INJURIES OF THE BRAIN AND SKULL

PART II

Edited by

P. J. VINKEN and G. W. BRUYN

in collaboration with

R. BRAAKMAN

Associate Editor

HAROLD L. KLAWANS JR.

NORTH-HOLLAND PUBLISHING COMPANY · AMSTERDAM · OXFORD

AMERICAN ELSEVIER PUBLISHING CO., INC. · NEW YORK

Library of Congress Catalog Card Number: 68-8297

ISBN for the complete set: 0 7204 7200 8

ISBN North-Holland for this volume: 0 7204 7224 5

ISBN American Elsevier: 0 444 10878 5

322 illustrations, 97 tables

PUBLISHED BY:

NORTH-HOLLAND PUBLISHING COMPANY · AMSTERDAM

NORTH-HOLLAND PUBLISHING COMPANY, LTD. · OXFORD

SOLE DISTRIBUTORS FOR THE U.S.A. AND CANADA:

AMERICAN ELSEVIER PUBLISHING COMPANY, INC.

52 VANDERBILT AVENUE

NEW YORK, N.Y. 10017

PRINTED IN GERMANY

Foreword to volumes 23 and 24

Head injuries have existed as long as mankind. Originally, they were exclusively the result of hunting accidents, falls, and, most commonly of all, hand-to-hand combat. (It is sad to have to recall that mankind has not progressed significantly beyond this aetiology.) Then, warriors took to wearing protective head outfits and even today, in this nuclear age, the helmet is still the symbol of the military profession. The first known records of man's surgical practice deal with the treatment of the wounds of the scalp, skull, and brain. Trephining of the skull is one of the oldest major surgical procedures.

However, during the centuries little progress in management was made and Guthrie's remarks (1847) that 'injuries of the head affecting the brain are difficult of distinction, treacherous in their course and, for the most part, fatal in their result' remained applicable to severe head injury until very recently. Management of penetrating injuries underwent signficant improvements in the latter part of World War II amongst the U.S., and particularly British, armed forces, due to the admission of victims in special head injury centres (for example, Oxford), where well-documented case histories were collected and studied. This lesson was not taken to heart insofar as civilian head injuries were, and are, concerned, most of which are still scattered in general surgical and neurological wards in regional hospitals. Truly such cases are the poor cousins. Even today they attract the attention of astonishingly few clinicians and scientists, whose international contact all too often is taken up with fruitless semantic discussions about terminology.

As a result of the growth in motor traffic in the last decade, there has been an enormous increase in the number of serious civilian head injuries, with the resultant large number of severely disabled survivors, and head injuries are now the most important cause of death in the first half of life in the developed countries of the Western world. This fact has given a new impetus to research, particularly into the phenomena occurring after the accident, such as changes of microcirculation-pattern, impaired autoregulation, and development of brain oedema. The nature of the persistent disability, the influence of rehabilitation on the rate and the degree of recovery, and the possibility to estimate the degree of attainable improvement in the early stages of recovery are amongst the large gaps in our current knowledge which still have to be filled.

Head injuries constitute a major health problem and do not attract the (limited) attention of neurologists only, but also that of bioengineers, and specialists in traffic medicine and public health. This has placed the Editors in a difficult position. They aim at complete coverage of the subject, but only from the clinical-neurological point of view. Accordingly, some borderline areas (e.g. prevention, social, and financial aspects) have been deliberately excluded. Some topics, splendidly dealt with in previous volumes, have been included again for the sake of completeness, although they have sometimes been written by other authorities. In the chapters on the post-traumatic syndromes, there has been a greater overlap than usual and differences of opinion were unavoidable, due to the strong convictions held by authors from various countries. The Editors were unable to arrange for the timely receipt of an acceptable chapter on birth injuries. Transplantation of kidneys would be impossible without the co-operation of the clinical neurologist. Consequently, chapters on brain death and its medico-legal aspects have also been included. During the preparation of his manuscript, Professor Verjaal of Leiden suddenly died. The Editors are indebted to Dr. van 't Hooft for having seen this chapter through to completion.

P.J.V.
G.W.B.
R.B.

Note:

Since this volume went to press, a particularly relevant work dealing with prognosis and outcome of CNS injuries has appeared: Ciba Foundation Symposium 34 (New Series) Amsterdam (1975).

Acknowledgement

Several illustrations and diagrams in this volume have been obtained from other publications. Some of the original figures have been slightly modified. In all cases reference is made to the original publications in the figure caption. The full sources can be found in the reference lists at the end of each chapter. The permission for the reproduction of this material is gratefully acknowledged.

List of contributors

G. Arfel

Institut National de la Santé et de Recherche Médicale, Suresnes 757

H. Binder

University Clinic of Neurology, Allgemeines Krankenhaus, Vienna 575

G. Blaauw

Department of Neurosurgery, Academic Hospital Dijkzigt and Erasmus University, Rotterdam 329

Reinder Braakman

Department of Neurosurgery, Academic Hospital Dijkzigt and Erasmus University, Rotterdam 105

A. Bricolo

Department of Neurosurgery, Civil Hospital, Verona 699

William F. Caveness

Laboratory of Experimental Neurology, National Institute of Neurological Diseases and Stroke, Bethesda, Md. 455

A. C. de Vet

The Hague 351

Harold Fishbone

Department of Surgery, The Brooklyn-Cumberland Medical Center, Brooklyn, N.Y. 179

G. Foroglou

Neurosurgical Department, Cantonal Hospital, University of Lausanne 231

J. B. Foster

Department of Neurology, The Regional Neurological Centre and Newcastle University Hospital Group, Newcastle upon Tyne 829

J. A. M. Frederiks

Department of Neurology, Catharina Ziekenhuis, Eindhoven 255, 487

F. Gerstenbrand

University Clinic of Neurology, Allgemeines Krankenhaus, Vienna 575

Flemming Gjerris

Neurosurgical Department, Bispebjerg Hospital, Copenhagen 27

J. R. W. Gleave

Department of Neurological Surgery and Neurology, Addenbrooke's Hospital, Hills Road, Cambridge 381

Emeric Gordon

Department of Neuroanaesthesia, Karolinska Hospital, Stockholm 599

Richard Gye

Department of Neurological Surgery, The Radcliffe Infirmary, Oxford 441

Eero Hillbom

Institute for the Rehabilitation of Brain Injured Veterans, Helsinki 501

Olle Höök

Institute of Rehabilitational Medicine, University of Göteborg 683

Reginald Hooper

East Melbourne, Vic. 637

Frederick E. Jackson

Department of Neurological Surgery and Research, Naval Hospital, Camp Pendleton, Calif. 381

Kenneth G. Jamieson

Brisbane, Qld. 261, 343

Ed Janon

Department of Neuroradiology, University of California, San Diego School of Medicine, La Jolla, Calif. 381

Bryan Jennett

Department of Neurosurgery, University of Glasgow and Institute of Neurological Sciences, Glasgow 445, 669

R. M. Kalbag

Department of Neurological Surgery, Newcastle General Hospital, Newcastle upon Tyne 369

S. Katinsky

Temple University Health Sciences Center, Philadelphia, Penn. 119

R. Kivelitz

Neurosurgical Clinic, University of the Saarland, Homburg/Saar 297

F. Loew

Neurosurgical Clinic, University of the Saarland, Homburg/Saar 297

Donlin M. Long

Department of Neurosurgery, University of Minnesota Medical School, Minneapolis, Minn. 627

Edwin E. MacGee

Kansas City, Mo. 183

James W. Markham

Department of Neurological Surgery, Stanford University, Stanford, Calif. 201

Robert E. Maxwell

Department of Neurosurgery, University of Minnesota Medical School, Minneapolis, Minn. 627

John E. McGillicuddy

Section of Neurosurgery, University of Michigan Medical Center, Ann Arbor, Mich. 141

Paul Mifka

Rehabilitation Centre Meidling, Vienna 517

J. Douglas Miller

University Department of Neurosurgery, Institute of Neurological Sciences, Glasgow 215

Urban Pontén

Department of Neurosurgery, University Hospital, Lund 599

John M. Potter

Department of Neurological Surgery and Accident Service, The Radcliffe Infirmary, Oxford 105

B. Ramamurthi

Institute of Neurology, Madras Medical College, Madras 275

Melville Roberts

University of Connecticut School of Medicine, Farmington, Conn. 59

Richard C. Schneider

Section of Neurosurgery, University of Michigan Medical Center, Ann Arbor, Mich. 141

W. Eugene Stern

Department of Surgery (Neurosurgery), UCLA School of Medicine, Los Angeles, Calif. 399

David Sumner

Leeds (St. James's) University Hospital, Leeds 1

J. U. Toglia

Temple University Health Sciences Center, Philadelphia, Penn. 119

H. A. H. van Till–D'Aulnis de Bourouill

The Hague 787

A. G. M. van Vliet

Department of Neurology, Erasmus University, Rotterdam 73

John Walsh

Department of Neurology, The Royal Prince Alfred Hospital, Sydney, N.S.W. 441

E. Zander

Neurosurgical Department, Cantonal Hospital, University of Lausanne 231

O. L. Zangwill

The Psychological Laboratory, University of Cambridge 477

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CHAPTER 1

Disturbance of the senses of smell and taste after head injuries

DAVID SUMNER

Leeds (St. James's) University Hospital, Leeds

Taste and smell are sometimes regarded as the poor relations of the special senses, not to be compared in importance with deafness, or more particularly, with blindness. Although the importance of the sense of smell has decreased with man's phylogenetic development so that we are now micro-osmic and not macro-osmic like the dog, anosmia, and to a lesser degree ageusia, will considerably diminish the pleasures of life, may bring professional disaster and can, on occasion, hazard life itself. At the same time, there is increasing awareness today of the importance of smell in our psycho-sexual life, both conscious and unconscious.

Bearing these facts in mind it is surprising that all anosmic patients do not complain spontaneously of loss of taste or smell after head injuries; it is mandatory to ask specific questions about these senses in anyone who has had a head injury however severe or however trivial.

For these reasons, together with the physiological and anatomical implications of post-traumatic anosmia and ageusia, the subject is important and fascinating.

Although loss of the senses of smell and taste after head injuries must have been observed since man existed, older writers on head injuries made no mention of this until 1864 when Hughlings Jackson described the case of a man who was struck from his horse by a Highway man 'all the worst results of concussion occurred – the sense of smell was lost forever'. This statement implies that the existence of post-traumatic anosmia was well recognised at that time if not reported.

Isolated case reports appeared over successive years and it is interesting how many of the very earliest writers on the subject made observations which were thereafter ignored for some considerable time. Notta in 1870 recorded two examples of post-traumatic anosmia, one of which followed a head injury with a fracture of the skull and one with neither fracture, loss of consciousness nor amnesia. He was the first to mention the possibility of recovery which he said might occur from 8 weeks to 7 months after the injury. Ogle (1870) described three cases and suggested that the anosmia was caused by tearing of the olfactory nerves at the cribriform plate and that it frequently followed occipital blows. He was the first to mention loss of taste and was quite definite that although this was a frequent complaint, primary taste, (that is for salty, sweet, sour and bitter substances) was intact. Mollière (1871) and Legg (1873) recorded further isolated cases, the latter describing the symptom of parosmia. Ferrier (1876) reported in detail an example of loss of both the sense of smell and of primary taste after a head injury with subsequent recovery of the ageusia but not the anosmia. He made the observation from this case which has continued to

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puzzle neurologists and anatomists to the present day: 'As regards causation, it is in the highest degree improbable that a blow on the head could cause a spontaneous rupture of such widely separated nerves as the olfactory, gustatory and glossopharyngeal'. He thought the site of the lesion to be in the lower part of the temporo-sphenoidal lobe. Rotch (1878) reported total anosmia and ageusia with complete recovery after an injury with a fractured base while Althaus in 1881, as did Grünstein (1882) and Gowers (1893), once again reiterated the view that there was always rupture of the olfactory filaments. Jacob in 1882 reported a further example of anosmia following a trivial occipital blow with recovery which he attributed to Galvanism.

ANOSMIA

Anatomy and physiology of olfaction

While the peripheral part of the olfactory pathway is clearly defined and widely accepted, the central connections are more widespread, diffuse and the subject of continuing debate.

The olfactory pathway begins in the yellowish brown olfactory mucous membrane situated high in the nasal cavity on the upper septum and superior turbinate. Here are to be found the primary receptor neurones which have two projections, one relatively coarse which projects towards the surface of the mucous membrane and from which come abundant, short olfactory cilia, while the other proximally directed projections of the olfactory cells are extremely fine (the finest fibres in the nervous system) and coalesce to form the olfactory filaments or nerves. These pass up through the perforations in the cribriform plate to enter the anterior part of the olfactory bulb, a flat, ovoid structure which lies immediately over the cribriform plate itself. The two bulbs, one on each side of the crista galli contain three types of cells, the large mitral cells, the smaller tufted cells and the granular cells of variable size. These granular cells have an associative function but fibres from the mitral and tufted cells pass distally to synapse with the fibres of the olfactory nerves, the sites of these synapses forming the so-called olfactory glomeruli. Axones from the tufted and mitral cells pass proximally to form the fibres of the two olfactory tracts (one from each bulb) which, lying on the floor of the anterior fossa, pass backwards to the anterior perforating substance. Here they divide into the fairly clearly defined lateral striae and the much less clear-cut medial striae.

The central pathway of the lateral striae is relatively simple, ending as it does in the pre-pyriform cortex and the amygdaloid nuclear complex. These structures form the primary olfactory cortex. Evidence of the primitive nature of the olfactory pathway is to be seen in the fact that olfactory impulses reach cortical levels without passing through the thalamus.

It is the proximal pathway and central connections of the medial striae which are more complex and less clearly understood. These fibres pass to the olfactory tubercle, the anterior commissure, the amygdala and thence to the striae terminalis. From these structures there are rich synaptic connections with the hippocampus, the ventral thalamic nuclei, the brain stem and the entire rhinencephalon. It is generally accepted that it is the primary olfactory cortex which is responsible for the conscious recognition and identification of odours (Brodal 1959). Although electrical stimulation of the olfactory bulb produced evoked potentials over a very wide range of structures, including not only the primary olfactory cortex but the hippocampus, limbic system, medial regions of the thalamus and the basal ganglia (Berry et al. 1952) a sensation of smell can be produced by stimulation only of the pre-pyriform cortex or amyglaloid nuclei and from no other structures (Penfield and Jasper 1954).

The central connections of the medial olfactory striae, however, seem to serve a psychic and emotional role (Brodal 1947). There is certainly little evidence that the hippocampus has any important relation to smell, a view supported by degeneration experiments (Meyer and Allison 1949; Adey and Meyer 1952; Allison 1954). Further support for this view is given by the fact that after ablation of the hippocampus there is neither loss of olfactory discrimination nor of olfactory conditioned reflexes (Swann 1934, 1935). The hippocampus is well developed in whales who lack olfactory bulbs and nerves and who are anosmic (Ries and Langworthy 1937). Equally no

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impairment of olfactory discrimination is produced following total bilateral degeneration of the ventral thalamic nuclei, although removal of the olfactory bulbs leads to loss of discrimination (Lashley and Sperry 1943). The whole problem of central connections of the olfactory pathways is reviewed in detail by MacLeod (1971).

Great problems still remain when the mechanism of odour reception and identification come to be considered and it is probably true that the last word has still to be said on almost any problem in this field.

Although traditionally the olfactory pigment has been assumed to play an important part in olfaction, there is little evidence now to support this view (Moulton 1971), and even the frequently accepted view that albino animals (deficient in olfactory pigment) have poor odour discrimination, has been shown to be equally untrue (Moulton 1960). The converse view that olfactory acuity is higher in those with dark skins (Ogle 1870) has not been upheld in more recent studies (McCartney 1968).

While it is generally agreed that the precise site for the interaction of an odour stimulus is the limiting membrane of the cilia (Moulton and Beidler 1967), most searching investigations with electron-microscopic techniques on the ultra structure of the olfactory mucous membrane have thrown little light on this aspect of the problem (Frisch 1969; Graziadei 1971).

Over the years many theories of olfactory reception have been propounded and decried. In 1951, Baradi and Bourne suggested enzyme mechanisms whereby the stimulus was involved in specific chemical reaction with the receptor cells. This has now been virtually abandoned because of the vast number of primary odours which exist, although as recently as 1970 Martin produced a more tenable approach to this mechanism. In 1954 Wright suggested a vibration theory which postulated that the molecular vibrations of specific odorant chemicals was in some way able to trigger off neuronal discharges from receptor cells whose own fibres were in phase, as it were, with the odour stimulus. This theory has not the widespread support that has been given to other concepts and, indeed, Davies (1971) is not even convinced that Wright's data support his conclusions.

A different mechanism to explain the firing of the receptor organ has been put forward by Davies (1953) who suggested that certain chemicals could 'penetrate and puncture' the membranes of the receptor cell and this would lead to cell membrane depolarisation and neuronal firing. This mechanism is not necessarily incompatible with the 'specific site' theory which, in one or other of its forms, is probably the most widely accepted hypothesis today. The general concept that odorant chemicals have their own individual molecular weight, size and configuration and that there are specific sites on the receptor organs for each 'primary odour' was first postulated by Amoore in 1952 and supported by Timmermans in 1954. This theory is well reviewed by Beets (1971) but one great problem is that there is no generally accepted theory of odour classification. Systems such as that described by Zwaardemaker (1895), who divided odours into ethereal, aromatic, fragrant, ambrosial, garlic, burnt, repulsive, nauseating and goat-like, have more entertainment value than use.

Amoore described seven primary odours in 1952. Harper and his co-workers (1968) suggested 44 classes but Amoore in 1969, expanding his original work, thought there were between twenty and thirty primary odours and felt that many of the 118 classes which had been described over the years could be reduced quite reasonably to 44 and possibly further. The view that discrimination takes place largely in the olfactory bulb rather than more peripherally, however, has been firmly advanced by Shepherd (1972) in an extremely good review of the organisation of this structure.

Electrical studies, as we have seen, have contributed largely to our understanding of the olfactory pathway but the contribution of the electro-olfactogram (EOG) has not been as great as might have been hoped. It has, however, made a significant contribution to our understanding of the olfactory receptor organ as a transducer and this aspect of olfactory physiology has been reviewed by Ottoson (1971).

It can be seen that the present views on the mechanism of olfaction are by no means unanimous and despite the vast amount of work that has been put into increasing our knowledge of the subject, the position was fairly accurately, if pessi-

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mistically, stated by Gesterland (1971) when he wrote, 'we know nothing about how the nervous system sorts smells into those categories which we feel must exist'.

Clinical methods

Unlike hearing and vision, the sense of smell is difficult to evaluate and attempts to quantify it have generally proved unrewarding. It is clear that to obtain accurate results, techniques of such complexity are required that they are more suitable for the research laboratory than for clinical practice (Jones 1954; Stuvier 1960).

Even relatively simple methods such as that described by Elsberg (1935) have been generally abandoned, not only because their routine use is too complex and time consuming, but because the results, ignoring as they do uncontrollable variables (Chavannaz et al. 1962), give a spurious sense of accuracy. Most clinicians content themselves today with using a few test substances which have a maximal olfactory and minimal trigeminal component (Sumner 1962; Hagen 1967). Caruso et al. (1969) puts in a strong plea to re-introduce the Proetz method (Proetz 1924) where increasingly strong solutions of odoriferous substances are used, correlating in his own experience the degree of hyposmia with the severity of the associated head injury.

Where there is apparent anosmia the clinical examination of the patient is not complete unless a strong solution of ammonia is used as well as the other test substances. The normal person will not only experience the irritation and reflex lacrimation produced by the trigeminal stimulating component of ammonia, but will also detect by his sense of smell the odour of ammonia itself. The patient with organic anosmia will experience all the trigeminal component but will not detect the odour of ammonia. The simulator will claim to experience neither the odour nor the irritation of the ammonia but, (providing the concentration of the ammonia is high enough) will not be able to mask the reflex lacrimation.

Where the need for objectivity is greater still, more complex techniques may be justified. Such techniques include polygraph methods (Rous and Synek 1967), psychogalvanic skin responses (Borsanyl and Blanchard 1962) and alpha blocking of the EEG with an olfactory stimulus (Sarteschi and Ardito 1960; Chavannaz et al. 1962). Gerin and his colleagues (1967) do, however, stress the limitations of EEG methods, pointing out that while it is possible to prove conclusively that anosmia is not present, it is impossible to prove that it is.

Apart from the difficulties in the quantification of the sense of smell, greater problems still are to be met when attempting to measure the range of odours to be detected. As we have seen, no generally acceptable classification of odours exists. Douek (1970) suggested that some patients had 'olfactory scotomata' which could be of diagnostic significance in clinical problems.

Quite apart from the significance of measuring the olfactory sensitivity to differing odours, there is, too, the question of the clinical significance of being able to identify, as well as detect, a range of odours. The difficulty is often compounded by the use of obscure substances or mixtures as test substances instead of those which are likely to be easily identified (Sumner 1962).

The problem of knowing whether the patient's skill or lack of it in identification is due to his intelligence or experience on the one hand or whether it is due to dysphasia, 'specific anosmia' (Amoore 1969), which may be inherited (Amoore 1971), 'smell agnosia' (Seydell 1932) or damage to the olfactory pathway on the other, has yet to be elucidated.

The incidence of post-traumatic anosmia

Although, as we have seen, isolated case reports of post-traumatic anosmia have been published since 1864, no suggestions as to the incidence were put forward until 1931 when Laemle found 17 patients with anosmia out of 26 (65%) who had suffered both concussion and a fracture of the base of the skull. The next reported incidence by Glaser and Shafer in 1932 was only 2.7% while Helsmoortel and his colleagues in 1933 found 18 in 43 severe head injuries (41%). Of these 18, 7 were 'partial', 3 had 'dysosmia', and 8 had complete anosmia. Goland (1937) described the finding of 6 anosmics in 38 (severe or moderately severe) head injuries (15%) while Knoflach and Scholl in 1937 found the incidence to be only

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6.4% in 387 head injuries. These authors made no reference, however, to the severity of the head injuries and relied entirely on their patients' statements rather than clinically testing the sense of smell.

The first large and unselected series appeared in 1943 when Leigh found 72 examples of anosmia in 1000 consecutive head injuries. The following year Freidman and Merrit (1944) reviewing the incidence of cranial nerve damage in closed head injuries, found the olfactory nerve to be the most commonly damaged (11 out of a total of 22 cranial nerve palsies) in a total of 430 patients (2.5%). Piacentini (1949) followed up 155 patients with severe or moderately severe head injuries, finding 15 with anosmia (9.7%); 12 of these 15 had sustained fractures of the skull. Mock (1950) however found no less than 38% of patients with a basal fracture had anosmia. Lewin (1954) found anosmia in 5% of 1000 head injuries (of whom 7.1% died), but the incidence rose to 78% of those who had cerebrospinal fluid (CSF) rhinorrhea. A similarly high incidence of anosmia in patients with CSF rhinorrhea was found by Gurdjian and Webster in 1958 who also pointed out that the incidence of anosmia rose from 40% to 80% if the associated dural tear had been repaired. Didier (1957), looking at the matter from another point of view, found in only 0.66% of patients attending an Ear, Nose and Throat clinic with anosmia was it due to a previous head injury. Graf (1961), reviewing the literature, suggested the overall incidence was about 5% while Klingler and Jost in 1963 examined 158 hospitalised patients with head injuries and found anosmia in 5%. Mifka (1964) reviewed 1,000 consecutive head injuries severe enough to be referred to an accident hospital and found 6.4% with anosmia. Of the 64 patients with anosmia, 54 (81%) had a fracture of the skull. The same year Sumner (1964), in another large series of 1,167 unselected head injuries (the only criterion for inclusion being that they had either a head injury severe enough to attend a hospital or their work's medical service), found an overall incidence of 7.1%. A very similar incidence of 7.3% in 1,000 cases of head injury was found by Rebattu and his colleagues in 1966, although Rauh in 1967 described anosmia in 18 of 115 injuries (15%). Taking the larger unselected series as a guide, therefore, it would seem that the overall incidence of anosmia after head injuries is of the order of 7%.

The wide variation in the reported incidence is probably related to the selection of the head injuries reviewed because, as we shall see, the incidence of anosmia varies enormously with the severity of the head injury.

The incidence of post-traumatic anosmia in relation to the severity of the associated head injury. Few authors have attempted to correlate the incidence of anosmia with the severity of the injury in unselected and consecutive series of head injuries. Many have described the incidence in a selected group of (usually) severe injuries (Laemle 1931; Helsmoortel 1933; Goland 1937). Others have stated their views without numerical analysis. Russell (1960), for example, stated that anosmia usually occurs after a severe head injury but may be seen in those patients in whom there has been a post-traumatic amnesia (PTA) of less than 1 hour while Leigh (1943), in his large series, remarked that although there was no direct correlation with severity, anosmia did appear to be associated with more severe injuries. Kindler (1936), on the other hand, claimed that the production of anosmia depended in no way on the severity of the injury.

Piacentini (1949) found 15 patients (3%) with anosmia in 502 head injuries, but stated that 12 out of the 15 (80%) had been associated with a fractured skull. Klingler and Jost (1963), finding 5% of patients with anosmia out of 155 who had been admitted to hospital, pointed out that the anosmia was associated in all cases with cerebral contusion (where the incidence was 16%) and, indeed, he claimed firmly that post-traumatic anosmia was only seen where cerebral contusion had been diagnosed by other means. Rebattu et al. (1966) referred to the significance of loss of consciousness at the time of the head injury and found that 75% of those patients with anosmia had been unconscious. His overall incidence of anosmia was 7.5% but where consciousness had been lost, it was 21%.

Mifka (1964), reviewing 1,000 patients whose head injuries were serious enough to warrant admission to hospital, found an overall incidence of

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