

Assuming, however, that the theories are correct, the records $n \geq 2$ must be interpreted as due to penetrating showers different from cascades and knock-on showers.

We have carried out experiments which suggest that these penetrating showers are connected with the extensive air showers. This is in agreement with the conclusions of Auger⁶ and with recent experiments of Wataghin⁷.

Though other interpretations cannot be excluded, it is plausible to assume that the observed showers contain several mesons simultaneously. The photographs of Braddick and Hensby⁷ appear to show examples of two associated mesons.

Comparison of the coincidence rates for $T = 0$ and $T \geq 1.8$ cm. shows that less than half the penetrating showers come from the air, while the rest are produced in the absorber T . It seems possible, therefore, that some of the observed showers are due to creation of mesons in the absorber T .

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⁶ Auger, P., Maze, R., and Grivet-Meyer, T., *C.R.*, **206**, 1723 (1938).

⁷ Wataghin, G., Souza Santos, M. de., and Pompena, P. A., *Phys. Rev.*, **57**, 61 (1940).

⁸ Braddick, H. J. J., and Hensby, G. S., *NATURE*, **144**, 1012 (1939).

Origin of Visual After-Images

It is sometimes disputed whether visual after-images are due to retinal or central processes. Their retinal origin appears to be capable of demonstration as follows. If one eye is pressed with the finger or a spring balance near the outer canthus for about thirty seconds, it will become temporarily blind. This effect, known to Thomas Young, Helmholtz and Donders, is attributed to retinal anoxæmia. The pressure should be about 250 gm., applied over an area of 1 sq. cm. The easiest method is to press on top of the lids, keeping them sufficiently far apart to permit vision. A brown mist will spread over the visual field, and finally all objects will disappear. The image of a bright light, such as a 60-watt bulb, at a distance of 1 m. or 2 m. should now be thrown on a fixed region of the retina in the blind eye.

The easiest method is to fixate the light with the other eye; the natural convergence of the two eyes will cause the other to remain stationary though the pressure will prevent the image from falling exactly on the fovea. Such fixation is maintained for two minutes; the normal eye may be closed for a second from time to time, to make sure that the other eye is still completely blind. If, finally, the head is turned away from the light and pressure removed an after-image of the light will, of course, be seen with the normal eye on any suitable surface, being dark on a bright background and bright on a dark one.

If the normal eye be closed and the other opened ten seconds or so after pressure has been released, its vision will be found restored, and an after-image

positive or negative, will be seen against a suitable background, though the stimulation from the primary image has never reached the lower and higher visual centres at all.

The image will usually be eccentrically placed in the visual field, through lack of normal fixation, and may be blurred or multiple owing to eye-movements. It may also be feebler at first owing to incomplete recovery of vision in the pressed eye. Under suitable conditions, however, its similarity to the image in the other eye leaves no doubt that both originate in the same way. This confirms the view that the positive after-image seen on a dark ground is due to retinal after-discharge, and the negative image on a bright ground to local reduction of retinal sensitivity. Since these effects occur despite anoxæmia, their photochemical origin is confirmed. The same result is obtained if the unpressed eye has been closed throughout, but fixation of the blind eye is then very difficult and there is no normal image for comparison. It is not denied that central processes may exert an inhibitory influence on these after-images.

Induced anoxæmia of the retina thus provides a useful method of blocking impulses between the retina and the visual centres and of isolating the contributions of different parts of the system to the sensory process.

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Metabolism of Tumours

IN a recent review¹ of the 1939 report of the British Empire Cancer Campaign, Dr. E. Boyland criticizes as "rather misleading" the following statement from the report of this Laboratory: "There appear at the present time to be two main points in which the metabolism of cancer differs from that of most normal tissues. Firstly the ability of cancer cells to form lactic acid persists even when the tissue is respiring, secondly cancer tissue has a respiratory quotient indicating that the oxidation of carbohydrate is abnormal".

This statement, far from being misleading, seems to me to be clear and correct: tumour tissue differs from most normal tissues in these two respects. In an earlier report² we had stated our view at length: following our findings³ that kidney medulla has a powerful aerobic glycolysis not only in Ringer solution but also, contrary to György *et al.*⁴, in serum, we said²: "In these somewhat rare cases, therefore, aerobic fermentation is certainly not associated with growth, still less with malignancy. Aerobic glycolysis is not specific for tumours, though practically all tumours have strong aerobic glycolysis". Later we studied cartilage⁵ which we found⁶ to have a similar type of metabolism. These are clearer examples than retina, since these tissues are probably no more damaged than in other tissue-slice experiments. Bone marrow glycolyses aerobically in Ringer's solution⁷, though the preparation used by Fujita² had low aerobic glycolysis in serum.

Some of Dr. Boyland's other examples are less satisfactory. Liver^{9,10}, smooth muscle¹⁰, diaphragm¹¹ and normal lymph glands^{12,13,10} have all been reported with insignificant aerobic glycolysis compared with that of tumours. In fact, the transformation of normal liver into a hepatoma is a striking example^{14,15}