to exist in edaphically favorable localities with strong isolation. In spite of that, the presence of *Ephedra* on Banks Island must involve considerable migration of the plant. It seems more likely that the species previously had a much wider distribution and that the successive Pleistocene glaciations eliminated it within the reach of the ice sheets.

As a conclusion, I suggest, on basis of palynological studies, that the long, warm interglacial periods such as Sangamon, Yarmouth, and Aftonian are probably represented by accumulation of organic deposits in the Far North and that further studies may disclose a much fuller sequence of Pleistocene deposits in the northern regions, outside the maximum extent of the Pleistocene ice sheets, than has been expected so far.

Table 1. Analysis of sample No. 4. This sample was collected from a bed of plant debris that was approximately 30 ft above sea level and 6 mi north of Cape Kellett.

Plant	Pollen grains identified (No.)
Picea	28
Pinus	43
Betula	32
Alnus	Abundant (local overrepresentation)
Tsuga heterophylla (?)	10
Ulmus	2
Tilia	2
Carya	1
Nonarboreal pollen, unidentified	17
Ericaceae	2
Polypodiaceae	1

Table 2. Analysis of sample No. 17. This sample was collected from a lens of plant debris that was approximately 20 ft above sea level and 7 miles east of Cape Kellett.

Plant	Pollen grains identified (No.)
Picea	6
Pinus	11
Betula	17
Alnus	16
Tsuga heterophylla	1
Tsuga mertensiana	1
Carya	3
Ulmus	1
Salix	1
Compare *Fagus*	1
Nonarboreal pollen, unidentified	32

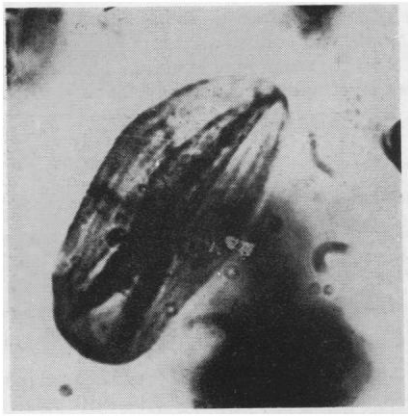

Fig. 1. Photomicrograph of a pollen grain of *Ephedra*, found as fossil in Pleistocene deposits on Banks Island. Longest axis of the pollen grain measures 48 μ.

Phytogeographical studies also would benefit by further palynological investigations, for these investigations greatly help to trace the migrations of plants during the past.

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References

1. S. T. Andersen, *Danmarks Geol. Undersøgelse II*, No. 80 (1954).
2. H. C. Cutler, *Ann. Missouri Botan. Garden.* 26 (1939).

17 October 1955

Conduction Block in Peripheral Nerve Produced by Oxygen at High Pressure

Paul Bert demonstrated inexcitability of frog sciatic nerve-muscle preparations that were exposed to 15 "superoxygenated atmospheres" for 2 hours (*1*). Hill and Macleod (*2*) and Bean and Bohr (*3*) extended these studies, confirming the toxic action of oxygen at high partial pressure (OHP) on the nerve-muscle preparation. Since the toxic effects noted in the nerve-muscle preparations (progressively decreasing contractile response to stimulation of the nerve and disappearance of the treppe phenomenon) could be attributed to either muscle, myoneural junction, or nerve dysfunction, singly or in combination, they offer no proof of neuronal poisoning by OHP. Accordingly, we have investigated the effects of OHP on peripheral nerve alone as a functioning unit.

Twenty-six experiments were performed on frog sciatic nerve. The nerves were excised, placed in a phosphate-buffered Ringer's solution (*4*), and suspended 1 hour later on an array of silver electrodes in a plastic block. This was placed in a pressure chamber (2 in. in diameter by 4 in. long) that was provided with electric feed-throughs for stimulating, voltage recording, and thermocouple leads. The nerves were kept moist by placing gauze sponges that had been soaked in Ringer's solution over the plastic block.

Usually the nerves were continuously stimulated throughout an experiment at 20 pulses/sec, 0.1 msec duration, beginning as soon as the chamber was sealed. Stimulus strength was just supramaximal for alpha fibers (generally 0.5 v), and the amplifier was adjusted so that the amplitude of each nerve action potential was displayed initially at 77 mm on an oscilloscope.

Control and experimental nerves were unpaired and selected at random, and the spike amplitude was measured every 5 minutes. After an observation period of 30 minutes, the chamber was flushed with the gas being investigated for 2 minutes, sealed, and brought to pressure in 5 to 10 minutes. Experiments were conducted at two levels, 1 and 12 atm (gage). The temperature during compression never increased more than 3°C and returned to precompression levels within 5 minutes. Figure 1 illustrates the extent of conduction block produced in peripheral nerve by pure oxygen under these conditions (complete conduction block being defined as the absence of the action potential in a nerve when stimulated with the initial electric stimulus parameters).

A standard t test (*5*) for significant differences in the means of the two groups (6 control and 9 experimental nerves) indicated that $p < 0.02$ at 3½ hours and $p < 0.01$ at 4 hours.

In a series of nerves exposed to a 5-percent CO_2–95-percent O_2 mixture, the stimulus threshold rose immediately upon compression; but if, after the chamber reached 12 atm, the stimulus voltage was increased approximately 100 percent (to about 1 v) so that the precompression spike amplitude was obtained, the nerves were blocked in the same fashion as ones at 12 atm of oxygen but with a significantly shorter latent period ($p < 0.01$). Mean time for block with 5-percent CO_2–95-percent O_2 mixture at 12 atm was 3 hours, compared with 4.5 hours for oxygen at 12 atm. Evidence was obtained that hyperoxic conduction block occurs at 10 atm and lower pressures with longer latencies, but a complete spectrum of pressures was not investigated.

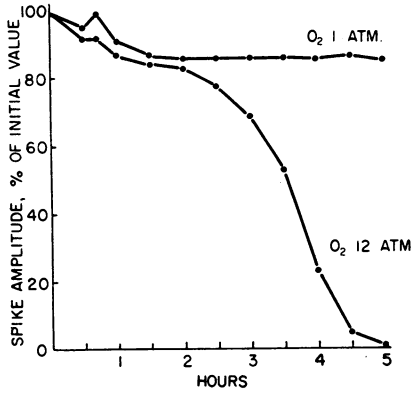

Fig. 1. Time course of conduction block produced in frog sciatic nerve stimulated with 0.5 v, 0.1-msec duration, 20/sec pulses in 12 atm of oxygen, compared with controls in 1 atm of oxygen that were stimulated in the same fashion. Spike amplitudes are mean values.

As in oxygen poisoning of muscle (*3*), peripheral nerve usually shows a partial recovery from hyperoxic conduction block when it is returned to air at 1 atm provided that the block is not maintained more than a few minutes. No complete recoveries occurred in our series. Nerves in which conduction was blocked by 5-percent CO_2–95-percent O_2 showed partial recovery sooner (within 10 minutes) and more completely upon decompression than those that were blocked by oxygen alone.

Nerve activity resulting from continuous stimulation neither seems to modify nor to be essential for the production of conduction block. A group of nerves was exposed to 12 atm of oxygen and stimulated every 30 minutes (for no more than 30 sec, just long enough to measure the spike amplitude). A second group of nerves was exposed to 12 atm of oxygen and continuously stimulated. No significant difference was observed between the two groups with respect to the time course of conduction block.

Hyperoxic conduction block in nerve may result from a direct toxic action of oxygen on the oxidative processes that are essential for maintenance of the membrane potential and impulse transmission. Inactivation of thiol (-SH group) enzymes (*6*) by increased formation of oxidizing free radicals (*7*) is a recently advanced explanation of this direct toxicity.

It has been reported that carbon dioxide decreases the latency of central nervous system manifestations of OHP (*8*), and it is interesting to note that it also decreases the latency of hyperoxic conduction block in peripheral nerve. Whether its efficacy in potentiating the block consists in changing the pH, increasing the membrane permeability, or involves some more obscure mechanism remains unknown. As yet, there is no evidence that OHP produces conduction block in the central nervous system, but hyperoxic conduction block may well play a role in the neural insult that initiates hyperoxic convulsions. The length of exposure and the pressure required to produce even mild nerve block in these experiments are greater than