Tinnitus: Facts, Theories, and Treatments

ISBN: 0-309-55433-0, 150 pages, 6 x 9, (1982)

Tinnitus

Facts, Theories, and Treatments

Dennis McFadden
Working Group 89
Committee on Hearing, Bioacoustics,
and Biomechanics
Commission on Behavioral and Social Sciences
and Education
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1982

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This work relates to Department of the Navy Contract N00014-80-C-0159 issued by the Office of Naval Research under Contract Authority NR 201-124. However, the content does not necessarily reflect the position or the policy of the Department of the Navy or the government, and no official endorsement should be inferred.

Library of Congress Cataloging in Publication Data
Main entry under title:
Tinnitus: facts, theories, and treatments.

Prepared by: Working Group 89, Committee on Hearing, Bioacoustics, and Biomechanics, Commission on Behavioral and Social Sciences and Education, National Research Council

Bibliography: p.

Includes index.

1. Tinnitus. I. McFadden, Dennis. II. National Research Council (U.S.). Working Group 89.
[DNLM: 1. Tinnitus. WV 272 M168t]
RF293.8.T56 1982 617.8 82-19028

ISBN 0-309-03328-4
Available from
NATIONAL ACADEMY PRESS
2101 Constitution Avenue, N.W.
Washington, D.C. 20418

Printed in the United States of America
First Printing, November 1982
Second Printing, August 1984

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Foreword

Many readers who work in fields related to hearing and deafness are familiar with the reports of working groups of the Committee on Hearing, Bioacoustics, and Biomechanics (CHABA). These reports are prepared by special study committees, known as working groups, set up by CHABA to advise federal agencies on issues of significant national need.

The original impetus for this report was a request brought to the National Academy of Sciences in 1980 by the Food and Drug Administration (FDA) of the U.S. Department of Health and Human Services. The FDA asked for assistance in evaluating the efficacy and safety of tinnitus masking units and tinnitus instruments (masker/hearing aid combinations). The National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) shared the FDA's interest in concerns about these matters and, in addition, had a broader interest in the status of knowledge about tinnitus. In response to the joint request from these two agencies, CHABA, with the approval of the Commission on Behavioral and Social Sciences and Education and the National Research Council, created Working Group 89 and charged it with the preparation of a general, critical review of the entire topic of tinnitus, including tinnitus maskers and instruments.

The performance of a working group depends strongly on the energy and wisdom of its chairman. Considerable skill and, often, diplomacy are required in preparing a final report that incorporates the scientific knowledge and opinions of a range of experts while providing the practical guidance needed by those who originally raised the question. In the case of tinnitus and tinnitus masking, it was felt that the person charged with the preparation of an evenhanded review and evaluation of current knowledge

should be an experienced auditory scientist who was not associated with a specific position on the issues to be studied. The broad scope of the following report, the balanced manner with which controversial questions are treated, and a light touch where many would have fallen into turgid scientific prose show how well this challenge was met by the chairman of Working Group 89, Dennis McFadden.

The members of CHABA deeply appreciate the efforts of the members of Working Group 89 and especially of its energetic chairman in providing this timely report.

CHARLES S. WATSON
CHAIRMAN
COMMITTEE ON HEARING, BIOACOUSTICS, AND BIOMECHANICS

Preface

Most of the work on this report was done during 1981 and early 1982, but it is not possible to specify a single cutoff date for the source materials used for the report. in some instances, no information more recent than several years old was uncovered; in other instances, preprints of articles or chapters to be published later in 1981 or 1982 were available and were used. It was not possible to be systematic, comprehensive, and up to date in all of the various literatures touched by this report, and the purely fortuitous way that some information was discovered makes it believable that important facts may have been missed. During the writing of the report, two symposium volumes on tinnitus appeared (CIBA Foundation, 1981; Shulman, 1981a); both are widely cited throughout this report, but the serious student of tinnitus is encouraged to examine the originals. In an attempt to provide as comprehensive a research bibliography as possible, some sources have been included even though they were not cited in the report for one reason or another.

Much of the information on tinnitus maskers/instruments comes from a single source--the University of Oregon tinnitus clinic. Because I wanted the review of that information to be as objective an possible, I tried to maintain a degree of distance between myself and personnel at the clinic, none of whom I have met. I did exchange several letters with Jack Vernon, the clinic's director, and he provided me with helpful comments on an early draft of the section on the efficacy of tinnitus maskers/ instruments.

I wish to thank the members of the working group for their splendid assistance in preparing this report. All were generous with their time and prompt in their replies to requests for comments on my successive drafts. Their

cogent comments about style, substance, and organization contributed greatly to the content and form of the report.

Beyond the members of the working group, many people contributed to the final form of this report. Milton Whitcomb, study director of the Committee on Hearing, Bioacoustics, and Biomechanics (CHABA), offered excellent counsel and guidance at critical points and, as always, proved to be an effective, genial, and unobtrusive administrator. I am indebted to CHABA members Donald H. Eldredge, William D. Neff, and Charles S. Watson for their valuable comments on the manuscript. Earleen Elkins of the National Institute of Neurological and Communicative Disorders and Stroke and Harry Sauberman of the Food and Drug Administration frequently provided me with the necessary perspective on the problem. Eugenia Grohman of the National Research Council was meticulous in her copy editing of the manuscript and did more than anyone else to render it readable and correct. Judy Searcy made preparation of the index as painless as such a job can be. And most important, the timely preparation and form of the report are due in large part to the word-processing skills of Lanier Bayliss, whose careful attention to detail and persistent good cheer are deeply appreciated.

A number of my friends have noted that tinnitus is an unlikely topic for me to be reviewing and critiquing, and I am the first to agree. I do not consider myself to be an expert on tinnitus: I have never done research on the topic, nor have I ever seen a tinnitus patient. I tried to read all the material that is available on tinnitus and to draft as organized, comprehensive, and critical a summary of the topic as I could. The realities of deadlines being what they are, not everything I hoped to do got done. However, as the reader will appreciate, any tinnitus report written in the foreseeable future will necessarily be only an interim report; far too little is known for it to be otherwise. I would appreciate hearing from readers about omissions, errors of fact or interpretation, and differences of opinion.

DENNIS McFADDEN
CHAIRMAN
WORKING GROUP 89

Contents

1 Introduction 1
Overview of the Report 1
A Definition of Tinnitus 5
Prevalence of Tinnitus 7

2 Facts, Theories, and Issues 10
Etiology of Tinnitus 10
Miscellaneous Unequivocal Sources of Tinnitus 11
Tumors of the Eighth Nerve 12
Noise Trauma and Presbycusis 13
Mechanisms of Tinnitus 14
Spontaneous Rates of Primary Fibers 15
Decoupling of Stereocilia 17
The Objective/Subjective Issue 18
Can Tinnitus Exist in the Absence of Hearing Loss? 23
Tinnitus in Children 24
Possible Experimental Models of Tinnitus 24
Meniere's Disease 27

3 Measurement Procedures 32
Quality of the Tinnitus 32
Spectral Location of the Tinnitus 35
Pitch Matching 35
Masking 36
Related Masking Results 37
Magnitude of the Tinnitus 42
Annoyance of the Tinnitus 45
Is the Tinnitus Monaural or Binaural? 48

The Issue of Beats with Tinnitus 51
Some Ways Tinnitus Is Not Like an External Sound 52
Summary of Measurement Procedures 54

4 Treatments 55
Psychological intervention 55
Surgery for Tinnitus 57
Exposure to Intense Sound 58
Drugs and Tinnitus 59
Drugs Causing Tinnitus 59
Salicylates 60
Quinine 62
Tobacco 63
Caffeine 63
Alcohol 63
Cocaine 63
Marijuana 63
Oral Contraceptives 63
Heavy Metals 63
Drug Therapy for Tinnitus 64
Niacin 66
Vitamin A 66
Lidocaine 67
Carbamazepine 72
Tocainide Hydrochloride 74
Phenytoin Sodium 75
Primidone 76
Sodium Fluoride 76
Sodium Valproate 77
Sodium Amylobarbitone 77
Alcohol 78
Miscellaneous Drugs 78
Conclusions About Drugs and Tinnitus 79
Allergy and Diet 81
Biofeedback 82
Hypnotherapy 83
Acupuncture 84
Electrical Stimulation 85
Alteration in Air Pressure 87
Tinnitus Maskers/Instruments 89
Efficacy of Tinnitus Maskers/Instruments 92
The Early Reports 93
The Later Reports 94
Other Reports 101
Conclusions 104

Residual Inhibition 104
Safety of Tinnitus Maskers/Instruments 107
Spectral Characteristics 107
Intensity of Tinnitus Maskers/Instruments and Duration of Use 110
Damage/Risk Criteria and Tinnitus Maskers/ Instruments 114

5 Standardizing Procedures 117
Medical Examination 117
Audiological Examination 119

References and Bibliography 123

Index 147

1

Introduction

Hearing sounds that do not originate in the world outside the body is an experience that probably every human has at one time or another in life. The sounds heard range from popping and clicking to intermittent roaring and buzzing to continuous pure-tones. In their diversity, these experiences have two things in common: they all originate, in one way or another, from inside the head, and they are all known as tinnitus.

Tinnitus can accompany a wide array of serious and minor disorders of the ear and of the body in general. As shall be seen, some causes are reasonably well understood, but most are not. Effective palliative treatment has been established for some forms of tinnitus, but for most, effectiveness of treatment continues to be unpredictable.

The following section provides an overview of the tinnitus problem and of the various topics that are discussed in detail in subsequent sections.

OVERVIEW OF THE REPORT

Tinnitus can be defined as the conscious experience of a sound that originates in the head of its owner. In some cases tinnitus exists because there is actually a source of acoustic energy located somewhere in the head and neck area —a contracting muscle, a clicking jaw, a defective vein or artery, etc.—that can also be heard by a second party, with or without the aid of special devices. However, the majority of tinnitus cases have no detectable acoustic basis, but instead arise from anomalies in one or more of the elements of the neural chain that constitutes the auditory nervous system. It is important to

emphasize at the outset that tinnitus is itself not a disease, but a symptom that is common to many maladies that afflict many different structures within and without the auditory system. A frequently drawn analogy is to fever and headache —symptoms that accompany many different disorders. Like those two symptoms, tinnitus can range in severity from mild and easily overlooked to severe and debilitating.

Existing estimates of the prevalence of tinnitus are all flawed in one way or another, but all are in accord over its ubiquity. A recent British survey indicates that about 1 percent of the general population has severe, occasionally debilitating tinnitus; were this percentage an accurate estimate of prevalence in the United States, there would be about 2.5 million Americans afflicted with severe tinnitus. At the other extreme, it may be that nearly everyone experiences a mild form of tinnitus at one time or another in life and thus that mild episodes of tinnitus are "normal" in the sense that an occasional backache or pimple is normal.

The majority of tinnitus cases are probably never reported as medical or auditory problems, but are simply accepted as normal phenomena or as occasional minor irritants. For some people, however, tinnitus can become as totally debilitating as any serious systemic disorder. It can be severe enough to turn an otherwise healthy, well-adjusted person into someone unable to work or socialize. Anecdotes persist about people committing, or threatening to commit, suicide because of severe tinnitus and of others begging to have their offending ear surgically destroyed in the hope of escaping a relentless tinnitus. Every experienced hearing specialist has seen less severe, but nevertheless serious, cases.

Given the multiple origins of tinnitus, it should be expected that no single treatment for tinnitus is likely to be found. Also, it should be no surprise that many forms of treatment have been attempted over the years. Unfortunately, few of these have had much success until recently. Indeed, until lately, hearing specialists have had little to offer tinnitus sufferers in the way of relief, and far and away the most common "treatment" even for severe tinnitus has been the statement that lots of other people have the problem, that there is nothing much that can be done about it, and that the patient will simply have to learn to live with it. This grim situation has brightened considerably, however, and the prospects now appear good that the symptom of severe

tinnitus will eventually be alleviated for a substantial fraction of its sufferers. The two most promising areas of advancement in the treatment of tinnitus involve drugs and masking.

In the past few years, several drugs have been identified as potent agents against several of the common forms of tinnitus. These include lidocaine, carbamazepine, and sodium amylobarbitone. So far each has drawbacks of one sort or another that prevent its immediate widespread use, but related drugs are being developed and studied.

Many tinnitus sufferers independently discover that sounds from the external world can cover up or mask their tinnitus. Such people realize relief in relatively noisy environments or from background sounds such as the interstation noise on a radio. Hearing specialists have known about the effectiveness of masking against tinnitus for decades, but not until recently was it offered in a systematic way as a palliative for tinnitus.

In the mid-1970s a group of hearing specialists at the University of Oregon Medical School developed a device for generating a masking sound that could be mounted in a standard hearing aid chassis. This tinnitus masker was later combined with a hearing aid in the same chassis, and this combination was called a tinnitus instrument. For about 5 years, tinnitus maskers and instruments have been prescribed and distributed to tinnitus patients coming to the Oregon tinnitus clinic. The primary question posed to CHABA Working Group 89 was to evaluate the efficacy and safety of tinnitus maskers/instruments.

Nearly all the available information on efficacy comes from follow-up questionnaire data collected, collated, and published by the Oregon group. Their series of reports contains some inconsistencies and some (perhaps understandable) exaggerations, but overall the outcomes are encouraging. It is important to remember in what follows that the Oregon group surely does not see a random selection of tinnitus sufferers, but rather, those severely enough afflicted to be motivated to travel to the clinic (and affluent enough to be able to). That is, the patients in the Oregon sample probably include some of the most severely afflicted tinnitus sufferers in the country, and the various success rates should be interpreted accordingly.

There are a number of reasonable ways to calculate estimates of success from the Oregon data; the basic issue is what number is most appropriate for use as the

denominator of the success ratio. About 25 percent of the people who appear at the Oregon clinic are sent away without a recommendation to try a masker, hearing aid, or instrument, and this number has been essentially constant over the years. If these people are included in the denominator when calculating a success ratio, the estimates obtained are very conservative ones. Less conservative estimates are obtained by excluding this 25 percent of the people from the denominator on the grounds that they were not regarded to be good candidates for masking and thus should not be "counted against" the success of the treatment. Still less conservative estimates involve excluding both this 25 percent and those people who were given a recommendation for one of the three devices—masker, aid, or instrument—but who did not purchase one after the trial period. Depending upon which of these increasingly less conservative denominators is used, between about 42 percent and 83 percent of the respondents to the Oregon clinic's questionnaire report either total or partial relief from their tinnitus through use of the recommended devices. Considering the presumed degree of tinnitus severity in this sample, such success rates are certainly encouraging. Masking of tinnitus is not a panacea, but from these statistics, and from the absence of other, equally effective treatments, masking must now be regarded as the treatment of first choice. The primary reason for caution regarding this conclusion is that at present essentially all of the data on efficacy originate from a single source.

The safety of tinnitus maskers/instruments is more difficult to evaluate at this time than is their efficacy. There are several reasons for this. First of all, there is essentially no information available about the sound-pressure levels (SPL) experienced by typical wearers of tinnitus maskers/instruments, nor about the temporal patterns of these exposures. Such information is crucial, of course, if safety is to be evaluated by consulting standard damage/risk criteria or exposure guidelines. Most tinnitus maskers/instruments have maximum outputs of 85–95 dBA, and some are rated as high as 105–110 dBA. The most widely used U.S. exposure guideline permits exposure to 90 dBA for only 8 hours per day when the exposures occur 5 days per week. Thus, nearly all currently available maskers/instruments are capable of exceeding common damage/risk criteria and therefore have the potential to produce hearing loss.

There exists a serious question, however, of whether tinnitus maskers/ instruments should be regulated on the basis of their presumed, or even demonstrated, risk to hearing. The issue is that for many thousands of people tinnitus is a severe, debilitating condition, and it can be argued that the risk, or even the inevitability, of some additional hearing loss caused by a masker/ instrument is a price worth paying for the relief from the tinnitus. An obvious parallel exists with hearing aids. Evidence is accumulating that the levels and durations experienced by many wearers of hearing aids may eventually cause additional hearing loss, yet few hearing professionals regard that risk (or inevitability) to be serious enough to warrant restricting the availability or use of hearing aids, nor presumably would hearing-impaired people be willing to forfeit their aids on these grounds. Similarly, certain drugs carry long-term risks (or inevitabilities) that do not rule out their use when they are all that is available for treatment of a serious malady.

It appears that for the moment the best policy to follow in regard to the safety of tinnitus maskers/ instruments is to make users explicitly aware of the virtues for hearing conservation of low masker levels and of intermittent patterns of exposure. Until more is known about the levels and durations of exposure experienced by typical users of tinnitus maskers/instruments, more restrictive policies are premature, and even when more is known, such policies may be judged inappropriate.

In summary, after years of neglect, tinnitus is rapidly coming to be a topic of active interest to clinicians, physiologists, psychoacousticians, and other hearing specialists. Much is left to be learned, but it is clear that tinnitus is now an established, legitimate research area; it is not just another buzz word.

In the rest of this report, the issues briefly mentioned here are examined in more detail, and the evidence on which current beliefs about tinnitus are based is presented and evaluated.

A DEFINITION OF TINNITUS

As is true for so many phenomena, a concise yet precise definition of tinnitus is difficult to achieve. As noted, one distinguishing feature of tinnitus is that the origin of the perceived sound is inside the head. In some cases there is an actual sound source—a vibrating body—underlying

the perception. Some examples are vascular anomalies, muscular contractions, clicking jaws, and even trapped insects. Obviously, the existence of such sound sources raises the potential for their being detected by others, with or without the aid of amplifying devices. Indeed, some truly remarkable cases have been reported. Glanville et al. (1971) discussed a case of a young child having a high-pitched tinnitus that could be heard at a distance of 4 feet. The child's father and one of its two siblings had similar, but less dramatic, emissions, and, interestingly, the father was unable to hear his own emission. Huizing and Spoor (1973) also reported on a patient whose ear emitted a high-pitched tone that was audible to all but herself. She, and the father mentioned previously, had circumscribed hearing losses in the spectral region of their emissions. Thus, technically speaking, neither had tinnitus, since they could not hear their own sounds. Other examples of intense emissions have been noted (see Zurek, 1981), one in a dog (Decker and Fritsch, 1982) and one in a cat (CIBA Foundation, 1981:133).

A long-standing distinction in the tinnitus literature is between those instances of tinnitus that have a vibratory origin and can be heard by others, as well as by the patient, and those instances in which the tinnitus is audible only to the patient, presumably because the site of origin is inside the nervous system and there is no vibratory concomitant. Various dichotomies have been proposed for these two forms of tinnitus: for example, vibratory/nonvibratory (Fowler, 1939, 1941), objective/ subjective, extrinsic/intrinsic (Atkinson, 1947), pseudo/ true (Jones and Knudsen, 1928). The intent behind the distinction, of course, is to partition instances of tinnitus into two broad categories as an initial aid to diagnosis and treatment.

Problems with all of these dichotomies have recently been spotlighted by the development of new procedures for monitoring acoustic activity in the outer ear canal (Kemp, 1978). The facts are discussed at length in the section "The Objective/Subjective Issue" in Chapter 2; all that need be noted here is that with these new techniques, weak acoustic activity has been found to underlie some instances of tinnitus that previously had been categorized as subjective or nonvibratory. The point is that there are a number of factors that can affect the ability of a person to detect a sound, and failure by a clinician to do so cannot reasonably be taken as evidence for the nonexistence of the sound. So, while these various dichotomies

are well intentioned, application of the criteria has been, and surely will continue to be, much more difficult than was initially apparent. Even more refined procedures may eventually reveal acoustic concomitants of other forms of tinnitus, thereby moving them from the subjective to the objective category, and conceivably, nonacoustic but nevertheless objective measures of some forms of tinnitus may be developed that will further muddy the distinction between objective and subjective forms of the malady. To the extent that treatment and scientific insight are dependent upon such categorizations, there is a need for a new appreciation of the shortcomings of the objective/subjective, vibratory/nonvibratory, and other dichotomies. While these distinctions are sometimes used in this report, our commitment throughout has been to the view that objective measures may eventually be developed for many, if not all, forms of tinnitus.

It should be noted that a recent major publication on tinnitus (CIBA Foundation, 1981:Appendix I) explicitly proposes to exclude from the definition of tinnitus those cases previously classified as objective. While the objective/subjective distinction may have become muddy in recent times and may deserve to be dropped (see "The Objective/Subjective Issue" in Chapter 2), there is no apparent justification for disregarding history and tradition by excluding from the definition of tinnitus those "head noises" that happen to have an acoustic concomitant.

In this report, then, the term tinnitus is used to describe the conscious experience of a sound that originates in the head, either acoustically or physiologically. For inclusion in the definition, no criterion of severity, loudness, annoyance, or other characteristic of the tinnitus need be met. A distinction not made by this definition, but which will eventually have to be drawn, is between the elementary sensory experiences of the tinnitus sufferer and the organized perceptual experiences of the hallucinating mental patient.

PREVALENCE OF TINNITUS

It is apparently widely believed that mild, occasional tinnitus is experienced by nearly everyone at some time or another and that these brief episodes are not necessarily associated with, or precursors to, auditory pathology. Given this belief, it is peculiar that most formal

attempts to estimate tinnitus prevalence do not effectively discriminate such instances of tinnitus from its severer, annoying, and protracted forms, nor do they discriminate between the tinnitus of normal-hearing and hearing-impaired subjects. The ambiguity of past questionnaires and interview questions surely is a significant source of variability in prevalence statistics. Nevertheless, over the years, the following prevalence statistics have been reported:

1. About 32 percent of all U.S. adults report having had tinnitus at one time or another, and about 6.4 percent of that same population characterizes the tinnitus as severe or debilitating (National Center for Health Statistics, 1968).
2. About 85 percent of 2,000 consecutive patients seen by an otologist complained of tinnitus (Fowler, 1944).
3. About 83 percent of 500 consecutive patients with acoustic neuromas had tinnitus (House and Brackmann, 1981).
4. About 79 percent of 190 patients with otosclerosis had tinnitus (Glasgold and Altmann, 1966).
5. About 75 percent of all cases of deafness report tinnitus (Heller, 1955).
6. About 13 percent of school children with audiometrically normal hearing report having tinnitus, at least on occasion (Nodar, 1972).
7. About one-half of a sample of deaf children had tinnitus (Graham, 1980).
8. Tinnitus prevalence increases with age up to about age 70 and declines thereafter (Reed, 1960).

A recent British survey (Institute of Hearing Research, 1981) has attempted to use less ambiguous questions than those used in the past in order to better discriminate between chronic, problem tinnitus and occasional, "normal" tinnitus. Of course, the information obtained may not generalize perfectly to the U.S. population. Nevertheless, preliminary analyses indicate that about 17 percent of the British population sampled had problem tinnitus, that about 1 percent had tinnitus that produced severe annoyance, and that about 0.5 percent had tinnitus that resulted in an inability to lead a normal life. About equal percentages (9–10 percent) reported tinnitus with and without apparent hearing impairment, fewer reported unilateral tinnitus than tinnitus in both ears or "in the

head," prevalence increased with age, and there was little sex difference in prevalence rate. (Note that if the 0.5–1 percent value does apply to the present U.S. population, it would mean that approximately 1.2–2.3 million people have severe or debilitating tinnitus.)

Some authors have attempted to estimate tinnitus prevalence in various subgroups of hearing-impaired people. These attempts are typically plagued by small sample size and other sampling problems. Nevertheless, Heller and Bergman (1953) studied 100 consecutive patients at a Veterans Administration audiology clinic and found that 73 percent had tinnitus. The preponderance of these (39/100) were diagnosed as perceptive deafness, with conductive deafness (13/100) and otosclerosis (8/100) being the two next most common categories. Reed (1960) reported that in a sample of 200 patients referred for severe tinnitus, 38 percent were diagnosed as suffering primarily from presbycusis, 16 percent primarily from acoustic trauma, 12 percent primarily from Meniere's Disease, 10 percent from idiopathic symptoms, and the remaining 24 percent from a variety of other maladies. J. T. Graham (1965) distilled some data from Heller showing that tinnitus is reported by 71 percent of those whose hearing impairment was diagnosed as perceptive, by 65 percent of those diagnosed as conductive, by 88 percent of those diagnosed as combined, and by 85 percent of those diagnosed as otosclerotic.

One unexpected fact has been found in some surveys of tinnitus. It appears that monaural tinnitus is about 1.5 times more likely to occur in the left ear than in the right (Hazell, 1981b; Institute of Hearing Research, 1981). Verification should precede attempts at explanation (see CIBA Foundation, 1981:31).

While the various official attempts to estimate tinnitus prevalence are subject to various procedural criticisms, the resulting estimates do imply two things: that the magnitude of the tinnitus problem has not been appreciated and that effective treatment of tinnitus has not been available. Whenever there is an ignored, afflicted population of this size, the potential for abuse by charlatans and misguided healers is great, as is the risk of exaggerating the effectiveness of any new treatment. Caution and critical restraint must be exercised when evaluating new or modified treatments, and that has been the attempt throughout the preparation of this report.

2

Facts, Theories, and Issues

ETIOLOGY OF TINNITUS

A student of tinnitus has no difficulty finding lists of conditions or mechanisms that cause, or are at least believed to cause, tinnitus. Often much more difficult to uncover is the evidence on which these beliefs are based. Simple intuition seems to be the most common basis. For example, it is intuitive that a localized lesion along the organ of Corti might cause an "irritation" that would in turn lead to continuous discharge in a small population of primary auditory fibers and, thus, produce a tonal or narrowband tinnitus. Similarly, localized damage in a small population of neurons in the cochlear nucleus or other auditory structure might be expected to produce a perceptual experience of sound where none exists. A "sensory epilepsy" due to an interruption in the afferent-efferent loop sounds like it might be responsible for a tinnitus experience in the spectral region of the interruption. And it sounds reasonable that emotional changes might produce changes in the composition of the inner-ear fluids and/or changes in the cochlear blood supply that might in turn produce differential neural activity that would produce auditory sensations.

But while these and dozens of other equally attractive proposals may be partially or entirely correct, there is little evidence to support any of them. To be sure, the etiology of a particular tinnitus has occasionally been established--particularly for those having clearly audible vibratory concomitants—but in the vast majority of cases the cause is unknown. There can be no doubt that reducing this ignorance about site of origin is desirable, for, presumably, type of treatment will eventually come to be based on diagnoses of specific causes. However, it is

doubtful that to date this ignorance has been a major deterrent to effective treatment, for so little is known about etiology and treatment that typically nearly everything is eventually tried if a patient persists in his or her complaints.

Over the years a truly staggering number of proposals have been made about the etiology of tinnitus in all of its various forms. It would serve little purpose to attempt a comprehensive review of these here; instead, a few selected proposals are discussed. The topic of drugs that cause tinnitus is considered in Chapter 4.

Miscellaneous Unequivocal Sources of Tinnitus

There are some unambiguously established causes of tinnitus. The lack of ambiguity is due to different factors in different cases, but some common factors are (1) sudden occurrence or appearance of both the cause and the tinnitus, (2) frequent or invariable associations between the cause and tinnitus, and (3) disappearance of the tinnitus following removal or treatment of the cause.

As might be expected, many of these unequivocal sources of tinnitus involve gross causes. Severe blows to the head, for example, can produce transient, long-term, or permanent tinnitus of various types (Shucart and Tenner, 1981). Overdoses of various drugs and general anesthetics used during surgery can initiate or exacerbate a tinnitus. Anemia, hypertension, hypothyroidism, and migraine have been linked with tinnitus (CIBA Foundation, 1981:232–236), as has multiple sclerosis (Shucart and Tenner, 1981). Partial or total immobilization of the middle ear structures, such as that produced by otosclerosis or even impacted cerumen (ear wax), can cause tinnitus. The onset of tinnitus sometimes coincides with pregnancy, but so does otosclerosis, so the latter may be the true culprit. Meniere's Disease has a characteristic tinnitus as one of its primary symptoms (see "Meniere's Disease" in this chapter). So-called sensorineural hearing loss such as that induced by chronic or acute exposure to intense noise is frequently accompanied by tinnitus. And, of course, already mentioned are the well-established causes of certain forms of objective tinnitus—anomalies of the vasculature or musculature of the head, neck, and jaw.

In regard to tinnitus caused by ear wax, Feldmann (CIBA Foundation, 1981:234) believes that simple occlusion of the canal is not the source of the tinnitus but that

attachment or contact of wax with the tympanic membrane is required. Coles (CIBA Foundation 1981:235) believes that the wax is serving to simply attenuate external sounds, thereby allowing a preexisting tinnitus to be revealed, an effect that can sometimes also be observed by inserting plugs into the ear canal.

It has been asserted that the tinnitus that accompanies otosclerosis is typically present only as long as the hearing loss is less than 50–60 dB (Lempert, 1946; Saltzman, 1949; J. T. Graham, 1965); beyond this value, it gradually diminishes and then disappears altogether. This assertion deserves confirmation, for, if true, it would have important theoretical implications. Specifically, it appears to be in accord with Kemp's (1981) ideas about the origin of so-called spontaneous otoacoustic emissions (see "The Objective/ Subjective Issue" in this chapter).

Among the types of tinnitus originating in the musculature of the head is the rapid clicking sound produced by involuntary, rhythmic contractions of the muscles of the soft palate. The condition is known as palatal myoclonus (see MacKinnon, 1968), and the actual sound source is believed to be the snapping together of the walls of the Eustachian tube.

Tumors of the Eighth Nerve

Unilateral tinnitus is frequently an early symptom of a tumor that affects the eighth nerve. Other—typically later-developing—symptoms are unilateral high-frequency hearing loss and vertigo. The overwhelming majority of these tumors are not malignant. Most are unilateral and most begin on the vestibular branch of the eighth nerve inside the internal auditory canal. (Because most eighth-nerve tumors are associated with the Schwann cells and are on the vestibular branch of the nerve, the term vestibular schwannoma is preferable to the older term acoustic neuroma or neurinoma.) The damage done by these tumors is primarily due to their compressing other tissues as they slowly grow in size.

Brackmann (1981a) reported that tinnitus was present in 83 percent of a group of 500 patients suffering from unilateral tumors of the eighth nerve and that tinnitus was the first symptom noticed by about 10 percent of that group. Ronis (1981) agreed with the latter figure but found tinnitus to be an initial symptom in 92 percent of

his cases. Postoperatively, "virtually all" of Ronis' patients had some tinnitus; Brackmann felt that the tinnitus was better postoperatively in about 40 percent of his patients and worse in about 50 percent (House and Brackmann, 1981). No information is offered about similarities and differences in tinnitus quality or magnitude, preoperative and postoperative. This lack of information makes it impossible to know if the "old tinnitus" persists or a "new tinnitus" is created by the surgical procedure.

A fair summary of this issue appears to be that: (1) unilateral tinnitus and hearing loss can arise in so many ways that they are poor predictors of eighth-nerve tumors, (2) the vast majority of people with an eighth-nerve tumor do report tinnitus either as the initial symptom or in combination with hearing loss and vertigo, and (3) even successful surgery to remove the tumor rarely eliminates the tinnitus. The clinician should remain alert to the possibility of an eighth-nerve tumor when unilateral tinnitus and hearing loss combine with vertigo, but it is far from inevitable that a tumor will prove to be the cause.

For some time, the prevailing wisdom was that early surgery is in order for all eighth-nerve tumors. Consequently, there was (and still is) concern that nonsurgical treatment of the tinnitus per se may work to the detriment of the patient by "covering up" an important symptom of a serious disorder (see "Medical Examination" in Chapter 5). In recent years however, there has been an increasing realization that many eighth-nerve tumors grow slowly and cause the patient only minimal discomfort and alteration of lifestyle. Thus, the feeling of urgency about tumor removal has been reduced somewhat; exceptions are tumors in children and young adults, which are still viewed as candidates for early removal. Further, the concern about "covering up" the tinnitus may be wholly unwarranted. It may well be that tinnitus caused by eighth-nerve tumors is refractory to most or all known treatments for tinnitus—that is, that this important symptom cannot be "covered up." At this point, essentially nothing is known about this possibility, notwithstanding its practical and diagnostic importance.

Noise Trauma and Presbycusis

Prolonged and repeated exposure to intense sound produces a distinctive pattern of hearing loss not unlike that associated with aging (presbycusis). The loss is typically

greatest at the highest frequencies and less at the lower frequencies, often with a reasonably sharp transition region between the affected and unaffected regions. Such patterns of hearing loss are very common and they are frequently accompanied by tinnitus. Feldmann (1971) estimated that about one-third of his patients followed this pattern and Schleuning's (1981) estimate was about one-half. Reed (1960) diagnosed 16 percent of his patients as having acoustic trauma and 38 percent as presbycusic. The accompanying tinnitus is reported to be of high pitch, and, when it is matched to a tone, the matching frequency is often located in the sharp transition between regions of greater and lesser hearing loss (Feldmann, 1971; Penner et al., 1981).

MECHANISMS OF TINNITUS

A distinction can be made between presumed or established causes of tinnitus and the actual mechanisms through which the tinnitus is produced. For example, what is the nature (and/or location) of the altered neural activity that produces tinnitus following noise exposure, drug overdose, or a blow to the head? It is intuitive that in many cases of tinnitus, knowledge of the mechanism will eventually prove more valuable for diagnosis and treatment than will knowledge of the cause.

Over the years, many proposals about underlying mechanisms have been offered (a number are mentioned by Durrant, 1981). No comprehensive review is attempted here. Instead, some general comments are made, and then the physiological evidence for two particular mechanisms are examined.

It is intuitive (which is not to say correct) that in order for a person to have an experience of a spectrally distinct sound for which there is no acoustical concomitant, something about the neural activity in that person's auditory pathway must be misleading higher auditory centers into the erroneous experience. That "something" might be a mechanical force—for example, a tumor or a blood clot causing a local compression of neural tissue—or a biochemical or biomechanical upset affecting a subset of neurons at some level in the auditory nervous system. (It is tempting to think of the resulting "tinnitus signal" as an increase in neural activity, but a marked decrease might be equally likely to be detected and to produce an erroneous experience.) Note that the upset could

logically manifest itself at a number of different places in the auditory system; for example, a set of abnormal fibers in the efferent system might produce aberrant behaviors in several disparate locations. A popular place to hypothesize tinnitus-producing upsets is the cochlea itself. It is a complex biochemical and biomechanical system, and it is relatively easy to imagine various specific malfunctions that might be associated with tinnitus.

Although numerous suggestions have been made over the years about possible mechanisms of tinnitus, an accurate characterization about the state of knowledge appears to be that nothing definite is yet known about the roles of biochemical or biomechanical effects operating in the cochlea or beyond to produce tinnitus. Attempts to establish such relations are obviously to be encouraged. One such line of research is discussed in the following section.

One point to keep in mind regarding mechanisms is that if the phenomenology of tinnitus is ever an accurate reflection of its origins, then some of those people with narrowband or tonal tinnitus may have highly localized lesions, while some of those with broadband or complex tinnitus may have numerous or widespread sites of tinnitus origin. The former is more difficult to imagine originating from a general systemic upset than is the latter, and the latter is more difficult to imagine originating from a localized lesion or anomaly.

Spontaneous Rates of Primary Fibers

A recurring idea in the recent tinnitus literature is that some cases of tinnitus may be traced to an abnormality in the resting (or "spontaneous") rates of firing of a localized set of primary auditory fibers. (The abnormality may originate in these primary fibers themselves or in more peripheral cells.) The suggestion has appeared in many forms, but typically the idea is that a small set of fibers is in a state of "irritation" and as a consequence is firing more rapidly than normal. In order to examine the evidence pertaining to this proposal, it is necessary to briefly review the facts of spontaneous firings in normal primary fibers.

The range of spontaneous firing rates is 0–100 action potentials per second in what are believed to be normal primary auditory fibers, but the distribution of rates is bimodal, not rectangular. About 25 percent of the fibers

(in the cat at least) have spontaneous rates below about 15 firings per second, and about 75 percent have spontaneous rates greater than 15 per second (see, e.g., Kim and Molnar, 1979). In order to test the idea of abnormal spontaneous rates underlying tinnitus, it is necessary to administer to animals stimuli that are known to produce tinnitus in humans and to presume that the same physiological changes have been induced, and thus, that the animals would also report tinnitus were thay able. Three such animal models of tinnitus have been used in attempts to determine changes in the spontaneous rate—kanamycin-and salicylate-induced hearing loss and noise-induced hearing loss.

Kiang et al. (1970) measured the spontaneous rates of primary auditory fibers following administration of kanamycin and the consequent destruction of much of the organ of Corti in the basal end of the cochlea. In those fibers with high and middle characteristic frequencies (CF) that could still be found, Kiang et al. observed both elevated thresholds of response and nearly total elimination of spontaneous firings. This led Kiang et al. to suggest that the tinnitus heard by patients who have received ototoxic drugs, and perhaps also by those with presbycusic and high-frequency, noise-induced hearing loss, is due not to an elevation in the spontaneous rates of a small set of neurons, but rather, to "the existence of distinctly different distributions of activity in tonotopically adjacent elements of the auditory nerve" (p. 264). That is, the absence of spontaneous activity in those still-functional neurons at the "edge" of a cochlear lesion might lead to a sensation of sound at some higher brain location. There is some psychophysical evidence that tinnitus does occur in the transition region between normal and impaired hearing—that is, at the edge of a pattern of loss (Penner et al., 1981).

Liberman and Kiang (1978) studied the spontaneous firing rates of primary auditory fibers in cats that had been exposed to intense noise bands for an hour or two. The effects differed in the different spectral regions surrounding the exposure, but in those regions where responsivity to sound was greatly reduced, the spontaneous rates were significantly depressed. Salvi et al. (1978) exposed chinchillas to an octave band of noise of sufficient intensity to produce about 40 dB of asymptotic threshold shift and then recorded from neurons in the cochlear nucleus (not primary fibers). The postexposure spontaneous rates were lower than normal in the frequency

regions in which behavioral thresholds were elevated and hair cell lesions were found.

Schmiedt et al. (1980) studied primary fibers in gerbils after administration of either kanamycin or noise exposure. In accord with Kiang et al. (1970), they reported a tendency toward lower-than-normal spontaneous rates in the kanamycin animals, but contrary to Salvi et al. (1978), they found a tendency toward higher-than-normal spontaneous rates in the animals exposed to noise. A curious aspect of this latter outcome is that the cells with increased spontaneous rates had low characteristic frequencies; tinnitus following noise exposure is typically high in frequency.

In contrast with Kiang et al. (1970), Dallos and Harris (1978) observed no differences in the spontaneous rates of primary fibers in chinchillas following kanamycin administration and consequent hair cell loss.

Evans et al. (1981) recorded from primary fibers in cats before and after administering salicylate and thus were able to report predrug and postdrug measures of spontaneous rates. They found essentially no change in the low spontaneous rate fibers, but the mean rate of fibers with high spontaneous levels increased significantly. Interpretation of this outcome is difficult, for the implication is that the effect did not apply differentially across cell CF, yet the tinnitus reported by salicylate users is typically of high pitch. Without a local, or a differential effect by CF, it is difficult to see how an "edge" could be set up.

Thus, while the idea of altered spontaneous rates in primary fibers is a plausible explanation of noise-or drug-induced tinnitus, the neurophysiological evidence is still very mixed. One reason for this may be species differences in the lesions produced by noise and ototoxic drugs (Dallos and Harris, 1978). The example does clearly point out the great need for an acceptable animal model of tinnitus.

Decoupling of Stereocilia

The tips of the stereocilia of the outer hair cells insert into small invaginations on the underside of the tectorial membrane; thus, as the traveling wave produced by an acoustic stimulus displaces the basilar membrane, a shearing action operates on the stereocilia. One possible consequence of noise exposure, and of exposure to certain

ototoxic drugs, is that the physical characteristics of the stereocilia may be altered. Tonndorf (1980) has argued that any ciliary change that alters the degree of coupling between the stereocilia and the tectorial membrane will alter the inherent noise level at that interface. Specifically, loss of stereociliary stiffness would lead to a partial decoupling at the hair cell/tectorial membrane interface and a consequent increase in the inherent noise level. Such partial decoupling over a relatively long section of the cochlea might produce a broadband tinnitus, while a localized decoupling might produce a narrow-band or tonal tinnitus. Tonndorf suggested that partial decoupling may be the underlying mechanism for tinnitus (and hearing loss) in Meniere's Disease and in acute noise exposure.

THE OBJECTIVE/SUBJECTIVE ISSUE

As noted previously, over the years some dramatic instances have been reported of loud sounds emanating from people's ears (Glanville et al., 1971; Huizing and Spoor, 1973). Since some of these passed unheard by their owners, they were not truly instances of tinnitus, and the old distinction between objective and subjective tinnitus has been further blurred by recent discoveries. Recall that, until now, the dichotomy had been based simply on the question of whether or not the examining professional could hear the subject's tinnitus, with or without the aid of a stethoscope. Obviously, the uncontrolled variables in such a situation are many. They include the intensity of the source, the amount of attenuation from source to receiver, the examiner's own hearing level in the frequency region of the source, and the ambient noise level in that frequency region.

Stimulated by the pioneering work of Kemp (1978, 1979a,b) on what has come to be called the evoked cochlear mechanical response (ECMR) or, more colloquially, the cochlear echo, several investigators have developed techniques for inserting sensitive miniature microphones into the external auditory meatus (Wilson, 1979, 1980a; Wilson and Sutton, 1981; Zurek, 1981). Somewhat to their surprise, they have discovered that it is quite common for normal ears to emit acoustic energy at one or more frequencies. These spontaneous otoacoustic emissions (OAEs) typically are too weak to be heard by an external observer, even using a stethoscope (sealing the auditory

canal provides amplification of these signals that previous observers did not have). In addition, most of these OAEs are also inaudible to the people who have them; thus, in most instances OAEs do not satisfy the technical definition of tinnitus—a conscious experience of a sound that originates in the head. However, in some instances a perceptual experience does correspond to an OAE, and it is those instances that most interest us here.

Before discussing details, we should emphasize that examples of all three logical possibilities for tinnitus are now known to exist. First, there are perceptions of sounds that have no (as yet detected) objective counterpart and that are presumably caused by abnormal activity at some place or places in the auditory nervous system (classical subjective tinnitus). Second, there are perceptions that do have an objective, vibratory concomitant originating from some structure in the middle or inner ear or elsewhere in the head and neck (classical objective tinnitus). Third, there are sounds—apparently emitted by the cochlea—that are physically detectable in the ear canal but that are not audible to the people who have them (unheard OAEs).

The levels of OAEs (measured in the occluded ear canal) vary from about 0–30 dB SPL, but the measured level is apparently not a good predictor of whether the OAE will be heard by its owner. According to Kemp's (1981) measurements, OAEs are not strictly tonal, but are noise bands, about 1.2–4.7 Hz in width (measured with a resolution of 0.16 Hz). Zurek (1981) and Wilson and Sutton (1981) have shown that OAEs can be suppressed by tones of other frequencies and, further, that the pattern of suppression shows a frequency selectivity that associates the phenomenon with cochlear activity. (Information about the time course of this suppression is provided by Kemp and Chum [1980] and Zurek and Clark [1981].) Thus, the narrowband emissions measured acoustically in the ear canal are generally believed to originate in the cochlea.

To date, there have been reports of about a half-dozen cases of people who experience a tonal tinnitus that corresponds to an OAE detectable in their ear canals (Kemp, 1981; Wilson and Sutton, 1981; Zurek, 1981). In contrast, easily 10 times this number of OAEs have been found that do not have a corresponding tinnitus. (Numerous subjects have had several unheard OAEs, sometimes accompanied by one that is heard.) In the most comprehensive survey to date, Tyler and Conrad-Armes (no date) studied 25 subjects with sensorineural hearing loss and tinnitus and found

only one OAE—and that was low frequency and did not match the subject's tinnitus. They also studied 20 normal-hearing subjects, and while five OAEs were found, none was audible to its owner. Zurek (1981) tested nearly three dozen people covering a wide range of age and auditory conditions; more than 20 OAEs were found, but none corresponded to a reported tinnitus. In addition, Zurek (personal communication) studied 16 people who complained of tinnitus of various sorts and found no emissions whatsoever.

In contrast to the negative results of these surveys, Wilson and Sutton (1981) did find an OAE corresponding to a reported tinnitus in 4 of 10 people studied; other, unheard OAEs were also present in these ears. Most of these people had apparently responded to a newspaper ad soliciting tinnitus sufferers, but it appears that none was afflicted with a particularly severe form. The 4 people having an OAE corresponding to a conscious experience may not have been actual "sufferers" so much as naturally careful introspectors, and perhaps with practice others might also come to hear their normally unheard OAEs.

In those four subjects for whom an OAE did correspond to a conscious experience, Wilson and Sutton (1981) demonstrated in a number of ways that the two phenomena are related, but not in all of the following ways for all subjects. Contralateral pitch matching has shown a good correspondence between the tinnitus pitch and the OAE frequency, the tinnitus and the OAE have both been cancelled by precise adjustment of the level and phase of an external tone, and changes in air pressure introduced into the outer-ear canal have shifted the frequency of the OAE upward and produced a corresponding increase in the pitch of the tinnitus. Elsewhere, Wilson (1980a) reported that the tinnitus and the OAE have been observed to undergo simultaneous fluctuations in magnitude. Further, heard OAEs are typically not described as tonal, but as rough or noisy (Wilson and Sutton, 1981; Zurek, 1981), in apparent accord with Kemp's (1981) demonstration that physically OAEs are noise bands, 1.2–4.7 Hz in width.

Kemp (1981) indicates that stimulation with sounds intense enough to produce temporary threshold shift (TTS) can produce diminutions in OAE magnitude that can last for several minutes, but it is not clear that the OAEs studied had a concomitant tinnitus that behaved similarly. Apparently not yet studied is whether an OAE and its associated tinnitus both undergo similar patterns of

residual inhibition (see "Residual Inhibitions" in Chapter 4).

Most OAEs detected to date have been in the low-to mid-frequency region, but this is surely due in large part to the high-frequency attenuation characteristic of the middle ear. Any conductive hearing loss would offer a similar impediment to detection of OAEs. Reported failures to detect an OAE in association with an existing tinnitus (e.g., Tyler and Conrad-Armes, no date) must always be evaluated with this present technical limitation in mind. That is, the dearth of OAEs detected above about 4000 Hz—where much reported tinnitus lies—should not yet be taken as evidence against OAEs being associated with high-frequency tinnitus.

To date, few OAEs have been found in nonhumans (see CIBA Foundation, 1981:133; Zurek and Clark, 1981). Decker and Fritsch (1982) described a dog that emitted a continuous narrow band of noise centered at about 10.3 kHz; since it was a pet, this animal was not fully studied physiologically, but the description suggests an OAE. Evans et al. (1981) did study a single guinea pig with a single OAE and found that: (1) the OAE was not affected by paralysis of the middle ear muscles; (2) it was raised in frequency by both increases and decreases of air pressure in the middle ear; (3) its acoustic level was reduced, but its round window magnitude was unaltered by changes in middle ear pressure; (4) it could be suppressed by introducing external tones of the appropriate frequency and intensity; (5) it was abolished under hypoxia and returned following restoration of a normal oxygen supply; and (6) it was basically unchanged by sectioning of the tendons of the middle ear muscles. Of great interest would be animal experiments on OAEs using some of the drugs that have been shown to be effective against tinnitus in humans (see "Drug Therapy for Tinnitus" in Chapter 4). Also of interest is whether and how OAEs might be produced in animal ears so that experimental study could proceed. Zurek and Clark (1981) were able to induce OAEs in only 2 of 17 chinchillas exposed to intense noise.

Much speculation surrounds the relationship between OAEs and the so-called cochlear echoes discovered by Kemp (1978). These echoes are acoustic energy detectable (using a microphone sealed in the outer-ear canal) several milliseconds following the presentation of a click or brief tone burst. The two effects may prove to originate from the same cochlea structure(s)—the echo being a normal consequence of cochlea design and the OAE being

the result of a local anomaly in the relevant structures. Whatever the eventual relationship of these two phenomena, the newcomer should be aware of their common historical origins.

Heard and unheard OAEs can be found in pathological ears of various sorts as well as in normal ears. It is as yet unclear whether an OAE of either type can exist in a spectral region showing substantial sensorineural hearing loss (an interesting point theoretically), but belief seems to be running against it. Rutten (1980) has shown that cochlear echoes can exist at low frequencies even when there is considerable hearing loss for frequencies above about 2000 Hz.

There is a final fact about OAEs that—while yet to be tied to tinnitus directly—is worthy of mention. Several investigators (Kemp, 1979a; Wilson, 1980a) have noted the relationship between OAEs and what has come to be known as the microstructure of the audiogram. Elliott (1958), Thomas (1975), Cohen (1982), and Kemp (1979a) have all shown that a person's audiogram can have numerous local inversions that are highly stable across long periods of time and that are predictable from certain acoustical measures of cochlear behavior (Kemp, 1979a). These peaks and troughs have been discussed as being normal consequences of the same mechanisms responsible for OAEs and cochlear echoes. Wilson (1980a) has shown that OAEs (heard and unheard) do not invariably occur at peaks or troughs in the audiogram, but interestingly, Glanville et al. (1971) and Huizing and Spoor (1973) did observe circumscribed regions of hearing loss in the region of tonal emission, and Flottorp (1953) believed that his idiotones always resided in a region of localized hearing loss. Also, Minton (1923) reported diminished sensitivity in the frequency region of a subjective tinnitus.

To summarize this topic, many--and perhaps the majority of—normal ears "spontaneously" emit continuous, narrow-band acoustic signals that appear to originate in the cochlea. These signals are low in level, and the vast majority exist unheard by those who emit them. The signals that are heard fit the standard definition of objective tinnitus, but it appears that few, if any, of these cases constitute a problem tinnitus. Several attempts have failed to find concomitant emissions in the ears of people who could truly be regarded as tinnitus sufferers, as opposed to careful introspectors. Further, OAEs do not share certain properties with the common forms of tinnitus (see "Some Ways Tinnitus Is Not Like an External

Sound" in Chapter 3). While it is disappointing to many who had momentarily hoped that some forms of tinnitus would prove to be objective and more easily studied (and perhaps more easily treated) than in the past, it appears that OAEs will not prove to be responsible for many instances of severe, problem tinnitus.

Students of hearing interested in the general topic of otoacoustic emissions are referred to the visionary paper by Gold (1947/1948). A broader review of otoacoustic emissions can be found in McFadden and Wightman (1983).

CAN TINNITUS EXIST IN THE ABSENCE OF HEARING LOSS?

It is common to see the assertion that most, but not all, sufferers from tinnitus have some hearing loss (85 percent according to Vernon et al., 1980; also see CIBA Foundation, 1981:29). Tinnitus without hearing loss is definitely a possibility—for example, when the cause of the tinnitus is a vascular anomaly—but when considering this issue, it is important to note two points.

First, hearing sensitivity is not typically measured above 8 kHz--indeed, on most commercial audiometers it cannot be measured above that frequency. Since hearing loss of various types often proceeds from high frequencies to low, it is very possible that some of those tinnitus sufferers thought to have normal hearing have in fact lost some hearing, but (so far) only in the untested region above 8 kHz. This is obviously consistent with the commonly reported observation that tinnitus is a frequent harbinger of hearing loss. Until it is demonstrated to be incorrect, it appears parsimonious to believe—particularly when the tinnitus is of very high frequency—that there is an accompanying hearing loss, although it may exist beyond the normal audiometric range. Support for this idea can be found in the data of Jacobson et al. (1969).

Second, pure-tone audiometry is typically done only at a set of standardized frequencies spaced at octave intervals. Thus, localized regions of hearing loss related to the tinnitus—above, below, or at the tinnitus frequency--might be missed by this sampling procedure (see Kemp, 1979a; Wilson, 1980).

The point is that even modern audiometers are poor research tools, and, when it comes to tinnitus research, a fixed-frequency audiometer is particularly inadequate.

TINNITUS IN CHILDREN

Two reports exist on the prevalence of tinnitus in children. Nodar (1972) asked a set of tinnitus questions during routine auditory screening of students in grades 5–12 (approximately ages 10–18). Of the approximately 6,000 students passing the screening, about 13 percent reported having tinnitus. This low percentage may reflect an ambiguity in the relevant question—it may have been unclear whether the question pertained to ever or to at that moment. Of those not passing the screening, about 59 percent reported tinnitus. Across the two groups, the most common characterizations of the tinnitus were "high" and "ringing." Since this was primarily a normal-hearing group, it is not surprising that the tinnitus inducing events mentioned were the same as are reported by normal-hearing adults—incidents of noise exposure, illness, stress, etc. From such evidence we might tentatively conclude that young ears are about as prone to short-term tinnitus as older ears, but that children have been less likely to report it.

More interesting perhaps are the observations of Graham (1981b), who questioned 158 partially and profoundly deaf students (ages 12–18) about tinnitus and found that about half had it on occasion. This is reported as being a great surprise to the hearing professionals who work with these children. Graham estimates that about two-thirds of the tinnitus sufferers had at least one episode a week. A curious finding was that the tinnitus was much more likely to be localized toward the better-hearing ear. The discussion following Graham's paper is recommended to those interested in the topic of tinnitus in children (CIBA Foundation, 1981:182–192).

POSSIBLE EXPERIMENTAL MODELS OF TINNITUS

A well-established research strategy in the medical sciences is to develop procedures for inducing the malady of interest in weakened or reversible form in otherwise healthy humans or in species other than man. Therapies that are successful on these "models" of the malady are then evaluated on volunteers actually suffering from the condition. The goal of developing experimental models of this sort is often not just the discovery of an effective treatment for ths malady of interest, but also insight into the basic physiological or neurophysiological mechanisms

involved. Experimental models of tinnitus appear possible, but little research has yet been done using them.

Tinnitus can be reversibly induced in otherwise normal ears in several ways —for example, administration of certain drugs, exposure to intense sounds, and exposure to waveforms with steep spectral skirts. This fact is important for several reasons: (1) it offers at least the possibility of developing procedures to study tinnitus in animals, with all of the usual gains in flexibility over human research, (2) studying (induced) tinnitus in normal human ears would allow certain within-subject controls that may prove valuable and would be otherwise not achievable, and (3) there is the potential for insight into the mechanisms underlying some forms of tinnitus. With very few exceptions, however, induced tinnitus in normal ears has yet to be studied as a model of tinnitus in pathological ears.

Two of the exceptions—Loeb and Smith (1967) and Atherly et al. (1968)—induced a short-term tinnitus through exposure to intense sound. One generalization emerges from these experiments: the induced tinnitus does not occur at the same frequency as the maximal temporary threshold shift (TTS), and where it does occur depends upon the nature of the exposure sound. Following exposure to octave-band noise, the tinnitus frequency was well below the maximum TTS frequency; with one-third octave bands, the tinnitus frequency continued to be lower than the maximum TTS frequency, but the difference was smaller than with octave bands; and with tonal exposure stimuli, the tinnitus frequency was higher than the maximum TTS frequency. From personal experience it is clear that monaural exposure to an intense sound can produce tinnitus in both ears. The qualities are typically different, with the experience in the exposed ear being more broadband and temporally complex, and the experience in the contralateral ear being more tonal. Thielgaard (1951) and Thompson and Gales (1961) mention this contralateral effect, but to our knowledge it has never been widely known or systematically studied. Its existence might be evidence that the efferent system is involved in tinnitus production.

Another procedure for inducing a short-term tinnitus involves the use of noise bands having steep spectral skirts. In our experience, listening for a few minutes to a relatively weak (35–50-dB spectrum level) noise band having skirts of about 400–500 dB per octave can produce

a high-pitched tonal tinnitus that will last several minutes (see McFadden and Plattsmier, 1982a). Lummis and Guttman (1972) used similar waveforms and found that the induced pitch was matched to a frequency about two-thirds octave higher than the edge of a low-pass noise band and about three-fourths octave lower than the edge of a high-pass noise band.

Finally, the tinnitus induced by aspirin and other common drugs also offers research opportunities not yet fully explored (see "Salicylates" in Chapter 4). For example, comparison of the onset and recovery times of the tinnitus and the hearing loss induced by aspirin could be revealing about the mechanics underlying both. Evans et al. (1981) did study the effects of salicylate and lidocaine (see "Lidocaine" in Chapter 4) on the response properties of primary auditory neurons in an attempt to find correlates to tinnitus, but ignorance about whether tinnitus was actually present and, if so, about its characteristics makes interpretation of their findings difficult.

All of the above procedures for inducing tinnitus could be utilized in connection with the ear-canal monitoring systems (mentioned in "The objective/ Subjective Issue" in this chapter) to examine whether the induced subjective experience has an objective counterpart and, if so, whether their spectral, temporal, and other characteristics are in accord. Zurek and Clark (1981) have taken the first step in this direction by inducing otoacoustic emissions (OAEs) in chinchillas by exposing them to noise. Evans et al. (1981) monitored a spontaneous OAE in a guinea pig while performing various psychophysical and physiological manipulations. Kemp (1982) noted some changes in the human cochlear echo (see "The Objective/Subjective Issue") immediately following exposure to intense sounds, and he argued that these may be related to the postexposure experience of tinnitus.

Three attempts to utilize the brain-stem-evoked response (BSER) with tinnitus have been reported (Berlin and Shearer, 1981; Dickter et al., 1981; Shulman and Seitz, 1981). Shulman and Seitz believe that people with tinnitus of central origin have BSERs different from those of people with normal hearing, but the effect requires confirmation. Dickter et al. demonstrate that patients with very similar audiometric configurations and tinnitus complaints can yield very different BSER data.

MENIERE'S DISEASE

The tinnitus associated with Meniere's Disease is being discussed in a separate section because it is more homogeneous and predictable in its character than is tinnitus associated with other disorders and because much is known about the constellation of physiological changes present in this disorder. Thus, the opportunities for isolating the origin(s) of this tinnitus appear great.

True Meniere's Disease consists of three primary symptoms—episodic vertigo, hearing loss, and tinnitus (see Barber et al., 1972). The onset of vertigo is frequently sudden and unanticipated. The episodes may last minutes or hours, but aftereffects often persist for several days. Severe attacks seem to alternate with milder ones in an unpredictable manner. The involvement is typically unilateral, particularly in the early stages of the disorder; estimates of bilateral Meniere's Disease range from 10 percent to 40 percent, although these may include some instances of other disorders that mimic true Meniere's Disease. W. F. House (1975) indicates that the disorder is more common in whites than in blacks (also see CIBA Foundations, 1981:28), is associated with industrialization and urbanization, and is believed to be absent in species other than humans.

The hearing loss experienced during an episode of Meniere's Disease is typically greater, if not exclusively, in the low-frequency region. Typically, the loss is unilateral, present in both air-conduction and bone-conduction tests, and accompanied by a feeling of fullness or pressure in the affected ear. The hearing loss is typically between 15 and 30 dB; it is usually described as fluctuating, since it varies with time and with the episodes of vertigo; and over repeated episodes there is typically a gradual buildup of permanent hearing loss. Recruitment is typically present, as is a diplacusis in the direction of raised pitch in the affected ear. Thus, the general pattern is much like that seen with a cochlear pathology. House (1975) indicates that the typical time course for the various symptoms is as follows: there is first an awareness of fullness or pressure in the ear and a buildup of tinnitus. Then, hearing loss develops, and finally, vertigo sets in. Recovery involves first a diminution in vertigo, then reduction in the feeling of fullness and the tinnitus, and, finally, a subjective return of hearing to normal.

In accord with the primary effects on hearing being in the low-frequency region, the tinnitus associated with an episode of Meniere's Disease is also typically low frequency in quality. Common descriptions are "roaring" and "buzzing." Objectively, Nodar and Graham (1965) found that all of a sample of 11 patients with confirmed Meniere's Disease matched their tinnitus to tones lower than 1000 Hz; the median was 320 Hz. Note that this is not to say that the tinnitus was tonal in character. Zurek (1981) could find no otoacoustic emission (see "The Objective/Subjective Issue" in this chapter) corresponding to the tinnitus heard by the one Meniere's sufferer he tested.

According to Goodhill (1979), current belief is that the primary cause of Meniere's Disease is an anatomical/physiological anomaly. The consequence is a dysfunction of the endolymphatic duct and sac system that somehow produces an excess accumulation (hydrops) of the endolymph. (Endolymphatic hydrops has in fact come to be used synonymously with Meniere's Disease.) In addition, certain other factors appear to be able to potentiate episodes of the disorder—for example, certain vascular conditions, allergies, endocrine deficiencies, and personality variables. The primary histological finding is of a distension of scala media, only occasionally accompanied by degeneration of the organ of Corti. More recently, anomalies of the vestibular aqueduct have also been reported.

Dozens of treatments of Meniere's Disease have been advanced (and criticized) over the years. Surgical treatments have ranged from the extremes of labyrinthectomy and vestibular neurectomy to more selective (and less destructive) procedures aimed at the endolymphatic sac. Arenberg and Bayer (1977) have recently argued that in the past many decisions for surgical intervention were based too heavily on the vertigo symptoms, with too little attention paid to the consequences for hearing. They claim that a large number of initially unilateral cases eventually come to be bilateral, and, thus, that the more destructive surgical procedures should be used sparingly. Arenberg and Bayer believe that recent modifications of the original Portmann procedure for decompressing the endolymphatic sac have yielded good-to-excellent results against vertigo as well as against the symptoms of tinnitus and pressure, without adverse effects on hearing, either immediately or in the long term. Thus, following Fisch (1976), they suggest that surgery of the endolymphatic

sac be accomplished as early as possible in the course of the disease--during the period when the hearing losses are fluctuating and reversible—and that vestibular neurectomy and excision of Scarpa's ganglion be done in the latter stages of the disease. In this way, existing hearing and long-term potential for hearing are maximally protected in both groups.

As an example of the diversity of procedures suggested for treating Meniere's Disease, consider Johnson (1954), who advocated various actions against the sympathetic nervous system. He believed in blocking the stellate ganglion (located along the sympathetic trunk at the level of the seventh cervical vertebrae) with an injection of procaine hydrochloride as early in a Meniere's episode as possible, and, if there were several recurrences, he felt that a partial dorsal sympathectomy was advised. He also performed stellate blocks on sufferers from tinnitus of other origin, and when the block was successful, he did dorsal sympathectomies on them as well. (Blocks were apparently ineffective against tinnitus arising from noise exposure.) These procedures were never widely adopted.

The second major form of treatment for Meniere's Disease is drug therapy. Over the decades, this has ranged from simple reduction of sodium in the diet to the use of diuretics, vasodilators, histamines, antihistamines, tranquilizers, etc. (for a review, see Arenberg and Bayer, 1977). This research has often been lacking in adequate controls, but some recent controlled research makes betahistine hydrochloride (Serc) appear highly effective against the vertigo, hearing loss, and tinnitus of Meniere's Disease (Frew and Menon, 1976). Glycerin does have the ability to produce dramatic short-term improvements in the hearing of some Meniere's patients and, in the process, to eliminate the tinnitus, but this substance is used primarily in diagnosis, not treatment (see Snyder, 1974; Klockhoff, 1975). Arenberg and Bayer (1977) conclude that no drug has yet proven to be satisfactory in its general ability to alter the natural course of the disease process, and—as previously indicated—they favor surgical intervention to accomplish this end.

As noted above, histology reveals a distension of scala media due to hydrops of the endolymph in sufferers from Meniere's Disease of long standing. This distension is primarily at the apical end of the cochlea, where the basilar membrane is most compliant. (Interestingly, it is widely believed that the hydrops appears not to be due

to an overproduction of endolymph, but rather to altered absorption characteristics in the stria vascularis that lead to fluid accumulation.) The twin facts that the loss of hearing is greatest at the low frequencies and that the tinnitus is of low frequency are, of course, in accord with this apical locus of disturbance. Exactly which consequence of the fluid accumulation and distension is responsible for the hearing loss and tinnitus produced by Meniere's Disease has been the object of some attention. An excess of fluid pressure could produce changes in the mechanics and micromechanics of the cochlear duct as well as possibly cause some compression of certain critical structures—such as the tectorial membrane, the hair cells, or the nerve fibers themselves—thus causing stimulation in the acoustic chain. An argument sometimes raised against the possibility of direct compression/stimulation is that histology reveals little or no hair cell or other damage at the apical end of the cochlea. This objection may not be cogent, for there is no apparent need for the compression/stimulation to be so great as to damage the cells, just great enough to activate them. Second, the common visual inspection techniques may overlook subtle effects. Tonndorf (1957) has given examples of changes in basilar-membrane mechanics that can occur with an endolymphatic overpressure; many of these are in accord with the symptoms of Meniere's Disease, but unfortunately no direct inference to tinnitus is possible. Recently, Tonndorf (1980) has argued that the assumption of diminished coupling between the hair cells and the tectorial membrane, due to stereo-ciliary dysfunction, might account for a number of commonly observed auditory anomalies, including sensorineural-type hearing loss and tinnitus. Electrophysiological recordings from animals in which endolymphatic hydrops has been experimentally induced (see, for example, Kimura, 1968) might shed light on the origin(s) of tinnitus in Meniere's Disease and other disorders as well.

Vernon et al. (1980) have reported that Meniere's sufferers constitute only 1 percent of all the cases they see at their Oregon tinnitus clinic. There are various possible explanations of this peculiar fact, but the field is narrowed by a second fact. The tinnitus described by these Meniere's sufferers is not the low-frequency roaring that typically accompanies acute episodes, but rather, it is middle-or high-pitched and frequently tonal in quality. The implication is that these patients were seeking relief from a tinnitus that

was either not, or was only indirectly, related to the Meniere's Disease. As it turns out, the tinnitus complained about was easily masked, and a hearing aid or tinnitus masker/instrument typically provided acceptable relief. It is unclear whether the characteristic roaring tinnitus of Meniere's Disease is responsive to masking. The data may not be easy to get, for a person in the throes of a Meniere's episode is typically in great distress and, thus, is not an ideal subject for psychophysical experimentation.

3

Measurement Procedures

For both theoretical and practical reasons, it is important to obtain characterizations of tinnitus—spectral location, degree of spectral complexity, magnitude, etc.—that are as accurate as possible. Unfortunately, little basic research has gone into the important psychophysical questions of: (1) comparability of the various existing measurement procedures, (2) their test-retest reliabilities, (3) their relative efficiencies with different types of patients, etc. Such research may be unexciting, but it is necessary as a basis for establishing a standardized test procedure.

The reader should keep in mind throughout this section on tinnitus measurement that for many people tinnitus is not constant in character, either within a day or across days. This raises two problems: (1) the psychophysical procedures, which are already unusual and troublesome for many naive subjects, can be made additionally difficult and frustrating by short-term fluctuations in the tinnitus; and (2) these short-term and long-term fluctuations necessarily create an uncertainty as to whether what is measured on a given day is truly representative of the tinnitus experienced by the sufferer.

Basically, there are three questions one might ask about a tinnitus: What is its quality? What is its spectral location? What is its magnitude? We consider these questions in turn.

QUALITY OF THE TINNITUS

What is the perceived quality of the tinnitus? This question pertains to both its temporal and its spectral characteristics. Does it sound continuous, fluctuating,

interrupted, etc.? Is it spectrally simple—tonal or reasonably narrowband—or is it spectrally complex—wideband, multiple but relatively discrete frequency regions, etc.? From where does it seem to emanate--one ear, both ears, somewhere inside the head, etc.? It is intuitive that different tinnitus qualities would be associated with different sites of origin and, thus, that information about quality would be important in diagnosis. Unfortunately, tinnitus quality is rarely an accurate guide to site of origin. Some exceptions are the pulsatile tinnitus of objective vascular origin and the low-frequency roaring tinnitus of Meniere's Disease and otosclerosis.

Most of what we know about the quality of tinnitus comes from patients' self-reports, a procedure that is fraught with problems. For example, we cannot be sure that when two patients use the same word to describe their tinnitus—(say) "chirping" or "chugging"—they mean the same thing. Different people have had different acoustic experiences and, as a consequence, may use words differently. What a "high-pitched squeal" is to a piccolo player and to a nonmusician who has considerable high-frequency hearing loss obviously could be very different. Further, the words chosen by a tinnitus sufferer as best describing his or her experience may or may not touch upon all of the dimensions of interest to the scientist, and, unfortunately, the questioner may not always ask the follow-up question(s) necessary to discover the omitted information. Standardized procedures for gathering information on tinnitus quality would be welcome and valuable. With the various problems of self-report procedures in mind, let us consider some results.

Heller and Bergman (1953) supplied a list of 39 words used by 80 normal-hearing and 100 hearing-impaired subjects to describe their tinnitus. For those with normal hearing, "hum," "buzz," "ring," and "pulse" were used by 18 percent, 15 percent, 12 percent, and 8 percent, respectively. For the hearing-impaired, "ring," "buzz," "hum," and "whistle" were used by 30 percent, 11 percent, 10 percent, and 9 percent, respectively. Reed (1960) reported that about 70 percent of his 200 patients used "steam," "ring," or "buzz"; he also noted that the patients' descriptions were not predictive of site of lesion--an unfortunate but common finding.

Some attempts have been made to acquire more objective information about the quality of tinnitus. Goodhill (1952) supplied patients with recordings of 27 different

sounds created to mimic previous descriptions of tinnitus. Patients were asked to indicate which sound was the most like their tinnitus, but only a few cases were reported. Hazell (1979b, 1981c) has developed a procedure that has much appeal. He uses a commercially available music synthesizer to create imitations of the patient's tinnitus. The procedure is said to be time-consuming—sometimes taking as long as 2 hours—but rewarding to the patient and informative to the experimenter. The patient is pleased to finally have a nonverbal way of communicating his experience to family, friends, and the clinician, and the experimenter discovers the wide range and richness of tinnitus experiences. One finding particularly worthy of note here is that tinnitus described as tonal by many patients is much more complex than a tone--a common product was a single frequency embedded in a less-intense narrow band of noise. To date about 200 patients have been studied in this way. Only preliminary findings are yet available, but these indicate that about 83 percent of the synthesized waveforms are nonpulsatile, about 52 percent involve a narrowband noise, and about 39 percent a tone, singly or in combination. Correlations of tinnitus quality with presumed site of origin have yet to be reported.

Related to this question of quality is the matter of the characteristics of the tinnitus in the two ears. Logically, there are multiple possibilities. The tinnitus might be strictly monaural. It might be binaural and very similar in the two ears. It might be binaural and of similar spectral and temporal characteristics but not of equal magnitudes in the two ears (see "Is the Tinnitus Monaural or Binaural?" in this chapter). It might be binaural and of quite different spectral and/or temporal characteristics in the two ears—(say) tonal in one ear at one frequency and rather broadband in the other ear at a different frequency. This issue of possible binaural differences in tinnitus has not been extensively studied, but clearly it is important (and may explain some of the failures of (monaural) devices like tinnitus maskers/instruments to alleviate tinnitus). Until more is known, it should be clear that the pitch-matching and masking procedures described below should be done with headphones, not free-field presentations, so that there is at least the opportunity for the patient and the examiner to detect different origins for different aspects of the tinnitus.

SPECTRAL LOCATION OF THE TINNITUS

If the tinnitus is reasonably narrowband or tonal, where is it located spectrally? Two basic procedures—with numerous major and minor variations on each—have been used to obtain measures of the spectral location of a tinnitus.

Pitch Matching

As the name implies, in pitch-matching procedures the objective is to obtain from the subject a match in pitch between the tinnitus and a sound supplied by the experimenter. Fowler (1928) and Josephson (1931) were among the first to use pitch-matching procedures. In the most common procedure, the matching sound is tonal; it is presented to the ear contralateral to the one the patient believes contains the tinnitus (see "Is the Tinnitus Monaural or Binaural?" in this chapter); it is interrupted at a regular rate; and it is not adjusted by the patient, but by the experimenter on the basis of responses made by the patient.

While pitch matching is perhaps the most intuitive procedure for ascertaining the spectral locus of a tinnitus, it is, unfortunately, subject to major problems. For one thing, tinnitus is often not strictly tonal in quality (see "Quality of the Tinnitus" in this chapter), which makes matching to tonal stimuli both very difficult and of questionable value once accomplished. Further, in the best of circumstances, pitch matching is a difficult task even for highly practiced subjects, let alone for relatively inexperienced patients. In both populations, it is common to see so-called octave errors—settings for a match that are actually twice or one-half the frequency of the sound being matched (see Vernon, 1977). Pitch-matching procedures have their place in tinnitus diagnosis, but the results must be interpreted with special care.

The problem of octave errors is particularly bothersome when interpreting pitch-matching data obtained from tinnitus patients. One reason is that tinnitus patients are often given only the sounds available from a standard audiometer for use in matching their tinnitus. That is, frequencies above about 6–8 kHz are often not available to the patient. Among the problems raised by this equipment limitation is the possibility that the tinnitus

frequency is being greatly underestimated in many cases of high-frequency tinnitus. As noted above, about 64 percent of tinnitus sufferers match their tinnitus to frequencies in the range 3–8 kHz. But, since the frequencies available on audiometers are rarely higher than about 8 kHz, it would be impossible for a patient with tinnitus at (say) 12 kHz to reveal that fact; his "best match" might be a 6-kHz tone or noise band. The use of standard audio oscillators or of the recently introduced tinnitus-measuring devices (e.g., Voroba, 1979a) would reduce this problem of underestimating tinnitus frequency. Furthermore, all tinnitus examination protocols should include a procedure to verify that any pitch match produced by a patient is not in error by one or more octaves in one direction or the other (Vernon and Meikle, 1981).

Penner (personal communication) has recently completed an extensive study using a pitch-matching procedure. Three sensorineural subjects with a history of noise exposure made approximately 80 matches over the course of 4 weeks. The surprising finding was that for all subjects the frequencies chosen as matches to the tinnitus had a tremendous range across sessions—from 2 to 5 kHz for different subjects. A similar effect was briefly noted by Voroba (1979b). If this finding is confirmed, it will have great theoretical and practical importance. Nearly all tinnitus sufferers comment on the fluctuating nature of their perception, but it seems safe to assert that few investigators realized that it varied so much in frequency. If it does, it has obvious implications for the design of tinnitus maskers/instruments (see "Tinnitus Maskers/Instruments" in Chapter 4).

Masking

The second basic procedure for estimating the spectral locus of tinnitus, masking, has the appearance of being free from some of the problems of the pitch-matching procedure. In masking, a relativaly narrowband (octave or one-third octave) stimulus is swept across the spectrum in successive steps and at each is adjusted in intensity until the tinnitus is just masked. The spectral location at which the least masker intensity is needed is taken as the locus of the tinnitus. This procedure is attractive because it appears to be more accurate than, and to lack some of the problems of, the pitch-matching procedure—the task seems to require less sophistication and practice

than pitch matching, and octave errors are less likely to occur. One problem noted with pitch matching applies to masking as well: if the tinnitus lies beyond the range of the equipment, such as a standard audiometer, it may be maskable, since lower frequencies can mask higher ones, but inferences drawn about the spectral locus (and the magnitude) of the tinnitus will be in error.

There is another problem that is not so much procedural as it is evidence of the great variety of maladies that can underlie tinnitus: in some patients the tinnitus cannot be masked, and in some others it can be masked by nearly any weak tone or noise band. Feldmann (1971) reported that the tinnitus in about 11 percent of his 200 patients was completely refractory to masking; they tended to be the patients with the most severe sensorineural losses. At the other extreme, about 32 percent of Feldmann's patients reported masking of their tinnitus when any weak sound was presented; these tended to be patients having flat hearing losses due to Meniere's Disease, sudden deafness, or otosclerosis. One must assume that, for this latter group, tinnitus must not have been much of an everyday problem, since environmental noise would presumably be adequate to mask the tinnitus. Vernon et al. (1980) confirm Feldmann's observations that masking of tinnitus is different from masking of real sounds in a number of ways. This issue is addressed in the section "Some Ways Tinnitus Is Not Like an External Sound" in this chapter.

Related Masking Results

Another interesting finding by Feldmann (1971, 1981) deserves note and further study. In some patients with monaural tinnitus, he claims to have been able to mask it with tones and noise bands delivered to the opposite ear (also noted by Josephson, 1931). Feldmann argues that the obvious interpretation of cross-conduction can be excluded; in fact, the intensity necessary to mask the tinnitus was often less when presented to the contralateral ear than to the ipsilateral ear. Apparently he saw this contralateral masking effect most regularly with Meniere's and sudden deafness patients, but it was also present in presbycusic and noise trauma patients.

Note that a contralateral masking effect can be explained in a least two ways:

1. The contralateral masker in some way produces a reduction in the tinnitus signal at its source. By way of an example, the efferent mechanism might be activated, thereby causing a change in the hair cells or primary fibers in the contralateral (tinnitus-producing) ear. This alternative is meant to encompass Feldmann's own suggestion of a "neural mechanism of contralateral inhibition."
2. The contralateral masker accomplishes a "true" masking at some neural level where information from the two ears is combined (the tinnitus might be originating at this neural level or at a more peripheral level and "just passing through"). This alternative shares some features with so-called central masking (Zwislocki et al., 1968), but the latter is typically smaller in magnitude than is contralateral masking of tinnitus. Curiously, the possibility of contralateral control of tinnitus has not been extensively explored by the advocates of tinnitus maskers/instruments (see "Tinnitus Maskers/ Instruments" in Chapter 4; CIBA Foundation, 1981:174–175).

An important finding recently rediscovered by Feldmann (1971) is that for some patients the tinnitus does not return immediately upon termination of the masker. Rather, termination is followed by a silent period and then a period of gradually returning tinnitus. (This effect was previously noted by Spaulding [1903] and by Josephson [1931].) In an example shown by Feldmann, a 500-msec masker produced anywhere from about 1.0 to about 2.5 seconds of posttermination silence, depending upon the maskers intensity and the ear to which the masker was delivered. He indicates that in other patients the silent period was much longer than this, and he raises the prospect of trying to "train" the mechanism underlying the effect. This period of silence or diminished tinnitus magnitude has since been named residual inhibition (Vernon, 1977). It is a topic to which we return in "Residual Inhibition" in Chapter 4.

Formby and Gjerdingen (1980) studied masking in a single tinnitus sufferer using a procedure similar to that widely used in experiments on the so-called psychophysical tuning curve. The tinnitus was viewed as a signal, and each of a set of pure-tone maskers was adjusted in turn until it "just masked" the tinnitus. When the signal is an external tone, the pattern of masker intensities obtained with such a procedure has a characteristic shape (see Small, 1959); relatively little

intensity is needed when the masker frequency equals or is close to that of the signal, and increasingly greater masker intensity is necessary for masker frequencies increasingly distant from the signal, with the slope being steeper on the high side than on the low side of the signal. Formby and Gjerdingen obtained this same general pattern when the signal to be masked was a tinnitus. This masking procedure has the virtue of providing a relatively precise estimate of the spectral locus of the tinnitus—be it tonal or narrowband—but it has the drawback of being relatively time-consuming and thus may not see wide application clinically. Relevant to the common assertion that tinnitus magnitude fluctuates greatly is the observation by Formby and Gjerdingen that substantially different masker levels were necessary in different sessions to mask the tinnitus. The range was about 40 dB in the most extreme case, and 15–20 dB was not uncommon.

One finding by Formby and Gjerdingen (1980) deserves further study, for it has important theoretical and practical implications. They found that when the tonal maskers were binaural, they had to be between 8 and 15 dB more intense in order to mask the tinnitus than when they were monaural. Such an outcome is reminiscent of a phenomenon obtained with external sounds, known as the masking-level difference or MLD (reviewed by McFadden, 1975). If verified to exist in some forms of tinnitus, the MLD might eventually prove useful in diagnosing the site of origin of the tinnitus.

Penner (personal communication) used both pitch matching and tonal masking (psychophysical tuning curves) across a number of sessions with the same subjects and found that the intersession variability for pitch matching was much higher than that for masking. There are a number of possible explanations for this outcome. One is that pitch matching is a less reliable procedure than masking for determining the spectral locus of tinnitus. Another is that the procedure is accurate but that tinnitus can somehow fluctuate in pitch without a concomitant fluctuation in its maskability. This matter deserves further attention.

Another intriguing feature of the relationship between masking and tinnitus has been discovered by Penner et al. (1981). Twenty patients were studied, all having tonal tinnitus and all diagnosed as having sensorineural hearing loss as a consequence of noise trauma or exposure (recall that, by Feldmann's [1971] account, such patients constitute

about 34 percent of tinnitus sufferers). The task was to continuously adjust the intensity of a broadband noise so as to keep masked either the tinnitus, in some runs, or, in other runs, a real tone of about 10 dB sensation level (SL)--decibels above the patient's own absolute threshold at a given frequency. As expected, for the real tone, 90 percent of the subjects needed essentially the same intensity throughout the 5-minute test period; that is, the signal-to-noise ratio remained the same. When the task was to continuously mask the tinnitus, however, all of the subjects showed a need for increasing intensity during the first few minutes of listening. The changes were considerable, averaging about 30 dB across subjects. The typical trend was for a rapid change in the necessary intensity over the first 10–15 minutes, followed by a flattening of the function. Penner et al. (1981) explain this curious outcome by noting that throughout the presentation of the masking noise and real tone, the firing rates of primary auditory neurons (and thus, perhaps, the rates of neurons throughout the auditory system) would decline with time, but because both masker and signal were being about equally affected, the noise intensity for equal masking would stay about the same. That the intensity does not remain the same when masking the tinnitus implies either that external sounds and peripheral tinnitus do not affect primary fibers in the same way or that in these subjects the tinnitus is originating at a site beyond the (adapting) primary fibers being activated by the masker.

As Penner et al. (1981) note, their outcome has important implications for one of the most puzzling problems of tinnitus--why its annoyance seems so out of proportion to its loudness. It may be that, for some patients, real-world sounds "fade" under continuous presentation, but the tinnitus does not and thus continues to annoy even in relatively high-noise backgrounds. The Penner et al. finding may also prove to be of great practical significance. Clearly, those tinnitus sufferers for whom continuous maskers lose effectiveness through time should not be treated with continuous maskers, but with intermittent maskers so that masker level—and, thereby, the risk of additional hearing loss—can be minimized. (Parenthetically, Penner [personal communication) finds that only about one-third of her (highly selected) tinnitus patients experiences residual inhibition following presentation of her maskers.)

Penner (1980) has introduced a variant on the basic masking procedure that is attractive because it offers the twin prospects of being more accurate and more informative about the tinnitus. The experimenter begins with a broadband noise, adjusts it to just mask the tinnitus, and then begins to (say) low-pass the noise in successive steps until the patient again hears the tinnitus. This value is noted, the noise is again made broadband and is now high-passed in successive steps until the tinnitus is again heard. The resulting pair of cutoff frequencies—the "masking interval"—is taken as the bandwidth of the tinnitus.

As noted above, the possibility of binaural tinnitus that is not spectrally or qualitatively similar in the two ears points out the need for masking measurements to be made using headphones, with monaural presentations made first to one ear and then the other, while the contralateral ear receives a broadband masker of reasonable intensity. This may, for the first time, allow the patient to identify the respective origins of a complex, binaural tinnitus.

Finally, a comment is necessary about people suffering from tinnitus who hear about masking as a treatment and wish to learn whether their tinnitus is of the maskable type. People may have heard about the common use of interstation FM noise as a masking source for tinnitus and may attempt to use it in a self-conducted maskability test. If people choose a small, inexpensive pocket radio for this test, however, they run the risk of reaching the wrong conclusion about the maskability of their tinnitus, because the speaker system on such a radio will not ordinarily be able to transduce the high frequencies at high levels. Thus, there may be failure to mask a high-frequency tinnitus, not because it is inherently unmaskable, but because sufficient high-frequency energy is not present. Tinnitus sufferers wishing to use interstation FM noise to test themselves for masking effectiveness should be encouraged to use a speaker system of reasonably high fidelity. Either for the purposes of this test or for the purposes of relief, use of a standard home stereo system is to be further preferred over a cheap radio, because a stereo system will allow the sufferer to selectively emphasize the high-or low-frequency regions to some extent, and thus it offers the prospects of effective masking at lower overall sound levels.

MAGNITUDE OF THE TINNITUS

What is the magnitude of the tinnitus? The masking procedures described above for estimating the spectral locus of the tinnitus might also be thought of as measures of tinnitus magnitude—the greater the intensity necessary for masking (at the frequency requiring the least masking), the greater the tinnitus strength must have been. Without denying this view, it must be noted that measures of tinnitus magnitude based upon masking procedures should be regarded as indirect at best, for a number of relevant factors are necessarily uncontrolled across patients, and these make conclusions and comparisons difficult. For example, the intensity necessary for masking will be affected by the relative widths of the noise band used for masking and the patient's critical band at the tinnitus frequency. Also, using a masking procedure to estimate tinnitus magnitude is questionable in those patients for whom maskers at any frequency are equally effective, and, of course, it is impossible in patients whose tinnitus cannot be masked.

Many auditory scientists believe that a more direct measure of tinnitus magnitude is obtained from loudness-matching procedures (see Scharf, 1983) than from masking procedures. Loudness matching is similar in concept to pitch matching, and, as shall be seen, they have some mutual problems. Minton (1923) and Fowler (1928) were among the first to use loudness-matching procedures.

In its purest and simplest form, loudness matching would proceed as follows. The tinnitus would be strictly monaural and either tonal or relatively restricted spectrally. A sound that is the best match possible to the quality and pitch of the tinnitus would be periodically presented to the ear opposite the tinnitus, and the patient would adjust its intensity (directly or indirectly by responses to the tester) until it matched the loudness of the tinnitus. There are several obstacles to this ideal case:

1. Quality, pitch, and loudness judgments are clearly all mutually interdependent—the best pitch match cannot be achieved until the loudness is known, etc. The solution would appear to be to first get rough measures of all three characteristics and then to use this information when getting more precise measures (see below).
2. Tinnitus is often binaural and not interaurally identical in magnitude or spectral locus, so the choice

of ear to receive the matching stimulus could be complicated.

3. During the contralateral presentation of the matching stimulus, two types of cues are available to the patient—loudness and localization cues—and it is not possible to know which is being used. Since it is not possible to interrupt the continuous (say) monaural tinnitus when the matching sound is presented, the situation is analogous to the task known as the simultaneous binaural loudness balance, which has been controversial over the years just because of this uncertainty about the stimulus basis for the subjects' responses (see Elliott and Fraser, 1970; Scharf, 1983). No obvious solution exists for this problem, since heterofrequency loudness matches—monaural and binaural—are generally regarded to be more difficult and thus more variable than homofrequency loudness matches (e.g., Goodwin and Johnson, 1980b).
4. According to Feldmann (1971), the contralateral presentation of the matching stimulus may produce a partial masking of the tinnitus (see above), the obvious consequence being an underestimate of the tinnitus magnitude. The problematic solution—a monaural, heterofrequency match—was mentioned above.
5. Recruitment or marked interaural differences in audibility may exist at the tinnitus frequency, either of which is capable of producing an incorrect estimate of tinnitus magnitude. Comparisons of estimates obtained by matching and masking procedures might reveal errors of this sort.

A procedural point needs to be made here. The Oregon group (e.g., Vernon et al., 1980) argues that it is important for loudness matches to be made using only ascending intensity series. In these, the intensity of the sound being adjusted to match the loudness of the tinnitus is gradually increased starting from a just-detectable level. In standard psychophysical practice, ascending series are interleaved with descending series (where the intensity would initially be considerably greater than the tinnitus magnitude). The problem with descending series is that the high intensities could produce partial or complete residual inhibition (see "Residual Inhibition" in Chapter 4), and, thus, a large underestimate of tinnitus magnitude, or even a total inability to measure it. The point is well taken and should be considered when developing standard procedures for measuring tinnitus magnitude.

With these comments in mind, let us examine some of the available data on tinnitus magnitude. After finding the best-quality matches and pitch matches that he could, Reed (1960) used a binaural matching procedure to estimate the magnitude of tinnitus. He found that about 69 percent of his patients needed 10 dB SL (sensation level) or less to match the loudness of their tinnitus, and only 5 percent needed more than 30 dB SL. While this widely cited survey is informative, interpretation of the data is hampered by obstacle 5 noted above; namely, when there are interaural differences in audibility in the spectral region of the tinnitus and the match is reported in terms of SL in the contralateral ear, there is necessarily uncertainty about the actual intensity needed for the match. Graham's (1960) data are similar to Reed's—about 75 percent of the patients needed 10 dB SL or less, and only about 4 percent needed more than 20 dB SL. Vernon et al. (1980) have asserted that patients are "inordinately reliable" in making loudness matches to their tinnitus; they claimed that test-retest comparisons rarely reveal differences of more than about 1 dB (also see Goodwin and Johnson, 1980b). This claim is remarkable and worthy of additional study, for highly practiced normal listeners show much greater variability than this in alternate binaural loudness balance (ABLB) tasks (e.g., McFadden and Plattsmier, 1982b). It is difficult to attribute this high reliability to the presence of recruitment, for even if small increment thresholds did accompany recruitment (see McFadden and Plattsmier, 1982a), Vernon and his colleagues see patients having tinnitus of various etiologies, and not all of these patients have recruitment.

For the sake of completeness, it should be noted that psychophysical methods other than masking and loudness matching might be adapted for use in estimating tinnitus magnitude. For example, with cross-modality matching (Stevens, 1966; Scharf, 1983), the subject would adjust the intensity of a stimulus presented to another modality—a light or a vibrator, say—until its magnitude equaled that of the tinnitus. To our knowledge, this has never been attempted with tinnitus. In other tasks reliable data have been obtained with this procedure; however, those subjects were typically rather well educated and sophisticated, and it is unclear whether cross-modality data from average people suffering from tinnitus would accurately represent tinnitus magnitude. One virtue of the procedure is that data can be obtained rapidly, so a simple test of the usefulness of the method should be easy

to implement. As another example, Goodwin and Johnson (1980a) have suggested reaction time as a measure of tinnitus magnitude.

ANNOYANCE OF THE TINNITUS

As we have seen, typical measures of tinnitus magnitude indicate that it is rarely matched to sounds greater than about 30 dB SL, and it is often difficult for the nonsufferer to understand how such apparently weak sensations can cause such great annoyance and distress to some tinnitus sufferers. At the root of this misunderstanding are two errors—presuming that near-threshold intensities cannot be perceived as loud and equating loudness with annoyance. Let us first consider the latter issue.

Numerous everyday examples testify to the lack of a simple relationship between loudness and annoyance. A buzzing fluorescent light can be extraordinarily irritating even though its level is below 30 dB SPL, while an air conditioner or heater bringing relief from the weather goes unnoticed at 50–60 dB SPL. A passing motorcycle that masks a segment of the evening news broadcast is more annoying than is the kitchen appliance being used to prepare the evening meal, even though the latter masks the same news segment. A neighbor's stereo system is more annoying than one's own even when 40 dB less intense. The barely audible crinkling of a cellophane candy wrapper can be highly distracting and annoying even during the loudest segment of a concerto with full orchestra.

Beyond everyday examples, the noncorrespondence between physical and psychophysical measures of sound and its capacity to annoy has long plagued scientists interested in quantifying annoyance (see, for example, Fidell, 1978; Schultz, 1978). The so-called "message of the noise"—while frequently difficult to measure beforehand—is far more predictive of annoyance than are physical measures of the sound involved. For example, airline employees or military dependents living at the foot of a jet runway are far less likely to complain about the aircraft noise than are neighbors whose livelihoods derive from other activities. And the noise from delivery trucks is far more annoying to neighbors than it is to the recipient of the delivery. There is evidence that in some situations annoyance is nearly synonymous with speech interference (Fidell, 1978), but this finding probably has little relevance to most tinnitus cases, where speech intelligibility is essentially unaffected by the tinnitus. Interference

with sleep is a common source of annoyance, but, somewhat surprisingly, tinnitus produces sleep difficulties for only about one-half of tinnitus sufferers (CIBA Foundation, 1981:27).

Various writers have revealed a misunderstanding about the loudness/annoyance relationship when discussing tinnitus. Fowler (1942, 1943) was among the first to note the poor correlation between annoyance and psychophysical measures of tinnitus magnitude. He talked of the "illusion of loudness" of tinnitus and claimed that through conscientious effort the clinician could eventually diminish or abolish the illusion. One suggested step was to have the patient perform a loudness match—which would invariably be achieved with a relatively weak sound—and then to present this sound while emphasizing to the patient that it obviously did "not correspond to his statements as to the severity of the symptom" (p. 397). The clear implication is that Fowler did not accept the possibility that a tinnitus could behave differently from a weak external sound in its ability to produce annoyance, an attitude that is not uncommon today.

More recent discussions of the discrepancy between the apparent magnitude and the annoyance of tinnitus have tried to emphasize the possibility of a basis for the effect other than a "psychological" one. The reader should recall here that tinnitus magnitude is typically reported in units of SL—decibels above the subject's own absolute threshold at that frequency. So, if hearing loss is substantial, a small value of SL will correspond to a large SPL. Appreciating this fact, Vernon (1976) suggested that the small tinnitus magnitudes reported might somehow be under the influence of a mechanism like the one functioning in certain pathological conditions to produce recruitment of loudness. Recruitment is defined as an abnormally rapid rate of growth of loudness; its effect is to render sounds well above (a pathology-elevated) threshold to be essentially normal in loudness even though weak sounds only 20–30 dB above threshold are depressed in loudness. Vernon (1976) suggested a "super-recruitment" might be operating on some forms of tinnitus to make them more loud, and thus more annoying, than they might seem. This explanation is unlikely to be universally applicable, for recruitment is present in only some of the pathological conditions that are routinely accompanied by annoying tinnitus, but the proposal is worthy of study.

Goodwin and Johnson (1980b) attempted a test of the Vernon proposal using a small sample of tinnitus sufferers

of different types. They compared loudness matches obtained with two procedures: a standard alternate binaural loudness balance (ABLB) and a monaural method that used a matching tone whose threshold was in the normal range (and thus was presumably free from recruitment even if the tinnitus frequency was not). The result was that the estimated magnitudes of tinnitus were, in every case, greater with the monaural than with the binaural procedure, and the conclusion was that tinnitus magnitude is frequently underestimated due to the contribution of recruitment. (While this may be a correct conclusion, it is also possible that for some subjects the results are complicated by the fact that, in order to find a matching frequency having a normal threshold, frequencies below about 1000 Hz had to be used, and these frequencies are known to have steeper loudness functions than tones between 1000 and 4000 Hz (Scharf, 1978]—that is, they have a "normal recruitment.") The Goodwin and Johnson results imply that the apparent discrepancy between the magnitude and the annoyance of tinnitus may be due in part to underestimation of the magnitude. Tyler and Conrad-Armes (no date) arrived at a similar conclusion using similar procedures.

Penner et al. (1981) have suggested that the high annoyance of tinnitus may in some cases be due to its failure to behave like an external sound once it gets into the central nervous system. In particular, Penner et al. point to their demonstration that a constant external sound can gradually lose its masking power over a tinnitus through the course of about 30 minutes of continuous listening, while it does not over a second external sound. Whatever the eventual neurophysiological explanation of this effect it is clear that the tinnitus is behaving aberrantly.

Expanding on the comment of Penner et al., it may be that this and other aberrant behaviors "bring the tinnitus to the attention" of certain neural mechanisms that persist in unsuccessful attempts to force the tinnitus to conform to the behavior of external sounds and that their ongoing failure reaches consciousness as annoyance. It is difficult to make this idea any more concrete at this time, but an analogy to sound localization comes to mind. When dichotic sounds are presented to a person's ears over headphones instead of in the normal, free-field manner, the sounds are not perceived as originating from external sources, but are perceived as having an intracranial origin. Various explanations of this phenomenon exist, but a long-standing one points to the fact that with headphones

the waveforms are not altered as they ought to be by normal head movements. The "expectations" of certain neural networks are not met following certain motor actions, and this gives rise to a unique perceptual experience—not annoyance, to be sure, but the analogy nevertheless appears relevant.

Finally, we return to "psychological" explanations of the apparently disproportionate annoyance of some tinnitus. Glass and Singer (1972) have studied the stress-inducing properties of noise exposure and have found evidence of much greater cognitive, emotional, and physiological effects when the schedule of noise presentations was perceived as being beyond the control of the subject. Perception of control greatly reduced the aversiveness and the aftereffects of the situation. This outcome is reminiscent of the comments made by some successful users of tinnitus maskers/instruments when attempting to explain their apparently paradoxical preference for one continuous sound (the masker) over another (the tinnitus). These patients often comment on the control they have over the masker. They can turn it on and off, change its intensity, etc., while their tinnitus is nearly totally out of their control—whatever changes it undergoes bear no immediately apparent relation to anything the patient has done. It is believable that the feeling of helplessness induced by this lack of control over their tinnitus is an important contributor to the annoyance reported by many tinnitus sufferers.

In summary, when attempting to comprehend the annoyance factor of tinnitus, it is necessary to consider several facts. First of all, since the loudness of real-world sounds is not a good predictor of their annoyance, it is not reasonable to expect that it would be for tinnitus either. Second, the low sensation level of most tinnitus does not mean it is not unpleasantly loud. Finally, tinnitus does not behave like external sounds in a number of ways, and this aberrant behavior may have various neuro-physiological and psychological consequences capable of producing high annoyance, directly or indirectly.

IS THE TINNITUS MONAURAL OR BINAURAL?

As noted above, about 37 percent of all tinnitus is believed to be monaural—a statistic based primarily upon patients' self-reports (Vernon, 1978a). It is possible that such reliance on self-reports is producing an underestimate of the incidence of binaural tinnitus, which may

in turn be leading to depressed success rates with treatments such as tinnitus maskers. The possibility of this error lies in the perceptual experience aroused by binaural stimulation. When external sounds that are similar spectrally and equal in intensity are presented to the two ears, the phenomenological experience is of a single fused image located in the center of the head. (In everyday listening, of course, sounds correctly appear to originate outside the head from locations in auditory space, but, with headphone listening and with self-generated sounds such as tinnitus, the sounds appear to originate from a location somewhere within the head; this difference between localization and lateralization, respectively, has never been fully explained—but see Schroeder [1975].) If an interaural difference in intensity is now introduced, the fused image appears to move in the head toward the ear receiving the more intense sound. The larger the interaural intensity difference, the more lateralized the acoustic image appears to be. Once the interaural difference gets to be about 15–20 dB, most listeners report the image to be so strongly lateralized toward the more intense ear that it is unclear whether the stimulus is binaural or monaural, and sometimes the presentation of a truly monaural stimulus is required to convince the listener that the previous stimulus was binaural but highly asymmetric.

The relevance of these facts to tinnitus should be clear. There is the danger that when a patient indicates that his tinnitus is present in one ear only, he may be in error: it may be originating at both ears, but more intensely at one of them, thereby producing a strongly lateralized image that misleads the patient into believing the problem is monaural. Note that the tinnitus need not be spectrally identical in the two ears for this to be a possible problem; real sounds that are several hundred Hertz apart can be fused and lateralized, and, in fact, the frequency limits for dichotic fusion grow with center frequency (Scharf, 1969, 1974; however, these experiments used brief stimuli and thus may not be relevant to the binaural tinnitus situation).

If the spectral regions of tinnitus origin are widely different in the two ears, or if the quality or complexity of the tinnitus is different in the two ears, the monaural/binaural question will be less of a worry than in patients whose configuration of hearing loss is similar in the two ears and whose tinnitus might thereby be expected to be spectrally similar in the two ears. There is also less concern when the matched intensity of the

tinnitus is about 10 dB SL or less (and the audibility is approximately equal in that frequency region in the two ears), for then the maximum possible interaural intensity difference would not be adequate to produce an acoustic image strongly lateralized to one side.

For completeness, it should be noted here that an effect not known for external sounds does occur with tinnitus. Some patients report their tinnitus to be similar or identical in the two ears but not fused—there is a (similar) image localized to each ear (Vernon, 1978a).

In the laboratory a simple test is available to a listener trying to decide whether an input is monaural or binaural—pulling the plug to one of the two earphones and observing any change in locus of the acoustic image—and while this option is obviously not available to a tinnitus sufferer, fortunately the means are available to test psychophysically for the possible binaural origin of tinnitus that is perceived to be monaural. Once a subject has matched his or her tinnitus for center frequency, complexity, and intensity, a similar waveform—but located in a spectral region adjacent to the tinnitus—could be presented binaurally and the subject asked to adjust the intensity in the ear opposite the perceived tinnitus until the image of this external sound appeared to originate from the same intracranial location as the tinnitus. If the intensity difference required for this match is small, it would be evidence for a binaural origin of the tinnitus; if it is relatively large, or if the patient is satisfied with a match to a monaural stimulus, it would argue for a monaural origin.

It is possible that some of the patients who have found tinnitus maskers to be of little long-term value might have been better satisfied had they been fitted binaurally. Similarly, at least some of the reports of tinnitus persisting after sectioning of the auditory nerve may be traceable to unrecognized binaural origin of the tinnitus prior to surgery.

Finally, there is a misunderstanding about the localization (lateralization is the correct term) of the tinnitus that must be corrected. Some authorities seem to believe that the locus of the source of a tinnitus is revealed by where the patient hears it. Reed (1960) and Goodhill (1979), for example, seem to believe that a tinnitus that sounds as if it originates at the ear(s) does so and that one that appears to be inside the head has a central origin. This simply does not follow.

THE ISSUE OF BEATS WITH TINNITUS

If some forms of tonal tinnitus were identical to an external tone in all ways but their origins, one might expect to be able to produce the experience of beats by introducing an external tone slightly different in frequency and similar in intensity to the tinnitus. In fact, reports of such beats are few. Wegel (1931) claimed to be able to hear an irregular, "mushy" beat when some single tones were introduced, but it is clear that Wegel appreciated how different his experience was from the normal beat heard when two external tones interact. For example, he found combinations of frequency and intensity of the external tone that should have produced beats but that instead produced "complete silence." Over the years, Wegel's report has been widely cited, but it has generally been viewed with skepticism and dismissed; however, recent developments have brought new attention and credibility to it.

Specifically, the spontaneous otoacoustic emissions (OAEs) discovered by Kemp (1979b) and studied by Wilson (1979, 1980) and Zurek (1981) have several features in common with Wegel's report, as well as with the reports on monaural diplacusis by Flottorp (1953) and Ward (1955). For example, in some subjects a heard QAE has been suppressed by an external tone of appropriate frequency and intensity; this is reminiscent of Wegel's "complete silence." OAEs have been observed, both perceptually and acoustically, to fluctuate in level (beat) when appropriate tones are introduced (Wilson, 1980; Zurek, 1981), and these fluctuations are often irregular, just like the "mushy" beats reported by Wegel (1931), Flottorp (1953), and Ward (1955). The fact that Flottorp and Ward did not have a tinnitus against which to beat, and Wegel did, appears to be a much-less-important difference since the discovery of heard and unheard OAEs. It is now possible to imagine that, in some of those rare instances in which beats against tinnitus were reported (Wegel, 1931; Wever, 1949; Vernon et al., 1980), what was being heard were interactions between the external tone and a heard OAE and that the beatlike effects observed by Flottorp and Ward were interactions with an unheard (perhaps narrow-band) OAE. (In this regard, it is sobering to see how close Flottorp was to discovering OAEs and how modern both his and Ward's discussions are.)

Some other reports of beats against tinnitus are apparently not explainable by appeal to OAEs. Stanaway

et al. (1970) reported on a single subject who could hear beats between an external tone and a tinnitus induced by exposure to an intense broadband noise, but no such interactions were reported by Loeb and Smith (1967) or Atherly et al. (1968). Lackner (1976) claims to have produced beats in patients whose tinnitus was of central rather than peripheral origin, but his arguments make no sense. For one thing, his procedure for producing beats was to introduce an external tone to the ear contralateral to the one in which the tinnitus was localized. Two external tones to different ears can produce beats—hey are called binaural beats—but such beats are different from monaural beats in many ways. Of primary interest here is the fact that binaural beats cannot be produced above about 800–1000 Hz using a single pure-tone to each ear (Licklider et al., 1950; Perrott and Nelson, 1969; but compare McFadden and Pasanen, 1975). Yet Lackner reports binaural beats at frequencies from about 2500 to 5000 Hz. It is conceivable that his patients were experiencing some other form of binaural interaction with their tinnitus—contralateral masking, for example, or perhaps the tinnitus had components in both ears and only appeared to be monaural (see "Is the Tinnitus Monaural or Binaural?" above), and the beat heard was a monaural one—but whatever the explanation, it is certain that these patients were not hearing what is ordinarily called a binaural beat with such high-frequency tones.

The weight of the evidence, then, favors the conclusion that tone like tinnitus does not ordinarily behave like an external tone when it comes to the issue of beats—monaural or binaural. There is a possibility that some of the reports of beats against tinnitus could be traced to an OAE underlying the tinnitus. The rarity of the reports of beats against tinnitus is in accord with this interpretation, for the growing belief is that relatively few cases of tinnitus have an OAE as their basis.

SOME WAYS TINNITUS IS NOT LIKE AN EXTERNAL SOUND

Some of the facts of tinnitus masking discussed in this report naturally raise the question of whether tinnitus masking should really be regarded as masking or whether it involves mechanisms fundamentally different from those involved when one external sound obscures another. An easy decision on this question is prevented by our ignorance

of the bases of the various forms of tinnitus. That is, in some instances the tinnitus and the masker may interact physiologically in much the same way as the physiological concomitants of two external sounds interact when masking occurs, whereas in other instances tinnitus "masking" may be accomplished via quite different physiological interactions. At this point all that can be done is to review and emphasize those features of successful tinnitus "masking" that have led some to question whether it is better called by another name in order to promote its better understanding (e.g., Vernon, 1981). In what follows it is important for the reader to remember that not all of these features of tinnitus masking are present in all patients, and exactly which features cluster as sets is not yet known. Further, some of these features may be refuted or modified by future research.

Tinnitus masking has shown itself to be unlike ordinary masking in several ways:

1. Sometimes tones of almost any frequency can "mask" tinnitus (Feldmann, 1971, 1981). For external sounds, masking can only be accomplished by sounds within certain spectral regions surrounding the signal; those regions do increase with increasing masker intensity, but they never reach the point of being several octaves above or below the signal, as has sometimes been reported for tinnitus "masking" (Vernon and Meikle, 1981).
2. When tinnitus can be masked, the intensity necessary for masking is often abnormally great or small relative to the signal-to-noise ratios required by external signals and maskers.
3. In order to keep some forms of tinnitus masked, it is necessary to gradually increase the intensity of the masker over the course of a half-hour listening session; this increase can amount to 30–45 dB (Penner et al., 1981). An external sound requires no such increase in the masker to remain at the same level of detectability.
4. It is claimed that sometimes tinnitus cannot be masked no matter how intense the masker is made (Vernon and Meikle, 1981). For external sounds this is never strictly true, although signals intense enough to be near the upper intensity limit of audibility—the so-called threshold of pain—may require maskers that are themselves dangerous to hearing.
5. In a reasonably large fraction of tinnitus cases, the tinnitus can be masked with sounds presented to the ear contralateral to the side of reported origin of the

tinnitus (Feldmann, 1971, 1981). In itself this is not unlike the behavior of external sounds—an external sound can mask a contralateral external signal via either cross-conduction or central masking. The peculiarity is that the intensity necessary for masking tinnitus often appears to be the same ipsilaterally and contralaterally.

6. Following the termination of an effective masker, ipsilateral or contralateral, there is sometimes a period of residual inhibition during which the tinnitus is absent or reduced in magnitude. In contrast, a masked external signal returns to audibility essentially immediately upon termination of the masker.
7. If spectrally similar sounds are presented to the two ears over headphones, they are perceived as a single fused sound having an apparent intracranial position that depends upon the relative levels and timing of the waveforms at the two ears. In contrast, tinnitus that is spectrally similar in the two ears will often not fuse, but will remain localized at the two ears (Vernon, 1978a).

SUMMARY OF MEASUREMENT PROCEDURES

This section has been concerned with psychophysical procedures and techniques for measuring and describing tinnitus. For the foreseeable future, such procedures will continue to be the primary source of clinical and experimental information about tinnitus, but it is believable that the long-term future will see the development and wide application of new and modified objective measures of tinnitus (or its correlates) useful in diagnosis and in choice of treatment. The recording of spontaneous otoacoustic emissions (OAEs) from the canal of the outer ear (Kemp, 1979b; Wilson and Sutton, 1981; Zurek, 1981) has already been discussed (see "The Objective/Subjective Issue" in Chapter 2), but other possibilities exist. For example, Shulman and Seitz (1981) have claimed that the brain-stem-evoked response (BSER) in patients having tinnitus of central origin is different in specifiable ways from that of people with normal hearing. Some other possibilities are that the response of tinnitus to manipulations of air pressure in the middle ear (see "Alteration in Air Pressure" in Chapter 4) may prove to have diagnostic value, as may its response to electrical stimulation (see" Electrical Stimulation" in Chapter 4) or to certain drugs (see "Drug Therapy for Tinnitus" in Chapter 4). The development and use of such diagnostic aids is clearly to be encouraged.

4

Treatments

Over the years the treatments attempted for tinnitus have covered the range from vitamins and drugs to hypnosis and masking, but far and away the most common treatment—and in terms of absolute numbers helped, probably the most effective one—has been simple reassurance from a hearing professional that mild tinnitus is not a rare disorder, not necessarily a harbinger of imminent deafness, not a symptom of serious brain disorder, etc. One specialist estimates that explaining the problem is a sufficient solution for 95 percent of the tinnitus sufferers he sees (CIBA Foundation, 1981:273), and this statistic presumably ignores those patients satisfied by the explanations of general practitioners or others prior to referral to specialists.

When they are being precise, medical specialists often reserve the use of the word treatment for actions taken to actually cure a disorder or disease, as opposed to actions taken only to relieve the pain or distress of the disorder or disease. The latter is technically termed palliation or palliative treatment. As will be seen, in these terms essentially every procedure described below should be viewed as (at most) a palliative, even though the word treatment may be used in discussing it.

PSYCHOLOGICAL INTERVENTION

As is true for many other serious ailments, tinnitus has the potential to create or to intensify psychological problems. Indeed, it might be argued that, in the past at least, tinnitus was more likely than many other maladies to have psychological concomitants because of the unresponsive and apparently uncaring reaction of medical

professionals to the problem. A patient diagnosed as having cancer, for example, may have a strong psychological response to the news, but typically he soon begins to receive medical treatments of various sorts for the problem. By contrast, all too often the tinnitus sufferer was offered no treatment of substance and no hope of one in the future. It is fully believable that after one or two experiences of this sort, the frustrated tinnitus sufferer would begin to evidence behaviors in accord with psychological interpretations of the origin of the tinnitus. That is, it is imperative to realize that the plight of tinnitus sufferers has, until very recently, been largely ignored, and, as a consequence, the discovery of variations from "normal" in their psychological profiles cannot be unequivocally identified as cause or effect of their tinnitus problem.

Considerable evidence and simple logic indicate that tinnitus is a symptom of various physiological anomalies of the auditory and other systems and that these conditions probably strike people of all personality types indiscriminately. This is not to say that, once present, a tinnitus will not be better handled by one person than another, nor that transient psychological difficulties cannot cause people to focus on or exaggerate a preexisting or new physiological malady; the point is that psychological makeup is probably a minor contributing factor to the underlying anomaly. Ambrosino (1981) agrees that there is no one personality type more likely to contract severe tinnitus, but that once contracted, tinnitus can affect personality. House (1981) studied the personalities of 150 seriously afflicted tinnitus patients, and Hazell (1981a) administered personality inventories to his 200 patients, but no attempts were made to partition cause from effect.

The possibility of interplay between psychological and physiological factors is obvious when stress is considered. (Many tinnitus sufferers report exacerbation of their problem during times of stress.) It is known that psychological stress in a person's private or professional life can produce concomitant physiological changes. If some of these changes are responsible for a new or an enhanced tinnitus, it is understandable that a person might come to focus on this obvious symptom as the source of all his or her problems. Untangling this web might be complicated indeed, and both too time-consuming for and beyond the training of the typical hearing professional. The temptation is often great to refer such cases to psychotherapists;

in the past this course has often been pursued too hastily—and ineffectively—since many patients balk at such treatment for what they strongly regard to be a physiological problem. A treatment procedure aimed at general relaxation—for example, biofeedback or just instructions to simply "slow down"—has appeal in such cases. Anecdotes abound about tinnitus symptoms diminishing or disappearing upon the natural disappearance of stress-inducing factors in a patient's life.

SURGERY FOR TINNITUS

Tinnitus is known to be a frequent, early symptom of tumors of the internal auditory meatus and the cerebellopontine angle and of a glomus jugulare. It also can herald the onset of otosclerosis or indicate the presence of vascular anomalies of various sorts. Clearly, several of these conditions require surgical attention, and in the process, the tinnitus might be alleviated. But such surgery should be distinguished from that performed solely as a treatment for a severe tinnitus.

Over the years, various surgical techniques have been attempted as a last resort to alleviate debilitating tinnitus. These include tonsillectomy, sectioning of the vestibulo-facial anastomoses (Fisch, 1970), excision of the tympanic plexus (tympanosympathectomy; Lempert, 1946), excision of the main trunk of the vestibular nerve (Fisch, 1970), sectioning of the cochlear nerve (Fisch, 1970), and dorsal sympathectomy (Johnson, 1954), to name a few. Results have been unpredictable at best, and success rates have been low.

House and Brackmann (1981) reviewed reports of the effects on tinnitus of various surgical procedures performed for tinnitus relief, as well as for reasons other than tinnitus relief. Following successful removal of an acoustic neuroma, about 40 percent of the patients felt that the tinnitus was better and about 50 percent felt it was worse. Following successful stapedectomy, 74 percent reported the tinnitus to be absent or reduced. Following translabyrinthine section of the eighth nerve, about 45 percent of the patients felt that the tinnitus was absent or improved.

As long ago as 1928, Jones and Knudsen were advising against sectioning of the eighth nerve and destruction of the cochlea as treatments for intractable tinnitus—not because of a concern for the loss of useful hearing, but

because of the general ineffectiveness of these procedures. Sectioning of the eighth nerve and labyrinthectomy are described as partly or wholly effective against tinnitus in about 50 percent of the cases (Hazell, 1979c), but it must be recalled that these percentages are presumably based upon small numbers of seriously debilitated patients. Thus, failure must be particularly disappointing to the patients. We have no way of estimating how many operations of these types are still performed, but they continue to appear to be ill advised solely as treatments for tinnitus. As Hazell (CIBA Foundation, 1981:213) has pointed out, cutting the eighth nerve may obviate the later use of other, possibly more effective treatments yet to be developed. (Surgical procedures advocated for Meniere's Disease and its attendant tinnitus were discussed in the section "Meniere's Disease" in Chapter 2.)

EXPOSURE TO INTENSE SOUND

From personal experience, it appears that any brainstorming session on tinnitus that includes people of varying levels of knowledge about the topic is likely to eventually produce a question of the following sort: Might it be possible to eliminate a narrowband tinnitus by selectively destroying the spectral region giving rise to it? The question often takes the more explicit form: Might exposure to a narrowband or tonal stimulus of high intensity be capable of "burning out" a tinnitus-producing spectral region? (The idea also occurred to Jones and Knudsen [1928].) It would be difficult indeed to enumerate the implicit assumptions underlying such a suggested treatment for tinnitus, and the likelihood is that, once enumerated, the prospects for success would remain sufficiently dubious that one would be reluctant to attempt the procedure even on well-informed volunteers. Fortunately, this difficult ethical predicament need not be faced by anyone, for a bold experimenter has already attempted the procedure on his own tinnitus.

I. M. Young had a unilateral, high-frequency tinnitus that was highly annoying; over a period of time, he administered various high-intensity sounds to himself and then carefully documented the aftereffects of these exposures (Young and Lowry, 1981, 1982). The upshot is that, even after heroic attempts, he has not been successful in eliminating his tinnitus, although he has produced some interesting effects that are difficult to explain. For

example, following one series of monaural exposures, the previously monaural tinnitus became permanently binaural. Then, following a monaural exposure of 121 dB SPL at 500 Hz for 21 minutes, the tinnitus frequency was greatly reduced in both ears and it remained so long after hearing had returned to normal. Over the course of weeks, it gradually returned to its original frequency, but with different recovery rates in the two ears.

From this one well-studied subject, it appears that tinnitus will not succumb to high-intensity sounds, but logically some form(s) of tinnitus of different etiology might be reduced by this procedure. Since exposure to intense sounds is known to produce both temporary and permanant tinnitus (as discussed in "Possible Experimental Models of Tinnitus" in Chapter 2), future investigators are encouraged to proceed cautiously even if using themselves as subjects.

DRUGS AND TINNITUS

Some drugs can cause or exacerbate tinnitus—and thus their termination can alleviate tinnitus. Other drugs can reduce or eliminate tinnitus—and thus are administered to alleviate tinnitus. Since termination and administration of drugs can both be considered forms of treatment, we discuss the two types of drugs here, in successive sections.

Drugs Causing Tinnitus

It is not uncommon for patients with various maladies to report tinnitus for the first time soon after beginning a new drug regimen. For any particular drug, the fraction of patients reporting new or enhanced tinnitus is typically small, but this should not lead us to disbelieve the reality of the reports. Given the large number of ways and locations in which tinnitus can arise, and given the large individual differences in reaction to drugs at the same dosage level, great heterogeneity of response ought to be the rule. Some of the drugs noted for their ability to induce or enhance tinnitus have been noted by Brown et al. (1981) and Goodey (1981). On the other hand, lists such as these and the one prepared by Drucker (1979) must necessarily exaggerate the number of drugs that actually produce tinnitus in a significant fraction of

their users. It must be remembered that tinnitus is a very common experience and thus might begin "spontaneously" while someone is taking a drug that should not itself be held responsible for the tinnitus. Considerable work will have to be done before we truly know which drugs possess the ability to induce tinnitus in normal and abnormal auditory systems.

Salicylates

Aspirin, of course, is the most commonly used drug known for its effects on hearing and tinnitus. After just 48 hours on a dosage of about 4.8 g/day, there is 10–15 dB of hearing loss (McCabe and Dey, 1965), and this can grow with continued use to as much as 40–50 dB (Myers and Bernstein, 1965). Typically, the hearing loss is essentially flat across frequency, but in some reports the high frequencies are more affected than the low (McCabe and Dey, 1965; McFadden and Plattsmier, 1982c). Upon termination of the drug, the hearing loss invariably recovers within 24–72 hours, depending upon the serum salicylate level achieved (Myers and Bernstein, 1965; McFadden and Plattsmier, 1982c).

At present there is contradictory evidence as to the site of action of salicylate in the peripheral auditory system. Gold and Wilpizeski (1966) observed diminutions in the whole-nerve response with weak acoustic stimuli but not with intense stimuli (unfortunately, they did not simultaneously record the cochlear microphonic). Silverstein et al. (1967) reported a strong depression in both the cochlear microphonic (CM) and the whole-nerve action potential (N_1) following intraperitoneal injections of sodium salicylate. A possible explanation of these two reports is that the salicylate affects the hair cells and that this effect is then reflected in the response of the eight-nerve fibers. However, Wilpizeski and Tanaka (1967) and Mitchell et al. (1973) found no change in CM response, only a diminution in N_1 following salicylate injection. Knowing whether or not the CM response is affected by salicylates is basic to insights about site(s) and mode(s) of action of this drug. Resolution of this discrepancy and integration of the outcome with the established effects of salicylates on the stria vascularis (see Woodford et al., 1978, for a review) would be welcome and valuable.

Tinnitus is commonly reported as a concomitant of heavy aspirin usage. Indeed, its presence is routinely used as a gauge of whether the salicylate level in the

blood has reached the value believed to be necessary for effective anti-inflammatory action. Thus, dosage is often increased until tinnitus is produced and then decreased until the tinnitus just disappears. (Mongan et al. [1973] warn that this procedure is very unreliable as a predictor of serum concentrations of salicylate, particularly in patients with preexisting, high-frequency hearing loss; many of these people never experience tinnitus, even at very high serum concentration levels.)

Aspirin-induced tinnitus is typically characterized as tonal and of high frequency, but no systematic investigations of its quality, pitch, and loudness as a function of blood salicylate level have been done. McCabe and Dey (1965) apparently did some informal pitch matching, which indicated tinnitus frequencies from 7 to 9 kHz. It has been reported that subjectively the tinnitus onset appears to precede the hearing-loss onset (Mongan et al., 1973), and there is an implication that it also recedes more rapidly than the hearing loss upon termination of the drug (in accord with the personal experience of the author, but see CIBA Foundation, 1981:130–131), but these timing relations are not well documented. Future experiments aimed at this timing issue should incorporate measures of relative hearing sensitivity at frequencies above the normal audiometric range.

No mention could be found in the literature of two features of aspirin-induced tinnitus that the author has personally observed. Attempts to match the pitch of an aspirin-induced tinnitus were frustrated by residual inhibition; a brief, relatively weak matching tone presented to either ear had the ability to eliminate the tinnitus for periods of 30–60 seconds as soon as its frequency approximated that of the tinnitus. Second, clenching of the jaw muscles would produce transient increases in the magnitude of the tinnitus (also see CIBA Foundation, 1981:202; Shulman, 1981a:184). Both of these effects were observed in three listeners taking approximately 5 g of aspirin per day. Unfortunately, the two effects do little to reduce the number of possible explanations of the origin of the aspirin-induced tinnitus. An intriguing but unlikely possibility is that this tinnitus might have as its basis a spontaneous otoacoustic emission (OAE; see "The Objective/Subjective Issue" in Chapter 2).

Great individual differences in serum salicylate levels and hearing loss have been reported for the same weight-corrected dose levels (Myers and Bernstein, 1965;

Mongan et al., 1973). Of both theoretical and practical importance would be information on the relationship between susceptibility to aspirin-induced hearing loss and tinnitus and susceptibility to temporary and permanent threshold shift. If eye color and melanin content (see "Lidocaine" in this chapter) are highly predictive of both, an important insight will be gained.

Finally, in regard to temporary threshold shift, Hamernik and Henderson (1976) have reported negative results from animal (chinchilla) experiments aimed at detecting potentiation of noise exposure by administration of sodium salicylate, but McCabe and Dey (1965) reported greater losses in absolute sensitivity following noise exposure when their (human) listeners were on a therapeutic dose of aspirin than when not (in accord with McFadden and Plattsmier, 1982c). This issue should be resolved, given the great quantities of aspirin consumed daily by workers in noisy environments.

Quinine

Quinine and other antimalarial drugs (e.g., quinidine, chloroquine, and hydroxychloroquine) have long been known for their ability to produce temporary hearing loss and tinnitus (e.g., Brummett, 1980; Brown et al., 1981). With the decline of malaria in the United States, however, these drugs have become infrequent sources of tinnitus in this country, and as a consequence little has been written about this form of tinnitus. Quinine-induced hearing loss and tinnitus are apparently of high frequency, and the tinnitus onset reportedly precedes the hearing loss. While the hearing loss is reported to be reversible in most cases, permanent loss has occurred—apparently following large doses or protracted administration. There appears to be a general belief that the mechanism underlying the hearing loss and the tinnitus is vasoconstriction, although the evidence is meager. Some reports indicate that in sensitive individuals the quinine contained in a single gin-and-tonic highball can be adequate to produce tinnitus.

The occurrence of tinnitus following administration of quinine sulfate is mentioned by Markham et al. (1967) and Segal et al. (1974), and the danger to hearing of chloroquine phosphate is highlighted by the report of Dwivedi and Mehra (1978). Chloroquine was shown to have an affinity for melanin by Dencker et al. (1973), which makes that compound, at least, different from salicylate.

Tobacco

Fowler (1942) asserted that smoking is a common cause of tinnitus and that at least a month's cessation is necessary to eliminate it as a causative factor. Whether or not Fowler was correct in this belief has yet to be satisfactorily established. Tyler (CIBA Foundation, 1981:235) indicates that new information on tobacco is forthcoming.

Caffeine

This agent is frequently mentioned for its ability to produce or exacerbate tinnitus (e.g., CIBA Foundation, 1981:235, 263), but no systematic studies of it were found.

Alcohol

Alcohol has the curious characteristic of being cited as both a cause and a treatment for tinnitus (see "Alcohol" in this chapter). Unfortunately, anecdotes are the primary source of this information at this time (e.g., CIBA Foundation, 1981:201,268).

Cocaine

Tinnitus is sometimes mentioned as a concomitant to cocaine use, and its vasoconstrictive actions make this claim believable. However, no information was found on the dose levels needed, the time course of onset and decline, etc.

Marijuana

It has been asserted that marijuana can markedly increase a preexisting tinnitus (CIBA Foundation, 1981:168), but no quantitative information appears to exist.

Oral Contraceptives

Some oral contraceptives can produce a hearing loss and an associated tinnitus (Brown et al., 1981). The effects are thought to be due to vascular changes. Detailed information was not found.

Heavy Metals

Tinnitus is a common side-effect of heavy-metal treatment for cancer (Merrin, 1978; Merrin et al., 1978; Von Hoff et al., 1979). For cis -platinum at least, the evidence indicates that the symptom is reversed upon withdrawal of the drug.

Drug Therapy for Tinnitus

A wide variety of drugs have been investigated as possible treatments for the symptom of tinnitus. The basis for interest in a particular drug or class of drugs is frequently not explicitly stated by the investigator, which suggests that it came to attention simply because patients receiving the drug for some other medical problem reported their tinnitus to be alleviated. Given the general level of ignorance about the sites of origin of and the mechanisms underlying tinnitus, this empirical origin of ideas about tinnitus-alleviating drugs should not surprise us. The fact is that greater success has been achieved with drugs discovered in this way than with drugs studied because of some theoretical view about the origins of tinnitus.

The list of drugs investigated at one time or another as a possible therapy against tinnitus is long (see Goodey, 1981), and no attempt is made here to be exhaustive. There are some general points about experimental design that the reader ought to keep in mind while reading the following synopses of the studies done on drug treatment of tinnitus.

1. Because tinnitus is not a single entity produced by a single or even a small number of causes, one should not expect any one drug to be universally effective against tinnitus. Further, the actual proportion of the sample of patients reporting relief in a particular study will depend heavily upon the makeup of that sample. If, for some reason, an investigator ended up with a sample heavily dominated by patients whose tinnitus arose from the same problem—(say) in the cochlea itself—and if the drug being studied proved to be extraordinarily effective against that problem, that investigator might have a very high success rate, while another investigator doing the same manipulations, but on a sample less heavily dominated by patients with that cochlear anomaly, might have a much lower success rate. Even in those experiments in which the audiological diagnoses on the patients are reported, one cannot be confident about homogeneity in the site(s) of origin of the tinnitus.
2. The severity of the tinnitus symptom varies greatly across the patients used in different studies and sometimes across those in the same study. In some studies, the patients used are referred to the investigator simply because they have tinnitus, of whatever magnitude; in

other studies, only severely afflicted patients are used as subjects. Comparison of the success rates across studies as different as these is problematic enough, let alone trying to generalize to the population of tinnitus sufferers at large.

3. Many of the drug studies of tinnitus do not include the controls usually regarded as necessary for unambiguous interpretation of an outcome. For example, few studies utilize single-or double-blind procedures, control and experimental groups, or cross-over designs.
4. Because of 2 and 3 above—the great heterogeneity in the makeup of the patients in different drug experiments and the absence of typical control procedures—it is difficult to evaluate the contribution of the so-called Hawthorne effect (Roethlisberger and Dickson, 1939) to the reported success rates. When some of the patients have been through months or years of frustration trying to get relief from their tinnitus, it may be that anyone's doing anything in an attempt to help them might be met with reports of success.
5. It is very rare in drug studies of tinnitus for an investigator to manipulate the dose level or the duration of the administration, either within or across subjects. Thus, the reader is always uncertain whether some of the patients in the "no relief" category might have moved to the "partial relief" category and/or some of the "partial relief" to the "complete relief" category had the dosage or the period of administration been changed.
6. Drug studies of tinnitus often do not include monitoring of, or reporting of, drug serum levels. This is an understandable omission in many cases, since the taking and analysis of numerous blood samples raises complications for both the patient and the experimenter. Without such information, however, it is impossible to know whether the difference between success and failure of treatment in individual patients might be due to differential compliance with the dose schedule and/or differential absorption of the drug, for whatever reasons. It is possible that some of the across-subject variability in effectiveness of some drugs may be traced to different "effective doses" reaching the relevant site, and while drug serum level is an imperfect control for this problem, it does constitute a necessary first step. A procedure that seems sensible is one in which fixed or weight-corrected doses are given to sufferers from a wide variety of tinnitus types in the early experiments on a particular drug, and then—given that there is some

evidence of effectiveness of the drug—the serum levels of a subset of the therapeutic successes and failures might be checked in a follow-up study.

Niacin

A member of the vitamin B complex, niacin has three common forms—niacinamide, nicotinic acid, and nicotinamide. The amino acid tryptophan can also be converted into niacin by the body. Niacin is a peripheral vasodilator and thus has been used in the treatment of peripheral vascular disorders and migraine headaches. It also has a long history in the treatment of some forms of Meniere's Disease. Atkinson (1944a) found that about 50 percent of his (selected) patients reported relatively long-term relief from their tinnitus while under chronic maintenance dosages of nicotinic acid—injections initially, then oral administration. Flottorp and Wille (1955) used balancing and masking procedures to estimate the magnitude of tinnitus before and during administration of daily nicotinic acid (again, injection initially, oral eventually). As with the Atkinson study, most of the Meniere's patients reported an improvement in their tinnitus. In addition, nearly all of the patients in a group having normal or near-normal hearing evidenced lower matching intensities as well as subjective improvement. Subsequent experience with niacin treatment has not been as positive, so while it is still occasionally used, it is not a routine component in the treatment of Meniere's Disease or of tinnitus in general.

Vitamin A

Graham (1965:Table 4) provides a summary of the studies concerned with vitamin A therapy. The early reports of both reductions in tinnitus and improvements in hearing sensitivity following massive intramuscular injection of vitamin A were not confirmed in later studies, and the issue seems to have been dropped. It is possible that people with dietary deficiencies of vitamin A do, in fact, realize both improvement in their hearing and reduction of tinnitus but that people with more normal diets cannot profit from increased intake of vitamin A. (A similar conclusion was reached by Ward and Glorig [1960] in a study of vitamin A and temporary threshold shift.) Because it is possible to overdose on vitamin A, it is not advisable for tinnitus sufferers to experiment with heavy doses of this agent without medical supervision.

Lidocaine

(also known as lignocaine; Xylocaine is a registered trademark) Several recent reports indicate a high rate of improvement in tinnitus following administration of lidocaine. This amide is commonly used as a local anesthetic in surgery of the middle ear and upper respiratory tract, but it is also a potent short-term anticonvulsant that has vasodilation as one of its effects. During routine systemic administration of this drug for controlling and isolating the origin of central pain, it was noticed that preexisting tinnitus was alleviated or eliminated. (Interestingly, a single administration has sometimes alleviated the tinnitus for up to 72 hours.) Subsequent research has substantiated its short-term effectiveness against long-standing, intractable tinnitus. Lidocaine itself is not suitable for use in a tinnitus management regimen because it must be administered intravenously, and it has a short half-life in the body. However, its use as a research tool has stimulated much interest in drug treatment for tinnitus, and, as a consequence, more suitable compounds are being developed and tested (e.g., see tocainide and carbamazepine below).

Gejrot (1963) may have been the first to note that lidocaine can affect tinnitus. He injected the drug during episodes of Meniere's Disease and found, among other things, that the tinnitus disappeared for about 20 minutes in all 11 patients described.

Melding et al. (1978) studied 78 patients with "incurable and intolerable" tinnitus. Prior to the single administration of lidocaine, each patient was given a standard audiological examination. Following administration (I.V. injection of 1–2 mg/kg over a period of 3–4 minutes), the audiometry was repeated, and the patients were asked to indicate any improvement in tinnitus by placing a mark along a 100-mm line to indicate what fraction of the original tinnitus remained. (Exactly when following the injection this judgment was made is not specified, but presumably it was within a few minutes of injection.) Approximately 35 percent of these severely affected patients reported total abolition of the tinnitus, and an additional 28 percent reported the tinnitus to be 0.3 or less of its initial magnitude; about 26 percent reported no relief from their tinnitus. This has to be viewed as a great success, given the ordinary refractoriness of severe tinnitus. The authors report that the relief "lasted between 10 minutes and three days," but without specification of (1) how the data for the longer intervals were collected or (2) how the return

of tinnitus progressed. These points are important, given the high likelihood that different patients were using different criteria for their judgments.

Some of the patients reporting relief from tinnitus claimed that it was accompanied by an improvement in hearing. There is as yet no evidence to support this impression; the postinjection pure-tone audiometry did differ in some of these subjects, but the differences appear to be within the typical test-retest range of variation. Nevertheless, claims of improved hearing are reasonably common in drug studies on tinnitus (see the discussion of Melding and Goodey [1979]; and Shea and Harell [1978] in the next section). It may be that these patients' subjective impressions of improved hearing stem solely from the elimination of the ordinarily present and distracting tinnitus, but it is worth investigating whether or not some aspect of auditory function other than pure-tone sensitivity—usable dynamic range, for example—is altered by lidocaine.

When Melding et al. (1978) partitioned their 78 patients into standard diagnostic categories, they discovered that those with sensorineural problems had the greatest chance of benefitting from the lidocaine injection. Of the 55 patients with "pure sensorineural hearing loss," three subgroups—constituting 41 patients—stand out, as shown in the following breakdown of patients reporting either a total abolition of tinnitus (excellent relief) or a reduction in it to 0.3 or less of its preinjection magnitude (good relief):

	Excellent Relief	Good Relief	Total
Endolymphatic hydrops	4/8	2/8	75%
Acoustic trauma/noiseinduced hearing loss	9/16	4/16	81%
Presbycusic hearing loss	11/17	4/17	88%

These samples are small and, as indicated above, highly selected for severity of tinnitus, but the suggestion appears strong that short-term relief is possible for the majority of people with sensorineural-based tinnitus. The mechanism(s) mediating this improvement remain unknown, but a valuable tool seems to have been added to the scientists' chests. (For additional lidocaine results, see the following section on carbamazepine.)

An exception exists to the general criticism about the lack of appropriate controls in drug studies of tinnitus;

Martin and Colman (1980) used a double-blind procedure in which each subject served as his or her own control (some-times known as a cross-over design). The subjects were patients referred by otologists because of their tinnitus, but apparently they were not as highly selected for severity of tinnitus as in other studies. They were given standard physical and audiometric exams, they matched their tinnitus in frequency and intensity, and their tinnitus was rated (by the experimenters) on a three-point scale of severity. At random, the subjects were given an intravenous dosage of lidocaine (1.5 mg/kg of body weight of a 2 percent solution) or of an equal volume of normal saline; then they again matched their tinnitus, had it rated, and indicated how much the tinnitus had been relieved. After a time interval corresponding to "more than double the physiological half-life of intravenous lidocaine" (presumably more than 200 minutes total), the second substance was injected and identical follow-up measures taken.

In the lidocaine condition, there were both objective and subjective improvements in tinnitus. Objectively, the lidocaine injection produced reductions in both the frequency and the intensity of the sounds chosen as matches to the tinnitus. The decreases in matched frequency appear to be real, but small; no explanation of these changes is provided or comes to mind. Following lidocaine, about 31 percent of the subjects matched their tinnitus to intensities smaller than those before injection by 7.5 dB or more (versus 6 percent following the saline injection); an additional 62 percent showed little or no change following lidocaine (versus 69 percent following saline). This is one of the few studies we know reporting an objective change in tinnitus following treatment (compare Donaldson [1978], discussed in "Sodium Amylobarbitone" in this chapter); the outcome is important and deserves further documentation, including study of its time course.

The subjective results in the Martin and Colman study are even more impressive than the objective results. One would expect that the reductions in the matched intensity of the tinnitus would produce concomitant reductions in the perceptibility or the annoyance of the tinnitus, and, in fact, 72 percent of the subjects judged their relief from tinnitus to be 50 percent or greater following the lidocaine injection (versus 9 percent following saline). This percentage is similar to the 61 percent reporting good or excellent relief in the Melding et al. (1978)

study. The percentage reporting total abolition of tinnitus following lidocaine was 35 percent in the Melding et al. study and 44 percent in the Martin and Colman study (recall that the former study used only subjects afflicted with severe tinnitus). Unfortunately, Martin and Colman provide no information on the time course of this temporary relief; such information could prove valuable.

Thus, Martin and Colman's use of a saline placebo seems to strengthen the case of I.V. lidocaine as an effective agent against tinnitus, and it is to be hoped that similar research designs will be adopted by other experimenters working in this area.

Emmett and Shea (1980) briefly reported the results of a study that points to a curious possibility. Intravenous lidocaine was administered to 592 patients having "extremely bothersome tinnitus." One minute after injection, the patients were asked to indicate the magnitude of tinnitus relief in percentage units. Unlike other studies, the data are reported separately for the unilateral and the bilateral tinnitus sufferers. It is interesting that the bilaterally afflicted were much more likely to report relief than were the unilaterally afflicted. About 73 percent (139/190) of those with bilateral tinnitus reported relief of 50 percent or greater from the lidocaine injection, while only about 49 percent (197/402) of those with unilateral tinnitus reported relief in that range. At the other extreme, no relief was reported by only about 6 percent of those with bilateral tinnitus and about 27 percent of those with unilateral tinnitus. This possible difference in lidocaine effectiveness against unilateral and bilateral tinnitus has both theoretical and practical importance, and thus it is necessary to verify it using tinnitus matching procedures.

The mechanism(s) underlying the effectiveness of lidocaine on tinnitus are being studied. Using peripheral nerve preparations, Condouris (1976) has shown that the primary effect of local anesthetics such as lidocaine is to prolong a neuron's refractory period. This in turn produces a reduced ability to transmit high rates of firing. The intuitive view of how local anesthetics act to reduce pain is thus confirmed; firing rate declines, and this is accompanied by a diminution in perceived pain. By implication, then, what lidocaine may accomplish in regard to tinnitus is a reduction in the rate of activation of some (primary?) auditory fiber(s) sending a misleading signal.

Supplementary information on mechanisms comes from other research. Comeau et al. (1973) showed that iontophoresis of a lidocaine solution from the middle ear cavity into the cochlea did not alter cochlear microphonic potentials recorded for frequencies from 100 Hz to 20,000 Hz. However, Hughes and Yagi (1975) reported that lidocaine injected into the middle ear cavity produced a "prompt and prolonged" depression in the cochlear microphonic, and Yagi et al. (1978) noted pronounced reduction in resting rate and in responsivity of primary vestibular neurons of cats. Gerke et al. (1977) have provided evidence that lidocaine blocks the uptake of adrenalin (but not noradrenalin) by sympathetic nerves surrounding the media of certain arteries. Rahm et al. (1962) warn that lidocaine hydrochloride alone can have highly unpredictable cochlear effects across individual animals, but that when mixed with a vasoconstrictor such as epinephrine, the effects are more homogeneous. (The commercial form of lidocaine—Xylocaine—can be obtained with or without epinephrine added.)

Perhaps the most interesting insights about the mechanisms underlying lidocaine action come from Lyttkens and his colleagues (1979a,b). They have reported that lidocaine (as well as bupivacaine and chlorpromazine) bind to melanin in the inner ear. Lyttkens et al. believe that the melanin is involved in energy transformation—mechanical to electrical—and that in some way lidocaine may act as an aid to this process in a defective ear, thereby reducing the tinnitus. Whatever the final explanation, the findings of Lyttkens et al. raise several interesting points regarding drug treatment of tinnitus.

This relationship between lidocaine and melanin is intriguing, for it is now well established that melanin content is strongly related to a person's susceptibility to both temporary (Hood et al., 1976) and permanent (Karsai et al., 1972) hearing loss due to noise exposure—the more heavily pigmented a person is, the greater is the resistance to hearing loss. One interpretation of this fact is that the melanin in the inner ear is in some way contributing to the ear's line of defense against damage from overstimulation. The demonstration by Lyttkens et al. that an agent apparently quite effective against some forms of tinnitus binds to the melanin in the inner ear raises the two questions of whether tinnitus incidence is also correlated with melanin content, and whether dose levels of lidocaine (and some other tinnitus-ameliorating agents) might better be tied to

skin or eye color than to body weight. It would be helpful to know whether a particular skin or eye color is disproportionately represented in the groups showing weak or no responses to lidocaine or carbamazepine (see following section). In a discussion at a recent conference (CIBA Foundation, 1981:27–28), several clinicians agreed that dark-skinned people were greatly underrepresented in their practices, and Coles indicated that the recent British survey of tinnitus did include eye color and race data that are now being analyzed. (W. F. House [1975] believes that Meniere's Disease is less prevalent in blacks than in whites, and, if this proves to be true, it may also be related to inner-ear melanin.) Melanin content appears to be a variable highly worthy of attention in future surveys and experimental studies of tinnitus.

Carbamazepine

(Tegretol is a registered trademark) This drug is an oral anticonvulsant and mild antidepressant that is best known in this country for its effective use on trigeminal and glossopharyngeal neuralgia; in Europe it has been used extensively against epilepsy.

Based on their view of the origin(s) of tinnitus—"an abnormal hyperactivity"—Melding and Goodey (1979) postulated that carbamazepine might be effective against tinnitus. As in the Melding et al. (1978) study discussed in the section on lidocaine, only patients with "incurable and intolerable" tinnitus were included in this study. As in the earlier work, a full audiological exam was performed prior to any drug treatment. Then lidocaine was administered and its effectiveness measured as described above. The intent of this phase of the procedure was to obtain the data necessary for later correlation of the effectiveness of lidocaine and carbamazepine. (It is not clear from the published report whether the outcome of this phase was known to the experimenters when conducting subsequent interviews and measurements and/or whether the patients were asked to, or dissuaded from, comparing the effectiveness of lidocaine with that of the carbamazepine they later received.) As an aside, it should be noted that the effectiveness of lidocaine was about What it was in the previous experiment—about 77 percent of the patients reported total abolition of their tinnitus or a reduction in its magnitude to 0.3 or less of its pre-injection value (although it is not clear whether this group of 125 patients included some or all of the 78 patients reported on in the previous experiment).

Regardless of how they fared on the lidocaine injection, all the patients studied by Melding and Goodey were offered treatment on carbamazepine. Dosage began at 100 mg three times daily and was gradually increased until therapeutic levels were reached (typically 600–1000 mg/day); dosage was then stabilized, and treatment continued for 2–3 months. Once dosage was stabilized, the patients scaled any improvements in tinnitus by making a mark along a 100-mm line. Of the patients reporting total abolition of tinnitus following a single I.V. injection of lidocaine, about 60 percent reported either total abolition of tinnitus or reduction in its magnitude to 0.3 of its original magnitude or less by the carbamazepine regimen. Of those reporting major reduction but not abolition of tinnitus from lidocaine, about 53 percent reported good-to-excellent relief from carbamazepine. In general, no patient for whom lidocaine failed to produce relief received relief from the carbamazepine regimen (but see below). As before (Melding et al., 1978), no significant changes in audibility could be measured even in those patients reporting an apparent improvement in hearing (see comment under "Lidocaine" in this chapter). Once the carbamazepine was withdrawn, the tinnitus returned to its original magnitude within 2–3 weeks.

Shea and Harell (1978) followed up on the Melding and Goodey demonstration with lidocaine and carbamazepine. Their patients were also highly selected for severe and intractable tinnitus and, again, about 80 percent of these reported significant improvement in their tinnitus following a single injection of lidocaine (the procedure for measuring tinnitus was not specified). Unlike the Melding and Goodey procedure, however, in this study only those patients showing relief from the lidocaine injection were allowed access to the carbamazepine regimen (27 patients); this screening was later shown to be of questionable value. Shea and Harell also used lower daily dosages of carbamazepine than did Melding and Goodey (100–400 mg/day in contrast with 600–1000 mg/day), with little apparent effect on the success rate; approximately 80 percent of the lidocaine-screened sample reported partial or complete tinnitus relief from carbamazepine (the same data are reported by Emmett and Shea [1980]). This is an encouraging finding, since the incidence of objectionable side-effects from carbamazepine is dose-related. Again, some patients reported a subjective improvement in hearing that could not be confirmed with the standard audiometric tests. Finally, Shea and Harell

report one case of particularly severe palatal myoclonus (rhythmic, rapid contractions of the soft palate) that succumbed to a daily regimen of a low dosage of carbamazepine.

An important outcome of the Shea and Harell (1978) experiment was that several patients who barely qualified for the carbamazepine phase, due to minimal response to the lidocaine injection, did profit from carbamazepine. This —plus the fact that some patients receiving excellent relief from lidocaine received only minimal relief from carbamazepine—seems to make screening on the basis of the lidocaine injection a questionable procedure. Given the current level of understanding about the actions of these drugs, future investigators may wish to consider eliminating such screening (but note that Goodey [1981] disagrees).

It must be noted that carbamazepine can produce a wide variety of side effects (Goodey, 1981) and carries a significant risk of hepatotoxicity; thus, careful monitoring is required of patients taking it. This obviously limits the usefulness of carbamazepine as a general treatment for severe tinnitus, but the experience of Shea and Harell shows that the problems may not be insurmountable.

Tocainide Hydrochloride

(Tocard is a registered trademark) Unlike lidocaine itself, which must be administered intravenously, this analog of lidocaine can be taken orally because of a difference in its metabolism by the liver. Additional attractions are that it has a physiological half-life of about 11 hours as compared to about 1.5 hours for lidocaine, and it has fewer side effects. Tocainide is currently being used experimentally as an oral anti-arrhythmic agent in cardiac patients.

Two reports exist of attempts to treat tinnitus with tocainide (Emmett and Shea, 1980; no date); since the former consists primarily of preliminary results of dose level, we shall discuss only the latter (also reviewed by Emmett, 1981). Subjects not showing at least 75 percent relief of tinnitus following I.V. administration of lidocaine were screened out; the remainder were assigned to one of three groups: 600 mg of tocainide at 6-hour intervals four times a day; that same dose or a placebo every other 6-hour interval (i.e., tocainide twice a day), or a placebo four times a day. The duration of the study was 2.5 days (10 dose intervals); it was double blind.

Of the 56 patients beginning the study, 11 (20 percent) dropped out because of nausea (two of these were receiving a placebo only). Ninety minutes following the last dose, the patients rated their relief from tinnitus. Relief was judged to be between 40 percent and 100 percent in 46 percent (6/13) of the patients receiving four doses/ day, in 41 percent (7/17) of those receiving two doses/ day, and in 7 percent (1/15) of those receiving only a placebo.

Emmett and Shea used a self-report procedure in an attempt to determine the duration of relief following termination of the drug. The group that received tocainide four times a day reported a return of the tinnitus to its initial loudness after an average of 59 hours, while the average for the two-a-day group was 20 hours (11 hours for the placebo group). That is, the higher dose level did produce longer-lasting relief even though it did not significantly increase the fraction of patients receiving relief in the first place (46 percent versus 41 percent in the four-a-day and two-a-day groups, respectively). This outcome is intriguing, but requires verification using tinnitus matching procedures. Emmett (1981) noted that some patients who did experience tinnitus relief from lidocaine did not from tocainide, but there is no information about the converse situation, since lidocaine was used to screen the patients for the tocainide trials. The advisability of such screening procedures was discussed under "Carbamazepine" above.

It appears, then, that tocainide is not as effective in reducing tinnitus as are lidocaine and carbamazopine. On the other hand, it is clear that clinicians and experimentalists do have an additional weapon in their arsenal against tinnitus. Finally, an intriguing possibility was raised by Hazell (CIBA Foundation, 1981:50); he reported that several patients for whom neither tocainide nor masking was effective did benefit from masking when it was combined with tocainide (also see Vernon and Meikle, 1981).

Phenytoin Sodium

(also known as diphenylhydantoin; Dilantin is a registered trademark) This drug is an oral anticonvulsant similar in action to carbamazepine. In the Melding and Goodey (1979) paper discussed above, brief mention is also made of Dilantin. It was apparently used on four patients who developed an allergic reaction to

carbamazepine, but the only indication as to how these patients fared is the comment that Dilantin is always less effective than carbamazepine in treating tinnitus (Goodey, 1981). Shea and Harell (1978) reported that none of 15 patients treated with this drug had any relief from tinnitus, even though all had some relief from the lidocaine injection.

Primidone

(Mysoline is a registered trademark) This anticonvulsant has been tried against trigeminal neuralgia with mixed results. Emmett and Shea (1980) briefly mention a study of its effectiveness against tinnitus. Patients initially received 250 mg twice daily; the dosage was increased monthly in increments of 250 mg/day up to a maximum of 2 g/day or until the tinnitus was relieved. Details are few, but apparently 27 percent (11/41) of the patients reported 80–100 percent relief, and an additional 59 percent (24/41) reported 20–80 percent relief from their tinnitus. Exactly when in the regimen these judgments were made is not revealed, yet this appears to be an important issue given the high incidence of side effects reported.

Sodium Fluoridi

This compound has come to be recognized for its ability to reverse the process of demineralization in the cochlear capsule that leads to the condition of otospongiosis. Since this condition is often accompanied by a sensorineural-type hearing loss and by tinnitus and vertigo, it is of theoretical as well as practical interest here that these latter symptoms are often diminished or abolished as the otospongiosis is reversed (Shambaugh, 1977). The explanation offered is that the otospongiotic focus gives off cytotoxic enzymes that then enter the perilymph, causing a deterioration of cochlear elements critical to the normal transduction process , and, as a side effect, producing a concomitant tinnitus. The administration of sodium fluoride cannot reverse the hearing loss (although according to Shambaugh it does arrest it), but it can eliminate the tinnitus and vertigo. The process of reversal of bone damage often takes several years of daily doses of 40 mg of sodium fluoride. No information about the time course of reversal of the tinnitus is given, nor apparently were any psychophysical measures of tinnitus taken.

Sodium Valproate

(Depakene, Epilim, and Ergenyl are registered trademarks) Goodey (1981) briefly mentions this drug, indicating that it reduces tinnitus in about the same proportion of subjects as does carbamazopine, but that, at the doses used to date, the amount of relief experienced is less than with carbamazepine. Apparently side effects are minor.

Sodium Amylobarbitone

Noting a relationship between drugs that are effective on trigeminal neuralgia and on tinnitus (e.g., carbamazepine), Donaldson (1978) selected this fast-acting barbiturate, known for its effectiveness on trigeminal neuralgia, for a study on tinnitus. Forty patients with tinnitus of varying severity were randomly assigned to the experimental or control groups. Prior to treatment, all patients were assessed audiometrically, were asked to match the pitch and loudness of their tinnitus, and were asked to rate their tinnitus on a four-point scale (from "only noticeable in quiet environments" to "interferes with sleep, and patient engages in some activity to distract attention from it"). The experimental group was then put on a regimen of 50 mg in the morning, 50 mg in the early afternoon, and 80 mg at night; tinnitus was reassessed after 6 and 12 weeks. The drug was withdrawn after 12 weeks, and a final assessment was made at 18 weeks. Apparently the control group received absolutely nothing, but these subjects were reassessed at 6 and 12 (but not 18) weeks. Thus, the study was not "blind," and the control group was primarily a control for the passage of time (and for the spontaneous remissions that might occur in that time). Prior to treatment, the majority (70 percent) of both the experimental and control groups matched their tinnitus to frequencies of 400–10,000 Hz, and 78 percent matched to intensities of 20 dB SL or less (in good accord with Reed, 1960). After 12 weeks, the control group changed very little on these measures, but marked changes occurred in the experimental group. Thirteen (of 20) in the experimental group (compared with 2 in the control group) matched to lower intensities—this included 4 subjects for whom the tinnitus was abolished—and many now matched to a lower frequency, a fact that stands unexplained (but was also obtained by Martin and Colman, 1980). Perhaps most important is the fact that, of the 12 experimental subjects who, prior to treatment, rated their tinnitus as being in one of the two most severe categories, only 1 persisted

in this rating at the 12-week reassessment. That is, 11 of the 12 most severe cases (92 percent) benefitted from the amylobarbitone regimen, and, for 2 of these, the tinnitus was abolished.

Thus, Donaldson's study provides evidence that sodium amylobarbitone may be quite effective in diminishing the disturbance caused by tinnitus. Its usefulness as a treatment for episodic or chronic tinnitus is somewhat limited by its potential for damaging the liver, but in the (apparently effective) dosage used by Donaldson, no such problems arose. One curious aspect of Donaldson's report is that at the 18-week reassessment—6 weeks after drug usage had ceased—there were "no significant alteration(s)" in the states of the experimental subjects, including no return of tinnitus in the four subjects who had reported it abolished. This implies a reversal of the conditions initially responsible for the tinnitus—an unlikely event—which makes this an outcome requiring verification through further study.

Alcohol

Excess alcohol consumption is frequently mentioned in anecdotes as a cause of tinnitus, but curiously, some tinnitus sufferers apparently find that small amounts of alcohol assist in tinnitus control (Goodey, 1981). Of theoretical and practical interest is the nature of the assistance. If the primary contribution of the alcohol is to general relaxation and improved coping ability, that is far different from an actual physiological effect on tinnitus magnitude. Psychophysical measurements are in order. However, the fact that large numbers of tinnitus sufferers have not independently discovered palliative effects in alcohol consumption implies that psychophysical measurements will fail to establish effects on the tinnitus itself.

Miscellaneous Drugs

Here we list drugs that have come to our attention, but about which little is known.

Hazell and Jackson (CIBA Foundation, 1981:278) mention informal studies using naftidrofuryl, and Jackson (CIBA Foundation, 1981:277) mentions lorcainide and bupivacaine.

Arlidin, a vasodilator, and chlortrimeton, an antihistamine, have been used singly and in combination with some success against some forms of tinnitus (Shulman, 1981b).

Goodey (1981) indicates that the tricyclics, especially nortriptyline, may be worth investigating as tinnitus-reducing agents.

Diazepam (Valium) is surely one of the most widely prescibed drugs for tinnitus and its psychological concomitants, but Goodey (1981) says that there is no evidence that it has any value in this regard, and it can make depressed patients worse.

McCormick and Thomas (1980) utilized a double-blind cross-over design in a study of mexilitine—a pharmacological relative of lidocaine that has an oral form. The patients' numerical estimates of the severity of their tinnitus were unaffected both by this drug and by the placebo.

It has been asserted that barbiturates do not cause tinnitus (CIBA Foundation, 1981:169; see "Sodium Amylobarbitone" in this chapter), and it has been suggested that the aminoglycosides may produce only a temporary tinnitus (CIBA Foundation, 1981:34).

Heparin is reported to have produced temporary relief from tinnitus in a number of heart patients (CIBA Foundation, 1981:170).

Trowbridge (1949) injected a 5 percent solution of ethylmorphine hydrochloride—an analgesic and vasodilator—directly through the tympanic membranes of patients suffering from tinnitus that he judged to be caused by structures of the middle ear (the tympanic plexus). He injected repeatedly at 4-day intervals and claimed reduction or elimination of tinnitus for the majority of patients so treated. He also claimed improved audiometric measures. No recent application of these procedures were found.

Conclusions About Drugs and Tinnitus

It is important to emphasize the particular need for appropriate controls and measurements in studies of drug treatment for tinnitus. The absence of balanced experiments and of blinds and the likely presence of the Hawthorne effect and a "flight to health" are a potentially troublesome mix when the goal is to determine efficacy of a procedure. Nevertheless, recent discoveries about the effectiveness of certain drugs against some forms of tinnitus are encouraging. Unfortunately, lidocaine, carbamazepine, and sodium amylobarbitone are all very potent drugs that do carry possibilities of serious side effects that limit their usefulness; however, there are some indications that relatively low doses may be effective against tinnitus (Shea and Harell, 1978), and related,

safer compounds may be identified. We clearly are not yet at the stage of being able to prescribe a safe and effective drug for every tinnitus sufferer, but the past several years have seen the establishment of a firm base of knowledge, and there is every reason to believe that further progress will be made. The effort is definitely worthwhile.

Some intriguing facts coming out of this research deserve verification and further study:

1. There is an interesting possibility that the mechanism(s) through which lidocaine works against cochlear-originating tinnitus involves melanin, a substance previously linked with susceptibility to noise-induced hearing loss. It is of theoretical interest to know if there is also a greater susceptibility to severe tinnitus among those less well endowed with melanin. Of both theoretical and practical significance are the questions of whether the effectiveness of drug (or other) therapy is related to melanin concentration, and whether susceptibility to the reversible tinnitus induced by drugs and exposure (see "Possible Experimental Models of Tinnitus" in Chapter 2) also varies with melanin concentration.
2. The drugs found to be effective against severe tinnitus have been reported to have other auditory effects. Many patients report a marked subjective improvement in everyday hearing; to date these impressions have not been confirmed by objective test (Melding et al., 1978; Melding and Goodey, 1979; Shea and Harell, 1978; Emmett and Shea, 1980; CIBA Foundation, 1981:190–191), but it is possible that audiometric measures other than pure-tone sensitivity would reveal improvements. Second, there is some evidence from matching procedures that, following certain drugs, the magnitude of the tinnitus is reduced (Donaldson, 1978; Martin and Colman, 1980), and its frequency is lowered (Donaldson, 1978; Martin and Colman, 1980). The former outcome is in accord with patients' reports of the distress from tinnitus being reduced, but the latter outcome is curious and yet to be explained. Both deserve experimental attention.
3. Goodey (CIBA Foundation, 1981:288) claims that following long-term drug therapy about one-half the tinnitus sufferers never again experience tinnitus at its previous level. Numerous explanations of this fact spring to mind, but first it is necessary to verify the assertion.

4. There exist hints that drugs can work in conjunction with masking therapy to produce a greater reduction in tinnitus than either can produce singly (CIBA Foundation, 1981:50).

ALLERGY AND DIET

It is believable that any allergic reaction that directly or indirectly affects the outer-, middle-, or inner-ear systems could also produce a concomitant tinnitus. For example, any food or inhalant allergy that caused a blockage of the Eustachian tube might produce tinnitus as a byproduct. So, in theory, various allergy-induced changes in the inner ear might produce tinnitus, but published evidence of such changes is scanty. Goodey (CIBA Foundation, 1981:234,263) comments on the role diet control plays in tinnitus management and notes that in his experience coffee, tea, tonic water, red wine, grain-based spirits, cheese, and chocolate have been the most common dietary sources of tinnitus. But beyond comments of this sort, nothing is published.

Allergies and their control seem to have been given the greatest attention in regard to Meniere's Disease. Belief persists among some clinicians that allergies, particularly food allergies, are responsible for, or are a significant secondary factor in, some cases of Meniere's Disease, and since this malady is typically accompanied by a low-frequency, roaring tinnitus, this work deserves mention.

There can be little doubt that occasionally a patient will be found who shows marked vestibular or auditory effects as part of a general allergic reaction to certain agents and that control of these agents controls the aftereffects (see Wilson, 1972). The issue is how representative such patients are. Shaver (1975) was only able to relieve the Meniere's symptoms of about one-third of his patients through allergy management, and the success rate of Endicott and Stucker (1977) was apparently even lower. Pulec (1972), Clemis (1975), and Powers (1975) report equally modest success rates. All of these authors comment on the great difficulties and uncertainties associated with isolating the allergy-producing agents in an individual's daily life.

Thus, it appears that allergy management may aid some tinnitus sufferers who also have Meniere's-like symptoms, but the published success rates indicate that unless

severe allergies are part of the patient's medical history, allergy management should be a course of last resort or only part of a general plan for managing tinnitus arising from Meniere's Disease (see, for example, Goodhill, 1979:541).

BIOFEEDBACK

In general, biofeedback procedures are designed to give a subject control of a physiological variable over which he or she previously had no conscious control. Common examples are blood pressure and heart rate. In a typical procedure, the relevant physiological variable is monitored, and changes in the desired direction are indicated (fed back) to the subject via a simple auditory or visual stimulus. The subject's task, of course, is to learn to produce the desired changes through any means available. The goal is to eventually be able to dispense with the monitoring and signaling devices, so that the subject can effect the relevant changes whenever desired or necessary in his or her everyday life.

Biofeedback has been attempted on patients suffering from tinnitus, but in no instance has the prototypical procedure just described been used. That is, the subject has not been trying to learn how to reduce the tinnitus itself; rather, the task has been to learn to "relax"--as defined by electromyographic changes in tension in (for example) the frontalis muscle. There are two reasons for this indirect approach: (1) the obvious one that in most cases the tinnitus has not been detectable by external sensors (human or electronic) and thus could not serve as the basis for a feedback signal, and (2) a line of reasoning that goes: tinnitus magnitude has been reported to co-vary with level of anxiety, anxiety is widely believed to be related to muscular tension, and therefore reduced muscular tension ought to reduce tinnitus (a view that surely oversimplifies the etiology of tinnitus). Apparently, few biofeedback practitioners argue that the tinnitus per se is affected by the biofeedback sessions (P. R. House, 1981, may be an exception), but rather that the (not to be denied) gain in control over general relaxation simply makes the tinnitus more tolerable. This does not invalidate the procedure, of course, but it does put it in a different category from procedures that may, or do, alter the source of the tinnitus. It is possible that a direct biofeedback procedure could be developed

for people whose tinnitus has an objective, acoustical basis, so that the patient could try to learn to control tinnitus magnitude in the same way that control of blood pressure and muscle tension are learned. To our knowledge, such a procedure has never been attempted.

Grossan (1976) reports the results of a typical biofeedback experiment. In that experiment, all 11 presbycusic patients "felt that their tinnitus was improved" in the sense that "they could now cope with it"; tinnitus magnitude, however, as measured by a matching procedure, was unchanged following the six 20-minute sessions. Fourteen of 18 patients with acoustic trauma reported some improvement following biofeedback training, but, just as with the presbycusic group, there was no major change in the tinnitus matching levels. Only 2 of 21 patients suffering from cranial or cervical trauma reported some relief from tinnitus, although 13 others did report improvement of neck symptoms.

J. W. House et al. (1979) studied only patients with severe tinnitus who were persistent in their searches for relief but who were "therapeutic failures." After 10–12 one-hour biofeedback sessions spent learning to relax the frontalis muscle and increase peripheral blood flow, about 10 percent of the 41 patients stated that their tinnitus was now absent, and about 80 percent felt that there had been some improvement in their tinnitus. Better sleep and less need for medication (tranquilizers and antidepressants) were also reported. No breakdown of these proportions by type of auditory pathology is provided.

Thus, biofeedback training does appear to offer hope to some tinnitus sufferers, but it is likely that whatever improvement results is due more to the patients' increased ability to cope with the symptom than to its having been reduced. It is possible that direct methods of biofeedback training could be developed in the future and that, with these, true reduction in some forms of tinnitus might be realized. Certainly biofeedback ought to be considered before more psychological treatments, such as formal counseling or psychotherapy, if only because patients are more likely to participate and cooperate.

HYPNOTHERAPY

We know of no large-scale study on the effectiveness of hypnosis on tinnitus. Marlowe (1973) reported on two cases in which relief was obtained following hypnosis.

In both instances, the tinnitus was of the sort accompanying noise-induced hearing loss—middle to high frequency and bilateral—and the primary problem was interference with sleep. Different approaches were used with the two patients, but no explanation is given of the basis for choice.

One patient was instructed in a "mind over matter" approach while under hypnosis; he was instructed to concentrate on the tinnitus and note that its magnitude was diminishing. Posthypnotic suggestion was given that the patient could do this himself at bedtime with the same effect. Over a 6-week span, at least, the patient did find the tinnitus to be less of a problem, both at bedtime and during the day.

The second patient was told, under hypnosis, to recall a particularly pleasureable musical passage whenever his tinnitus became troublesome. The stated intent of this was to create an internally generated masking sound for the tinnitus, and, while it appears somewhat bizarre, the procedure apparently was very effective for this patient. During a 6-month follow-up period, the patient reported no instances of tinnitus interfering with his sleep—although he did on occasion notice some "strange music" at bedtime!

It is certainly believable that hypnosis could serve as an additional form of assurance to a patient or could produce a feeling of control over the symptom, thus supplying a sense of relief. It seems to be worth trying on some patients, for example, prior to beginning a program of psychotherapy.

ACUPUNCTURE

It probably comes as no surprise that acupuncture has been attempted as a treatment for tinnitus. Largely undocumented claims of success can be found, but at least one author believes that only about 5 percent or less of patients are helped by acupuncture (Mann, 1974). Given that such values are probably in the range of spontaneous remissions and given that higher success rates are achieved with other procedures, there appears to be little justification for resorting to acupuncture for the treatment of tinnitus.

ELECTRICAL STIMULATION

Hatton et al. (1960) found that 15 of an initial sample of 33 patients suffering from tinnitus reported increases or decreases in its magnitude under electrical stimulation applied through electrodes on the head and body. The positions of the stimulating and reference electrodes were systematically rotated in such a way that each subject was tested in eight conditions (two polarity arrangements for each of four pairs of electrode positions).

Consider first those patients, of the subset of 15, having monaural tinnitus. When Hatton et al. located "the anode" on the same side of the patient's body as the tinnitus, the magnitude of the tinnitus was decreased, sometimes to the point of total suppression, as current strength was increased. With the electrode polarity reversed, these patients reported an increase in tinnitus magnitude as the current was increased. (Residual inhibition was not tested for or spontaneously noted.) In the patients with bilateral tinnitus, the same pattern obtained— the tinnitus on the side of the body with the anode" decreased and that on the side with "the cathode" increased as current strength was increased.

The patients whose hearing loss Hatton et al. traced to noise trauma and to presbycusis were notably unresponsive to the electrical stimulation. Apparently neither increases nor decreases in tinnitus were observed by these patients. If confirmed, the outcome will be an important one, for it has strong implications about the site of action of the electrical stimulation.

Tonndorf (CIBA Foundation, 1981:226) reported that J. D. Hood observed a suppression of tinnitus during and following electrical stimulation to the mastoids. Bilger (1977:169) reported that some patients fitted with an auditory prosthesis (cochlear implant) experience alteration of their tinnitus when the stimulating device is turned on; for some of these people the tinnitus is exacerbated, for others it is reduced or eliminated. For some patients these effects diminished with time since implantation. Brackmann (1981b) generally confirms Bilger's findings and implies that many implant patients experience pronounced residual inhibition following use of their stimulators. Brackmann also asserts that stimulation from the implant can reduce or eliminate contralateral as well as ipsilateral tinnitus and that the implantation operation itself reduced or eliminated preexisting tinnitus in about 80 percent of a particular sample of 29 patients.

Cazals et al. (1978) have confirmed earlier reports by others (e.g., Graham and Hazell, 1977; Field, 1893) that tinnitus can sometimes be abolished through the transtympanic application of brief electrical pulses to the cochlea (also see Portmann et al., 1979). Cazals et al. dealt only with patients having severe to profound hearing loss. The reference electrode was a silver disk attached to the earlobe; the active electrode was a ball of silver attached to the round window or promontory (although Aran and Cazals [1981] now urge the use of a platinum-iridium wire). The stimuli were electrical pulses of variable intensity, duration, frequency, and polarity. When "negative current" was applied to the active electrode, 81 percent (13/16) of the subjects were able to perceive auditory sensations of some sort with some combination of stimulus values; this effect has previously been reported and studied, of course, and is the basis on which hope for a cochlear implant prosthesis is built (e.g., Merzenich, 1975).

When the polarity of the electrical pulses was positive at the active electrode, auditory sensations were rarely reported, and, when they were, they were brief and they occurred at pulse offset—i.e., during a negative-going portion of the stimulus. However, in the subset of six patients who experienced tinnitus, five reported that it was suppressed by positive pulses. (Indeed, one of these subjects was not aware that he normally had a continuous tinnitus until it was eliminated by positive pulses.) As might be expected, the intensity necessary to achieve suppression varied across subjects (by a factor greater than 10); not so expected, perhaps, is the fact that frequency of stimulation appeared not to be important as long as it was higher than some "critical" value that did vary across subjects. The one subject not experiencing tinnitus suppression from positive pulses had had cranial trauma with pontine concussion, and thus it is possible that his tinnitus originated from a central location not affected by this electrode placement. An interesting report from one of the subjects who did have some residual hearing was that his understanding of speech was not obviously degraded by simultaneous presentation of the positive electrical pulses.

In a later report, Aran and Cazals (1981) make some additional points. The electrical stimulation was only effective in suppressing tinnitus clearly localized in the ipsilateral ear—never in the contralateral ear (compare Hatton et al., 1960). While no constant clinical

features could be identified as predictive of the effectiveness of electrical stimulation, Aran and Cazals believe that the suppressible instances had a peripheral origin and the nonsuppressible instances had a central origin. In some patients the electrical stimulation was maintained for as long as an hour, with no diminution in effectiveness. Apparently some patients did report periods of relief extending beyond the duration of the electrical stimulation (so-called residual inhibition), but Aran and Cazals are clearly dubious about the reality of this effect. (Graham and Hazell [1977] once observed a 4-hour-long suppression following a 30-second stimulation.)

Cazals et al. (1978) suggested that the electrical stimulation with positive pulses produces the equivalent of hyperpolarization in neural elements of the cochlea or beyond. The idea is obviously in accord with the fact that reversing the polarity of the pulses—thereby presumably causing the equivalent of depolarization—produced auditory sensations in the same subjects. It does not explain why pulses are effective and steady-state (DC) stimulation apparently is not nor why there is a "critical" frequency of pulse stimulation. A test of the idea with animals might be informative.

Thus, electrical stimulation appears to be worthy of further investigation as a treatment for severe tinnitus. The next step might be to try the procedure of Cazals et al. (1978) on volunteer tinnitus sufferers who have less hearing loss than did those patients. The reports from the Aran group raise the intriguing possibility of chronic implantation of both an electrode on the round window and a stimulator delivering pulses of the correct polarity for patients with otherwise intractable tinnitus. As Aran (1981) has noted, suppression of tinnitus with electrical stimulation may eventually prove to have diagnostic value even if it does not prove useful itself as a treatment.

A related matter deserves note here. Among mental health professionals there exist anecdotes about electroconvulsive therapy (ECT) producing tinnitus, sometimes in a severe form. No published reports could be found on this topic, but the relationship was recently noted (CIBA Foundation, 1981:230).

ALTERATION IN AIR PRESSURE

Vernon et al. (1980) briefly describe a device developed by R. Sandlin that has produced short-term relief in a

single patient with a roaring, low-frequency tinnitus and a history of otosclerosis. Apparently the device fits tightly into the ear canal and is equipped with a syringe and a valve that allow the patient to increase the resting air pressure in the space between the device and the tympanic membrane. The procedure is described as awkward but very effective for this patient. It is asserted that it is fairly common to observe relief from tinnitus as a consequence of a change in the resting air pressure, as occurs in routine tympanometry (see, for example, Johnson and Goodwin, 1981).

Tjernstrom (1977) manipulated the relative air pressure in the middle and outer ears of patients with Meniere's Disease and found that an overpressure in the middle ear produced decreases in the tinnitus, vertigo, and low-frequency hearing loss that are characteristic of this disease, as well as decreases in the feelings of nausea and fullness. It is reported that attempts to incorporate such pressure changes into a treatment regimen for Meniere's Disease is being attempted in Tjernstrom's laboratory in Malmo, Sweden (J. Tonndorf, personal communication).

Wilson (1980a: Figure 12) has reported being able to reliably produce a low-frequency tonal tinnitus in himself under conditions of body tilt. His explanation involves the changes in acoustic impedance offered by the middle ear (stapes) to the cochlea that alterations in body orientation would produce. In normal body orientation, the impedance of the middle-/inner-ear boundary can be thought of as mass dominated, but, as body orientation is inverted and hydrostatic pressure altered, the impedance comes to be stiffness dominated. Wilson notes that the polarity of a reflection off a stiffness-dominated termination is opposite to that of a mass-dominated termination, and he argues that his low-frequency tinnitus becomes audible under body inversion because the output of an existing "active process" in his cochlea is driven into oscillation by the stiffness-terminated reflections. By Wilson's argument, his orientation-induced tinnitus ought to have an objectively detectable counterpart (a spontaneous otoacoustic emission; see "The Objective/Subjective Issue" in Chapter 2) that does not exist under normal orientation, but he has so far been unable to isolate it.

Wilson and Sutton (1981) also introduced pressure changes directly into the outer ears of a number of people having spontaneous emissions. Both increases and

decreases in pressure generally produced upward shifts in frequency of the emission that were experienced (by the subjects whose emission was audible) as increases in pitch and as alterations in the tonal character of their tinnitus. The sound-pressure levels of the emissions could either increase or decrease with pressure change, but no concomitant changes in tinnitus magnitude were reported. Apparently no actual psychophysical measurements of tinnitus were made in these conditions.

Evans et al. (1981) worked with a guinea pig that had a spontaneous otoacoustic emission. They were also able to shift the frequency of the emission upward by introducing small (0.5 percent) increases or decreases in outer-ear pressure. Unlike the Wilson and Sutton (1981) demonstration, however, only reductions in the level of the emission were observed under conditions of altered pressure. Of course, we cannot know from this whether there were concomitant changes in subjective experience.

Thus, we have one brief report from Vernon et al. (1980) of a patient whose tinnitus can be reduced by altering the air pressure in the outer-ear canal, and, on the other side, we have Wilson and Sutton's (1981) report of no obvious perceptual change in tinnitus magnitude following air-pressure manipulations that produce increases and decreases in spontaneous otoacoustic emissions. Ignorance of the cause of the tinnitus in the Vernon et al. patient permits us to view these outcomes as not necessarily contradictory. That and the recurrence of anecdote about air-pressure changes and tinnitus reduction imply that mechanical attempts to relieve tinnitus may deserve greater attention for purposes of both diagnosis and treatment.

TINNITUS MASKERS/INSTRUMENTS

The idea of using an external sound to mask an obtrusive tinnitus dates back at least to Jones and Knudsen (1928), although Saltzman and Ersner (1947) are typically given credit for first implementing the idea. To a nonsufferer, there may seem to be something inconsistent in a sufferer's willingness to substitute one continuous sound for another--a point to which we shall return—but its effectiveness for some tinnitus sufferers is not to be denied. Indeed, many sufferers independently discover similar self-treatment strategies, e.g., mistuning an FM receiver and using the interstation noise as a masker

while sleeping, choosing an office with relatively high background noise instead of a quiet one, etc. But while some tinnitus sufferers and some hearing professionals have long been aware of the value of masking, it was not until relatively recently that there was any serious attempt to try to capitalize on masking as a general treatment for relief of tinnitus (Vernon, 1977).

Why substituting a masking noise for the tinnitus serves as effective relief for some patients has been explained in a number of ways. Some patients comment that their tonal or narrowband tinnitus is simply more annoying than the broadband masker they choose. An important feature for some patients is the knowledge that the external masker is "a real sound" and under their control if they choose to alter it in some way—clearly not a feature of the tinnitus. This issue of potential control as it relates to annoyance has been studied by Glass and Singer, 1972; see "Annoyance of the Tinnitus" in Chapter 3.

The observation made by Saltzman and Ersner (1947) was that a goodly number of patients experienced relief from their tinnitus when fitted with a hearing aid. Their explanation was that the now-amplified external sounds acted to "drown out" the tinnitus. This observation is, of course, in perfect accord with the self-administrations practiced by many tinnitus sufferers and noted above—the use of broadband maskers such as FM interstation noise, the preference for a relatively noisy working environment over a quiet one, etc.

Nearly 30 years after the Saltzman and Ersner report, Vernon (1977) took their observation an additional step and suggested not just amplifying ambient sounds to achieve masking, but deliberately generating sounds for that purpose. The idea apparently originated in part out of frustration in treating tinnitus sufferers having reasonably normal hearing over the standard audiometric frequencies. These patients often had tinnitus at such high frequencies that masking it by using hearing aids to amplify ambient sounds was either impossible—due to the bandwidth limitations of the aids—or impractical—due to the consequent amplification-induced distortion in the low-and mid-frequency regions in which hearing was normal. But while the procedure was apparently originally conceived for use with these special cases of tinnitus, its use was soon extended so that tinnitus masking has now been attempted on sufferers having tinnitus of a wide variety of types.

In order to simplify the process of tinnitus masking in the everyday lives of their patients, Vernon and his collaborators developed sound-generating circuitry that could be housed in a standard hearing-aid chassis. This device was called a tinnitus masker. In a later development, tinnitus masking circuitry was housed in the same case with a standard hearing aid. This combination was called a tinnitus instrument; it has proved to be a useful device because many tinnitus sufferers also have hearing loss that is responsive to amplification. It is estimated that 10,000 tinnitus maskers and instruments are now in use in the United States (Vernon and Meikle, 1981).

If we presume for a moment that tinnitus will behave like an external sound when it comes to masking (see "Some Ways Tinnitus Is Not Like an External Sound" in Chapter 3), then, ideally, the sound generated to mask a tonal or narrowband tinnitus would itself be reasonably narrowband, and it would be located as accurately as possible in the spectral region of the tinnitus. A masker with these characteristics would be maximally efficient in that the least intensity would be required, and, thus, the least interference with other external sounds would result. While this ideal is nearly achievable with modern technology, to date it has not been vigorously pursued. Instead, the masking sounds generated by all tinnitus maskers/instruments are rather broadband, with limited energy in the region above about 6 kHz (see Vernon et al., 1977). Thus, for high-frequency tinnitus, greater intensity will be required for masking—if masking can even be achieved--and consequently there will be greater interference with the perception of real-world sounds than would be true if the masker were narrower and spectrally better located. Broadband or spectrally complex tinnitus, of course, raises the same problem in a slightly different form: in order to mask the tinnitus, other real-world sounds may also be partially or wholly masked, and it becomes increasingly difficult to decide whether the symptom or its treatment is the less desirable.

A recent finding by Penner (personal communication) is relevant to this issue of the ideal bandwidth for a tinnitus masker. Recall that she obtained daily pitch matches over the course of a month from three sensorineural subjects and that the range of settings covered thousands of Hertz for every subject (see "Pitch Matching" in Chapter 3). If this result is an accurate

reflection of a long-term variability in tinnitus pitch, it might be the case that a narrowband masker fixed in frequency would not be the optimal tinnitus masker for some subjects; a tunable narrowband masker or a masker broadband enough to cover the range of frequency variation might be preferable. This matter clearly deserves further experimental attention.

Efficacy of Tinnitus Maskers/Instruments

The early reports on the efficacy of tinnitus maskers were very optimistic (Vernon et al., 1977; Vernon, 1978b; Vernon and Schleuning, 1978), perhaps misleadingly so, and, as a consequence, maskers began to receive much attention from auditory specialists and the popular press (Galton, 1979). Unfortunately, later reports (Schleuning et al., 1980; Roeser and Price, 1980; Rose, 1980) have been somewhat less encouraging, and this seems to have produced a widespread feeling that tinnitus masking is of little or no value (e.g., Loavenbruck, 1980). As will be seen, the truth lies somewhere between the extremes of panacea and worthlessness.

The reader is cautioned at the outset that at present there exists only one substantial set of data on the effectiveness of tinnitus maskers/instruments—the data from the Oregon group. This means that, even after a careful analysis of the data, erroneous conclusions are possible, for unreported procedural details or aspects of the data-reduction process may be instrumental in producing an undetected bias in one direction or another. We have, through private correspondence with the Oregon group, attempted to verify that the details of the published accounts are complete and, where not, to ascertain those details. Nevertheless, the risk remains that the conclusions reached here are appropriate, given the available data, but incorrect due to procedural or analysis details unrecognized for their biasing influence. Only additional, large-scale studies will reveal whether the following assessment of the effectiveness of tinnitus maskers/instruments is correct.

A procedural note is also in order at this point. Any effort to evaluate a new treatment for a long-neglected malady is subject to bias in the direction of overestimating its effectiveness. This bias stems from the natural gratitude and hopeful expectations of the previously neglected patients, and it is surely magnified when the

patients also make a financial investment in the treatment, as, for example, the purchase of a masker, instrument, or aid. Ideally, attempts to estimate effectiveness should be made only after enough time has passed to allow the patients to make objective judgments about effectiveness that are relatively free from this bias. Of course, ideal conditions cannot reasonably be expected in real-world research, so it becomes the reader's job to remain alert to the possible contribution of this bias in what follows.

The Early Reports

In a 1977 article, the establishment of a tinnitus clinic at the University of Oregon Medical School was announced, along with some preliminary results (Vernon et al., 1977). At that time, 80 tinnitus patients had been seen; masking as provided by hearing aids had been recommended to 48 (60 percent) of these, and tinnitus maskers had been recommended to 29 (36 percent). (Tinnitus instruments did not become available until late 1978.) Of the patients accepting the recommendations, about 68 percent of the hearing-aid users and about 76 percent of the masker users "experienced total relief from their tinnitus" when the device was worn. About two-thirds of this latter group were also reported to have residual inhibition of their tinnitus. The implication given in this report is that the average patient routinely experienced 35–40 minutes of residual inhibition following a day's use of the tinnitus masker; at the other extreme, one patient had once experienced 16 hours of relief, and another, 18 hours.

By 1978, 158 patients had been seen at the Oregon tinnitus clinic (Vernon and Schleuning, 1978), and a third category of recommended treatments had been added—so-called FM masking, which is simply the use of interstation radio noise as a background when working quietly or trying to sleep. By then, about 52 percent of them had been recommended hearing aids, about 24 percent tinnitus maskers, and about 6 percent FM masking. (Thus, the proportion apparently receiving no recommendation was up from 4 percent in 1977 to 18 percent in 1978—a dramatic change, given that the 80 patients of 1977 were presumably included in the 158 of 1978.) According to the 1978 report, the percentage of patients who "obtained complete relief of their tinnitus" was about 67 percent (28/42) of those who accepted the recommendation of a hearing aid, about 81 percent (26/32) of those who accepted the recommendation

of the tinnitus masker, and about 67 percent (6/9) of those who accepted the recommendation of FM masking. Vernon and Schleuning (1978) calculated that overall about 72 percent of their patients had been relieved of their tinnitus by masking in one form or another. Unfortunately, this 1978 report contains no update on the proportion of patients showing residual inhibition, but two cases are related in which the patients' residual inhibition gradually increased with continued use of the tinnitus masker, until the tinnitus eventually disappeared completely, causing the patients to return the masking units. Both of these "cures" involved tinnitus of several years' duration.

Later in 1978 Vernon (1978a) reported some summary statistics on the 513 patients who had, by then, been seen in the Oregon tinnitus clinic. Unfortunately, not all of the percentages of interest here were presented, but two trends did persist: the percentage to whom hearing aids were recommended dropped further, to 48 percent, and the percentage to whom no recommendation was made rose further, to 23 percent. Also of interest is the fact that about 78 percent of all patients seen exhibited either partial (43 percent) or complete (35 percent) residual inhibition in Vernon's standard test for residual inhibition (discussed under "Residual Inhibition" in this chapter).

Thus, the early evidence indicated that effective relief from tinnitus can be obtained through the application of masking. It must be appreciated that the patients seen at the Oregon tinnitus clinic were, by and large, an extreme sample—people with long-standing or particularly troublesome tinnitus—a fact that contributed greatly to the significance attached to the positive results by auditory specialists and laymen alike. It is fair to say, however, that many hearing specialists remained unconvinced. In part this skepticism stemmed from a reaction to the way the data were being released and to certain actions of the American Tinnitus Association (see McFadden, 1982). Nevertheless, the evidence for the effectiveness of masking against tinnitus continued to accumulate.

The Later Reports

By 1980, the Oregon tinnitus clinic had seen about 1,000 patients having tinnitus as their primary complaint. In an attempt to evaluate the longterm

effectiveness of the various masking schemes that had been prescribed, questionnaires were sent to about 750 of these patients, and the 598 (80 percent) that were returned were discussed by Schleuning et al. (1980). For a number of justifiable reasons, these authors chose to discuss separately the data from patients seen from 1976 to 1978 and those seen in early 1979; these two subsets consisted of 493 and 105 returned questionnaires, respectively. Because it was based on a full-fledged questionnaire, this 1980 report is obviously more detailed than were the previous ones, and, while this detail is welcome, it does make comparison with the early reports difficult in places. Obviously, neither Schleuning et al. nor we can know whether, or how, the data would change if all the questionnaires had been returned. Summaries of the data reported by Schleuning et al. and discussed below can be found in Tables 1 and 2.

Of the 493 patients from the 1976–1978 period who returned the questionnaires, only about 27 percent (132) had been recommended hearing aids; this percentage is down from about 52 percent of the 158 patients described in the Vernon and Schleuning (1978) report and down from about 48 percent in the Vernon (1978) report, a discrepancy that is not discussed. (Specifically, in the Vernon [1978] report on 513 patients, 246 [48 percent] had been recommended hearing aids, yet in the Schleuning et al. report of the 493 patients in the 1976–1978 group, only 132 [29 percent] had been recommended hearing aids. Vernon [personal communications] could not account for this fall in absolute numbers, except to note that the two samples were not perfectly overlapping. The impression given in the various Oregon reports is that they are a series of updates and that the statistics are cumulative. Apparently this is not strictly true.) Approximately 41 percent of the 493 patients had been recommended tinnitus maskers, and an additional 9 percent had been recommended the newly developed tinnitus instruments; this total of 50 percent is more than double the 24 percent cited by Vernon and Schleuning (1978). Finally, about 23 percent were given no recommendation—very similar to the proportions reported earlier—but it is unclear whether or not this category now contains the patients who were recommended FM masking, a category used in the earlier report by Vernon and Schleuning (1978).

It is possible to estimate the success of these various devices in a number of ways. One is by examining the fraction of patients still using the device after a fixed

TABLE 1 Results of Follow-up Questionnaire Returned by 493 Patients Seen 1976-1978[a]

	Maskers			Hearing Aids			Instruments			Totals			
	No.	Percent of Purchased	Percent of Recommended	No.	Percent of Purchased	Percent of Recommended	No.	Percent of Purchased	Percent of Recommended	Summed No.	Percent Summed Purchased	Percent Summed Recommended	Percent Total Respondents
Recommended for trial purchase	**204**	–	100	**132**	–	100	**44**	–	100	**380**	–	100	77
Did purchase device	93	100	46	91	100	69	32	100	73	216	100	57	44
(Currently wearing device)	(61)	(66)	(30)	(68)	(75)	(52)	(29)	(91)	(66)	(158)	(73)	(42)	(32)
Tried but did not purchase	58	62	28	17	19	13	5	16	11	80	37	21	16
Did not try or purchase	53	–	26	24	–	18	7	–	16	84	–	22	17
No recommendation	–	–	–	–	–	–	–	–	–	**113**	–	–	23
RELIEF													
No. of purchasers	93	100	46	91	100	69	32	100	73	216	100	57	44
Total relief	6	6	3	2	2	2	5[b]	16	11	13	6	3	3
Partial relief	69	74	34	39	43	30	22	69	50	130	60	34	26
No relief	18	19	9	50	55	38	5	16	11	73	34	19	15

[a]Adapted from Table 2 of Schleuning et al. (1980). Some column totals ignore rounding errors.
[b]By inference.

TABLE 2 Results of Follow-up Questionnaire Returned by 105 Patients Seen in Early 1979[a]

	Maskers			Hearing Aids			Instruments			Totals			
	No.	Percent of Purchased	Percent of Recommended	No.	Percent of Purchased	Percent of Recommended	No.	Percent of Purchased	Percent of Recommended	Summed No.	Percent Summed Purchased	Percent Summed Recommended	Percent Total Respondents
Recommended for trial purchase	**36**	–	100	**17**	–	100	**25**	–	100	**78**	–	100	74
Did purchase device	23	100	64	14	100	82	16	100	64	53	100	68	51
(Currently wearing device)	(21)	(91)	(58)	(12)	(86)	(71)	(16)	(100)	(64)	(49)	(92)	(63)	(47)
Tried but did not purchase	10	44	28	1	7	6	5	31	20	16	30	20	15
Did not try or purchase	3	–	8	2	–	12	4	–	16	9	–	12	9
No recommendation	–	–	–	–	–	–	–	–	–	**27**	–	–	26
RELIEF													
No. of purchasers	23	100	64	14	100	82	16	100	64	53	100	68	51
Total relief	5	22	14	1	7	6	2	12	8	8	15	10	8
Partial relief	14	61	39	8	57	47	14	88	56	36	68	46	34
No relief	4	17	11	5	36	29	0	0	0	9	17	12	9

[a]Adapted from Table 3 of Schleuning et al. (1980).

period of time. Of the 216 patients in the 1976–1978 subset who did purchase the device recommended, 73 percent (158) claimed to be still using it at the time of the questionnaire. Another way to estimate success is the one used in the early reports—simply asking the patients whether or not the device provided relief from their tinnitus. In the early reports, the only positive outcome category was "complete relief"; on the questionnaire, however, only 6 percent of the 1976–1978 subset reported "total" relief, with an additional 60 percent reporting "partial" relief (these percentages are calculated across the three devices—aids, maskers, and instruments—but they are much the same for each). Schleuning et al. noted that the interpretation of this outcome is unclear; it may have been that some patients had complete relief from tinnitus during the wearing of their devices (so-called active relief) but that they had acquired high expectations of pronounced residual inhibition that were not realized, causing them to regard their overall relief as only "partial."

The percentages cited above were only for the 1976–1978 subset of respondents to the questionnaire; the data for the 105 patients in the 1979 subset are different in a number of ways (compare Tables 1 and 2). For one thing, more tinnitus instruments and fewer hearing aids were recommended—about 24 percent and 16 percent, respectively, as compared with about 9 percent and 27 percent in the 1976–1978 subset. The explanation of this shift is straightforward—the tinnitus instrument was not commercially available until late in the 1976–78 period, and, since most of the patients did have some hearing loss accompanying their tinnitus, it just made good sense to recommend the combined aid/masker units once they became available. About 34 percent of the 1979 subset had only tinnitus maskers recommended to them, down from about 41 percent in the 1976–1978 subset. No recommendation was made for about 26 percent of the 1979 subset, up from about 23 percent in the 1976–1978 period. On the two measures of success, the 1979 subset surpassed the 1976–1978 group. Overall, about 92 percent of the patients who did purchase the recommended device claimed to be still using it at the time of the questionnaire—compared with 73 percent in the 1976–1978 subset—and about 15 percent reported "total" and 68 percent "partial" relief—compared with 6 percent and 60 percent, respectively.

Schleuning et al. (1980) cited a number of factors contributing to the improvement in the success figures

from 1976–1978 to 1979, but they seemed to regard technological improvements in the tinnitus instruments as the primary one. Of course, an important variable operating to confound unambiguous interpretation of the apparent improvement is the time factor; at the time of the questionnaire, the 1976–1978 group had had more time than the 1979 group in which to become disenchanted with their masking devices. A possibly overriding factor has surfaced, however, that deserves special attention. Not mentioned in the Schleuning et al. article is a change in the Oregon group's procedures for making recommendations that was apparently instituted in late 1978 or early 1979 and thus differentially affected the success statistics of the 1979 group (Vernon, personal communication). The change was to make only a "tentative" recommendation after the initial examination. The patient took that initial recommendation to a hearing-aid dispenser and tried the recommended device for a month before deciding to purchase or return the unit. An unspecified number of patients returned to the clinic with complaints during this 1-month period, and some received new "tentative" recommendations. Apparently, a recommendation only became "final" once a patient had accepted or rejected the "tentative" device and/or had ceased returning to the clinic. Thus, with the new procedure patients who would presumably have been categorized as unsuccessful under the 1976–1978 procedures were, in some cases, being reexamined, given alternative recommendations, and eventually counted as successful. No one interested in good care for the patient can dispute the virtues of this flexible, staged procedure for making recommendations; it is unquestionably a laudable change. But the change does create a statistical problem for those interested in evaluating the efficacy of masking for tinnitus. Specifically, the improvement seen in the success measures obtained from Tables 1 and 2 must now be viewed with some reservation. Given the modified procedures, it would be astonishing if the success figures did not improve from 1976–1978 to 1979—in the latter group the final recommendations sometimes came only after the failure of one or more tentative recommendations, and presumably some very risky cases were given tentative recommendations but still appear in the no recommendation category in Table 2. The reader is encouraged to keep this important procedural change in mind while reading the remainder of this section.

Since they are currently the primary source of information on the effectiveness of masking on tinnitus, the

questionnaire statistics of Schleuning et al. deserve a final summary. Questionnaires were sent to 750 patients who had been seen since the inception of the Oregon tinnitus clinic in 1976. Replies were received from 598 of them (an 80 percent return rate). The authors argued that their procedures and instrumentation and the products available to their patients had changed so much by the beginning of 1979 that the survey data from the patients seen since then should be analyzed separately from those of the patients treated during the initial 3 years of the clinic. When the data are partitioned in this way, they support the contention that the tinnitus clinic has become more effective in recommending and/or fitting devices capable of alleviating tinnitus. Looking across the three types of device recommended—maskers, aids, and instruments—about 42 percent of the 1976–1978 subset and about 63 percent of the 1979 subset were using the recommended device at the time of the survey (see Tables 1 and 2); these numbers become 73 percent and 92 percent, respectively, when the denominator of the fraction is changed from simply the number of patients given a recommendation to the number of patients who actually purchased the respective device after the trial period. This improvement in use holds within, as well as across, the three categories of recommended device. Also indicative of improved procedures and recommendations from the 1976–1978 period to the 1979 period are the percentages of patients reporting either partial or total relief from their tinnitus. In the 1976–1978 subset, this group was 66 percent of the patients who actually purchased the recommended device (about 38 percent of the total to whom recommendations were made), and in the 1979 subset it was 83 percent (about 56 percent of the total receiving recommendations).

All of these indications of procedural improvements must be interpreted with an eye to the fact that the percentage of patients to whom no recommendation was made went up slightly, from 21 percent in 1976–1978 to 26 percent in 1979; possibly some of the diagnostically more risky patients who were given recommendations in the early years of the clinic were not later on, and this would naturally contribute somewhat to the higher success rates. Even though the sample size is still quite small, note should be taken of the greater apparent success of tinnitus instruments than of tinnitus maskers (Table 2), a trend that is worth watching. A final indication of improved procedures and success can be extracted from the

Schleuning et al. data. In the 1976–1978 subset, about 22 percent of the patients for whom a device was recommended simply did nothing—not even use the device during a trial period (see Table 1). This fraction dropped to about 11 percent in the 1979 subset (Table 2). Greater persuasiveness may be the primary factor in this change, but it is certainly also in accord with a presumption of improved procedures or products. Vernon (personal communication) also credits this improvement partly to the increasing willingness of medical insurance companies to pay for tinnitus maskers/instruments.

From these questionnaire data, then, there emerges a picture that, if not as optimistic as the early reports would have led us to expect, is still encouraging. Masking does contribute some relief to about one-half (56 percent) of the patients for whom it is recommended. This is not the three-fourths (72 percent) claimed at one point (Vernon and Schleuning, 1978), but it is still significant.

The right-most column in Tables 1 and 2 is presented in an attempt to give perspective to the overall value of masking as a treatment for tinnitus. For these calculations, the denominator used was the total number of questionnaires returned; that is, it is an attempt to take into account the fact that no recommendation of masking is given to many patients following examination. Thus, it can be seen that the typical patient appearing at the doors of the Oregon clinic has about a 42 percent (8 percent + 34 percent) chance of realizing either partial or total relief from his or her tinnitus. Given the apparent severity of the symptoms in the patients referred to the clinic, this is a laudable success rate.

Other investigators of tinnitus masking have reported lower success rates than the ones of the Oregon group, making those reports important to examine before reaching a conclusion about the efficacy of masking as a treatment for tinnitus.

Other Reports

The article by Roeser and Price (1980) is frequently cited as containing evidence on the efficacy of tinnitus maskers that is contrary to that of the Oregon group. As shall be seen, this conclusion is open to question. Roeser and Price studied 52 patients referred to them because of serious tinnitus. Following audiological examination, one of three recommendations

was made: a trial period with a tinnitus masker (65 percent of the patients), a trial period with a hearing aid (21 percent), or no trial period with either device (13 percent). (These percentages are revisions of those given by Roeser and Price, because they chose to include in the no recommendation category five patients who were in fact candidates for a masker unit, but who chose not to participate in a trial period after hearing a masker and judging it to be less tolerable than their tinnitus; including these cases in the no recommendation category compromises the concept of "recommendation" when the intent is to evaluate efficacy.) Unfortunately, tinnitus instruments were not included in this evaluation. Of the 23 patients who did use a tinnitus masker on a trial basis, 20 returned a questionnaire sent them at some time following the trial period (an 87 percent return rate). Roeser and Price report data on three items from their questionnaire, but unfortunately only for the 20 patients encouraged to try tinnitus maskers; no information is supplied on the 11 patients who tried hearing aids. Fifteen of the 20 respondents (75 percent) answered yes to "Does the masking unit totally cover your ringing?"; 6 of 20 (30 percent) answered yes to "Do you find the noise that the masking unit produces more tolerable than your ringing?"; and 5 of 20 (25 percent) answered yes to "Is your ringing reduced or absent when the masking unit is removed?" The last question is obviously aimed at the issue of residual inhibition and is easily interpreted. However, when reaching a conclusion about active relief, Roeser and Price choose to emphasize the second question rather than the first. Thus, they conclude that 30 percent (6/20) of the respondents are receiving active relief. This interpretation is unclear; the question most directly related to active relief would seem to be whether or not the tinnitus was "covered" by the masker, and to this question, 75 percent of the respondents answered affirmatively. The matter might have been made less ambiguous had Roeser and Price asked a version of the question asked by Schleuning et al (1980): "Are you currently using the device as a source of relief from your tinnitus?" The authors do reveal that 55 percent (11/20) of their respondents did purchase a masker following the trial period.

Thus, the conclusion reached by Roeser and Price (1980)—that the tinnitus masker is much less effective than the Oregon group indicates—is not strongly supported by the data they present. Further, the Oregon

group has criticized that study on a number of grounds, including failure to include tinnitus instruments in the evaluation and utilization of only a limited number of early models of tinnitus masker. The issue of the relative tolerability of masking noise and tinnitus—addressed by Roeser and Price's second question—is an interesting one and worthy of additional study, but adequate evaluation of the issue requires high confidence that the masking sound being used in the comparison is the optimal one.

Rose (1980) has published a brief note on his experience prescribing tinnitus maskers. Unfortunately, the presentation is too vague about important details (total sample size, severity of tinnitus, bases for recommendations, effectiveness of hearing aids, etc.) to be of much help in evaluating efficacy of masking on tinnitus, but some facts are worth noting. Of the 31 patients who participated in a 1-month trial period, 32 percent later purchased a masking unit; of these, about 38 percent were still using the device, regularly or irregularly, at the time of the follow-up interview. Just as with the Roeser and Price study, it is believable that these percentages would have been higher if more masker models or tinnitus instruments had been offered to the patients.

Pulec et al. (1978) found that about 7 percent of the 950 new patients seen by them in an 18-month period had tinnitus as a major complaint. Twenty-eight of these patients were given a thorough tinnitus evaluation, which revealed that 23 (82 percent) had tinnitus that was maskable. Several case studies are presented, but no follow-up statistics are presented to aid in evaluating long-term efficacy.

Miller (1981) indicates that a controlled study of tinnitus maskers/ instruments is under way.

Finally, a member of this working group (A. Shulman) is now engaged in a long-term study of tinnitus maskers/ instruments. Several hundred tinnitus sufferers have been seen, but to date only preliminary results have been published (CIBA Foundation, 1981:257–258). Preliminary analyses indicate that about 73 percent of those for whom a device (masker, instrument, or aid) was recommended did purchase it after a trial period (compared with the 68 percent of Schleuning et al.; Table 2 above) and that only about 11 percent of all patients were given no recommendation (compared with 26 percent in Table 2), but the follow-up data on current use and reported relief had yet to be analyzed at the time of this writing.

Conclusions

So, while some investigators feel that effectiveness of masking on tinnitus has been overestimated by the Oregon group, we find that there are procedural and instrumentation features of these studies that could easily have led to an underestimate of effectiveness, rendering this "negative evidence" less than compelling. On the other hand, essentially all of the positive evidence does come from only one group at this time, a situation that makes any cautious scientist uneasy. Some inconsistencies have surfaced in the Oregon group's statistical summaries, and some of their procedural changes have surely contributed to enhanced success figures. If we accept their data, it appears that when reasonable care is taken in the choice and fitting of an aid, masker, or instrument, about 83 percent (15 percent + 68 percent) of tinnitus sufferers who actually purchase the recommended device will appreciate partial or total relief from their symptoms (Schleuning et al., 1980; patients in 1979 subset). This corresponds to about 56 percent of those for whom a masking device of some sort is recommended and about 42 percent of all patients seen (see Table 2). Such percentages are clearly attractive given the state of affairs prior to the introduction of tinnitus maskers/ instruments. However, the reader is cautioned to draw only interim and conservative conclusions at this time, in recognition of the weaknesses in the currently available data. A conclusion that appears consonant with the available data, yet conservative, is that tinnitus maskers/ instruments appear to offer hope of at least partial relief to a substantial fraction of tinnitus sufferers. Stronger conclusions must await additional data from both the Oregon group and others.

Residual Inhibition

Recall that one of Feldmann's (1971) many interesting observations about tinnitus and masking was that, following termination of an effective masker, the tinnitus often did not immediately reappear at its premasking magnitude; frequently several seconds of silence were realized prior to the return of the tinnitus (see "Related Masking Results" in Chapter 3). Feldmann appreciated the possibility this effect offered; he explicitly mentioned the prospect of possibly "training" the underlying mechanism so as to provide the tinnitus sufferer with periods of relief.

In the early reports from the Oregon group, this (now-named) residual inhibition was given much attention, and there seems to have been great hope among some members of the group that residual inhibition would frequently prove to be protracted or trainable and thus very valuable to a large number of tinnitus sufferers. Reports of patients who showed extended periods of residual inhibition—hours, days, and even "cures"—were repeated and picked up by the press (Galton, 1979). As a consequence, unrealistic expectations were possibly created among both tinnitus sufferers and hearing professionals. This surely was unintentional, but it is believable that some patients for whom masking was reasonably effective may have forsaken its use once it was realized that—contrary to expectations—prolonged periods of residual inhibition were not being produced. There is no doubt that very long periods of relief and even "cures" have been realized by a few patients following masker use, but they are rare (Vernon and Meikle, 1981). Fortunately, the later reports have tended to play down the more sensational instances of residual inhibition and instead have emphasized its value as a predictor of success of masking as a therapy, although the goal of possibly prolonging residual inhibition to the point of making it permanent is still mentioned (Vernon et al., 1980). The techniques that are being, or have been, tried to accomplish this end have not been specified (see Vernon and Meikle, 1981).

The experience of the Oregon group is that patients who show residual inhibition are more likely to profit from masking of some sort than are patients who do not show residual inhibition. A standard procedure to measure residual inhibition is obviously desirable, and the Oregon group claims to have developed one, although the details of its development are not clear from the published reports. First, masking of the tinnitus is attempted with tones and narrowband waveforms. Then these maskers are presented for 60 seconds at a level 10 dB higher than the level that was effective in masking the tinnitus. Upon termination of the masker, the patient apparently reports periodically on the magnitude of his tinnitus. The "usual" length of the residual inhibition period is said to be 25–45 seconds following a 60-second masking interval (Vernon et al., 1977), with about 35 percent of their patients showing complete residual inhibition for at least part of this time and an additional 43 percent showing only partial residual inhibition (Vernon et al., 1980). Exactly how this information is used for

making recommendations is not clear. However, the absence of residual inhibition is not taken as definite evidence of lack of effectiveness of masking therapy (Vernon, 1981), and it does not invariably lead to the patient being placed in the no recommendation category (Vernon, personal communication).

Not only are statistics about the relationship between residual inhibition and recommendation category not available, but information about residual inhibition as a predictor of masker effectiveness is also lacking. For example, the latest report summarizing follow-up data on 598 patients of the Oregon clinic (Schleuning et al., 1980) does not present any correlations between residual inhibition as measured on the standardized procedure and later success with a tinnitus masker/instrument. It seems reasonable to expect that the magnitude and duration of residual inhibition will have at least some relation to the magnitude and duration of the masker producing it. The choices of 10 dB above masking level and 60 seconds duration are not explained in detail and may be worthy of further examination. Also of interest would be objective data on the return of tinnitus magnitude following masker termination—using (say) loudness matching of some sort instead of verbal reports.

Some aspects of residual inhibition deserve further study. It is commonly asserted that Feldmann (1971, 1981) frequently observed residual inhibition when the masker was presented to the contralateral ear, while the Oregon group never does (Vernon et al., 1977; Vernon and Schleuning, 1978; Shulman, 1981:212). The truth apparently lies somewhere in between. Vernon (personal communication) claims that Feldmann observes contralateral residual inhibition in only about 10 percent of his patients and that that fraction is in good accord with the experience of the Oregon group. Since the two groups use maskers of markedly different intensity and duration, this agreement is encouraging.

Vernon (CIBA Foundation, 1981:282; Vernon and Meikle, 1981) has noted that following a period of complete residual inhibition, some patients report that their tinnitus does not return gradually and monotonically, but rather it "bounces back" in intermittent spurts; this fact obviously has important implications for the underlying mechanisms.

Goodey (CIBA Foundation, 1981:288) has reported an effect analogous to residual inhibition following drug treatment of tinnitus. Upon withdrawal of a drug that

has been effective against their tinnitus, about half the patients find that the tinnitus never returns to its previous level.

For the sake of completeness, the experience of Penner (personal communication) with residual inhibition should be noted. Recall that she works exclusively with patients who are categorized as having sensorineural hearing loss. In this select sample she finds that, following exposure to the masker, about one-third report no change, about one-third report decreased tinnitus (residual inhibition), and about one-third report increased tinnitus. Recently, the Oregon group also mentioned seeing increased tinnitus following exposure in a substantial fraction of their patients (Vernon and Meikle, 1981). The effect has been named "residual facilitation," but nothing about its diagnostic value has been presented.

Safety of Tinnitus Maskers/Instruments

The concern most frequently expressed about tinnitus masters and instruments is that their use may be producing hearing loss, or additional hearing loss. When considering the problem of hearing loss induced by exposure to intense sound, one must attend to several features of the stimulating waveform (see Kryter et al., 1966): its spectral makeup; its overall intensity and the distribution of intensity across the frequency regions present; the duration of exposure; whether the exposure sound is continuous or interrupted; and, if the latter, the nature of the intermittency. In all applications described to date, tinnitus maskers/instruments supply continuous sound, so the contribution of intermittency to exposure-induced hearing loss is a problem that can be ignored for the time being. This leaves the issues of the spectral characteristics, intensity, and duration of the exposure, about which the following generalization is relevant: for a noise with any given spectral characteristics, the greater the intensity and/or the greater the duration of daily exposure, the greater is the risk of an exposure-induced hearing loss. The question, then, is how great is the risk of wearing a tinnitus masker/ instrument?

Spectral Characteristics

Many long-known and well-documented facts of masking lead to the belief that the

most efficient masker for a given sound—the one that will require the least intensity—will be one that is located in the same spectral region as, and has a bandwidth similar to (if a little wider than), that of the sound to be masked. It must be remembered that, for the most part, these basic data were collected on normal-hearing subjects and that Feldmann (1971), Penner (1980), Penner et al. (1981), and Vernon et al. (1980) have argued that at least some forms of tinnitus do not behave in all respects like "real" sounds when it comes to masking (see "Some Ways Tinnitus Is Not Like an External Sound" in Chapter 3). Nevertheless, explicitly stated goals of the masker/ instrument-fitting procedure are to determine and prescribe the masking waveform that best masks the tinnitus (Vernon et al., 1977; Vernon and Scheuning, 1978; Vernon and Meikle, 1981). One obvious practical reason for these goals is the belief that the narrower the bandwidth of the tinnitus masker, the less interference it will produce with the perception of important real-world sounds, such as speech. But to date, these goals have been partially frustrated by lack of availability of test equipment and of tinnitus maskers/instruments capable of generating waveforms with a wide variety of bandwidths and center frequencies. In this regard, tinnitus test equipment has improved more rapidly than have the tinnitus maskers/ instruments (Voroba, 1979b).

The spectral characteristics of several available tinnitus maskers were shown by Agnew (1979b) and Vernon and Meikle (1981); while they do offer somewhat different distributions of intensity across frequency, all are quite wideband in their outputs. This feature of present maskers/instruments is troublesome in a number of ways. Most obvious is that wide bandwidth maskers have more potential to interfere with the perception of important everyday sounds than do narrower maskers; this factor acts to hold down the intensity at which the user sets the masker/instrument and, thus, possibly diminishes its effectiveness as a tinnitus masker. Second, all things being equal, the more wideband the waveform is, the less efficient a masker it will be. This factor acts to increase the overall intensity at which the user sets the masker/ instrument, for the less efficient the masker is—again, all things being equal—the greater its intensity will have to be for a fixed level of masking. And, the greater the intensity, the greater is the concern about exposure-induced hearing loss.

Two engineering aspects to the problem of individually tailoring a masking waveform to a particular patient deserve comment. One difficulty has been in the actual delivery of high-frequency energy to the eardrum (the high-frequency response of hearing aids has been similarly limited by the lack of availability of small microphones or speakers with efficient high-frequency responses). However, recent developments promise resolution of this aspect of the problem; Killion (1981), for example, has developed behind-the-ear and in-the-ear systems having a nominal 16-kHz bandwidth. Utilization of these systems in tinnitus maskers/instruments is to be encouraged.

The second aspect of the problem of individual tailoring of tinnitus maskers involves the process of obtaining the masker waveform. Until recently, the most common procedure for obtaining a narrow band of frequencies was to begin with a relatively wideband source and then filter out all frequencies but those in the spectral region of interest. Filtering schemes have evolved over the years, but it is still the case that a filter capable of supplying a reasonably narrowband waveform at a high center frequency is much too cumbersome to incorporate into an everyday device such as a masker/instrument. And even if this were possible, in order to permit true individual tailoring of maskers, the manufacturers would have to make available either a large number of devices with different masker center frequencies or else a wide array of (say) plug-in filter modules. An obvious solution to this problem is not to generate a wideband source and then filter it to obtain a narrow band, but to generate a narrow band in the first place. With digital synthesis and manipulation of waveforms, such a possibility exists, but manufacturers of tinnitus maskers/instruments have yet to take advantage of this approach. A small, easily wearable device could be produced that is programmable by the manufacturer or the dispenser to generate a waveform with the specific spectral characteristics determined to be necessary to produce the most efficient masker of that patient's tinnitus—that is, a "master" tinnitus masker/instrument. It is likely that the manufacturers have (rightly) been cautious about developing such devices because of uncertainty about the long-term efficacy of masking for relief of tinnitus. The results reviewed here suggest that such development may no longer be premature.

In regard to the issue of tailored narrowband maskers for tinnitus, the reader is reminded of the pitch-matching data of Penner and Voroba (see "Pitch Matching" in Chapter 3) implying that tinnitus frequency may vary greatly across time. If confirmed, this finding suggests that the ideal tinnitus masker/instrument might be either narrowband but user-adjustable in frequency, or else just wideband enough to "cover" the frequency excursions of the tinnitus of the particular patient.

Manufacturers contemplating development of new maskers/instruments that capitalize on digital synthesis procedures should be aware of recent evidence that indicts waveforms with very steep spectral skirts (McFadden and Pasanen, 1980; McFadden and Plattsmier, 1981a). Even if subsequent reports do not confirm that such waveforms are dangerous to the physiological mechanisms underlying frequency resolution, they should still be avoided because of their demonstrated ability to induce a short-term tinnitus (see "Possible Experimental Models of Tinnitus" in Chapter 2), whose cumulative and long-term characteristics are unknown. It would be ironic indeed if a new generation of tinnitus maskers/instruments were themselves responsible for inducing additional tinnitus. The evidence is still scanty, but it appears that narrowband waveforms having attenuation rates of about -70 to about -200 dB per octave at their "edges" are reasonably safe, but that slopes of -400 dB per octave (McFadden and Pasanen, 1980) and -600 dB per octave (Lummis and Guttman, 1972) should be avoided.

Intensity of Tinnitus Maskers/Instruments and Duration of Use

On the basis of survey and experimental research on the permanent hearing loss caused by sounds of various intensities, durations, etc., so-called damage/risk criteria (DRC) have been established (Kryter et al., 1966). For a fixed level of risk (proportion of population manifesting losses of particular magnitude at particular frequencies), the DRC specify the combination of sound intensity (measured on the "A-weighted" scale) and duration of exposure that must not be exceeded during an 8-hour work day in order to satisfy that risk criterion. For many occupational purposes, the U.S. government has designated 90 dBA and lower as "safe" for a working lifetime of daily exposures (Occupational Safety and Health Administration, 1974). If the continuous noise exposure is 95 dBA, the maximum allowable daily exposure is limited to 4 hours per

day, and, if it is 100, 105, 110, or 115 dBA, the maximum allowable daily exposures are 2, 1, 0.5, and 0.25 hours, respectively. Ward et al. (1976), to take one example, seem to view these as acceptably safe values, but some investigators feel that a more conservative definition of "safe"—a "lower fence"—is necessary to adequately protect workers. For example, Passchier-Vermeer (1974) argues for 80 dBA for steady-state noises and daily 8-hour exposures (also see Burns and Robinson, 1970; Berger et al., 1978). Obviously, if the exposures are for periods greater than 8 hours or for more than 5 days per week, the specified intensities would have to be correspondingly decreased in order to maintain the same average loss of hearing. This is an important point, for apparently many users of tinnitus maskers/instruments wear them throughout their waking hours (Vernon and Meikle, 1981), and some even sleep with them on. (Contrary to general expectation, perhaps, is the fact that only about one-half of the people with serious tinnitus have difficulty falling asleep, and this seems to bear no simple relationship to the matched intensity of the tinnitus [see Vernon, 1977].) Thus, it is not possible to reach a decision about the safety of tinnitus maskers/instruments by simply comparing their maximum overall intensities with the standard damage/risk criteria—longer daily exposures and more successive days per week are involved.

A report issued by the U.S. Environmental Protection Agency (1974) includes procedures for translating the 8-hour occupational standards into corresponding levels for 24-hour exposures. As noted above, an important consideration is the intermittency of the exposure. Let us first consider an extreme, hypothetical case—a patient who uses a tinnitus masker/instrument continuously at the same level throughout each 24-hour period. For this no-intermittency situation, the translation procedure yields a value of 66.4 dB as the maximum permissible (in order to protect virtually every person from more than 5 dB of permanent hearing loss at 4000 Hz after 40 years of exposure). If the hypothetical patient did not keep the masker adjusted to the same level throughout the 24-hour period, but occasionally reduced its level or turned it off--that is, introduced intermittency—then the maximum permissible level could be greater than 66.4 dB. To take one example, assume that for at least 10 percent of each 1-hour period the patient sets the masker level to 65 dBA or less; the maximum permissible level now rises to 71.4 dB. The maximum permissible level rises to 73 dB if the

masker is continuous for only 8 consecutive hours per day and is 60 dB or less for the remaining 16 hours. Various other examples are possible but are not necessary for current purposes. We see that somewhere in the vicinity of 66 to 73 dBA would be the maximum allowable level of a tinnitus masker that was worn regularly if existing damage/risk criteria were the relevant standard for regulation. It is to this range of levels, then, that one might want to compare the levels typically experienced by wearers of tinnitus maskers/instruments. Unfortunately, the comparison is not easily made, given the available data. (It must be emphasized that these EPA translations are viewed as unnecessarily conservative by many experts who believe that "effective quiet" may lie at or somewhere above 75 dB. Without getting into this controversy, it can be noted that, even if the safe level for continuous 24-hour exposure were to rise by, say, 15 dB from the 66–73-dBA range cited, none of the following arguments would be qualitatively altered.)

When dealing with pathological ears, the standard way to express sound level is relative to the patient's absolute sensitivity (absolute threshold) for that sound in that ear; sound levels so referenced are designated as decibels sensation level (dB SL). This usage is sensible for many purposes, for it often better communicates a sense of stimulus strength, or perceptual magnitude, in pathological ears than does a unit with a fixed reference such as decibels sound-pressure level (dB SPL). The problem this raises for us, however, is that DRC, effective quiet, etc., traditionally are specified in units analogous to dB SPL, while the intensities necessary to mask a tinnitus are typically given in dB SL without the necessary information to make the transformation to dB SPL. Thus, while we know from numerous reports that the measured loudness of tinnitus is typically low—only rarely is it matched to sounds greater than about 20 dB SL (see "Magnitude of the Tinnitus" in Chapter 3)—we do not know the overall sound-pressure level typically necessary to mask it. About all we presently have to go on when evaluating the safety of tinnitus maskers/instruments is the manufacturers' specifications of maximum level.

The manufacturers' specifications sheets for many models of tinnitus masker state a minimum overall output level of 40–45 dB SPL (note, not "A-weighted") and a maximum overall output level of between 85 and 95 dB SPL, and some models specify maxima in the range of 105–110 dB SPL. These measurements are sometimes made using a standard

2-cc coupler (e.g., HA-1), but they are frequently also made using a Zwislocki coupler (with the dB 110 adapter), which better reflects the high-frequency characteristics of the typical ear canal. This latter procedure is to be preferred, particularly when dealing with maskers/instruments specifically designed to mask high-frequency tinnitus, for the standard coupler underestimates the level of high frequencies.

Therefore, essentially every masker/instrument now available is capable of producing sound levels in excess of the 66–73 dBA that is derived as safe from existing standards, and some are capable of producing levels far in excess of that value. (Recall that current OSHA regulations would allow only about 0.5 hour per day per 5-day week of the 110 dB that some maskers/instruments claim to be capable of.) Thus, the potential for damage, or additional damage, to hearing clearly exists. At this time it is impossible to estimate how much risk is involved—or how much damage has already been done—because there are no systematic data yet available on the levels at which satisfied, chronic users of maskers/instruments routinely set their devices, on the temporal patterns of use, or on changes in hearing sensitivity after prolonged use of these devices. It may be that the typical user sets the masker/instrument to a level below the nominal 66–73 dBA or sets it to different values throughout the day, thereby reducing the overall risk. Data on these issues are important to acquire, and, given the large and growing number of users of maskers/instruments, they should not be difficult to obtain. People with bilateral hearing loss but monaural tinnitus would be particularly interesting, since the nontinnitus ear would serve as a within-subjects control for additional loss.

It should be noted that, if manufacturers do begin producing tinnitus maskers/instruments capable of generating narrowband waveforms, it will raise a further complication to the use of existing DRC and other guidelines as standards for regulating maskers/instruments. Most of what is known about exposure-induced hearing loss comes from exposures to relatively broadband waveforms, and it is that knowledge upon which DRC have been based. However, concentrating all the energy in a relatively narrow spectral region can create a potentially more damaging waveform than one with the same overall level but greater bandwidth. The magnitude of the additional risk is unknown, however. For such noises EPA guidelines in dBA are not appropriate, and criteria for octave or one-third

octave bands such as those described by Kryter et al. (1966) should be used. To the extent that tinnitus behaves like an external sound when it comes to masking (cf. Feldmann, 1971; Vernon et al., 1980; Penner et al., 1981), the narrowband masker is to be preferred, for the overall level required to achieve masking of the tinnitus with a narrowband waveform should be lower than that required with a wide one—with external sounds it is the spectrum level that matters for masking, not the overall level. Even when the tinnitus does not act like an external signal, the narrowband masker has a virtue: it puts a smaller segment of the basilar membrane at risk of damage than does a wider-band masker. While there is some knowledge about the relative dangers of noise bands of different center frequency (Kryter et al., 1966), almost nothing is known about frequency regions above about 4000 Hz, so, if narrowband tinnitus maskers are developed for use at very high frequencies, existing DRC will be of questionable applicability.

Damage/Risk Criteria and Tinnitus Maskers/Instruments

Much of the above discussion may be irrelevant. It is extremely important to appreciate that compliance or noncompliance with current or future DRC may not be an appropriate basis on which to judge or to regulate tinnitus maskers/instruments. For many people, tinnitus is a severe, debilitating affliction, and it could easily be argued that relief from this affliction is, in many cases, worth the risk, or even the inevitability, of some hearing loss. Many commonly used drugs carry risks (or inevitabilities) of negative effects of various sorts, and even hearing aids themselves are capable of producing sound levels far in excess of both the 66–73-dBA and the 90-dBA values cited, depending upon the ambient levels in which the user lives. The implicit risks attendant to the use of such drugs, and of hearing aids, are generally judged by medical professionals, laymen, and users to be worth the benefits accrued. Tinnitus maskers/instruments appear to be indistinguishable from common drugs and hearing aids in this regard. That is, it can be argued that, if tinnitus sufferers are made aware of the potential risks to hearing involved in using a tinnitus masker/instrument at high intensities and/or for prolonged periods of time, and they still choose to wear the device, perhaps that decision should be theirs to make. It is clear that in the past "the consent" of masker/instrument

users has not been as "informed" as it might have been, but this is true for users of hearing aids as well, and the solution appears simple. The practitioner, the dispenser, or the manufacturer could supply a brief written document that explains the situation and emphasizes the virtues of always using as weak a masker as possible and of introducing as much intermittency as possible (advice that seems equally desirable for users of hearing aids).

In this regard, the outcome of Penner et al. (1981) should be recalled; for some forms of tinnitus, continuous maskers lose their effectiveness relatively rapidly, and increasingly higher sound levels are necessary to accomplish the same result. Regular intermittency in the use of the tinnitus masker/instrument worn by such people would appear to be highly desirable. The ability to use lower masker levels ought to make the masker/instrument less obtrusive for the wearer, and it certainly ought to reduce the risk of masker-induced hearing loss. At this time no systematic studies have been done on the periodicity and duration of the intermittency necessary to control masker level in these patients.

Related to this matter of intermittency is another point. It might be possible to capitalize on the residual inhibition effect to gain greater safety and reduced inconvenience in tinnitus maskers/instruments. A member of the Working Group (L. Kaufman) realized that for some people it might be possible to periodically interrupt or to amplitude-modulate (AM) the masker wave-form at a rate that would allow residual inhibition to tide the person over until the next masker interval (or half-cycle of high intensity). The basic idea, of course, is to have the masker present only long enough and at adequate intensity to institute a subsequent period of residual inhibition. Interruption or AM rates of about two cycles per minute ought to be about right, according to the residual inhibition evidence reported by the Oregon group. The advantages to the user would be that: (1) about half the time the masker would be at a low level or absent and thus should be less of an impediment to the processing of real-world sounds such as speech, and (2) as a consequence, the average daily noise exposure would be reduced. The variants of the idea that come to mind appear to require more elaborate hardware than does simple AM, but obviously other temporal sequences of masker level would be possible with microprocessor-based maskers/instruments. Presumably, relatively long rise-decay times would always be important. A possible problem is that a slowly varying

or regularly intermittent masker might be more annoying than a steady one, but the idea is definitely worth trying with some patients for whom residual inhibition is strong.

Hearing professionals have had a long-standing concern about the possibility that hearing aids induce additional hearing loss (sometimes known as induced deterioration or overamplification). Berry (1939) and Holmgren (1939) were among the first to raise the issue, which has yet to be satisfactorily resolved. To cite just a few of the published papers on the topic, Kinney (1961), Macrae and Farrant (1965), and Jerger and Lewis (1975) all concluded that there is evidence for temporary or permanent deterioration of hearing induced by hearing aids set for high amplification, while Naunton (1967), Bellefleur and Van Dyke (1968), Derbyshire (1976), and Titche et al. (1977) all concluded the opposite. A reading of this literature makes it believable that the disagreement stems in part from lack of adequate control of such important factors as actual level of amplification of the aid, length of its use, type of impairment, measurement procedures, and ambient environmental levels. In a recent article, Humes and Bess (1981) argue that—even using very conservative estimates of such critical factors as exposure level and duration—most hearing-aid users probably are at risk of substantial additional hearing loss from daily use of their aids. Their calculations are worthy of careful study by anyone interested in this important problem. For our purposes here, it must be concluded that, while the safety aspects of tinnitus maskers/instruments would appear to have much in common with those of hearing aids, too little is known about the latter to help us reach a decision about the former.

5

Standardizing Procedures

A desirable goal is widespread adoption of a standard procedure for measuring and evaluating tinnitus, and it would be particularly nice to be able to put forth that standard procedure here. Unfortunately, such action is premature. Too little is known about crucial aspects of tinnitus behavior to be able to specify the best procedures for assessing it. Instead, we can offer only some suggestions about what a standard procedure of the future might include.

MEDICAL EXAMINATION

As has been noted several times in this report, tinnitus is itself not a discrete disease entity; rather, it is a symptom that is produced by many known and unknown causes and diseases. For this reason, it is wrong to begin by treating the symptom, whether with drugs, tinnitus maskers/instruments, or whatever. The first step should always be to try to diagnose and treat the underlying cause. Of course, some causes of tinnitus are themselves not treatable, given current knowledge, and palliatives for the tinnitus are the only recourse. This fact does not exempt the clinician from the obligation to try to establish the underlying disorder. This section is intended only to indicate the wide variety of possible causes to be considered by the responsible physician—and should not be taken as a complete guide to diagnosis or treatment.

Ever since the introduction of tinnitus maskers/instruments as a palliative for problem tinnitus there has been concern in some quarters that eliminating this symptom may be "covering up" an important sign of a serious

medical problem—for example, an eighth-nerve tumor. This concern may be exaggerated, because no present treatment of tinnitus eliminates it permanently; the concern also appears to be based on logical considerations, not on widespread experience or even anecdotes about such occurrences (see, for example, Miller, 1981). However, even if documented instances of a serious illness being obscured by effective treatment of tinnitus are rare, the point is well taken. Just as patients complaining of tinnitus deserve complete audiological examinations, they also deserve medical examinations that emphasize those general bodily problems that are known to be associated with tinnitus. Of course, all of the general physiological abnormalities that may produce tinnitus have yet to be identified, but we are able to indicate certain items that deserve attention.

1. Blood pressure. The commonly observed variations in tinnitus with stress and the blood pressure changes known to accompany stress imply that altered microcirculation in the cochlea may be the basis for some tinnitus. Treating the tinnitus with a masker/instrument or other agent may be less direct than treating the blood pressure problem itself.
2. Kidney function. It is commonly asserted that the ear is like the kidney in that both structures are concerned with maintaining normal electrolyte concentration gradients. This similarity between the two structures causes them to react similarly to certain agents. Thus, verification of normal kidney function seems advisable in sufferers from severe tinnitus.
3. Drugs. The medical examination should establish what drugs—both physician-prescribed and self-prescribed—the patient takes, and on what schedule(s). Aspirin and other salicylate-bearing agents are obvious ones to eliminate if possible, but other drugs, or combinations of drugs, may be responsible for a particular individual's tinnitus, and attempts to relate the onset of tinnitus episodes to patterns of drug usage may be worthwhile. If there is a suspicion of the tinnitus being drug induced, a further step would be to prescribe substitute drugs for short periods of time and observe any effects on the tinnitus.
4. Diet and allergy. These are probably very infrequent causes of tinnitus, but verification of a balanced diet, and suggestion to the patient to be alert to possible temporal associations between tinnitus episodes and exposures to common allergy-producing agents, cannot hurt.

5. Other conditions. Goodey (1981) and Schleuning (1981) argue that a general medical examination for a tinnitus sufferer should also include tests for infections of the head, neck, and teeth; hypothyroidism; hyperthyroidism; early diabetes; hypoglycemia; disturbance of serum lipids; high blood viscosity; autoimmune disease; vasospastic disease; meningitis; multiple sclerosis; and migraine.

AUDIOLOGICAL EXAMINATION

Many hearing specialists believe that a complete audiological examination should precede the prescribing of any form of treatment for tinnitus, but this is not universal. Experts also differ in their strength of feeling about the necessity for a complete audiological examination when the primary symptom is tinnitus (Shulman, 1981:205-207). A complete examination would include, but not necessarily be limited to, air-and bone-conducted pure-tone threshold measures, speech threshold and discrimination tests, tympanometry and reflex testing, and site-of-lesion tests if indicated.

Following these standard tests, the tinnitus itself should be documented. The measures made should include, but not necessarily be limited to, pitch and loudness matches, a test of maskability, and a test for residual inhibition. At present there exist numerous procedures for making these various measures, and, unfortunately, they do not always produce the same outcome. It may be that particular methods will eventually prove more reliable and valid than others for particular forms of tinnitus, but at the moment it is premature to specify particular psychophysical procedures for the various measures or for different forms of tinnitus. Nevertheless, it is possible to make certain general recommendations about measures and procedures.

1. Some tinnitus is said to be "masked" by sounds from numerous locations across the spectrum and some by both ipsilateral and contralateral waveforms. The use of several tonal or narrowband maskers, both ipsilaterally and contralaterally, would thus seem worthwhile, for it may be that some tinnitus sufferers would be better served by a masker waveform different from that most effective against an external sound of the same frequency and band width.

2. Similarly, residual inhibition should probably be studied with several maskers, both ipsilaterally and contralaterally.
3. It follows from the previous two recommendations that testing should not be done using loudspeakers to present the stimuli, but with headphones, and preferably with headphones having a good seal, good high-frequency response, and high interaural attenuation.
4. It is important that pitch matches be verified with tests at octave frequencies above and below the matched frequency, and since pitch matches are so difficult for unpracticed subjects, it would be advisable to have a repeat determination either later in the examination session or at another time.
5. Because tinnitus is notorious for its variability, both within and across days, it is important that the current status of the tinnitus be established at the time of the tinnitus examination. Specifically, the patient should be asked whether the tinnitus being experienced is typical, and, if not, how it differs and how greatly it differs from the typical problem day. If the tinnitus of the current day is judged to be sufficiently atypical, the patient should probably be asked to return during a more typical episode; otherwise, the measurements made could be very misleading to the clinician attempting to prescribe for the problem. In today's busy clinics, such a procedure may be very inconvenient both for the patient and the clinician, but the inconvenience and delay ought to be offset in the long run by better diagnosis and treatment.
6. A procedural suggestion made by the Oregon group appears well advised: measures of tinnitus magnitude should be made using procedures that involve only successive increases in intensity, for the premature introduction of high intensities could produce a residual inhibition that would invalidate later measures. The suggestion applies, of course, to measures of tinnitus other than magnitude measures; in general, stimuli of long duration and relatively high intensity should be avoided throughout the tinnitus examination.
7. Whenever possible, it would be advisable to report loudness matches in both SL and SPL units.
8. Attempts to determine the monaural or binaural nature of the tinnitus appear worthwhile. As noted in the section "Is the Tinnitus Monaural or Binaural?" in Chapter 3, a binaural but asymmetric tinnitus can appear to the patient as having a monaural origin, but such a

tinnitus will in many instances be much more resistant to a (monaural) masker/instrument than will a truly monaural tinnitus. One procedure for attempting to determine the monaural/binaural nature of the tinnitus has already been described (see "Is the Tinnitus Monaural or Binaural?" in Chapter 3), and others could surely be developed. The examiner must always remember that a person's tinnitus may be composed of many different spectral components (see "Quality of the Tinnitus" in Chapter 3) and that some of these may be monaural, some binaural and approximately equal in magnitude, and some binaural and quite discrepant in magnitude. Ideally, the monaural or binaural nature of each of the components would be determined along with their relative annoyances, so that an optimal masker program could be chosen.

Additional measures and procedures that would be desirable will undoubtedly become clear as our knowledge of the origins of tinnitus increases.

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Index

A

Acoustic neuroma, see Tumors of the eighth nerve
Acoustic trauma, 9, 13-14
Acupuncture, see Treatment of tinnitus, acupuncture
Aging and tinnitus, 8
Air pressure changes, see Treatment of tinnitus, manipulation of air pressure
Alcohol, 63, 78
Allergies, see Treatment of tinnitus, allergies
Alternate binaural loudness balance (ABLB), 44, 47
American Tinnitus Association, 94
Aminoglycosides, 79
Anemia, 11
Anesthetics, 11
Animal models of tinnitus, 16, 17
Annoyance of tinnitus, see Tinnitus, annoyance of
Arlidin, 78
Aspirin 16, 17, 26, 60, 61, 118
Audiogram, microstructure, see Microstructure of audiogram

B

Beats with tinnitus, 51-52
Betahistine hydrochloride (Serc), 29
Biofeedback, see Treatment of tinnitus, biofeedback
Blow to the head, 11, 14
Brain-stem-evoked response (BSER), 26, 54
Bupivacaine, 78

C

Caffeine, 63
Carbamazepine 72-74, 75, 77, 79
Causes of tinnitus, see Etiology of tinnitus
Central masking, 38, 54
Children and tinnitus, 8, 24
Chlortrimeton, 78
cis-Platinum, 63
Cocaine, 63
Cochlear echo, see Otoacoustic emissions
Cochlear emissions, see Otoacoustic emissions
Cochlear implants, 85, 86
Cross-modality matching, 44

D

Depakene, see Sodium valproate
Diabetes, 119
Diazepam, see Volium
Diet, see Treatment of tinnitus, diet
Dilantin, see Phenytoin sodium
Diplacusis, 51
Drug overdoses, 11

Drugs causing tinnitus, 59-63
also see by name
Drug treatment of tinnitus, see Treatment of tinnitus, drugs

E

Ear wax, 11
Efferent system, 15, 25, 38
Eighth-nerve fibers, see Primary auditory fibers
Electrical stimulation, see Treatment of tinnitus, electrical stimulation
Electroconvulsive therapy (ECT), 87
Emissions, see Otoacoustic emissions
Endolymphatic hydrops, 28-30, 68
Epilim, see Sodium valproate
Ergenyl, see Sodium valproate
Ethylmorphine hydrochloride, 79
Etiology of tinnitus, 10-11
Eustachian tube, 12, 81
Evoked cochlear mechanical response (ECMR), 18
also see Otoacoustic emissions

G

Glycerin 29

H

Hair cells, 17-18, 30, 60
Hawthorne effect, 65, 79
Hearing aids, safety of, 114, 116
Heavy metals, 63
Heparin, 79
Hypertension, 11
Hyperthyroidism, 119
Hypnosis, see Treatment of tinnitus, hypnotherapy
Hypoglycemia, 119
Hypothyroidism, 11, 119

K

Kanamycin, 16
Kidney function, 118

L

Lidocaine, 3, 26, 67-75, 79
Lignocaine, see Lidocaine
Lorcainide, 78
Loudness-matching procedures, 42-45, 46, 119
high reliability of, 44

M

Magnitude of tinnitus, see Tinnitus, magnitude of
Marijuana, 63
Masking-level difference (MLD), 39
Masking of tinnitus, 3, 36-45, 89-116
"adaptation" and, 39, 40, 53, 115
contralateral, 37-38, 41, 52-54
"covering up" tumors, 13, 117-118
different from external sounds, 37, 40, 47, 108-110
drugs and, 54-81
efficacy, 3, 4
hearing aids, 90
interstation radio noise, 41, 89, 90, 93
Meniere's Disease, 27-31
monaural versus binaural, 41
safety, 4, 5
success estimates, 3-4
Measurement of tinnitus, 32-54
Mechanisms of tinnitus, 14-20, 30, 52, 53
also see Etiology of tinnitus
Melanin connection, 62, 71-72, 80
Meniere's Disease, 9, 11, 18, 27-31, 37, 66, 67, 72, 81, 88
Meningitis, 119
Mexilitine, 79
Microstructure of audiogram, 22
Migraine, 11, 119
Multiple sclerosis, 11, 119
Music synthesizer, 34
Mysoline, see Primidone

N

Naftidrofuryl, 78
Niacin, 66
Nicotinic acid, 66
Noise exposure, see Acoustic trauma
Nortriptyline, 78

O

Objective tinnitus, 1, 5-7, 18, 19
  in nonhuman species, 6, 21
  unheard by owner, 6, 18-23
Octave errors, 35, 37, 120
Oral contraceptives, 63
Otoacoustic emissions (OAEs), 6, 12, 18-23, 26, 28, 51, 54, 61, 88 , 89
  cochlear echo, 18, 21, 22, 26
  in nonhuman species, 21
  Meniere's Disease and, 28
  physical characteristics, 19-21
  spontaneous OAEs, 12, 18-19, 26, 51, 54, 61, 88, 89
  also see Objective tinnitus
Otosclerosis, 8, 9, 11, 12, 37, 88
Otospongiosis, 76

P

Palatal myoclonus, 12, 74
Personality and tinnitus, 55-57
Phenytoin sodium, 75-76
Pitch of tinnitus, see Tinnitus, spectral location of
Pitch-matching procedure, 35-37, 39, 91, 120
Pregnancy, 11
Presbycusis, 9, 11, 13, 14, 37, 68, 83
Prevalence of tinnitus, 2, 7-9, 13, 14
  acoustic trauma and presbycusis, 13, 14
  eighth-nerve tumor, 12-13, 57-58
  in children, 8, 24
  in hearing-impaired, 9
  monaural versus binaural, 9
Primary auditory fibers, 30, 40, 60, 70
  kanamycin and spontaneous rates, 15-17
Primidone, 76
Procaine hydrochloride, 29
Psychological problems and tinnitus, see Treatment of tinnitus, psychological
Psychophysical tuning curves for tinnitus, 38-39

Q

Quality of tinnitus, see Tinnitus, quality of
Quinine 62

R

Racial differences, 27, 71
  also see Melanin connection
Reaction time, 44
Recruitment, 27, 43, 44, 46, 47
Residual facilitation, 107
Residual inhibition, 20-21, 38, 40, 43, 54, 61, 85, 87, 93, 94, 98 , 102, 104-107, 115, 119, 120,
  contralateral effects, 106
  drugs and, 106-107
  also see Residual facilitation

S

Safety of tinnitus maskers/ instruments, see Tinnitus, maskers/instruments, safety of
Salicylate, see Aspirin
Sensation level (SL)
  definition, 40, 46
  versus sound-pressure level (SPL), 44-46, 112, 120
Sleep interference, see Tinnitus, interference with sleep
Sodium amylobarbitone, 3, 77-78, 79
Sodium fluoride, 76
Sodium valproate, 77

Spectral locus of tinnitus, see Tinnitus, spectral location of
Stereocilia, 17-18, 30
Stress, 48, 56, 82, 118
Surgery for tinnitus, see Treatment of tinnitus, surgery

T

Temporary threshold shift (TTS), 20, 25, 59, 62, 66, 71
Tegretol, see Carbamazepine
Tinnitus
  annoyance of, 40, 45-48, 69, 90
  as a symptom, 2, 56, 117
  definition, 1, 5-7, 19
  different from external sound, 51, 52-54, 108
  "edge effect," 16, 17
  experimental models, 24-26
  hearing loss and, 23, 24
  induced by noise exposure, 16-18, 25, 26, 29
  interference with sleep, 45-46, 83, 84, 89-90, 111
  magnitude of, 41-45, 67-69, 73-75, 77, 83, 85, 112, 120
  monaural versus binaural, 27, 26, 34, 39, 42, 48-50, 70, 120, 121
  objective/subjective issue, 6, 7, 18-23
  perceptual location, 50
  quality of, 32-34
  spectral location of 28, 35-41
  variability in, 32, 36, 39, 91, 110, 120
Tinnitus maskers/instruments, 3, 4, 31, 34, 36, 38, 48, 50, 89-116 , 117, 121
  damage/risk criteria (DRC) and, 4, 5, 110
  efficacy, 92-104
  levels of, 4, 5, 111-114
  masker waveforms, 91, 108-110
  "master" units, 109
  production of hearing loss by, 40, 107, 110-116
  regulation of production of hearing loss by, 5
  safety of, 107-116
  sound-pressure levels of, 4, 5, 111-113
  success estimates, 95-104
Tobacco, 63
Tocainide hydrochloride, 74-75
Tocard, see Tocainide hydrochloride
Treatment of tinnitus, 2
  acupuncture, 84
  allergies, 81-82, 118
  audiological examination, 119
  biofeedback, 57, 82-83
  diet 81-82, 118
  drugs, 3, 54, 59-81, 118, also see by name
  electrical stimulation, 54, 85-87
  exposure to intense sound, 58-59
  hypnotherapy, 83-84
  manipulation of air pressure, 54, 87-89
  medical examination, 117-119
  palliative versus treatment, 55, 117
  psychological, 55, 56
  reassurance, 55
  surgery, 13, 57-58
Tumors of the eighth nerve, 12-13, 57-58

U

University of Oregon tinnitus clinic, 3, 4, 30, 93-107

V

Valium, 79
Vestibular schwannoma, see Tumors of the eighth nerve
Vitamin A, 66

X

Xylocaine, see Lidocaine