eighty-eight days. The gastrocnemius muscle weighed the same as that on the control side, and reacted equally well. Moreover, the animal had lost all trace of the limp which was so prominent for some weeks after the operation. Unfortunately, the histological examination was incomplete, as the branches to the gastrocnemius were not examined to see how far they had suffered and how far recovery was due to a true nerve regeneration. In Experiments 13, 15, and 16, however, the investigations were more complete; in 13 (sixty-four days) regeneration was shown histologically to be well advanced, and the muscles had so far recovered that, examined by the Leduc current, they reacted in a manner quite different from the typical reaction of degeneration. In 15 (sixty-one days) and 16 (fifty days) regeneration was much less advanced. In neither of these could any new axones be found in the peripheral branches, and the muscles gave the complete R.D. It is probable that this backwardness, contrasted with 13, is dependent on the greater degree of fibrosis produced by the alcohol in the former, the fibrous tissue forming, as I have found in other experiments, an effective barrier against the downgrowth of the regenerating axones. There is every reason to believe, however, that in due time complete recovery of power would occur in all muscles which had been paralysed by such injections. In other words, there does not seem to be any real danger of permanent paralysis in these cases, though in man the time taken for complete recovery might be a matter of a year or more. This is a matter of some interest, in view of the cases of peroneal paralysis recorded by Schlösser and others (v.a.) following the treatment of sciatica by these means, though such a procedure is, I fancy, no longer likely to be advocated at the present time.

CONCLUSIONS.

1. Alcohol, injected into the trunk of a peripheral nerve, produces a more or less complete local necrosis of the nerve at the point of injection.

2. The change is not an "ascending" one, the nerve above the point of injection remaining normal; the cells of origin of the fibres may show some degree of chromatolysis, but do not exhibit signs of permanent injury.

3. The conditions produced by such injection are more favourable to regeneration than those resulting from simple section without suture. The anatomical continuity of the nerve trunk favours rapid regeneration, though this is to some extent retarded by the fibrosis which occurs to a greater or less extent in every case of alcohol injection.

4. It is apparently impossible by a single injection of alcohol to produce complete necrosis of the Gasserian ganglion, its dense texture preventing complete infiltration. The alcohol tends to find its way under the sheath of the ganglion towards the proximal root, which is affected to a greater degree than the actual ganglionic cells.

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- ¹ Schlösser, *Klin. Monatsblatt f. Augenheilkunde*, 1903, Bd. 41 (2), s. 244.
² *Ibid.*, *Versammlung d. Deutscher Ophthal. Gesellsch.*, Heidelberg, September, 1903. ³ *Ibid.*, *Verhandlung des Congresses f. Innere Medizin*, Wiesbaden, 1907, s. 49. ⁴ Finkelnburg, *ibid.* ⁵ W. Harris, *Lancet*, 1912, vol. i, p. 218. ⁶ O. May, *Brain*, 1911, vol. xxxiv, p. 272.

GREY HAIR ASSOCIATED WITH NERVE LESIONS.

By G. LENTHAL CHEATLE.

In previous papers¹ I have pointed out that when the sites of the points of incidence and areas of occupation are taken into consideration, then there is reason to believe a nervous influence exists which can induce greyiness of hair, at least in some cases. The particular cases to which I drew attention were of two classes.

Class 1. Persons having had grey patches of hair ever since they could remember.

Class 2. Persons who gradually become grey with advancing age. This class of case must again be subdivided into:

(a) The initial occurrence of patches of grey hair, which gradually fuse with the general greyiness of the scalp and beard.

(b) The gradual appearance of scattered grey hairs beginning in the temporal regions. In this subdivision there is an absence of patches.

It is those cases in Class 1, and (a) of Class 2, which show by the points of incidence a nervous genetic influence. In Class 1 the grey patches are often hereditary as well.

In the present article I wish to indicate a large class of cases which acquire greyiness of hair in association with other nerve lesions, and therefore probably connected with nervous changes.

Fig. 1 shows the nerve lesions on the right side of a case of Sir Victor Horsley's, to whom I owe my thanks for allowing me to publish it. The clear area on the forehead and frontal region of this scalp was anaesthetic, the skin was bleached and the hair was grey. The dotted area posterior to it was hyperaesthetic and the hair was not grey. In both areas there was slight overlapping of the left side, which otherwise was normal with regard to sensation and the colour of the hair. The case suffered from leucoderma.

Fig. 1.

Fig. 2.

Fig. 2 shows the state of things in a case of my own, an out-patient of King's College Hospital, aged 36 years. A subcutaneous neurofibroma growing on each of the sites marked by a cross on the cheek and forehead of the right side, and which were noticed about the same time ten years ago, when the hair also began to grow grey.

The state of the greyiness of hair was as follows: On the mid-orbital maximum point of Dr. Henry Head, there was a patch of normal brown colour, which appeared as an island in the completely grey V-shaped area seen in Fig. 2, behind which, and limited behind by the posterior boundary of the fifth nerve, was an area of scattered grey hairs. There was no disturbance of sensation. Tactile, heat, cold, and pain sensations were normal.

It would be of immense theoretical and practical interest to discuss the state of greyiness of hair, together with the protopathic nerve influence described by Dr. Head. The discussion would be very discursive, owing to the fact that our knowledge is not sufficient to enable us to state what makes hair appear grey; and although one may have opinions upon the subject they are not sufficiently matured for dogmatic pronouncement.

All I want to do now is to show that here is an epithelial change in state, which takes place *pari passu* with a change in the nervous system, and, therefore, probably due to it.

Although greyiness of hair is of itself an unimportant epithelial change, it appears at a time of life common to another and important epithelial change—namely, cancer. The genesis and etiology of an unimportant change in a tissue may illuminate the genesis and etiology of an important change in the same tissue.

I am, at any rate, assuming a correct attitude in saying that because a nerve influence can be shown at work in the genesis of one epithelial change, it ought, therefore, to be considered in the genesis and etiology of another epithelial change—that is, cancer.

Greyiness of hair may be said to be a degenerative change and cancer a proliferative change. The proliferative change in cancer can hardly be considered an object lesson in regeneration; its products are nearer degeneration products, so far as size, shape, and function of the cells are concerned.

REFERENCE.

- ¹ The Incidence of the Hair's Greyiness, *BRITISH MEDICAL JOURNAL*, July 22nd, 1905. The Mental Nerve Area and its Relation to Greyiness of Hair, *BRITISH MEDICAL JOURNAL*, July 4th, 1908.