CHANGES IN THE FIELD PROPERTIES OF MICE WITH TRANSPLANTED TUMORS*

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In a series of previous papers, it has been shown that the onset of an atypical growth in mice produces significant changes in the electrodynamic fields. This was true in the case of spontaneous tumors (Burr, Smith, and Strong[1]), in tumors induced by methylcolanthrene (Burr, Strong, and Smith[2]), and tumors induced by external application of benzpyrene (Burr, Smith, and Strong[3]). In all cases these changes were significant and independent of local phenomena such as those of inflammation.

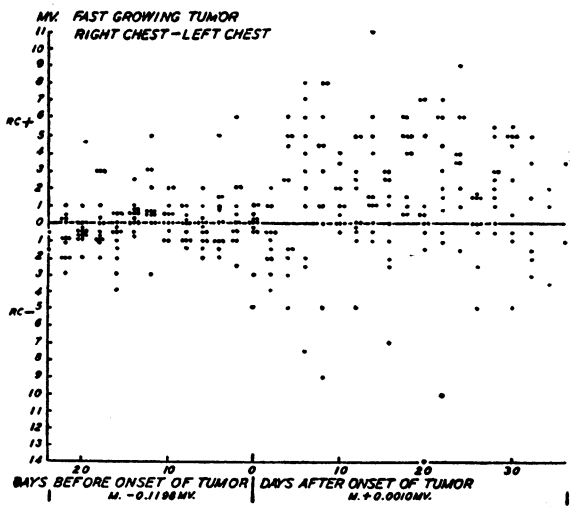

FIG. 1. Scatter-diagram of voltage gradients between right chest (*RC*) and left chest (*LC*) in millivolts for successive days in mice injected with fast-growing tumor.

In the present study, 36 mice of the Strong Strain A were used; 12 succumbed before the end of the experiment. The mice were divided into two groups. The 16 animals of Group I were injected with Yale Tumor No. 1; of these, 11 survived and were used as a basis for a statistical study. In Group II, 20 animals were injected with transplantable tumor No. 139,658A (Strong). Thirteen of these survived and were likewise studied statistically. In both groups the injections were made

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in the right axilla when the animals were approximately eight weeks old. As in previous series, the non-anesthetized animals were strapped, dorsum down, to a corkboard and voltage gradients were determined, by the Burr-Lane-Nims technic, between the sternum and the right and left chest, and between the sternum and the pubis. Determinations were made almost daily.

The mice of Group I were injected with a fast-growing Yale Tumor No. 1. The duration of the experiment was approximately 28 days, records were made for 11 days, on the average, prior to the appearance of the tumor, and for 17 days following. A scatter-diagram of the voltage gradients across the chest before and after the onset of tumor is shown in Figure 1. The means of the determinations are also included. Two important things should be noted. First, prior to the appearance of the tumor, the right chest was predominantly negative 120 microvolts, with a fair degree of clustering about the mean. After the appearance of the tumor, the right chest went positive 10 microvolts with a great deal of scatter from the mean. The means show a shift in the voltage gradient of more than 120 microvolts to a positive polarity. Subjecting the data to an analysis of variance with disproportionate sub-class numbers (Snedecor[4]), this difference is highly significant, the F value being 2.6878, with 1.92 required for high significance. The F value for differences in sub-classes with respect to time is

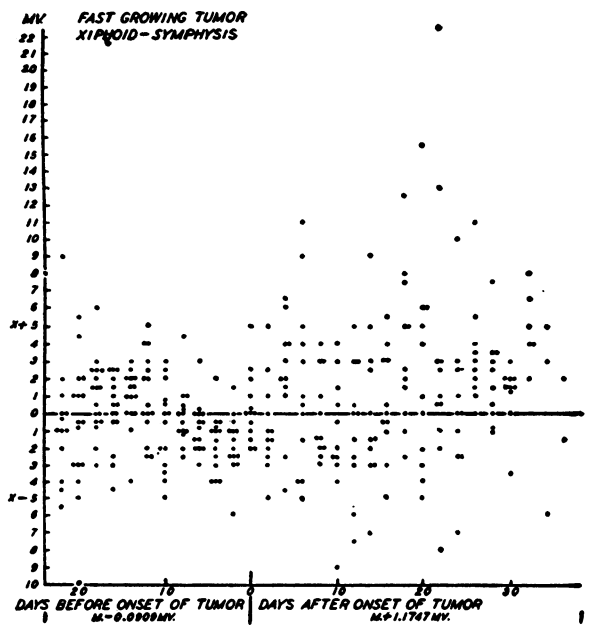

FIG. 2. Scatter-diagram for sternum (X)-pubis voltage gradients of fast-growing tumor.

14.346, with 6.7 requisite for high significance. These cross-chest readings indicate, therefore, a high degree of effect of the tumor upon the local field properties, changing the polarity of the mean and introducing the factor of great variability.

The sternum pubis recordings are given in Figure 2. It will be noted that before the tumor appeared, the sternum was negative 90 microvolts and after the onset of the tumor it went positive to a mean of 1175 microvolts; whereas in the chest the means of the sternum show a shift to the positive side but of somewhat lesser magnitude. The F value for sub-classes is 2.359 with 1.92 for high significance. The F value for sub-classes with respect to time is 7.792, with 6.7 necessary for high significance. Here again, with the advent of tumor, there is a significant shift of the sternum to a positive potential with respect to the symphysis and a marked increase in the variability of the determinations. These two sets of findings indicate that the presence of the tumor produces not only a local effect in the chest region but a general systemic effect as well, for the changes are noted in the over-all longitudinal measurement of the sternum-pubis as well as in the cross-chest measurement.

In Group II, 20 animals were injected with a slow-growing tumor and studied over a period of 45 days. The average number of days required before the appearance of tumor was 15. Figure 3

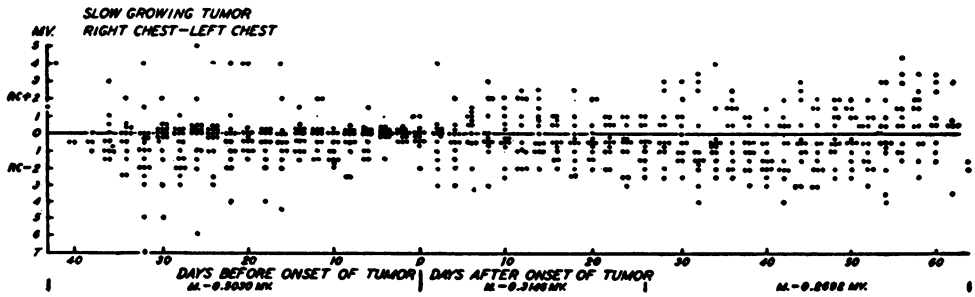

FIG. 3. Scatter-diagram for cross-chest voltage gradients in mice with slow-growing tumor.

is a scatter-diagram of the voltage gradients across the chest. The mean of all the cross-chest measurements taken during the interval between the injection of the tumor and the appearance of a new growth showed the right chest to be negative 503 microvolts. With the appearance of the tumor, readings of the first 15 days showed a

drop to 315 microvolts, to be followed in the second 15-day period by a further drop to 269 microvolts. Here again, it will be seen that the values tend to cluster about the mean prior to the appearance of the new tumor, whereas, following it, the voltages tend to scatter. Subjecting these data to the Snedecor analysis, it is clear that the means differ significantly and that, in general, the right chest tends to become more positive after the appearance of the tumor. The effect of the slow-growing tumor on the longitudinal voltage gradients is shown in Figure 4. Unlike the previous records, here there

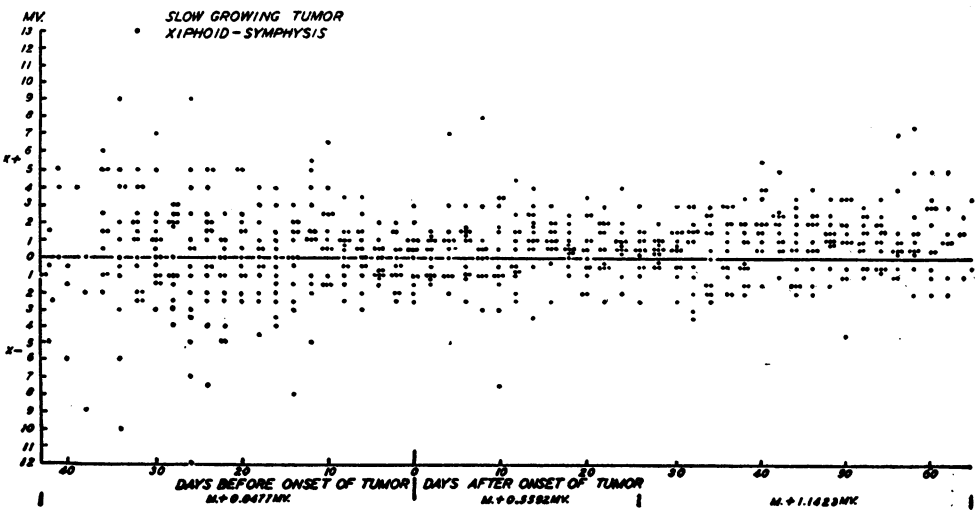

FIG. 4. Scatter-diagram for sternum (X)-pubis voltage gradients of slow-growing tumor.

seems to be variability in the animals before the onset of tumor, with an increasing stability following its appearance. However, it will be noted that in both sets of measurements in the group having the slow-growing tumor, the impact of the tumor is drawn out over a longer period of time with the probability that the organism makes a somewhat more satisfactory adjustment to the new growth, at least in so far as the voltage gradients are concerned.

It will be clear from the above that the fast-growing tumor produces a considerable disturbance in the electrical fields, whereas the slow-growing tumor produces a similar disturbance over a longer period of time. In both instances the site of the new growth becomes

significantly positive with respect to the left chest, and the sternum positive with respect to the pubis.

Needless to say, these studies should be extended to problems of tumor growth in man. During the last three years, more than 10,000 such determinations have been made in the Tumor Clinic of the New Haven Hospital. Significant differences have been found between groups of normal individuals and groups of patients suffering from chronic cystic mastitis, from carcinoma of the breast, and from carcinoma of the lip. However, these studies are as yet incomplete and no conclusions can be drawn from them.

The results of all electrometric studies of atypical growth in mice point to the fact that the new growth disturbs in significant fashion the electrical field of the organism. Moreover, since it has been shown that fields exist in a manner that can be measured electrometrically, and that the fields appear before the morphological pattern is established (Burr, in press), it would seem probable that in cancer there exists a disturbance in a field control of the patterns or organization of growing cells. In addition, since the fields exist in the unfertilized egg, the conclusion is forced that the fields are bound up with the inherited mechanisms of the organism. If this be true, cancer would then result from the impact of some untoward environmental circumstances upon a defective field, which is passed on from one generation to the next. This means, of course, that cancer is the result of a constitutional defect and is not caused by a specific agent acting by itself. Somewhere, in certain organisms, the mechanism controlling the pattern of organization is unable to withstand the impact of certain kinds of environmental factors and, as a result, its controls break down and cancer results. Such an assumption would explain why a number of external agents seem to be associated with the onset of neoplastic growth. It would seem, therefore, that the search for the cause of cancer should be directed toward an analysis of the mechanisms which control growth and development, as well as for the specific chemical agents or similar insults which bring about the breakdown in the orderly processes of growth and differentiation. These findings suggest that one of the profitable avenues by which we can reach an understanding of cancer is through a more rigorous and careful analysis of the fundamental processes underlying growth and development.

References

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