ANOSMIA.*

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The peripheral nerve-cells of the mucous membrane of the regio olfactoria are connected by means of the non-medullated nervi olfactorii with the bulbus olfactorius, which again is connected with the brain through the tractus olfactorius. The lateral root of the olfactory tract ends in the gyrus uncinatus, some fibres run to the temporal lobe; the median root of the tract ends in the gyrus fornicatus, one bundle of the fibres passes to the commissura anterior, thence the greater portion of these fibres passes by the lower part of the lenticular nucleus, between putamen and globus pallidus, and is lost in the temporal lobe, whilst the smaller portion of these fibres runs in the region of the internal capsule and ends in the optic thalamus. The commissural fibres also contain crossed routes. The central terminations in the cortex are situated in the gyrus hippocampi, gyrus uncinatus, and the anterior end of the gyrus fornicatus. The centres for smell are connected through association fibres with the cortical centres of the trigeminus and glosso-pharyngeus. We have, unfortunately, very few clinical and pathologico-anatomical observations to support these histological facts. Most clinical and post-mortem observations are limited to the anterior cranial fossa, to lesions of the olfactory bulb and tract. Quite unique are those cases in which, anosmia having been present during life, pathological lesions have been found in the brain and cortex post-mortem. Further, the few anatomico-pathological facts have not revealed isolated foci, which would enable us to locate precisely a cerebral olfactory centre. Examinations of cases of cerebral tabes have demonstrated atrophy of the roots of the olfactory bulb, of the olfactory nerves, and a loss of fibres in the anatomico-histological centres of smell. In cases of cerebellar as well as of cerebral tumours atrophy of the olfactory nerves has been observed, and this has been regarded as a secondary change due either to increased intracranial pressure or to chronic inflammation of the pia. Loss of fibres has been noted in the gyrus uncinatus and gyrus hippocampi in cases of paralysis progressiva. In a case of anosmia Schäffer and Frey found atrophy of the olfactory tract and a great loss of fibres in the gyrus uncinatus and cornu ammonis. In a case of kakosmia a tumour of the right gyrus hippocampi was found. In tumours of the gyrus uncinatus

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and the gyrus fornicatus hallucinations of smell have been noted; so also anosmia in cases of hæmorrhage, embolism and lesions of the temporal lobes. In one case crossed anosmia was found along with aphasia and right hemiplegia. It is to be seen, then, that our knowledge of the nerves of smell, of the course of their fibres, and of their centres, has so far been but little increased by pathological observation. Nevertheless, the few facts thus obtained can be brought into harmony with those derived from comparative anatomy, and justify the conclusion that in man the centre for smell is probably situated in the gyrus hippocampi and gyrus uncinatus. Clinical experience shows that lesions in the regions of the above-described nerve-routes can cause disturbance or loss of smell, that a partial decussation of the nerves of smell takes place in the cerebrum, and that an association between the centres of smell and the cortical trigeminus centres is, at the least, possible.

Let us now examine the ætiology and attempt a classification of the different clinical forms of anosmia. As to the ætiology of anosmia, cases can be so classified as to give us (1) One group in which either pathological changes in the region of the sense of smell or injurious influences are recorded; and (2) a second group in which changes cannot be discovered and are not to be expected, but in which the loss of smell is rather to be referred to mechanical interference with nasal respiration or to functional disturbances. We divide anosmias into the following classes: (1) Essential or true anosmia—this can be central or peripheral according to the part of the regio olfactoria that is affected; (2) mechanical or respiratory anosmia; (3) functional anosmia.

Amongst the ætiological causes of peripheral true anosmia are found inflammation of the peripheral regio olfactoria leading to olfactory neuritis, also atrophy of the nerve of smell. Inflammation of the peripheral regio olfactoria can arise in connection with inflammation of the nasal mucous membrane as the result of influenza. It may be a temporary affair or may end in atrophy. Syphilitic processes, high-lying, latent or recurring polypi, also chronic ethmoiditis, may produce pathological changes in the peripheral regio olfactoria. Again, this region may be affected by the atrophy of ozæna, or by senile atrophy, though the latter is generally central in origin. As for central true anosmia, the causes may be cerebral tumours in the anterior cranial fossa, abscess, hydrops ventriculorum, sclerosis, emboli or hæmorrhages, syringomyelia, cerebral tabes, paralysis progressiva, senile atrophy; further, lesions directly affecting the bulbus and tractus olfactorius, such as cerebro-spinal meningitis, syphilitic and tubercular lesions,

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traumata causing stretching or tearing of the nerves, bulbs or tracts. To this group also belong intoxications with tobacco, cocaine, mercury, lead, morphia, atropine, sulphuric acid, also over-stimulation and exhaustion of the sense of smell. Irritating gases, cocaine and tobacco have a peripheral, as well as a central, action. In this group again are to be reckoned the toxic influences of influenza, blood-poisoning and erysipelas, also congenital anosmia due to defects in, or absence of, bulb, tract, or cortical olfactory centres, lastly pigmentary atrophy, which probably affects the ganglion cells.

Respiratory or mechanical anosmia is caused by anything that interferes with nasal respiration, congenital atresia of the nares, acquired narrowing and synechiæ of the nares, a well-marked asymmetry of the nasal skeleton, deflections of the septum, tumours of the nasal fossæ, especially polypi, inflammatory swellings, hypertrophy of the turbinal bodies, foreign bodies.

The common cause of functional anosmia is hysteria, but various reflexes from distant parts may produce it, *e.g.*, after ovariotomy, during menstruation, after cauterization of the inferior turbinals and extirpation of the Gasserian ganglion. In this group must be included intermittent anosmia, without evident cause. The anosmia accompanying psoriasis buccalis, and disappearing when the latter is cured, is also functional.

Hallucinations of smell, various parosmias and kakosmias are frequent precursors of anosmia.

Our knowledge of anosmia is extremely meagre, and can only be extended by exact clinical observation and pathological examination.

NOTES.

It would be difficult to overrate the importance of the discussion at Ipswich on the Bacteriological Diagnosis of Diphtheria, and we are glad to be able to publish a full report which has appeared in the Journal of the British Medical Association.

Messrs. J. and A. Churchill, of Great Marlborough Street, London, will publish in the course of the present month a revised edition of the Descriptive Catalogue of the Museum of the Sixth International Otological Congress, compiled and edited by Jobson Horne and Arthur H. Cheatle. Copies will be presented to all foreign and colonial members of the Congress.

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