The question now arises, What is the relation of the motor nerves during periods of long quiescence? For example, during the interval between two lactations, or after the menopause. Is it possible that the nerve has also some inhibitory influence, and that while it does not drive the cells in one direction it prevents them from developing in another. Prevents them, for example, from exercising that inherent and intrinsic power of growth the possession of which seems to be indicated by their mode of origin. If this is so, abolition of the nerve (for example, its withering) at the menopause might leave the cells at liberty to exercise this faculty of growth, unless it also was abolished at the same time. What results such an unfettered mode of growth would give we have not the least idea. But it might lead to the formation of gametoid cells, or of cells which could not resist the entrance of leucocytes (according to the views of Dr. Forbes-Ross). Such symbiosis resulting in the formation of a new entity, with new endowments, something after the fashion of the formation of a lichen from the combination of a fungus with an alga.

There are other ways in which a new mode of life might conceivably be imparted to cells. Suppose these failed to develop in a certain way, either from their own degradation or from partial failure of nerve stimulation, the motor nerve might be able to make them grow, but not in a shape which would enable them to secrete. In case it be held that the sympathetic and not the chorda tympani is the trophic nerve of the submaxillary gland, my arguments would not be affected, but we should only have to substitute its name for that of the chorda tympani.

The continual and unrestricted growth of inoculated cancer cells indicates that these bodies possess, as Dr. Bashford points out, properties hitherto unknown in the higher animal kingdom. But the cancer cell, though a living entity, is not, strictly speaking, a viable one *per se*. The inoculated cancer mass when it lives and grows is not a mass of cancer cells only. But of these *plus* a new growth of connective tissue furnished by the host.

We can scarcely do otherwise than suppose that when, as in a certain percentage of cases, the inoculation fails, it does so because the new host refuses to supply the new connective tissue.

If we suppose a failure of physiological condition to be the cause of cancer, we are at once led to turn our attention to the law of senility. The term is synonymous with "old age," but, as commonly used, it connotes those bodily changes which occur in old age. Scanty or decolorised cranial hair, irretentive brain, loss of teeth, piping voice, impairment of hearing and vision, tremulous handwriting, stooping figure, tottering gait, shrivelled and inelastic skin, and so on, are all indications of diminished functional activity and corresponding impairment of structure. For example, who can suppose that the irretentive brain of old age is structurally identical with the retentive brain of youth? Such a proposition is unthinkable.

So far, then, from it being improbable that some cells and nerves of a gland may undergo senile deprivation, it is almost certain that some of them will. Worth notice is the fact that in spite of the general impoverishment of the hair, in not a few cases the eyebrows and the hair about the ears present a redundant growth. Senile changes are progressive, but not continuously so nor uniformly so in all parts of the body, nor is it common for all the signs to be present together in one person.

The fact of cancer being a disease of senility, which is itself a period of degradation of tissue, tallies with the supposition that cancer may be primarily due to a degradation of the tissue in which it arises. It is to be noted that no other explanation of the "law of senility" in cancer has as yet been offered. But besides general senility there is that form which affects only individual parts, such as the thymus at an early age and the female reproductive organs at or about the menopause.

The frequency of cancer in these latter organs at this period seems to me to enforce still more powerfully the idea that the disease has its prime origin in some structural or vital default, either in the cells in which it is first manifested or in the nerves which dominate these cells, possibly in both these combined. It seems to me quite certain that underlying the first visible evidence of cancer development, whether this be the gametoid cells of Messrs. Farmer, Moore, and Walker, or the secreting cells invaded

by leucocytes of Dr. Forbes-Ross, there must be some invisible antecedent influence leading to these changes.

The first-named authorities say: "We feel that the evidence justifies us in deliberately correlating the appearance of these neoplasms" (the gametoid cells) "with the result of a stimulus which has changed the normal somatic course of cell development into that characteristic of reproductive tissue. We look, then, upon this transformation as representing the immediate cause of the development of the malignant growth, but the remote cause must be sought for amongst those various stimuli, some of which—for example, continuous irritation—are known to favour their development." We here have a frank suggestion that something has to be added to the part to produce cancer, and this idea is, I fancy, pretty generally entertained.

"Irritation" is always sooner or later followed by exhaustion, so that events which appear to, and really do, follow irritation may be due to it only indirectly and directly to the subsequent exhaustion—that is, to the "something" taken away.

I submit that there is good reason to suspect that the origin of cancer may be intimately connected with senile changes in the nerve endings or in the cells, or perhaps with both these combined.

NOTE ON THE TRANSMISSIBILITY OF SYPHILIS TO APES.

BY

ALBERT S. GRÜNBAUM and RALPH D. SMEDLEY,

M.A., M.D., F.R.C.P.

M.A., M.B.

(FROM THE PATHOLOGICAL LABORATORY, UNIVERSITY OF LEEDS.)

ALTHOUGH this note contains only a preliminary step in further investigations, we think it of some slight interest, since we have not seen that any confirmation of the work of Metchnikoff, Neisser, and others regarding the susceptibility of anthropoids to syphilis has yet appeared in this country, to place the following observation on record.

Fig. 1.—Head of chimpanzee showing chancre on right eyebrow.

A male chimpanzee was inoculated on the eyebrow by rubbing into it a portion of a chancre freshly excised. Signs of infection, indicated by some duskeness of the skin and a small nodule, appeared on the sixteenth day. For some days later these signs seemed to regress, but then general swelling and induration of the eyebrow with duskeness of skin became very evident. Later still exfoliation of the skin, leaving a surface yielding a serous or seropurulent secretion, has occurred. The cervical glands on the same side are enlarged. The general health of the animal seems unaffected, and there is no rise of temperature.

Although sought for, the *Spirochaeta pallida* was not found until the thirty-seventh day after inoculation.

Fig. 2.—Three spirochaetes from lesion, showing pointed ends $\times 1,300$.

Our thanks are due to Mr. Walter Thompson, Lecturer on Operative Surgery, who was good enough to give us the material for inoculation, and to Mr. Littlewood, Professor of Surgery in the University, who kindly confirmed the diagnosis in the ape from a clinical point of view.