Results of clinical olfactometric studies

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SUMMARY

A modification of a flow olfactometer with a new application apparatus, with which "quasi-free" nasal respiration allows the elimination of adaptation without a special testing room, subsequent results using this device to examine olfactory thresholds before and after septum operations, as well as reference to threshold increases in 57 post-operative cases of cheilognathopalatoschisis are reported. An esthesio-neuroblastoma as well as the deformity syndrome with cheilognathopalatoschisis and encephalodystrophy are used as examples for combined olfactory transmission and perception disorders. Studies of 55 smokers with primary neurosensory disorders demonstrated a threefold increase in the olfactory threshold and an up to 50% decrease "fatique-time". A mean acetone deviation factor of 1.93 was seen in 100 students from 20-27 years of age before and after eating. Correspondingly, after a substantial breakfast and lunch, the olfactory threshold attained its maximum daily value within 90 minutes, much more pronounced than after intake of 80 grams of glucose solution. In contrast to the literature, the olfactory threshold was seen to continuously increase, dependent on age. Studies of the perceptive and recognition threshold on 100 normal individuals and 28 patients with hyposmia exhibited with 3 σ, a significant difference. In patients with hyposmia, the absolute values for the two threshold types vary greatly, however not their deviation factors. More importance should be attached to the sense of smell as the so-called lesser senses give us the greatest pleasures.

ADVANCES in olfactometry in the past few years have led to a greater differentiation in the studies of physiological and pathological olfactory functions.

Clinically, the question is continually raised as to the presence of respiratory or essential neuro-sensory disorders. The last mentioned of these can be further subdivided into pre-bulbar, bulbar and post-bulbar disorders. Respiratory, as well as neurosensory variations in the olfactory capacity can be more precisely explained clinically and quantitatively through the determination of a reduction in the threshold sensitivity in the sense of a hyposmia or anosmia.

A flow olfactometer with free nasal respiration, better suited for many studies than the unphysiological insufflation method was employed. A special experimental chamber with rapid and complete exchange of the air was used to elimi-

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nate "adaptation" so strongly present with the sence of smell. Because of the great expense in procurement, the apparatus used up until now was modified so that the application device permits "quasi-free" nasal breathing. As a result of the large volume of the nasal olive, the flow disturbances due to pathological turbulence in the nose were held to a minimum. With limited dead space, rapid changes in concentration can be achieved and adsorption to the wall can be held to a minimum by providing sufficient quantities of air supplied with scent molecules applied immediately in front of the connecting olive. Most important, however, is that only a minimally lower pressure is present in the olfactory olive with respect to the external pressure, so that no scent molecules escape. This is achieved by a fan which can additionally and controllably suck in air through a variable opening.

Olfactometric threshold studies demonstrate constantly strong variations among individuals. Therefore, the question as to whether a true hyperosmia exists is, at least, questionable. As the olfactory impulse is centrally and bilaterally transmitted across the anterior commissura, unilateral, total loss of olfactory sensation should be seen as occuring peripherally.

Respiratory disorders or disorders of the transmission of olfactory sensation, that is the prevention of the transmission of the air or gas treated with scent molecules are caused by anatomical alterations of the nasal structure with the resulting pathological flow relationship as are observed in extreme cases of bilateral nasal entrance or choanal atresia and other anomalies. All other ventilation obstructions of the nose, for example, foreign body obstructions including rhinoliths, various tumors and cysts, as well as mucosal swelling including hyperplasias (polyps) also cause an increase in the respiratory olfactory threshold. As a rule, these can be remedied even in more serious cases.

Studies of olfactory thresholds in combination with the measurements of nasal resistance before and after septum operations were carried out by Kittel and Waller (1973) on 52 patients. It was shown that in cases of at least unilateral olfactory sense reduction, a significant post-operative improvement in the threshold with simultaneous reduction of the nasal resistance could be seen.

Respiratory hyposmias can also be improved non-operatively, for example by using antiphlogistic and other agents which reduce the swelling. Even in cases of loss of the nose in lupus vulgaris and an improvement in the olfactory threshold using plastic nasal prosthesis or through respective nasal inserts which direct the inspiratory flow, could be achieved.

The precision of several olfactometers discovers even the finer, most frequently overlooked, olfactory threshold changes.

Studies of the olfactory capability of 57 children with previously operated cheilognathopalatoschisis ranging in age from between 6 to 12 years, demonstrated increases in the respiratory threshold due to frequent pathological nasal respiratory relationships, the often encountered nasopharyngitis and rhinitis and to not infrequently seen chronic catarrh with mucosal hyperplasia (Kittel, Moser, and Schneider, 1975).

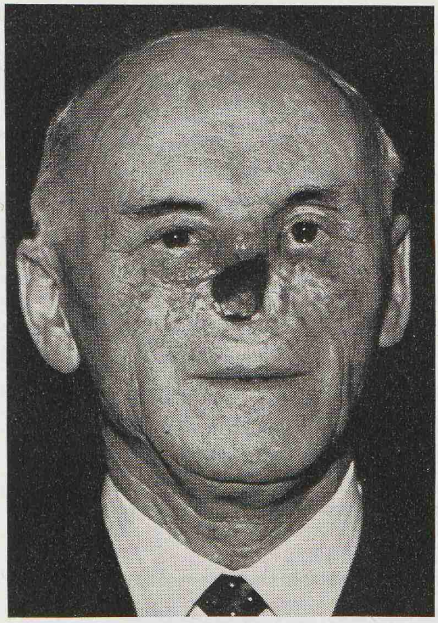


Figure 1. "Destructive Lupus Vulgaris".

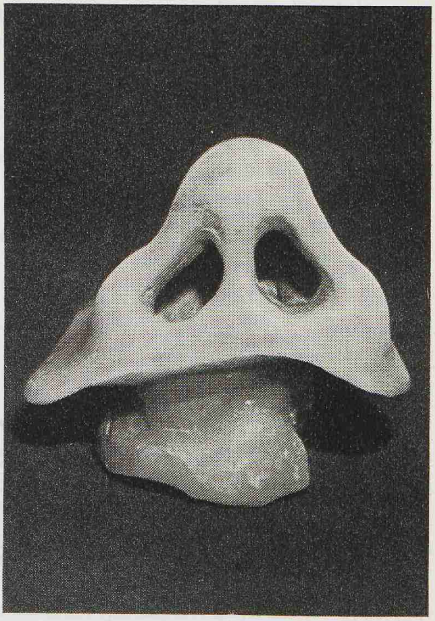


Figure 2. "Nasal Insert directing the Respiratory Flow".

Figure 3. "Plastic Nasal Prosthesis with attachment to the Eyeglasses".

The bi-rhinal mean of cleft palate patients was 8.43 x 14^{14} molecules/ml of air. That of a control group of 66 children was 7.27 x 10^{14}. With a standard error of the mean of $3\sigma = 0.66 \times 10^{14}$, the difference is significant. This, however, was insignificant in cases of isolated cleft palate, although a recognizable tendency for an increased threshold was present. The threshold was correspondingly high with continuous cleft in all three methods of stimulus application, (bi-rhinal, mono-rhinal - right and left). From this an at least relative hyposmia could be deduced, in as much as a threshold of 30 x 10^{14} is required for an absolute hyposmia. On the cleft side, the olfactory threshold values were more frequently and more strongly increased. In this patient collective, they were the highest in five cleft palata children with a large, still open, connection between the nose and mouth.

Combined olfactory transmission and olfactory sensitivity disturbances as described by Kittel (1968) in an analogous manner to auditory transmission and sensory disturbances, can be caused by nasal tumors as well as inflammation with swelling and intoxication in so far as the CNS-olfactory epithelia are also damaged. A classic example for combined damage is seen in the infrequently encountered esthesioneuroblastoma.

The malformation syndrome with cheilognathopalatoschisis and nasal deformities on the one hand, as well as encephalodystrophies with frontal lobe defects on the other hand, also cause combined disturbances. They also frequently occur in chemical workers and smokers as a result of the epithelial irritation with swelling and olfacto-toxic effects.

These two groups also most frequently display neuro-sensory damage and olfactory sensitivity disorders due to their exposure to sulfurous and halogen compounds, spray paint aerosols, including epoxy resins and with numerous other substances. Thus these olfactory disorders are in a sense analogous to the hearing disorders encountered in workers exposed to a great deal of noise. These workers should also be subjected to yearly olfactometric and odorimetric studies.

Although smoking also causes combined disorders as a result of chronic inflammatory mucosal swelling from smoker's catarrh, the pyridines and colloidins in the smoke specifically damage the receptor cells. Furthermore, nicotine causes synaptic blocking.

Threshold determinations and fatique time measurements were carried out on 55 smokers of nearly identical age divided into five groups with a relatively constant daily cigarette consumption. These showed that dependent on consumption with free nasal passage, repeated application of zero stimuli, light sniffles and a stimulus duration of two seconds, a continuous olfactory stimulus increase and reduction of the fatique time occurs (Kittel, 1970). The fatique time was defined as the period between initial perception and extinction of the olfactory sensation, in each instance with a fifty-fold increased threshold concentration. Heavy smokers exhibit a threefold higher threshold than non-smokers; their fatique time is only about half that of non-smokers.

Threshold increase and fatique time shortening express the loss of "fine taste".

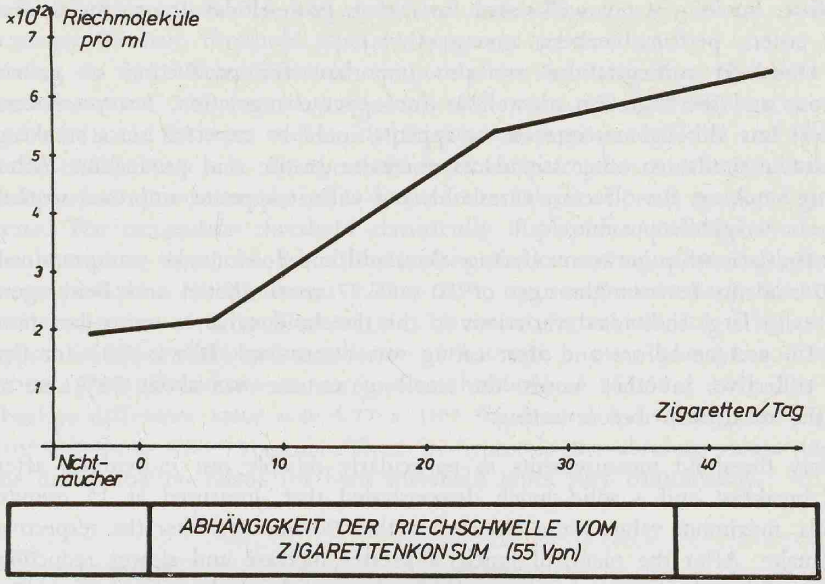
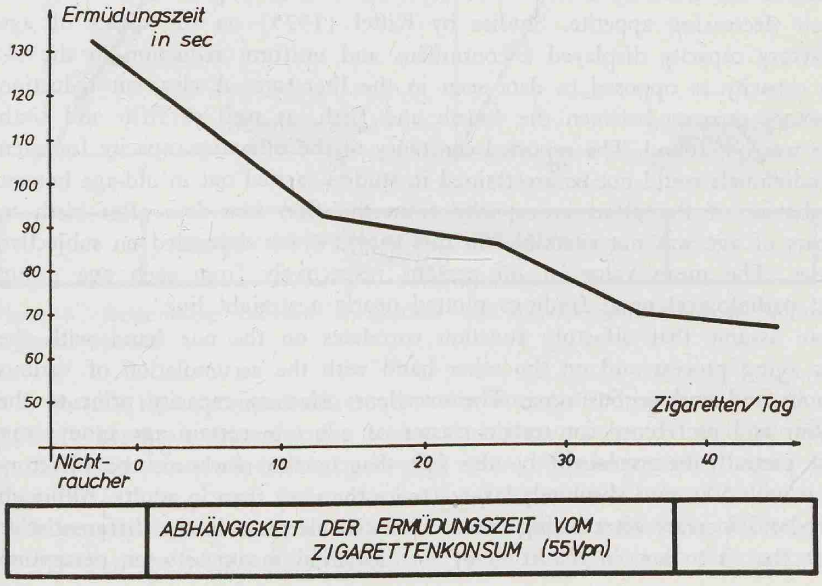


Figure 4. Relationship between olfactory threshold and cigarette consumption (55 persons).

Figure 5. Relationship between fatique time and cigarette consumption (55 persons).



Therefore, smoking is not well-suited for certain professional groups (wine and coffee tasters, perfume makers, among others).
Since threshold concentrations are also important for production of gastric secretions and the digestion as well as for appetite regulation, heavy smokers often eat less although an increase in appetite could be expected since smoking in a matter similar to other stimulants, increases gastric acid production. After stopping smoking, the olfactory threshold, and with it appetite, improves so that a gain in weight is possible.
The interrelationship between olfactory threshold and food intake was examined in 100 students between the ages of 20 and 27 years (Kittel and Reitberger, 1970) with large individual variations in the threshold value, a mean deviation factor for acetone before and after eating was determined. It was 1.93 for the entire collective; in other words, the smell of acetone was about 50% worse after the meal than before eating.

Olfactory threshold measurements in particularly reliable test individuals after a full breakfast and a solid lunch demonstrated that, measured at 15 minute intervals, maximum values were observed within 90 minutes after the respective food intake. After the plentiful lunch, a greater increase and slower reduction was seen although the threshold was lower than in the early morning prior to eating with its probable nervous-humeral influences and often less marked hunger sensation. After oral intake of 80 g of 50% glucose solution, the sensitivity variations were not as pronounced as after a normal meal.

Whether olfactory threshold increases are only the result or partially the cause of the satiation, does not, with the regularity of its increase, in any way change the possibility of using the deviation factor as a measure for human satiation. Increases in the olfactory threshold in older individuals might also be a factor for their decreasing appetite. Studies by Kittel (1975) on the effect of age on olfactory capacity displayed a continuous and uniform reduction in the olfactory capacity as opposed to data seen in the literature. A clear-cut reduction in olfactory capacity between the fourth and fifth, as well as fifth and sixth decades was not found. The reported constancy of the olfactory capacity for even older individuals could not be ascertained in studies carried out in old-age homes. The behavior of the olfactory capacity from the first few days after birth to five years of age was not examined in this study, which depended on subjective responses. The mean value of 40 persons respectively from each age group without pathological nasal findings plotted nearly a straight line.
One can assume that olfactory function correlates on the one hand with the organic aging process and on the other hand with the accumulation of various exogenous and endogenous noxi. The excellent olfactory capacity prior to the sixth year and preference for certain classes of odors in certain age groups can at least partially be explained by the fact that in the newborn, the olfactory region is with 500 mm^2 absolutely larger (twice the sice) than in adults. Although the threshold increase after eating causes a relative decrease in the differentiative capacity, that is to say, in reduction of the deviation factor between perception

and recognition threshold, the age-related threshold increases do not cause any tendency for both threshold types to approach one another.

Studies on the differentiation of the perception and recognition threshold were carried out on 100 normal individuals (62 males, 38 females) between the ages of 18 and 28 years and on 28 patients with hyposmia (Kittel and Wendelstein, 1971). As a rule, the threshold level for perception and recognition were independent of whether lesser to greater concentrations were measured or vice versa. The recognition threshold consistently displayed a marker increase over the perception threshold. For all the investigations it was significant with 3α. The mean perception threshold was 7.77 x 10^{14}, the recognition threshold 12.54 x 10^{14} molecules of acetone/ml of air. Deviation factor was 1.6. The cases of hyposmia had a differentiation factor of 1.8 with a mean perception threshold of 88.47 x 10^{14} and a mean recognition threshold of 161.68 x 10^{14}. The mean absolute difference value was 4.77 x 10^{14} for normal individuals, and 73.21 x 10^{14} for those with hyposmia. Thus, in hyposmia the absolute values and not the differentiation factor for both threshold types vary considerably.

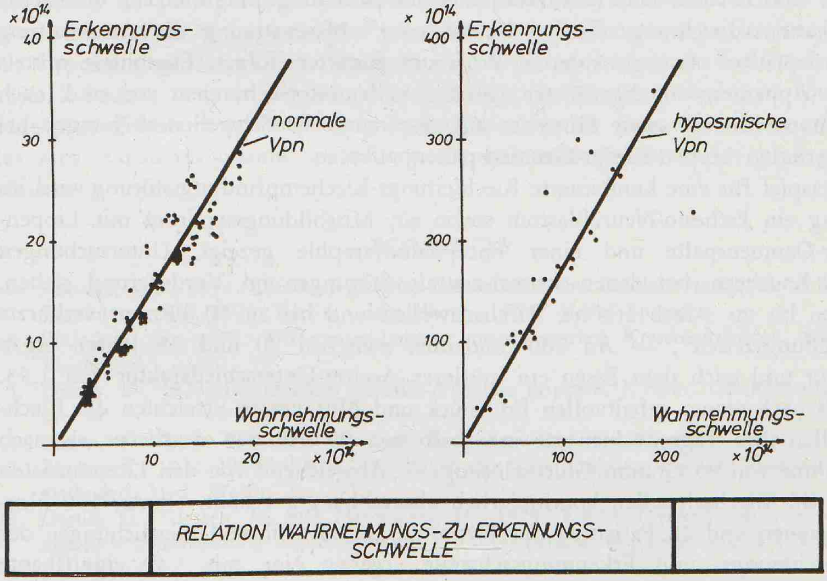


Figure 6. Relationship between recognition- and perception-threshold in normal and hyposmic persons.

It is possible that the minor differences sometimes noticed between both threshold types in hyposmia represent the recruitment phenomenon. If such an olfactory recruitment exists at all, it can, by its very nature, only be present in neuro-sensory disorders.

Neurosensory hypo- or anosmia can be caused by a number of factors besides damage to the olfactory epithelium as a result of exposure to olfacto-toxic sub-

stances and cellular metabolic waste as is not infrequently seen in influenza with its pathological feelings of tiredness or with herpes or ozena. Among these factors are pressure, inflammation or chemotoxic damage of the fila and central olfactory pathway, trauma with filal tears or with basal frontal lobe lesions, intracranial tumors such as meningioma of olfactory bulb, metabolic and hormonal disturbances, idiopathic defects or deformity syndrome, pigmentation disorders, numerous neurological and psychiatric syndromes, the most varied heredictory syndromes, meningitis and encephalitis, and other intracranial processes as well as physiologically with advancing age. Clinical experience with olfactory disorders is so varied that only a small part of all experimental results can be reported here. However, the afore-mentioned results once again make clear that the olfactory presents numerous interesting aspects from both a physiological and pathological viewpoint, and in no way should its designation as a latter or secondary sense be associated with insignificance. Finally, according to an earlier expression, it is precisely these lesser senses which give man his greatest pleasures.

ZUSAMMENFASSUNG

Einem Bericht über die Modifikation eines Strömungsolfaktometers mit neuer Applikationsvorrichtung, die bei "quasi-freier" Nasenatmung die Ausschaltung der Adaptation ohne gesonderten Prüfraum gestattet, folgen Ergebnisse mittels dieser Apparatur durchgeführter Riechschwellenuntersuchungen vor und nach Septumoperationen, sowie Hinweise auf respiratorische Schwellenerhöhungen bei 57 operierten Lippen-Kiefer-Gaumenspaltenpatienten.

Als Beispiel für eine kombinierte Riechleitungs-Riechempfindungsstörung wird im Vortrag ein Esthesio-Neuroblastom sowie ein Missbildungssyndrom mit Lippen-Kiefer-Gaumenspalte und einer Encephalodysraphie gezeigt. Untersuchungen an 55 Rauchern, bei denen sensori-neurale Störungen im Vordergrund stehen, ergaben bis zu 3-fach erhöhte Riechschwellen und bis zu 50 Prozent verkürzte "Ermüdungszeiten". — An 100 Studenten zwischen 20 und 27 Jahren ergab sich vor und nach dem Essen ein mittlerer Aceton-Unterschiedsfaktor von 1,93. Jeweils nach einem gehaltvollen Frühstück und Mittagessen erreichten die Riechschwellen ihre Tageshöchstwerte innerhalb von 90 Minuten deutlicher als nach Aufnahme von 80 Gramm Glucoselösung. — Abweichend von den Literaturdaten waren die Riechschwellen kontinuierlich altersabhängig erhöht. — An 100 Normalpersonen und 28 Patienten mit Hyposmie durchgeführte Untersuchungen der Wahrnehmungs- und Erkennungsschwelle ergaben eine mit 3 @ signifikante Differenz. Bei Hyposmien wichen zwar die absoluten Werte beträchtlich, nicht jedoch der Unterschiedsfaktor für die beiden Schwellenarten auseinander. — Man sollte dem Geruchsinn wieder mehr Bedeutung beimessen, zumal gerade die sogenannten niederen Sinne die höchsten Genüsse bedingen.

RÉSUMÉ

Les auteurs utilisent un olfactomètre dont les caractéristiques — système d'application du stimulus et ventilation nasale quasi libre — permettent d'éliminer le facteur adaptation, sans l'emploi d'une chambre d'examen spéciale.

Ils rapportent les résultats d'une étude des seuils olfactifs mesurés avant et après septoplastie. Ils constatent une élévation des seuils chez 57 patients opérés de bec de lièvre et fente palatine.
Un estésioneuroblastome, un syndrome malformatif avec bec de lièvre et fente palatine, une encéphalodystrophie sont présentés comme exemples d'une atteinte olfactive mixte de transmission et de perception.
L'étude de 55 fumeurs démontre une élévation du seuil olfactif qui est 3 fois supérieur aux valeurs normales, ainsi qu'une diminution d'environ 50% de la période de fatigue c'est-à-dire du temps écoulé entre la perception initiale et la fin de la sensation olfactive.
Chez 100 étudiants âgés de 20 à 27 ans, le seuil olfactif pour l'acétone apparaît plus élevé, en moyenne d'environ 50%, après un repas. Les valeurs maxima du seuil olfactif sont mesurées 90 minutes après l'ingestion d'un repas copieux. Les modifications du seuil sont plus importantes qu'àprès l'ingestion d'une solution contenant 80 gr de glucose.
A l'opposé des données de la littérature, le seuil olfactif apparaît en augmentation progressive avec l'âge.
Les seuils de perception et d'identification olfactives mesurés chez 100 sujets normaux et chez 28 hyposmiques présentent des différences significatives. Chez les hyposmiques, les valeurs absolues des deux seuils varient fortement d'un sujet à l'autre, mais le rapport entre les deux seuils est relativement constant. Une plus grande importance devrait être accordée au sens olfactif car ce sont les sens considérés comme les plus petits qui nous procurent les plus grands plaisirs.

REFERENCES

1. Amoore, J. E., 1971: Olfactory genetics and anosmia. In: Beidler: Handbook of Sensory Physiology, Vol. IV/1. Berlin - Heidelberg - New York: Springer.
2. Bodechtel, G., 1974: Differentialdiagnose neurologischer Krankheitsbilder. Stuttgart: Thieme.
3. Dimov, D., 1973: Les troubles olfactifs d'origine grippale. J. franç. Oto-Rhino-Laryng., 22, 53.
4. Engen, T., 1973: The sense of smell. Ann. Rev. Psychol., 24, 187.
5. Fischer, R., 1972: Physikalische Grundlagen der Nasenfunktion und ihre Untersuchungsmethoden. Med. Wschr. 26, 271.
6. Gerull, G., Giessen, M. and Mrowinski, D., 1974: E.R.O. (evoked response olfaktometry) - Untersuchung der Adaptation für olfaktorische Impulsreizung. HNO (Berl.) 22, 233.
7. Hansen, D., 1971: Zur Pathogenese von Geruchs- und Geschmacksstörungen. Dtsch. med. Wschr. 96, 1196.
8. Harper, R., 1972: Human Senses in Action. Edinburgh - London: Churchill Livingstone.
9. Herberhold, C., 1975: Funktionsprüfungen und Störungen des Geruchssinnes. Arch. Oto-Rhino-Laryng. Kongressbericht. 210, 67.
10. Hommerich, K. W., 1964: Störungen des Gehörs und des Geruchs bei endokraniellen Erkrankungen. Arch. Ohr.-Nas.-KehlkHeilk. 183, 86.
11. Joyner, R. E., 1964: Effect of cigarette smoking on olfaktory acuity. Arch. Otolaryng. 80, 576.
12. Kittel, G., 1961: Bilaterale Taubheit mit Vestibularausfall und Anosmie nach stumpfem Schädeltrauma. Z. Laryng. Rhinol. 40, 515.

13. Kittel, G., 1968: Die moderne Olfaktometrie und Odorimetrie. Z. Laryng. Rhinol. 47, 893.
14. Kittel, G., 1970: Möglichkeiten der Olfaktometry. Ermüdungsmessungen bei Rauchern. Z. Laryng. Rhinol. 49, 376.
15. Kittel, G., 1975: Olfaktometrie. HNO 23, 20.
16. Kittel, G., Moser, M. and Schneider, S.: Der Lippen-Kiefer-Gaumenspaltträger und sein Geruchsinn. (in Druck).
17. Kittel, G. and Reitberger, U., 1970: Wechselwirkungen zwischen Riechschwellen und Nahrungsaufnahme. Arch. klin. exp. Ohr.-Nas.-KehlkHeilk. 196, 381.
18. Kittel, G. and Waller, G., 1973: Der geruchsverbessernde Effekt durch Septumoperationen nach Cottle. Z. Laryng. Rhinol. 52, 280.
19. Kittel, G. and Wendelstein, P. G. J., 1971: Zur Differenzierung der olfactiven Wahrnehmungs und Erkennungsschwelle. Arch. klin. exp. Ohr.-Nas.-KehlkHeilk. 199, 683.
20. Koch, U., 1971: Das Ästhesioneuroepitheliom des Olfaktorius. Z. Laryng. Rhinol. 50, 667.
21. Lehnhardt, E. and Rollin, H., 1969: Berufsbedingte Riechstörungen, HNO (Berl.) 17, 104.
22. Matzker, J., 1965: Riechen und Lebensalter-Riechen und Rauchen. Arch. Ohr.-Nas.-KehlkHeilk. 185, 755.
23. Mc. Cartney, W., 1968: Olfaction and Odours. Berlin - Heidelberg - New York: Springer.
24. Petrides, P., 1972: Geruchs- und Geschmacksstörungen bei Diabetes mellitus. Münch. med. Wschr. 114, 265.
25. Rauh, Ch., 1969: Zur Pathophysiologie des Geruchssinns. In: Aktuelle Themen der Oto-Rhino-Laryg. Berlin: VEB Verlag Volk u. Gesundheit.
26. Schaupp, H., 1971: Geruchsstörungen nach Grippe. Arch. klin. exp. Ohr.-Nas.-Kehlk Heilk., 199, 692.
27. Zilstorff-Pedersen, K., 1972: Über Altersveränderungen des Geruchssinnes. HNO (Berl.) 20, 124.

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