MIGRAINE CAUSATION: THEORY AND RESEARCH

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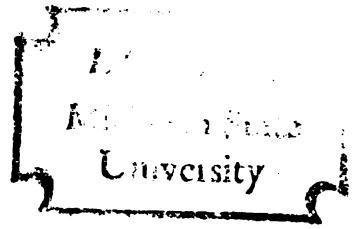

ABSTRACT

MIGRAINE CAUSATION: THEORY AND RESEARCH

By

David Morris Schnarch

Since the early writings of Freud, migraine has commonly been accepted in clinical practice as a psychosomatic disorder. Yet, the transition between psyche and soma has largely remained a mystery, although psychoanalytically-oriented inferences about 'repression' abound. Moreover, many laymen and mental health professionals misdiagnose any severe headache as 'migraine.' It was the purpose of the present study to focus attention on these areas of professional interest by presenting a critical analysis of the available literature on migraine theory and research.

In the first chapter the symptomatology of migraine, the physiological events which underlie the attack, and the basis for differential diagnosis from other headache phenomena were presented. For example, migraine is caused by constriction and dilation of the walls of the cranial arteries, and headpain is often unilateral (hemicranial) at onset. Tension headache is caused by hypertonia

of the striated muscles of the face and neck, and is always bilateral. The role of genetic involvement in migraine causation was also considered.

Several different explanations of the trigger for migraine attacks were considered. Psychologically oriented theories (especially psychoanalysis) suggest that susceptibility to migraine results from a long term tendency to translate hostile impulses into self-punishment, or to react to hostility by unconsciously withdrawing from personal responsibilities and interpersonal confrontations. Attacks supposedly occur when the person has to repress feelings of hostility which are generally unacceptable to him. Constitutional theories suggest that any strong affective response or physiological change that evokes heightened sympathetic nervous system activity (sufficient to significantly change serotonin levels) should precipitate an attack. Migraine susceptibility develops from a genetically transmitted hypersensitivity of the cranial arteries to fluctuations in serotonin level in the blood stream.

A novel suggestion of how a genetically transmitted migraine-disposed physiology might account for the personality traits that psychoanalysts often report for their migrainous clients was offered. It was suggested that the partial maternal deprivation experienced by children of migrainous mothers was formative in the later personality

development of the child. Moreover, it was suggested that this 'migrainous personality' may develop in people who had a migrainous mother, but who do not have migraine attacks themselves.

The research literature on migraine was critically reviewed: i.e., incidence and duration of attacks, age of onset, sex differences, E.E.G. abnormalities, relation to epilepsy, stress reactions, conditioning, as well as the personality and case history studies.

Overall, the research on migraine was notable in its inability to critically evaluate any of the conceptual models of the trigger for the attack. Personality and case studies were inconclusive due to failure to include control groups in their design. Moreover, since authors often did not distinguish between their empirical observations and theoretical conclusions, it is likely that their conceptual biases determined which portion of a case history would be considered 'significant' and which portion would be ignored. The inconclusiveness and outright conflicts between reported results also made it difficult to evaluate the different conceptual models. This was particularly true in regard to the reports of psychosexual development and sexual functioning from the personality and case studies.

In areas where research reports were highly consistent, such as duration and frequency of attacks, this data offered little

discrimination between theoretical models: all models were consistent with the reported data.

It was concluded that currently available migraine research does not permit evaluation of the various theoretical models of the trigger for migraine attacks with any degree of finality. Several research designs which would discriminate among the theories was were presented.

MIGRAINE CAUSATION: THEORY AND RESEARCH

By

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DEDICATION

In memory of Bill Kell, who was my sponsor during the days when I was striving to become a therapist and an adult, and who served as chairman of this thesis until the time of his death.

He knew a little about migraine.

He taught me the tools of my trade.

He taught me a lot about life.

ACKNOWLEDGMENTS

In the process of devoting five years to completing a Master's Thesis, I have accrued many debts of gratitude. I am particularly grateful to Dr. Jack Hunter, who stood behind me thru the last three of the four years I devoted to the first thesis I wrote. Without his emotional support and considerable research proficiency, I would have come away from the experience convinced of my own inadequacy. Instead, I have learned something about quality research, and also that the emotional prices involved in making 'expedient' and 'pragmatic' concessions often far exceed the effort needed to stand behind my own convictions. In addition, Jack served as chairman of this current thesis since its formative stages. The reader will be unable to fully appreciate how drastically my thoughts about migraine have changed and clarified thru the massive investment in time and energy that Jack has offered to this second thesis topic.

I also wish to thank Dr. Al Rabin and Dr. Don Grummon, who served as committee members for this thesis. In particular, Dr. Rabin's comments on the effects of maternal deprivation helped to stimulate and sharpen some of the ideas that appear in Chapter V.

There are considerable emotional costs involved in receiving one Master's degree for the price of two Master's theses, and I have been fortunate to have friends who were willing to bear with me while I was paying them. Dr. Mike Barnat spent several long nights helping me reword the first thesis, as well as several long nights commiserating with me when I decided to put it aside.

It is hard to properly acknowledge people like Barry and Sara Lester, who were willing to live with me during all the craziness. Many nights we sat around the dinner table drinking toasts of water to a thesis that I thought would never be finished.

For my wife Neil, this whole experience may have been a trial upon her, but not anything out of the ordinary. She has been willing and able to stick out some tests to our friendship during the last 10 years that I look back upon in amazement. The experience of being with Barry, Sara, and Neil, has taught me that other people can live with me. That Neil still prefers to causes me to look at her in wonderment.

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INTRODUCTION

There are several factors that make the study of migraine worthwhile. First, there is the humanitarian aspect. By one estimate (Moench, 1951) two to eight million people suffer the excruciating pain and bizarre symptomatology of migraine attacks. Moreover, it is common for migraine sufferers to experience attacks for periods over 10 years, and it is not uncommon to have attacks for over 30 years (Furmanski, 1952). Many studies have found Ss who have attacks daily.

Secondly, there is the economic aspect. Migraine is sufficiently severe to cause all ongoing activity of the individual to stop. This creates difficulty in completing work and loss of income to the individual. Likewise, migraine reduces the productivity of industrial labor forces, particularly of administrators and other individuals in positions of responsibility and authority.

Third, the treatment of migraine has not been noticeably successful. Chemotherapy has been somewhat helpful, especially where ergotamine tartrate has been used (Dalessio, 1970). But chemotherapy only offers management of current attacks and provides little in the prevention of recurrence. Psychoanalysis has been used as a

prophylaxsis, but it has produced only limited success (Mitchell & Mitchell, 1971) and is beyond the financial and intellectual grasp of a large segment of the population. New treatment modalities may evolve from critical examination of our theories of migraine etiology.

Fourth, migraine is of intellectual and professional interest to those individuals who explore the interaction of mind and body. Since the early writing of Freud (1915) migraine has commonly been accepted in clinical practice as a psychosomatic disorder. Yet, the transition between psyche and soma has largely remained a mystery. A better understanding of the "how" and "why" of migraine could add much to the field of psychosomatic medicine.

Fifth, and finally, it is hoped that the current study of migraine will bring some much needed clarity to mental health professionals in understanding the symptomatology of migrainous clients. It is all too easy to construct psychodynamic interpretations for symptoms actually arising from physiological origins. Likewise, it is possible to accept behaviors as being physiologically determined simply from lack of familiarity with biological data.

Many laymen and therapists are in the habit of labeling any severe headache a 'migraine.' It is important for the therapist to be able to ask knowledgeable questions to differentiate between tension headache, migraine headache, and brain tumor. Clearly the failure to

do so can bring more lethal results than simply the premature termination of therapy because the client shows no improvement.

It is the purpose of the present study to explore and examine the available literature on migraine theory and research. In the next chapter, the symptomatology of migraine and the basis for differential diagnosis from other headache phenomena, the physiological events which underlie the migraine attack, and the role of genetic involvement will be considered. In the chapters that follow, various attempts to specify the 'trigger' for migraine attacks will be presented. This will include both psychological and constitutional models of causation. In addition, an attempt to outline how physiology may determine the personality characteristics of migrainous individuals is presented in Chapter IV.

Following the presentation of theoretical considerations of migraine causation, a critical review of migraine research is presented in Chapter V. An evaluation of previously presented migraine theories, in light of the research evidence, will also be offered at this time. Finally, some brief conclusions and implications for future research appear in Chapter VI.

CHAPTER I

THE MIGRAINE SYNDROME

The Migraine Experience

The experience of a severe migraine attack can be conspicuously painful and painfully conspicuous. A person may recognize the warning signs that characteristically precede his migraine attacks, and wait for several hours in anticipation of his next one. He may notice blind spots in his vision, or experience visual hallucinations of bright flashes of light, geometric designs, and stars. Parts of his face, hands, or even his feet may become numb.

During an attack, a person may suffer terrible headache pain, and often look quite ill and dispondent. His hands and feet may be cold, his breath foul, and he may reek of stale sweat. His hands and face may become puffy and swell to the point that jewelry cannot be removed from his fingers. He may moan or cry, and speak weakly without vigor. The slightest movement, loud sound, or bright light may increase the intensity of his headache. The person with a migraine headache may find it impossible to concentrate or recall anything, and

can become extremely hostile and impulsive. He may have prolonged vomiting attacks of such intensity that dry retching may leave him weakened and shaking for several hours after the headache terminates. For several hours after, his face and especially his eyelids may remain puffed and 'blotchy.'

On the other hand, migraine attacks can be so mild that the person can go about his daily routine as he would with any mild headache. Beyond the headache, he may experience nothing more than a sense of extreme tiredness. His associates may notice little more than an increase in his irritability. Although most people associate migraine only with an excruciating headache, migraine is actually found to occur with widely diverse symptomatology.

Clinical Definition of Migraine

Migraine is usually defined as an episodic disturbance of cerebral functioning, associated with incapacitating unilateral headache (Mitchell & Mitchell, 1971; Wolff, 1972). Although headpain is the most prominent feature, diagnosis is based on occurrence of several of the following symptoms: a sense of 'warning' of impending attacks (prodromes); headache is recurrent, throbbing, and usually unilateral at onset; relatively 'perfect health' between attacks; nausia, vomiting, and irritability, particularly at the height of the attack;

extreme visual sensitivity to light immediately preceding or during the attack; vertigo, tremors, sweating, dryness of the mouth, pallor of the skin, and chills during the attack (Alexander, 1950; Moench, 1951; Lance, Anthony & Gonski, 1967; Wolff, 1972).

Visual 'blind spots' or flashes (scotoma), speech difficulties and occasional parasthesias are 'prodromal' disturbances that typically signal the onset of the migraine attack. Scotoma are usually short duration and range in size from a scarcely noticeable blind spot to total impairment. Cranial artery dilation occurs during the attack, but permanent structural damage is exceedingly rare (Wolff, 1972).

The location and duration of migraine headache is unstable. Migraine sometimes is reported in small patches, and sometimes the whole hemicranial area. Location may shift during the attack, and has been reported to sometimes become bilateral after onset. Migraine pattern is usually stereotyped for a given individual, but varies widely from person to person.

For research purposes investigators, such as Marcussen and Wolff (1949) utilize the individual's reaction to ergotamine tartrate as a test for a headache being migrainous in nature. Injections of ergotamine tartrate will abruptly and reliably terminate an actual migraine attack, but it will have little effect on non-migrainous headaches. The reason for this effect will be explained in the next chapter.

Subclassification of Migraine Types

Wolff (1972) subdivided migraine headaches into five basic types: 'classic' migraine; 'common' migraine; 'cluster' migraine; 'hemiplegic' and 'ophthalmoplegic' migraine; and 'lower-half' migraine. All of these subtypes involve short term distention of cranial arteries, but differ in several ways.

Classic migraine is the basic vascular headache described above, involving transient visual, sensory, and/or motor prodromal symptoms. Common migraine is a vascular headache that lacks the striking prodromal disturbances, and is often less unilateral. The classic and common migraine headaches are usually undifferentiated in the migraine research literature, and collectively referred to as 'migraine' proper. The current paper will follow this convention. The remaining migraine subtypes are generally differentiated from the above group by their distinctive sensory or temporal features.

Cluster migraine is a vascular headache in which the absence of visual or other prodromal symptoms such as nausea or vomiting is conspicuous. The name for this type of migraine is derived from its tempo and brevity. Many attacks occur in close-packed groups, sometimes several occurring in a 24-hour period. Each attack may last only an hour or less. Clusters are often separated by long periods of remission. Cluster migraine is also distinctive in that it is

always unilateral and occurs on the same side of the head for a given individual. Cluster migraine displays a predilection for males; if common and classic migraine displays any sex differential, it is more common in females. Cluster migraine is sometimes referred to as 'histamine' migraine (Alexander, 1950). Cluster migraine is commonly caused by an allergic reaction to ingesting vasodilator agents such as alcohol, nitroglycerine, and histamine.

Hemiplegic and ophthalmoplegic migraine are vascular headaches displaying outstanding sensory and motor disruptions that persist during and after the headache itself. Incomplete motor paralysis and anesthesias on one side of the body denote hemiplegic migraine. These symptoms sometimes last for days or weeks following the attack, although recovery is usually complete. Ophthalmoplegic migraine displays a palsy or paralysis of the eye muscles following the headache, which also may last for several days or weeks. Visual distortions during the headache are quite pronounced. Roy (1953) reported that ophthalmoplegic migraine may be caused by refractive errors in the eye and can sometimes be relieved by corrective lenses.

Other Types of Headaches

Migraine can be distinguished from other forms of headache by behavior and physiology. Migraine is less common than tension

headaches. There is no prodromal sequence or vomiting usually associated with tension headaches. Migraine is usually unilateral whereas tension headache is bilateral. Migraine results from constriction and dilation of the cranial arteries, whereas tension headaches result from prolonged and excessive constriction of the head, face, neck, and shoulder musculature (hypertonia). In tension headaches, the muscles of the neck are usually tender and limited in motion due to contractions which produce dull, persistent pain often continuously sustained for weeks or months. Migraine is always reported as discontinuous and episodic. Phenominologically, migraine is usually more intense pain than is tension headache.

Severe head pain is not always of the migraine type, and it is important for each case to be carefully diagnosed. Non-migrainous headaches that involve cranial arteries can be caused by infections, and are identified by the presence of high fever. Carbon monoxide poisoning, convulsive or concussive reactions, and hypoglycemia can also cause non-migrainous vascular headaches. They differ from true migraine by their non-recurrent nature. Tumors, hematomas, absecesses, post-lumbar punctures, and general cranial inflamations can cause severe headache attacks. Likewise, noxious stimulation of ocular, aural, nasal, sinus, and dental structures must be ruled out as a source of headache pain before concluding that the headache is of the migraine type. Generally, the lack of prodromal warning, and

lack of recurrence interspersed by periods of remission, differentiate the above causes from the true migraine attack.

Migraine Incidence

One of the problems in determining actual incidence of migraine is the lack of standardized criteria utilized by various researchers in establishing the diagnosis. For example, Weider, Mittlemann, Weschler, and Wolff (1944) used the criteria of "severe, frequent, incapacitating headache," which likely included severe headache other than migraine. Waters and O'Connor (1970) used the criteria of "unilateral headache with warning, and nausea and vomiting." The variance in diagnostic criteria may account for some of the difference in reported migraine incidence.

Estimates of migraine incidence generally range from five to eight per cent. However, these figures may not accurately reflect migraine incidence in the general population. For many people, the headache is a necessary part of the working solution to an emotional problem. Often poor people either do not seek treatment, or find their way into understaffed hospital clinics where they are misdiagnosed (Sperling, 1952).

Waters and O'Connor (1970) point out that conventional estimates of incidence, based on treatment records of private physicians

TABLE 1

INCIDENCE OF MIGRAINE

Study	Sample Size	Percentage of Incidence
Balyeat & Rinkel (1931)	2984	5
Stein (1933)[1]	2222	5.2
Grimes (1937)	15000	8
Lennox (1941)	1000	5.4
Weider et al., (1944)	10000	9
Lennox & Lennox (1960)[1]	1000	6.3
Waters & O'Connor (1970)	2933	6.7

[1]Cited by Wolff, 1972.

and hospitals, do not include migraine sufferers whose attacks are sufficiently mild or infrequent that medical treatment is not sought. Using an epidemiological questionnaire and subsequent clinical interviews, Waters & O'Connor (1970) found that 46 per cent of the people diagnosed as having migraine had never seen a doctor at any point in their lives for their headaches.

Duration and Frequency of Attacks

Wolff (1972) reported that the duration of migraine attacks are highly variable. Attacks have been reported to last from 20 minutes to several days. Although the results of a single study are hardly conclusive, the following data collected by Touraine and Draper (1934) is presented as a guideline to the distribution of headache duration. Touraine and Draper (1934) noted a strong tendency for men to report attacks of much shorter duration than women. Of the 13 males in their study, 77 per cent reported attacks in the 6 to 11 hour category. In contrast, women made up almost the entire group of patients having headaches last as long as a day or two.

The frequency of attacks varies widely between migraine sufferers, and also may vary for a given person at different times in his life. Mitchell and Mitchell (1971) reported that Ss in their treatment study had an average of about 6 attacks per month, although this varied

TABLE 2

DISTRIBUTION OF ATTACK DURATION
(from Touraine & Draper, 1934)

Duration of Attack	Percentage of Sample (N = 50)
6 to 11 hours	28
12 to 23 hours	32
24 to 48 hours	10
more than 2 days	26
variable, up to 3 days	4
	100 per cent

widely between individuals (range = 2 to 28 attacks per month). Wolff (1972) reported that attacks may occur only four or five times in a lifetime for some people, once a month, or several times a week. Moreover, Wolff (1972) reported that a patient may have attacks once or twice a week for a period of six months, and then be free from attacks for the next five years. Sometimes after a high frequency of attacks for a year, the person may never have another attack again.

Summary

In this chapter, the phenominological experience of migraine, and the criteria for establishing a clinical diagnosis was presented. For review, migraine is an episodic, unilateral headache, sometimes becoming bilateral after onset, and usually involves intense headache pain. Visual distortion, nausea or vomiting, and other sensory-motor dysfunctions are commonly present.

Migraine is generally divided into five subtypes (Wolff, 1972). The usage of the term 'migraine' in the present paper will refer to 'classic' and 'common' migraine only. 'Cluster,' 'hemiplegic,' and 'ophthalmoplegic' migraine are distinguished from the classic and common types by their distinctive (and generally more extreme) sensory and motor disruptions. Other causes of severe headache were presented,

and the means by which they were distinguishable from migraine headache were discussed.

The incidence of migraine is generally accepted to be five to eight per cent. However, several indications were presented that this estimate is not representative of actual migraine incidence.

The duration and frequency of attacks is highly variable between individuals, and may also vary for a given person at different times of his life. Migraine attacks may occur 4 times in a person's life, or may happen at the rate of several per week. One study (Mitchell & Mitchell, 1971) reported an average of about six attacks per month. A separate study (Touraine & Draper, 1934) reported the majority of attacks last between 6 and 23 hours. Migraine attacks have been known to vary from 20 minutes to several days.

CHAPTER II

PHYSIOLOGICAL INVOLVEMENT IN MIGRAINE

Historical Perspective

Occurrences of unilateral head pain were first recorded about the first century A.D. From that time until the mid-1800's, speculations about 'humors' and 'black bile' were predominant physiological explanations for migraine causation. Riley's article (1932) provides an extensive review of the early developments in migraine theory thru the early 1900's. As late as the 1920's migraine causation was relatively undifferentiated from epilepsy, and was suggested to result from metabolic disturbances, brain lesions, and kidney disease (Collier, 1928).

Riley (1932) noted that suggestions of arterial involvement in migraine etiology was made in the earliest documents (e.g. Galen, A.D. 131-201). The interest in arterial involvement was a by-product of early explorations of the human circulatory system. Galen (cited in Riley, 1932) suggested that humors and vapors built up inside the cranium by traveling along the cranial blood vessels to cause the

headache. Anhalt (1724) thought that changes in the cranial blood vessels gave rise to the pain of migraine. Anhalt could not decide whether constriction or dilation of the vessels occurred. Wepfer (1726) suggested that vasodilation caused 'stagnation of the blood,' which in turn caused the migraine pain. However, Wepfer had no demonstrable structural basis for his theory, since no vaso-oscillation mechanism had been recognized at that time.

Advances in neuroanatomy and neurophysiology which occurred in the 1800's were applied to theoretical models of migraine etiology. Claude Bernard (1858) published a report on his investigation of the sympathetic nervous system. Dubois-Reymond (1860) adopted Bernard's results into his own theory, in which he suggested that migraine was caused by constriction of the cranial blood vessels. Dubois-Reymond reasoned that this 'vasomotor neurosis' was produced by irritation of the cervical sympathetic nerves, located in the neck. This was perhaps the earliest suggestion of a vaso-motor mechanism. It was not until much later that the theory of vaso-motor involvement was experimentally confirmed, although the reasoning behind these early hypotheses was disproven.

Ergotamine Research

In the early 1900's, physicians experimented with a wide range of drugs, trying to find something that would provide effective relief from the pain of migraine. Caffeine, histamine, epinephrine, pitressin, amyl nitrate, and calcium gluconate were some of the drugs tried (O'Sullivan, 1936). Sandoz Chemical Company produced a new drug during this period, known by 'Gynergen' and other trade names, that proved to be very effective in migraine pain reduction. This drug was a synthetic form of ergotamine tartrate. Sandoz Company began to offer financial support to independent researchers to encourage research on the effects of ergotamine tartrate (Gynergen).

In 1935, Lennox and von Storch published their results in treating 120 migraine patients with ergotamine tartrate. Of the entire group, 107 Ss (89 percent) experienced abrupt and complete cessation of headache pain upon administration of ergotamine tartrate. Ss were chronic migraine sufferers, who had not been helped previously by other forms of treatment.

The beneficial action of ergotamine was rather specific to migraine headache. In 45 Ss with non-migraine type headaches, only seven derived any benefit from the drug. While the authors recognized that ergotamine was more effective than any previously reported form

of treatment, Lennox and von Storch (1935) reported that the mechanism by which relief was obtained was yet unknown.

The following year, O'Sullivan (1936) reported that she had administered ergotamine tartrate for treatment of 97 patients having a collective total of 1,132 migraine headaches over a two-year period. The drug abruptly and completely terminated headache pain in 1,042 attacks in 89 Ss. Ergotamine had no effect on the frequency of attacks, but was effective in stopping attacks once they had begun. Although O'Sullivan was uncertain of the action of ergotamine tartrate, she suggested it involved the vascular system. Moreover, she felt that its effect was not merely an analgesic action. Several Ss who were suffering from other pain as well as migraine (e.g. toothache, gunshot wound in one hand) found ergotamine effective for the migraine, but sustained the pain in the other locations.

The observations that ergotamine tartrate promptly terminated migraine pain (Lennox & von Storch, 1935; O'Sullivan, 1936) allowed Graham and Wolff (1938) to experimentally study the mechanism of the migraine headache. Graham and Wolff (1938) used photographic records to show that ergotamine tartrate diminished the intensity of migraine pain by reducing the amplitude of pulsations of superficial carotid arteries, located in the surface of the scalp, by 50 per cent. The rapidity with which the pulsation amplitude diminished, which varied

widely between Ss, determined the speed with which pain relief was experienced. When response to ergotamine was rapid, relief was swift.

Graham and Wolff (1938) also found that this drug did not alter the threshold for the perception of pain. Migraine headaches experimentally induced by injections of a vasodilator (which increased arterial pulsations) following injections of ergotamine tartrate, were still extremely painful to Ss. Further, ergotamine tartrate did not block the response of the smooth muscle of the artery wall to further impulses from the sympathetic nervous system, which created increased arterial pulsations.

These same investigators found that reduction of arterial pulsations could be accomplished mechanically. Manual pressure on the carotid artery on the painful side of the head reduced pulsations in the temporal artery and brought pain relief. In some Ss total abolition of pain was accomplished by manual pressure on the temporal and occipital arteries alone. No relief was derived from similar pressure on the unaffected side of the head.

The results of Graham and Wolff's (1938) research tended to confirm the suggestion of vasomotor involvement that had been hypothesized 162 years previous (Wepfer, 1776). Graham and Wolff (1938) suggested that migraine pain results from the distention (vasodilation) of cranial arteries. Moreover, ergotamine tartrate was found

to reduce head pain by causing the constriction of these arteries and thereby reducing the amplitude of pulsations.

A conclusion the authors failed to draw, although evident in their results, was that a unilateral migraine attack results from amplified pulsations of the carotid artery, which occur on only one side of the head. The commonly reported progression from unilateral to bilateral pain after onset probably results from the pulsations eventually spreading to the same arteries on both sides of the skull.

It is important to remember that Graham and Wolff (1938) explored vasomotor functioning in regard to migraine pain, which actually occurs rather late in the migraine sequence. All their experimental procedures were performed after the prodromal phenomena, which characterize the onset of the attack, had subsided. Thus, their results have no bearing on the physiological changes which mark the inception of migraine, and concern only the origin of migraine pain. It was to the area of the physiological 'trigger' that later researchers addressed themselves.

Serotonin Involvement in Migraine

Lance, Anthony, and Gonsky (1967) found that serotonin, a natural component of blood plasma, effected the diameter of blood vessels in the body. High serotonin levels were found to cause vasodilation

of cranial arteries. In a study of plasma serotonin levels of migrainous Ss during attacks of migraine and attack-free periods, Curran, Hinterberger and Lance (1967) found that serotonin levels were lower during initial periods of migraine attacks in 80 per cent of Ss. Serotonin decrease occurred at the onset of the attack and persisted for most of the duration of the headache. This finding was replicated in a similar study of 15 migrainous Ss during 21 migraine attacks (Anthony, Hinterberger & Lance, 1967). From their results, Anthony et al. (1967) concluded that a fall in total plasma serotonin was a specific feature of the migraine attack, and probably was one of the triggering mechanisms of the attack itself.

Lance, Anthony, and Hinterberger (1967) reported that migraine could be precipitated by artificially lowering serotonin levels by injecting Ss with reserpine. Conversely, migraine could be relieved by increasing plasma serotonin levels thru direct injections of serotonin. The authors concluded that onset of migraine was specifically related to drops in plasma serotonin. Since reserpine is not naturally found in the blood stream, they speculated that some endogenous substance with a similar serotonin-reducing action is liberated at the onset of the migraine attack; the consequent decrease in plasma serotonin initiates the vascular changes responsible for migraine headache and the prodromal symptoms.

It has been known (Blaschke & Philpot, 1939) that monoamine oxidase (MAO) is a normal component of blood plasma that effectively decreases serotonin level by de-aminating it. Kimball, Friedman and Vallejo (1960) demonstrated that headaches in migrainous Ss decreased in severity and frequency when MAO production was inhibited, to increase and stabilize their serotonin level. Lance (1969) has also reported that 14 of 19 migrainous Ss became either headache free or showed substantial reductions in frequency when MAO production was chemically inhibited. In all 14 Ss, plasma serotonin levels increased more than 20 per cent above comparable pre-treatment levels. The alleviation of migraine thru MAO restriction suggests that MAO activity is involved in migraine episodes by causing fluctuations in normal plasma serotonin levels.

Overview of Chemical and Physiological Changes in Migraine

To sum up the experimental findings on the chemistry and physiology of migraine attacks, changes in serotonin and MAO levels in the blood plasma have been demonstrated to cause vaso-constriction, which leads to the migraine headache. Specifically, increases in MAO, relative to serotonin levels appear to effectively decrease the serotonin content in blood plasma. A decrease in serotonin level, 45 to 60

per cent below normal level creates vasoconstriction and marks the inception of the attack by the appearance of the prodromal symptoms (Anthony, Hinterberger & Lance, 1967). Normalization of serotonin levels (spontaneously or by experimental means) effectively terminates the migraine attack if administered during the early prodromal signs.

If the serotonin level is not stabilized early, the body attempts to correct the vasoconstriction condition by increasing serotonin levels in the blood plasma. However, this actually leads to an over-compensation which creates a vasodilated condition in the cranial arteries. This marks the final phase of migraine: massive head pain. At this late stage, administration of ergotamine tartrate, a known vasoconstrictor, increases muscle tone in the arterial walls and decreases the amplitude of arterial pulsations. If medical intervention does not occur, the body eventually re-establishes the serotonin-MAO balance.

The above model of chemical disruption of vascular functioning is undisputedly the physiological mechanism behind migraine headache. However, it lacks a 'trigger' mechanism that would account for the initial decrease in serotonin. In the search for the precursor of the physiological changes that underlie the migraine attack, investigators have turned to the psychological characteristics of the individual for the answer. In the following chapters, the prominent theories of

migraine causation will be examined and compared. Finally, these theories will be examined in light of the available research data.

It is important to note that the model of vascular functioning presented above is fully compatible with all of the theories to follow. All of these theories, which were formerly written: ". . . and this causes migraine" would now simply be written: ". . . and this causes the blood plasma changes which in turn cause the vaso-oscillations of migraine." In fact, most theories of migraine deliberately left gaps for the physiological data that was unknown at the time.

Genetic Involvement

Another aspect of migraine that has received much attention is the role of genetic transmission of a pre-disposing migraine physiology. The observation that migraine often occurred along family lines has repeatedly appeared in the literature (Collier, 1928; Martin, 1928; Grimes, 1931; Touraine & Draper, 1934; Murphy, 1954; Goodell, Lewontin & Wolff, 1954; etc.). From their review of six relevant studies, Goodell, Lewontin and Wolff (1954) noticed the possibility that the migrainous individual inherited a genetic predisposition from a relative appeared in 75 per cent of cases reported. Touraine and Draper (1934) arrived at similar conclusions from their own review of the literature. The supposition of genetic transmission of migraine

has become so entrenched that the presence of migrainous relatives is often a criterion used for diagnosis of migraine in the patient.

Of the 50 Ss in one study (Touraine & Draper, 1934), 41 Ss (82 per cent) reported recurrent headaches in another family member of the preceding generation. In nine cases, there was no evidence of migraine in parents, siblings, or distant relatives. Migraine in the father was reported in only five cases; both parents were migrainous in only two cases. Thirty-two Ss reported migraine coming thru maternal lines. In 30 cases, migraine occurred in the mother while in two cases it was reported only in the maternal grandmother. In one of the two latter families, the grandmother had been a member of the S's household.

Although this data is suggestive of genetic transmission, the authors caution against making conclusions from their study due to a shortcoming common to all the yet reported genetic studies: it is impossible to be sure that the headaches of family members were actually migraine. Diagnosis based on behavioral reports by Ss are liable to considerable inaccuracy.

The caution expressed by Touraine and Draper seems well-founded in two respects. For one, they failed to collect data on the chance frequency of obtaining reports from non-migrainous people of relatives having "migraine" headaches, by which to establish a baseline for examining reports of migrainous Ss. For another, their estimates

of 82 per cent incidence of migraine in biological relatives is in considerable disparity with later studies.

Using similar criteria to the above study, Lance and Anthony (1967) established a baseline of 18 per cent for reports of "migraine" in family members from non-migrainous Ss (tension headache patients). In contrast, the authors found that 46 per cent of 500 migrainous patients reported "migraine" in family members. The differences between migraine and tension headache patients in family incidence of migraine was statistically significant ($p < .001$). On the other hand, the 46 per cent found by Lance and Anthony (1967) is in sharp disagreement with the 82 per cent found by Touraine and Draper (1934). Part of this discrepancy may be due to Touraine and Draper's failure to clearly differentiate between migraine and tension headache.

The study by Goodell, Lewontin and Wolff (1954) directly focused on the heredity issue, and utilized a control group. In 65 clinic patients, diagnosis of migraine was based on clearly defined criteria: presence of typical migraine symptoms as well as a previous history of attack relief from ergotamine tartrate. (This last criterion assured that Ss suffered from vasodilation headache, rather than tension headache or hysterical symptoms.) An additional 79 Ss, receiving private treatment for migraine from the third author (diagnostic criteria unknown), were added to the experimental population. Thirty-four of the clinic patients and 25 of the private clients

received personal interviews to collect data for the study. Thirty-one of the clinic patients and 54 of the private patients were unavailable for interviews, and their information was collected by questionnaire. In collecting their data, the authors relied upon the previously reported traits of perfectionism, and the need to perform with exactitude and meticulous attention to detail in migrainous Ss, to make their reports reliable. This is a highly questionable methodological procedure.

The 119 migrainous Ss in this study reported 343 relatives as migrainous. Twenty Ss had no migrainous relatives; 66 had one to three migrainous relatives; 22 Ss had four to seven migrainous relatives; and eleven Ss had eight to nineteen migrainous relatives. In the families of Ss reporting at least one migrainous relative, there was a total of 832 offspring. Of these offspring, 265 children had no migrainous parents; 76 of these children (29 per cent) were reported migrainous; 502 children had one migrainous parent; of these, 222 were reported migrainous (44 per cent); 65 children had two migrainous parents; of these children, 45 were reported migrainous (69 per cent).

In considering the observed and expected frequency of affected and nonaffected offspring of migrainous parents, the authors found statistical support for the notion that the trait of migraine is inherited ($X^2 = 39.35$, $df = 2$, $p < .001$). Goodell et al. (1954) concluded that migraine is due to a recessive gene that manifests itself in headache attacks in 70 per cent of the people with the genetic trait.

Although this study was methodologically more sound than those previously discussed, several shortcomings still exist. For one, the population was treated as homogeneous, without establishing equivalent diagnostic criteria. For another, the authors themselves may be cited:

> It was recognized that genetic control was lacking. It was appreciated that an identical heredity equipment might give expression to headache in one environment and not in another. Also, the mere fact of having one or both parents with migraine might be an environmental influence conducive to migraine in the offspring.
>
> Furthermore, the method of obtaining information about the occurance of migraine headache in other family members, by asking patients to recall such illness in relatives both temporally and spatially distant, may be seriously unreliable, especially since the complaint of pain in the head from a variety of causes can be elicited from approximately 85 per cent of the population.

Moreover, studies relying on Ss' report of their relatives' behavior are open to further distortion. The above authors, and others, have noted that headaches of family members tend to be very similar in clinical expression. Thus, studies like those presented here, cannot rule out alternative explanations such as imitation and learning. Since migrainous people are very likely to visit a number of doctors for diagnosis and treatment, bias is introduced. As he is likely to have been told that migraine runs in families, he is more likely to interpret the behavior of relatives in this direction.

In conclusion, there is some evidence to support a widely held belief that at least part of migraine causation is genetic in nature. In examining the theoretical models of migraine causation, it will be

of interest to note to what degree each considered the possibility of genetic involvement in the determination of migraine headaches. The fact of unilateral head pain is very hard for most psychological theories to explain, but is quite plausible that only one side of the skull would contain a genetic weakness in the arteries.

Summary

The occurrence of unilateral headpain has received attention as early as the first century A.D. However, it was not until the mid-1900's that vaso-motor involvement of the cranial arteries was experimentally confirmed thru research into the effects of ergotamine tartrate in migraine pain reduction.

The onset of the prodromal symptoms of the migraine attack is contiguous with vaso-constriction of the cranial arteries. This vaso-constriction is caused by a 50 per cent reduction of serotonin level in blood plasma. The body attempts to correct the vaso-constricted condition by increasing serotonin plasma levels. An over-compensation actually occurs, yielding a vaso-dialated condition due to heightened serotonin levels. Headache pain normally occurs when cranial arteries are in a vaso-dialated condition.

Reports of genetic transmission of a pre-disposing migraine physiology are quite widespread in the literature. Several studies

regarding familial occurrence of migraine were presented. Their results tended to support the notion of a genetic pre-disposition.

CHAPTER III

PSYCHOLOGICAL THEORIES OF MIGRAINE CAUSATION

Background of the Psychological Theories

Suggestions of psychological involvement have appeared in the earliest papers on migraine (Moench, 1924; Riley, 1932). Early formulations of migraine suggested that the migrainous individual was emotionally and psychologically 'normal' between attacks. Personality manifestations were originally thought to be prodromes or side effects of the actual attack.

However, as the amount of migraine research began to increase, particularly in the 1930's, certain characteristics of migrainous individuals were reported with considerable regularity: perfectionism, worrysome, chronically tense, preoccupied with success and achievement, hard driving, resentful, rigid, highly competitive, jealous, sarcastic, highly ambivalent, moralistic, capable of establishing only superficial interpersonal relationships, and unsatisfying sexual contact (Touraine & Draper, 1934; Wolff, 1937). As the life histories of migrainous people were more closely examined, psychoanalytically

trained investigators began to consider the hypothesis that personality structure and emotions played a basic etiological role in migraine causation. Moreover, the lack of any previously established conceptual framework for migraine further encouraged psychoanalytic interpretations.

Weber (1932) noted that some authors at that time considered the psychological factors in migraine causation to be relatively minor, and attributed psychological characteristics to other underlying physical conditions. Moreover, other authors were taking an intermediate view in which the psychological factor was co-contributor, along with a hereditary predisposition and the current physical state of the individual.

Weber (1932) presented a case study of a 56-year-old migrainous woman whom she had treated in psychoanalysis, to illustrate her contention that psychological factors such as neurotic conflicts, were the primary causal agent in migraine attacks. She suggested that migraine was caused by repressed feelings of rage and humiliation that were striving for expression in the Unconscious of her patient. Moreover, Weber suggested that the purpose of the attacks was to preserve the client's infantile dependency on her mother and bolster the client's feelings of omnipotence. Thru the attacks, Weber suggested, the client was able to stay at home when she would otherwise be

expected to be outside and independent. The attacks also made the client's mother very attentive to her.

However, because Weber only studied a single S, she was unable to come to any substantive conclusions. Although the case history was suggestive of primary psychological causation, it was consistent with a cranial pressure increase theory, a vasomotor theory, a metabolic theory, an ocular theory, and endocrine and digestive theories. Weber (1932) did not offer any concrete method by which the psychological elements of her client might be converted into somatic expression. Rather, she only suggested that her client's background and presenting problems were readily integrated and consistent with psychoanalytic theory.

Touraine and Draper (1934) conducted the first large sample (N = 50) case study of migrainous individuals. These observers noted difficulties with personal insecurity and interpersonal relationships to be quite common among Ss. Moreover, Ss were commonly observed to be perfectionistic and intolerant of criticism. Most Ss were also quite ambivalent in their feelings towards their mother. They displayed a strong conflict between a desire to escape her influence and a compulsion not to leave her. Touraine and Draper (1934) suggested that a trigger for a migraine attack might be any event which threatened to change the patient-mother relationship.

However, Touraine and Draper (1934) also believed that susceptability to the attack was inherited. For this reason, a more detailed examination of their research will be presented when other interactional theories are considered in the next chapter. Touraine and Draper's report of personality characteristics and dynamics were the springboard for psychoanalytic formulations that followed within several years. More often than not, the psychoanalytically trained investigators tended to overlook the suggestion of genetic-environment interaction that Touraine and Draper presented in their article (1934), and focused on the parent-child dynamics that were more suggestive of psychodynamic involvement.

Psychoanalytic Theory of Migraine

Frieda Fromm-Reichmann (1937) took up the notion of ambivalence and developed a more orthodox psychoanalytic theory. She hypothesized that migrainous people suffer from an unresolved ambivalence: they cannot tolerate being aware of their hostility towards consciously beloved persons. Thus, they repress their overt hostility such that it must eventually emerge in the physical symptoms of migraine. The suggestion of hostility in the child for the parent had appeared before in psychoanalytic theory. Fromm-Reichmann suggested that when it occurred in particularly tightly-knit family in which parents use

withdrawal as punishment, the child represses his anger in fear of parental abandonment. Her theoretical formulations were based on her experiences with a small group (N = 8) of migrainous people with strong intellectual strivings, including two physicians and a scientist. From her contact with this highly select population Fromm-Reichmann (1937) became the first author to suggest that the location of pain in the head was of symbolic significance:

> This general mechanism is that migraine patients primarily want to destroy . . . [the other person's] intelligence and brilliancy, respectively their brain and head, as the concrete representation of their mental capacity. This mental castration of another person is not allowed and therefore, according to the analytically well-known unconscious mechanism, is turned back towards the patient himself; he does to himself by these means what he wanted to do to his partner, thus punishing himself for his forbidden tendencies (1937, p. 27).

Fromm-Reichmann hypothesized that repressed anger is expressed in spasmadic contractions of smooth muscle tissue in the cranial artery walls, which produces migraine. She argued that since conscious anger is normally expressed in movements of striated muscle under voluntary control (e.g. arms or legs), repressed anger is expressed via smooth muscle tissue that is less responsive to conscious control.

> If we remember our well-known analytic experience that the organism is able to give unconscious utterances to repressed feelings by involuntary movements, then we understand that a patient suffering from

> migraine, that is from the results of unconscious spasmodic contractions of the involuntary muscular system, is unconsciously expressing his repressed hostility against a beloved person on the same principle as another would express his conscious hatred.
>
> The average person feeling conscious hatred against an adversary uses contractions of his voluntary skeletal muscles which obey conscious impulses as a normal conscious means of expressing his hostility. The migraine patient who represses his hostility against consciously beloved persons, produces as his unconscious expression of this repressed hatred spasmodic contractions of involuntary smooth muscles which obey unconscious impulses (1937, p. 28).

[This same argument is sometimes used to suggest that migraine is indicative of more extensive repression of impulses than tension headache, since migraine involves smooth muscle activity, whereas tension headache results from hypertonia of the striated muscle of the face and neck.]

However, Fromm-Reichmann's hypothesis regarding unconscious impulses and smooth muscle activity is not convincing. There is no reason why smooth muscle activity would not be involved in anger as well. In fact, when Alexander's article (1950) is considered subsequently, it will be seen that Alexander considered smooth muscle activity to be an integral part of conscious rage reactions. Smooth muscle activity is not solely the province of repressed impulses. Moreover, the psychoanalytic suggestion that unconscious impulses are expressed in smooth muscle tissue is not unique to migraine. A

similar rationale is used to explain gastric ulcer and essential hypertension.

The question of why it is the head that is affected is a critical one for Fromm-Reichmann's theory. She suggested that symptoms were localized in the head because migrainous people were rivals of their beloved adversaries, and felt resentful of their intellectual capabilities, unconsciously wanting to destroy or feel superior to them. However, an examination of the occupations of 400 migrainous and 1000 non-migrainous Ss (Allen, 1927) revealed occupation played no decisive factor in migraine incidence. This result may indicate that neither the level of intellectual capability or the level of intellectual strivings of the migrainous person may be important in migraine etiology. Even if Fromm-Reichmann's hypothesis is tentatively accepted for her highly select Ss, it would still not explain localization of symptoms in the head for the majority of sufferers. Since migraine appears to be randomly distributed across socio-economic and educational levels, there are many migrainous people of average intelligence or less, pursuing non-intellectual goals such as homemaking and raising families, for whom Fromm-Reichmann's theory would not be adequate.

Finally, the author implied that vascular changes were brought about by the intra-psychic process of introjection. However, this would not explain the fact of unilateral onset.

Furmanski (1952) suggested that migrainous people were extremely narcissistic and possessed strongly developed aggressive impulses. He hypothesized that this developed from a lack of demonstration of affection by the parents, or excessive strictness in child-rearing practices. Moreover, because of the resulting frustration of the child's needs, the child becomes highly ambivalent between performing to gain love and approval, and expressing the hostility. The hypothesized frustrations lead to anal and/or oral fixations, which result in the eventual development of ultramoral and rigid superego. The migrainous person is unable to express hostility because of the massive guilt it would generate. Furmanski (1952) suggested that the migraine attack occurs when the individual cannot tolerate any more frustration and hence cannot suppress the hostility frustration evokes. He conceptualized migraine as the physiological manifestation of suppressed or repressed hostility, initially directed toward the family, and later to frustrations in general.

Basically, this formulation is a re-integration of the previous writings of Fromm-Reichmann. Perhaps the only new contribution offered by Furmanski was an emphasis on the role of frustration in the etiology of migraine. Furmanski (1952) suggested that frustration is the cause of early psycho-sexual fixations which in turn develop a character structure that is highly intolerant of expression of hostility. Moreover, previous authors suggested that inhibition of aggression

produced migraine; Furmanski (1952) extended the chain of events one step further: frustration evokes hostility which eventually exceed the individual's suppressive or repressive capabilities. The migraine, rather than the hostility, is then emitted.

Perhaps the clearest statement of the classical psychoanalytic model is that by Sperling (1952):

> To be fully conscious of this rage is considered dangerous by the patient who fears that he cannot resist the urge for destruction of the frustrating object or of himself. Repression of this rage and of the impulse to kill serves to protect both the object in the outer world and the patient himself. At the same time, gratification of the impulse is achieved unconsciously in the symptom. Every successive headache in the migrainous patient is such a repetitive killing of the frustrating object. There is no guilt feelings, the punishment being inflicted by the patient upon himself in the physical pain and suffering. The choice of the specific symptom--headache--is determined by the specific impulse to kill the object by an attack upon the head (as an expedient and primitive way of killing), whether this be choking, bashing in of the head, shooting through or crushing the head, etc. The accompanying manifestations of nausea, vomiting, diarrhea, represent attempts of the patient to rid himself of his destructive impulse somatically (1952, pp. 161-2).

Alexander (1950) also postulated that migraine is caused by the repression and suppression of hostile and aggressive impulses. In examining both reported personality traits, and the specific mechanisms behind migraine, he wrote:

> The relevance of the characteristic personality features . . . consist merely in that personality types that are likely to repress their hostility have a greater inclination toward migraine attacks (1950, p. 160).

Alexander suggested two new reasons for the head being the site of the pain. First, migraine is caused by the repression of hostility at the conceptual phase (i.e. its planning and imagery). Since the head is guilty of the hostility, the self-punitive attack is directed at the head.

Second, the repression and suppression of hostility has physiological concommitants that produce migraine. The inhibition of rage precludes muscle action, but the increase in blood flow that is part of the rage reaction still occurs. Alexander suggested that blood flow to the muscles and skull increases in anticipation of any violent activity. But since blood flow to the muscles does not occur in inhibited rage, the balance is shifted to the cranium. Indeed, he assumed that this cranial flow exceeds even the normal increment associated with violent emotions that are not restricted.

Basically, Alexander (1950) suggested that migraine resulted from high cranial blood flow, and that anger is one of many things that cause an increment in blood flow. His model is psychoanalytic in his explanation of why anger causes migraine in some individuals and not in others. That is, the more repressed the expression of anger is in the individual, the more likely he is to have psychosomatic ailments. If hostility is repressed at the conceptual phase, the self-punishment is directed at the head. Hostility suppressed at the behavioral level results in arthritis.

As was true of previous psychoanalytically oriented writers, Alexander's model does not explain unilateral onset of migraine pain. However, it does suggest a physiological mechanism by which intra-psychic processes could be transmitted into physiological changes leading to the migraine attack. In this respect, Alexander's model goes beyond those previously presented.

The suggestion of secondary grains in migraine attacks is unique to psychological models of causation. In the intra-psychic sphere, hostile impulses are punished by the migraine pain, and guilt for the impulses is accordingly reduced. Awareness of the hostile impulses is preempted by directing attention toward the attack itself. The attack also reduces anxiety about parental abandonment, by presenting the migrainous individual in a regressive, dependent state. "Magical" reasoning may occur along the line of: "they won't be able to leave me when I show them how much I need them." The person's hostile impulses are less likely to be expressed directly against the parents, because they are vented in the attack upon the self.

The attack also offers psychological advantages in coping with unrealistic perfectionistic tendencies. For one, the attack offers the individual a convenient rationalization for not meeting his own achievement expectations. For another, the person who will not risk confrontation with his parents by openly rejecting their performance

expectations may unconsciously reject them under the guise of being too ill to perform.

The attack may also offer some realistic advantages in interpersonal relations. It facilitates obtaining dependency relationships with other people, and diminishes the probability of other people expressing hostility for any reason. Moreover, the migrainous person is usually exempt from any performance or independence expectations from others during an attack.

Several general characteristics of the psychoanalytic model of migraine are evident. For one, the model is affect specific. That is, it postulates the involvement of particular feelings, especially hostility, ambivalence, and guilt.

A second characteristic is the involvement of intra-psychic defense mechanisms, directed against the experience and expression of these affects. Authors do not agree on the relative degree of involvement of repression or suppression. However, formulations are generally biased in the direction of repression, since this facilitates the argument (clearly presented by Fromm-Reichmann, 1937) that migraine results from the disruption of physiological processes unavailable to conscious control.

A third characteristic is the belief that the occurrence of the attack is psychologically determined. That is, hostility and the repression of hostility causes migraine. Moreover, the location of

the attack in the head of the person results from redirecting hostile feelings against the self, rather than being physiologically determined. Psychoanalytic models also suggest that migraine may be sustained by the secondary gains derived from the attack. That is, the person may unconsciously enjoy the 'benefits' (e.g. the dependency role) from the migraine.

Finally, psychoanalytic theory suggests that migrainous people are fixated at the anal and/or oral stages of psycho-sexual development. These pre-genital fixations make the migrainous individual particularly susceptable to the hostile and ambivalent feelings that trigger the migraine attack.

Another Psychological Model

Psychoanalytic formulations were the earliest and most extreme of the psychological theories of migraine causation. Later theories, such as that presented below by Marcussen and Wolff (1949) tended to be more moderate in the theoretical constructs invoked. Yet the impact of the psychoanalytic approach is clearly evident.

Marcussen and Wolff (1949) suggested that migrainous individuals need not be neurotic, but may have made an 'adequate adjustment' to life. However, they are rigid, ambitious, perfectionistic, and hard driving people. Migrainous people seek the rewards that

clearly superior performance brings them, but at the cost of great expenditures of energy. Events of daily living produce gradually mounting tension because these individuals are unable to meet rigid, unrealistic standards of performance which they set for themselves. They exhaust themselves in attempting flawless performance in all undertakings, and disregard their needs for rest and relaxation. The outcome is a life characterized by feelings of resentment, tension, fatigue, and exhaustion. If while fatigued and exhausted, an event occurs which evokes rage and resentment, then a migraine attack occurs.

Marcussen and Wolff (1949) predict the attack to occur when the stamina of the physiological regulating processes (unspecified in their article) are overtaxed. Unconsciously induced vascular headache pain occurs to force the individual to withdraw from the frustrating and life-threatening situation, allowing restoration of energy reserves. If the individual had been relaxed and rested, the same incident might have evoked the same rage. However, it probably would not have set off the chain of bodily changes leading to migraine.

It is important to note that the concept of "energy" as Marcussen and Wolff use it, is strictly in a metaphysical sense. It refers more to the capacity for certain psychological operations, than the capacity for purely physical activity. For instance, a person who plays tennis for relaxation in his leisure hours is burning far more calories than when he is sitting tense and overburdened at

his desk job. Yet the authors suggest that the tennis player is restoring his 'energy reserves' that he drained while being tense at his desk in the office. Thus, Marcussen and Wolff (1949) cannot mean "energy" in the usual scientific sense.

Thus, the fact that they refer to their theory as an underlying "physiological protective mechanism" is very misleading. Their use of the word "energy" is distinctively intrapsychic. Marcussen and Wolff (1949) suggest that the individual is capable of coping with only finite amounts of tension and hostility without release. Indeed it is emotional turmoil more than physical labor which drains energy reserves.

In their model, the withdrawal from the frustrating and fatiguing life situation during the migraine attack is a non-volitional or unconscious malingering. That is, the attack occurs for the sole purpose of avoiding those particular tasks that are especially emotionally threatening or draining, and challenging to the individual's performance expectations. This is in no way inconsistent with the idea of a tendency to withdraw into migraine in reaction to the experience of hostility. Confrontation would also classify as a very high 'energy use' activity. Suppression involves 'hard mental work' and thus is another energy drain that potentiates a migraine attack. The depleted individual who expends the energy to suppress the anger that a frustrating situation evokes will have that much less energy to expend in

fulfillment of his performance expectations. Advocates of Freud's economic model of psychic energy will recognize this viewpoint as a special case of the psychoanalytic model of migraine causation.

Comparison of the Psychological Theories

Marcussen and Wolff's (1959) formulation does not invoke the concept of repression. However, they do utilize the idea of suppression and link it specifically to hostile impulses. Moreover, they suggest that guilt is often a prominent feature and perhaps an integral part of migraine precipitation, although guilt without frustration and hostility is not causal. Psychoanalytic writings suggest that hostility and resentment, together with guilt for the impulses themselves, precipitates the attack.

Both models suggest a secondary gain attached to the migraine attack. In the psychoanalytic model, the seondary gain is the expression and self-punishment for hostile impulses, thus expiating guilt feelings. In the Marcussen and Wolff model, the gain is opportunity to rest without guilt and the avoidance of compulsive performance expectations.

Both models predict the occurrence of the attack on a defense or overload basis in response to ongoing events. In the psychoanalytic model, the attack occurs when there is insufficient energy to maintain

the repression and suppression of unacceptable impulses. Marcussen and Wolff (1949) predict the attack will occur at those times when the individual is hostile, frustrated, and thus has insufficient energy to maintain his perfectionistic performance.

There is only one way that the two models differ, and that seems to be only a semantic difference. Psychoanalytic theory suggests that migrainous individuals are neurotic, whereas the Marcussen and Wolff (1949) model states they are not. Yet, Marcussen and Wolff would not argue that the migrainous person's performance and achievement expectations are realistic. Nor would they deny that migraine attacks are a grossly inappropriate way of coping with unrealistic performance demands. Rather, they might describe them as a 'self-defeating life style.'

Summary

A substantial number of articles have been written regarding the psychoanalytic theory of migraine causation. This theory suggested that attacks were psychologically determined by the repression of hostility for consciously-beloved persons.

The theory proposed by Marcussen and Wolff (1949) was also presented. Marcussen and Wolff suggested that attacks result from

unconscious attempts to withdraw from frustrating and threatening situations when the individual's 'energy reserves' are depleted.

The psychological theories of migraine causation considered in this chapter utilize the concepts of intrapsychic defense mechanisms, and conservation of psychic energy. The psychological theories also stress the importance of hostile impulses and secondary gain in the etiology of the migraine attack.

CHAPTER IV

CONSTITUTIONAL THEORIES OF CAUSATION

Early Constitutional Theories

Grimes (1931) suggested that migraine attacks occur only in those individuals with a predisposing 'migraine instability.' He thought this instability was an inherited genetic trait that varied in strength between individuals. Grimes suggested that a migraine attack occurred when stress, the immediate precipitating factor, exceeded the migraine 'stability' of the individual. If the instability was great, a slight stress might be sufficient to trigger an attack. If the instability was minor, considerable stress would be needed to trigger an attack.

Grimes (1931) thought that stress could be either physiological or psychological in nature. However, he thought that physiological stress was responsible for more attacks than was psychological stress.

Grimes also thought that age of onset was related to the degree of migraine instability. If the instability was great, onset

tended to occur early in life; if the instability was slight, onset occurred when the individual was an adult. Logically, the less tolerant of stress a person is, the sooner in his life is he likely to have his genetic predisposition manifest itself in a clinical problem of migraine attacks.

As mentioned earlier, the theory of Touraine and Draper (1934) is more properly excluded from the models of direct psychological causation. Specifically, it is their postulation of a basic genetic factor in the physiological makeup of the migrainous individual that separates it from the other theories considered in the previous chapter. However, since their data did not fit any simple genetic model, they rejected a purely genetic explanation of migraine. That is, they assumed that although the constitutional capacity for migraine was transmitted genetically, a person who is susceptible to migraine will not actually suffer migraine unless that person has been subjected to a certain pattern of emotional experiences. Touraine and Draper (1934) sought to find the "essential environment" for migraine by looking for common elements in their patients' case histories.

They came to the conclusion that the immediate trigger for a migraine attack is strong anxiety generated by the individual's response to his immediate surroundings. After examining the early relationship that 50 migrainous people had with their mothers, they suggested that the migrainous individual experiences a conflict

between a desire to escape from the mother's influence and a compulsion not to leave her. Any event which threatens to alter the balance of the ambivalent feelings of the child for the mother generates anxiety that triggers the predisposed migrainous physiology. Thus, an increase in 'smother-love' or any threat that a separation is about to occur triggers an attack.

Touraine and Draper also noted that most of their patients suffered from inadequate sexual functioning. This was consistent with their model. If anxiety brings on an attack, and if sexual inadequacy produces anxiety, then migrainous persons with sexual problems should be much more likely to have a large number of attacks. That is, constitutionally migrainous individuals with sexual difficulties are much more likely to come to clinical attention.

It is pertinent to note that the authors were writing in 1934, when sexuality was a major emotional problem and frigidity was widespread. Thus, it is not surprising that sexual difficulties were commonly identified among migrainous individuals. Moreover, it is likely that the individuals who failed to make an emotional separation from their parents would also have greater difficulty in adjusting to married life.

Although Touraine and Draper's report of parent-child interactions may bring to mind superficial similarities with the psychological theories of the previous chapter, their significant differences

should be pointed out. For one, Touraine and Draper hypothesized a genetic predisposition, without which a person will not have migraine regardless of postnatal experiences. Psychological theories claim that migraine can develop solely from developmental experiences. For another, Touraine and Draper identified the parent-child relationship and sexual concerns as common triggers for the underlying genetic predisposition. They did not suggest that either problem was indicative of an underlying personality structure that was unique or causal of migraine attack. In these respects, the Touraine and Draper (1934) theory differs from the psychoanalytic theory. Other differences between the psychological and constitutional theories will be considered at the conclusion of this chapter.

Wolff (1937)[1] presented a view of psychological involvement similar to the Touraine and Draper (1934) model of genetic and environmental interaction:

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[1]The work of the late Harold G. Wolff is outstanding in the field of migraine research. He is credited with 14 publications of his own, and participated as second or third author in perhaps 100 more. The fact that Wolff is cited as advocating a constitutional theory in the current chapter, while cited for a psychological model in the previous chapter (Marcussen & Wolff, 1949) is not inconsistent with his contributions. From a small sampling of his work, it is apparent that Wolff considered many diverse approaches to the problem of migraine: the role of genetic inheritance of migraine predisposition (Goodell, Lewontin & Wolff, 1954), vaso-oscillations and the effects of ergotamine tartrate (Graham & Wolff, 1938), the role of emotions in triggering genetic predispositions to migraine (Wolff, 1937), and specific emotional patterns being causal in migraine (Marcussen and Wolff, 1949).

> The [migrainous] Ss studied presented common personality features which are in no sense pathognomic of migraine, nor are they associated with migraine alone. However, these personality features in certain life situations are especially prone to call forth pernicious emotional reactions. In Ss predisposed to migraine such reactions may precipitate attacks of migraine; hence the personality features of these persons become important (1937, p. 896).

Wolff (1937) observed the common characteristics of perfectionism, inflexibility, indecisiveness, and difficulties in social and sexual relationships in his case study of 46 migrainous people. But Wolff (1937) was interested in delineating these traits for indications of what types of life situations might be related to the frequency and intensity of migraine attacks. He was not suggesting that these traits were indicative of underlying psychodynamics that produce migraine:

> The evidence indicates, and it is well to emphasize it, that there are a multiplicity of personality features, life situations and emotional reactions which are of importance in migraine. Therefore, it is futile and fallacious to reduce the problem to this or that element in the psychobiologic constellation.
>
> Hence, in these particular Ss, with special predisposition and psychobiological equipment that tended to create sustained pernicious emotional states and fatigue, it may be reasonable to postulate that labile physiological mechanisms within the cranium were set off which ended in the untoward chain of events constituting the attack of migraine.

Wolff's position is quite similar to that arrived at by O'Sullivan (1936), following her work in treating migrainous Ss by administration of ergotamine tartrate. O'Sullivan wrote:

> That psychic factors can precipitate attacks in a migrainous person, most of us who have had any experience with the syndrome will not deny. That they are the only factors in the production of the episode is not in accordance with the observations of this clinic. That psychic factors alone can completely check 1,000 full-blown migraine attacks within from fifteen minutes to two hours, I challenge (1936, p. 121).

A Recent Constitutional Theory

In recent years, Mitchell and Mitchell (1971) have presented a model that shares the emphasis on genetics and physiology suggested by previous authors. Moreover, this recent work incorporates the documented involvement of changes in blood chemistry and vascular activity that earlier authors could only hypothesize.

Mitchell and Mitchell (1971) suggest that the trait sought after in earlier genetic studies, and postulated by advocates of a 'migrainous constitution,' is an inherited hyper-sensitivity of the cranial arteries to fluctuations in serotonin and MAO levels. In explaining the genesis of the specific attack, the authors point out that MAO and serotonin are released thru the sympathetic nervous system. If the genetic hypersensitivity is present, Mitchell and Mitchell (1971) predict that anything that produces heightened or chronic sympathetic nervous system activity (SNSA) will produce a migraine attack. Since extreme emotional reactions, such as rage, anxiety, etc., are the most common cause of heightened SNSA, migraine

is normally triggered in susceptable individuals by extreme emotional responses to daily living.

Mitchell and Mitchell go on to suggest that migrainous individuals have certain personality characteristics which predispose them to states of heightened SNSA. Because these individuals are prone to 'low uncertainty thresholds,' migrainous people are inordinately susceptable to anxious feelings of insecurity in coping with their environments. The low uncertainty thresholds increase the likelihood of perceiving one's surroundings as ambiguous and uncertain. Moreover, the authors suggest that the overt reactions of migrainous people tend to be restricted in these same situations. Both these developments result in increased SNSA. Although Mitchell and Mitchell offer no rationale for this inhibition of behavior, one may speculate that an individual may hesitate to commit himself to a behavioral response if he doubts its appropriateness, i.e., if he is uncertain about his environment.

While their notion of "low uncertainty thresholds" (Mitchell & Mitchell, 1971) adds clarity to the psychological components of their theory, the authors may have actually weakened their model by extending themselves into this area. Their notion of uncertainty threshold is not central to their theory, but is an 'add on' assumption which seeks to explain the personality traits reported by other writers. If research shows these traits not to be characteristic of migrainous

individuals, then Mitchell and Mitchell's theory would be unnecessarily disconfirmed.

The authors' theory is still cohesive even if random personality traits are postulated. In people with the inherited vascular sensitivity, migraine could result from normal reactions to an environment which presents a high frequency of situations capable of evoking extreme emotional reactions. That is, the inherited physiological reactivity would be an attractive answer to future research findings that some individuals with similar psychological traits develop migraine while others do not.

Comparison of the Constitutional Theories

At this point, several basic similarities of the models proposed by Touraine and Draper (1934), Wolff (1937), and Mitchell and Mitchell (1971) should be evident. All models postulate a basic underlying genetic factor that predisposes a person to migraine attacks. Moreover, the immediate trigger is any strong emotional reaction, such as anxiety or hostility. However, the content of the affect is not important. The personality structure determines those areas that are most likely to provide the strong emotional response. Touraine and Draper (1934) focused on ambivalence, Wolff (1937) pointed to frustration and resentment, and Mitchell and Mitchell (1971) looked

at ambiguity. All models suggest a constitution-environment interaction, thru which the etiology of an attack can be traced.

Because of these basic similarities, these models will be considered as one general theory of migraine causation. It will be referred to as the model of constitutional causation.

Comparison of Psychological and Constitutional Theories

There are several comparisons between the psychological and constitutional models available. Both models assume that the immediate cause of a migraine attack is an extreme emotional reaction. However, for the psychological model, the emotion must be either overt or repressed hostility. For the constitutional model, any strong emotion should precipitate an attack. In fact, even non-emotional causes of SNSA such as injections of adrenalin should precipitate an attack.

The models differ even more strongly in the postulation of the ultimate cause of migraine attacks. The constitutional theory suggests that the migrainous individual's susceptibility to attacks following a strong emotional reaction results from genetically transmitted arterial weaknesses and is unrelated to personality. For the psychological theory, the susceptibility to attacks stems from a long term disposition to either translate hostility into self-punishment (Psychoanalysis) or to react to hostility by unconsciously withdrawing

from responsibilities and confrontation (Marcussen & Wolff, 1949). Thus, the psychological theories not only require particular traits in the migrainous individual, but are also dependent upon the theory of the unconscious and secondary processes. As mentioned previously, the psychological model cannot account for unilateral onset of head pain in migraine. The constitutional model can account for unilateral onset as well as the shift to bilateral pain after onset. That is, the genetically transmitted sensitivity may be particularly high in a localized area of the cranial arteries on one side of the head. Moreover, in some pre-disposed individuals, the cranial arteries on the other side of the head may be somewhat sensitized, allowing the vaso-oscillations to generalize more readily after unilateral onset.

Both models implicate the family in migraine etiology. The constitutional model makes predictions about the family as a gene pool. The psychological model makes predictions about the family as a source of childhood experiences of a particular nature.

Summary

Several constitutional theories of migraine were presented, including a recent proposal from Mitchell and Mitchell (1971). Constitutional theories postulate a genetic predisposition at the root of migraine etiology. The suggested immediate trigger for an attack

is heightened sympathetic nervous system activity. The SNSA may result from chemical changes in the body, or from strong emotional reactions.

Several comparisons between constitutional and psychological theories were offered. To repeat a few, psychological theories invoke intrapsychic processes (repression) and specific affective responses (hostility). Constitutional theories do not utilize intrapsychic processes, and suggest that any strong affective response, regardless of content, can evoke a migraine attack. Psychological theories do not consider genetic involvement in migraine etiology, and differ from constitutional theories in this respect.

CHAPTER V

THE MATERNAL DEPRIVATION HYPOTHESIS

Background

All of the theories considered thus far have implicated personality as an agent in the causation of migraine. The constitutional theorists assume that an extreme emotional response triggers the attack. Thus, they predict that within the population of genotypic migrainous people, those least adjusted (for whatever reason) will suffer migraine as a clinical problem. The psychological theorists assume that unresolved psychosexual fixations are responsible for the characteristics reported to be common to migrainous individuals: perfectionistic, preoccupied with success and achievement, rigid, resentful, unable to express hostility, and establishing only superficial interpersonal relationships. These traits are sometimes collectively referred to as the 'migrainous personality.' Moreover, the psychological theorists assume that personality structure produces the chronic migraine condition, and overt or repressed hostility acts as the trigger for a particular attack.

This chapter will be devoted to the opposite question: does migraine have a causal influence on personality? The chapter will conclude with a discussion of the specific predictions about personality and migraine which will allow this theory to be experimentally examined.

The specific hypotheses to be developed below assume that migraine is, in fact, genetically determined. Moreover, they utilize previous suggestions in the research literature that women are 2-1/2 times as likely to have migraine as men (e.g. Barolin, 1970). If this is true, then it is quite likely that if a person is migrainous, then his mother was also migrainous. If a person's mother was migrainous, then her patterns of child-rearing behavior were strongly affected. This in turn would have effects on the child.

The Maternal Deprivation Hypothesis

Assume that a given mother is migrainous. When mother is having an attack, she is unavailable and unresponsive. This has certain immediate implications for a single episode. Even stronger implications stem from the fact that the deprivation experience is episodic and hence exists in the context of ordinary and presumably warm mother-child contacts.

Consider first a single episode from the child's point of view. Mother is emotionally cold. If the child is seeking contact, then mother is 'rejecting' him. If the child is afraid, then mother does not comfort him and tell him that his fears are unjustified. She leaves his fears unchallenged. Even if the child feels hostile, there is no retaliation. Thus, during the attack the child is emotionally a non-person. What of the child's behavior? His affiliative behavior is not returned and the mother's failure to respond acts as punishment. On the other hand, his distructive behavior goes unpunished. That is, the child receives neither positive nor negative reinforcement during his mother's migraine attack.

In considering the long range implications of the mother's migraine episodes, the critical fact is that these episodes stand in sharp contrast to the mother's behavior when she is well. That is, the child receives both acceptance and rejection, both reward and punishment. Only when the mother is well is there any relationship between her response and the child's feelings or actions. Furthermore, the child does not differentiate in any cause-inferring way between his mother's behavior when sick and her behavior when well. The child merely sees a large <u>random</u> element in his mother's response to him. This might be expected to have the well-known effects of partial reinforcement.

First consider the positive things which are rewarded part of the time and ignored part of the time. The child's love for his mother is partially reinforced and hence should resist extinction. Thus the child may have great difficulty in separating from his mother in later life. The child's affiliative behavior is partially reinforced and hence should also resist extinction. Thus, in later life, the child should be 'compulsively' polite and ingratiating. In an achievement-oriented home, he would become an obsessive performer. Finally, it is important to note that the element of uncertainty in the child's expectations of other's behavior will produce anxiety even when he is behaving in an approved way. This anxiety interferes with thought and lowers alertness. Thus, it is likely that the child or the later adult will (1) not be sensitive to changing circumstances (i.e., his good behavior will be perseverative) and (2) he will miss many of the subtle cues that show how his peers actually react to him.

Next consider the negative behaviors which are sometimes punished and sometimes ignored. A prime candidate is his hostility and aggression toward his mother. Partial negative reinforcement of these feelings and behaviors will make it very difficult for the child to do anything which his parents <u>might</u> disapprove of, and hence make it difficult for him to express his disagreements with them. That is, the later adolescent will have difficulty 'establishing his independence.' Furthermore, the element of uncertainty in his mother's

responses means that in later life he will not be sensitive to the fact that people's reactions vary as a function of circumstances. Thus, his social inhibitions will not vary with circumstances and he will appear to be 'rigid' and 'moralistic.'

In a nutshell, partial deprivation predisposes the child to experience acute anxiety, and develop excessive needs for love and its display. The craving for love is often expressed in the child's insistent demand for praise and attention from his parents and other people. On the other hand, his uncertainty in regard to punishment causes him to be rigidly wedded to 'correct' procedures and obsessed with self-control.

There is an additional possibility. If the child interprets his mother's behavior as 'abandonment,' then he may withdraw from overt intimate contact, and hence cease to receive close warmth even when his mother is well. Since there is no particular reason for his mother having abandoned him, he has no reason to suppose that other people will be any different. Such a child would become a person who finds it extremely difficult to be dependent upon other people. Other people would seem unreliable and inconsistent to him. Thus, he may 'overcompensate' for numerous suddenly discontinuous experiences with mother by rejecting intimate interpersonal relationships with other people.

There is one other possibility of an extreme reaction on the part of the child. If the child identifies himself as the 'cause' of his mother's attacks, he may never risk voicing his anger or resentment because of the guilt and fears of further abandonment that it may generate. As in the case of guilt over death, these feelings are never voiced and hence are never subject to disconfirmation. The child could be subject to severe guilt over its mother's migraines without the mother ever knowing what's happening.

One other point deserves mention in this case. It is likely that the child's hypothesis of personal causation would arise late enough in life that the above-described effects of maternal deprivation would already be set. However, if the child formed this hypothesis at an early stage, then the child would have a basis for differentiating between the mother's behavior when sick and when well. That is, such a child would <u>not</u> be subject to the partial reinforcement effects described above. For example, such a child need not become an over-achiever. Indeed, his guilt may instead produce depression that leads to under-achievement. Thus, such a child may not have a 'migrainous' personality. Of course deep and abiding guilt produces its own problems.

These predictions stand in sharp contrast to those made by Bowlby (1951). He predicted that maternal deprivation would lead to the development of a psychopathic personality: i.e. the very

antithesis of the love-craving, inhibited, and rigid 'migrainous' personality. Actually the contrast is misleading. Bowlby was concerned with the total deprivation of institutionalization, not the partial deprivation described above. For Bowlby's subjects, there is only the rejection, never the acceptance. Thus his boys and girls are simply alienated rather than obsessed with an uncertain acceptance from others. Bowlby's subjects were rarely punished and thus grew up uninhibited rather than obsessed with uncertain sanctions from others.

However, Bowlby (1951) does extrapolate to other cases. He suggests that the effects of deprivation will be proportional to their degree. Thus he would predict that the child of the migrainous mother would be intermediate in character development between the psychopath produced by 100 per cent deprivation, and the strong, well-adjusted morality of a child from a perfect home with 100 per cent alert response from mother. This prediction is in direct contrast with that made by the maternal deprivation hypothesis above.

Zero per cent deprivation means that all the reinforcement contengencies are perfectly consistent. Thus the prior reasoning would essentially agree with Bowlby that the normal home produces a moral, well-adjusted child. The reasoning behind the maternal deprivation model does not, however, postulate a monotonic relationship between deprivation and warmth-morality. If the relationship is in fact

continuous (and it may not be), then it is the reasoning of this paper that the relationship is U-shaped going from warm-moral-secure at zero deprivation to proper-moralistic-insecure at partial deprivation, to cold-immoral-secure at 100 percent deprivation.

Finally, it is important to note that the personality predictions above stem from the migraine of the mother, not the child. In short, the child of the migrainous mother would develop the 'migrainous personality' as a result of partial maternal deprivation. However, this does not mean that this person will also have migraine. This will only be true if he has also inherited the genetic predisposition.

Learning Deficits Related to Partial Maternal Deprivation

It was suggested above that the child of the migrainous mother would not express hostility during childhood or later in life. The observation that migrainous individuals do not express hostility is widely reported by therapists, and forms the backbone of the psychoanalytic theory: repressed hostility causes migraine. A recent report from Nemiah and Sifnos (1970) suggests an explanation for this observation that is relevant to the maternal deprivation hypothesis.

Nemiah and Sifnos (1970) reported that persons undergoing psychoanalysis for psychosomatic disorders generally manifested a

total unawareness of feelings, and were unable to put their subjective experiences into words. Psychoanalysts would explain this in terms of repression. Nemiah and Sifnos propose an alternative hypothesis: as children, these individuals failed to learn appropriate cognitive labels for identification of their feelings.

Since the mother-child contact is curtailed by the mother's migraine attacks, there is an increased likelihood that the learning of appropriate cognitive labels will not occur. Moreover, the migrainous mother may not welcome her child's anger with her when she has migraine episodes. At these times when the child is expressing his dissatisfactions, she may help him to mis-label his feelings, i.e.: "You're not angry, you're tired; go to bed!" Note too that the child soon learns that expression of anger brings loss of contact with mother. Finally, if the migrainous mother also had a migrainous mother, then she herself may not adequately label her affective states. How can a mother teach her child to recognize affect if she herself cannot?

Augmenting the Constitutional Theory of Migraine Causation

Thus, it is quite possible that partial maternal deprivation, stemming from the familial transmission of a sex-linked genetic factor,

may cause the development of specific personality features in the child. These include an inability to recognize and deal appropriately with feelings, an inability to tolerate dependency relationships, an inability to express anger, and the development of perfectionistic achievement goals.

According to the constitutional theory, any strong emotional reaction should be capable of creating heightened SNSA to trigger a migraine attack. Thus, if the above maternal deprivation hypothesis is correct, the constitutional theory leads us to the conclusion that an event which touches upon the frustrated childhood dependency needs should trigger an attack. Relevant events might be the death, divorce, or any threat of loss of the parent. A more common, and developmentally inevitable circumstance would be that period during late adolescence when the child recognizes the realistic limitations of the parent, as well as the impending separation from the family unit. Contributing to this is the psychological reaction to sexual maturation, school or job demands, and peer-group expectations for increasingly intimate relationships. All of this closes the door on returning to the home and opportunities for child-like dependency.

Even if the <u>cause</u> of migraine is genetic, the <u>result</u> of the migraine attack is secondary gain. Thus, it may be seen that the migraine attack facilitates dependency on other people, and enforces

the dependent role upon a migrainous person who might not be willing to accept it under normal circumstances.

One may ask why maternal deprivation, rather than paternal, has been specified in the deprivation model. This is simply a reflection of child-rearing practices in our contemporary society. Mothers usually have primary responsibility for the child during the early years of life, whereas fathers usually leave home during the day to go to their place of employment. Thus, debilitating headaches in the mother will provide more partial deprivation experiences for the child than will be the case if the father is migrainous. Moreover, it would be easier for the mother to compensate for the father's incapacitation in the evening after work, than for the father to compensate for the mother's attacks during the day when the child is awake.

Comparison with the Psychoanalytic Theory of Migraine

At this time, the maternal deprivation hypothesis and the psychoanalytic model may be compared. The models are similar since both identify the same important secondary gains that may be derived from the migraine attack. However, the models differ in more ways than they agree. The psychoanalytic theory hypothesizes attacks are

caused by unconscious intrapsychic processes. The deprivation model assumes that attacks are caused by genetically inherited weakness in the cranial arteries. Psychoanalysis suggests that personality determines physiological activity, whereas the deprivation hypothesis suggests that physiology determines (in part) character development.

It is acknowledged that the maternal deprivation hypothesis may seem speculative, but it is no more conjectural than the psychoanalytic theory as to why certain commonly reported personality traits might develop. The psychoanalytic theory assumes that the child fears not being able to control his hostility toward his parents for their denial of immediate gratification of his narcissitic demands. Hence the child represses his hostility to protect his parents and ultimately himself from his 'overwhelming' anger. Clearly, speculation is heavily indulged in here too.

In another respect, the maternal deprivation hypothesis is less conjectural than the psychoanalytic theory. The maternal deprivation hypothesis presents itself as only one of a large number of triggers for migraine attacks, within the constitutional model. It does not present itself as <u>the</u> unique or common trigger for attacks as does the psychoanalytic theory.

Predictions Based on the Maternal Deprivation Hypothesis

At this time, several specific hypotheses based on the maternal deprivation model can be offered:

1) The child of a migrainous mother will have the 'migrainous personality,' regardless of whether the child has migraine or not.

2) Migrainous children of migrainous fathers are much less likely to have the 'migrainous personality.'

3) The 'migrainous personality' should also be found among the children of mothers who are alcoholic, or epileptic, or more generally in the children of any mothers whose response to her children is arbitrarily interrupted at frequently occurring intervals.

Summary

Theories previously considered have implicated personality as an agent in the causation of migraine. The maternal deprivation hypothesis suggests that migraine may influence the development of personality. Relevant portions of Bowlby's work on maternal deprivation effects (1951) were presented. Learning deficits in

connecting affect with cognitive labels, that may result from partial deprivation were also considered.

It was suggested that the child of a migrainous mother might receive discontinuous care (partial maternal deprivation) as a result of mother's migraine attacks. If this occurred, it might have consequences on the future personality development of the child. Specifically, the child may be predisposed to develop the personality traits collectively referred to as the 'migrainous personality.' The utility of augmenting the constitutional theory of migraine causation with the maternal deprivation hypothesis was explored. A comparison between the maternal deprivation hypothesis and the psychoanalytic theory of migraine causation was offered.

CHAPTER VI

ADDITIONAL RESULTS OF MIGRAINE RESEARCH

Information regarding the frequency of migraine incidence and duration of attacks was presented in Chapter I. Moreover, research reports on the physio-chemical and genetic aspects of migraine were considered in Chapter II. The present chapter will present a review of migraine parameters that have been explored.[1] Both the merits of the research designs and the validity of the resulting conclusions will be considered.

Several alternative conceptual models of migraine causation have been presented in Chapters III, IV, and V. The research implications for support and validation of these migraine theories will be considered in this chapter.

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[1] A more comprehensive listing of migraine research is offered by Wolff (1972). Wolff's book contains a collection of publications by Wolff and other authors, some of which are cited in the present paper by their original individual articles. Rather than duplicate the scope of the review provided by Wolff (1972), the present chapter offers a more critical analysis of the important studies in the field. The presentation in this chapter is basically consistent with Wolff (1972).

Age of Onset

Martin (1928) reported that onset in 80 to 90 per cent of migraine cases occurs before age 25. Moench (1961) reported that the majority of reported cases start between the ages of 16 and 35.

In two studies of migraine in college students and staff, Mitchell and Mitchell (1971) reported mean age of onset to be 14 (N = 17) and age 19 (N = 20). In Furmanski's study of 100 migrainous adults (1952), initial attacks appeared before puberty in seven per cent, during puberty in 35 per cent, during "young adulthood" in 48 per cent, and after age 40 in ten per cent. In the study by Touraine and Draper (1934) of 50 migrainous Ss, onset occurred before age ten in 22 per cent, between ages 10 and 14 in 20 per cent, between ages 15-19 in 26 per cent, between ages 20 and 29 in 28 per cent, and between ages 30 and 39 in four per cent. Knopf (1952) found age of onset in 30 migrainous Ss to occur before age 10 in 20 per cent, between ages 11 and 19 in 50 per cent, between ages 20 and 29 in 17 per cent, between ages 30 and 39 in 10 per cent, and after age 40 in three per cent.

In review, onset most commonly occurs during late adolescence and the following years of the twenties (Wolff, 1972). Onset in the late 30's and 40's is relatively rare. Two studies indicate that

childhood migraine is more common than suggested by other reports. Knopf (1952) questioned the validity of her own data:

> We must be cautious, however, when questioning patients with regard to the time of onset of their illness; they are frequently so convinced that they have suffered from it for a long time that they are only too ready to say: "I have had it for ever since I can remember."

The reports of distribution of age of onset of attacks appears inconsistent with a hormonal or physiological maturation theory. Some cases exist showing onset at every part of the life cycle. Moreover, the bulk of onset occurs several years after onset of puberty.

The information on age of onset offers little discrimination between various conceptual models of causation. The results are quite in keeping with the psychological models outlined earlier: performance expectations and personal responsibilities increase markedly during the years of early adulthood. The model of Marcussen and Wolff (1949) is still viable here. These findings on onset also appear congruent with the psychoanalytic formulations about the importance of separation threats, frustrated dependency needs, and repressed hostility. Reports of onset beyond age 40 may indicate those cases where onset was triggered by the death of a parent whom the migrainous person took care of into old age.

The constitutional models are also compatible with these results. Touraine and Draper (1934) suggested that anything which

threatens the emotional balance in the ambivalent relationship with mother should trigger an attack, such as the individuation and separation from the parents that normally occurs during late adolescence and young adulthood. The model of Mitchell and Mitchell (1971) makes similar predictions since the increased performance expectations and personal responsibilities of adolescence can be extremely anxiety provoking. However, information on developmental physiology, which might show that SNSA increases in adolescence (or doesn't) has yet to be collected.

The data is also compatible with the maternal deprivation model. The realization of decreasing opportunity for child-like dependency as the individual approaches adulthood may tap into the developmental experiences of the pre-migrainous child to evoke SNSA changes that regularly exceed the vascular stability for the first time. Depending on the socio-economic status, age of marriage, level of education, the years from 14 to 30 are those in which the individual may foresee his long range failure to attain fundamental goals.

It should be noted that onset does not refer to the first headache, but rather the first series of headaches that are identified as a problem (i.e. the onset of <u>chronic</u> headaches). Thus, the 14 to 30 age period may mark the onset of <u>chronic</u> feelings of anxiety, anger, and frustration, which lead to frequent migraine attacks that are identified as onset.

Sex Differences in Incidence

Godinova (1967) reported migraine to be four times as common in females as in males. Childs and Sweetman (1961) found migraine incidence to be 2-2/3 times as great in women than in men (N = 1607). This finding was similar to a previous report by Moench (1951), who wrote that migraine incidence was 2-1/2 times as frequent in women. Barolin (1970) concluded that migraine was "clearly more common" in women. In examining twelve previous studies, involving 1930 migrainous Ss, Allen (1927) noted that females outnumbered the male Ss by a ratio of about 2.5 to 1.

Support for the notion of sexual differences in incidence is usually derived indirectly from the balance of men and women who seek treatment. Only one study (Allen, 1927) directly explored the sex differences in incidence, without relying on treatment records. Allen (1927) studied migraine incidence in the members of a county medical society and their wives (103 males and 92 females). Allen found migraine incidence to be equally common among men and women. He suggested that women were more apt to come to clinical attention because their headaches were more severe. However, this report is highly questionable. Allen reported overall migraine incidence to be 52 per cent, in contrast to the widely accepted estimate of eight per cent incidence in the total population. The author's inflated estimate of

incidence may have resulted from his use of sophisticated Ss as well as inaccurate diagnostic criteria.

Of the Ss in studies which drew their samples from applicants to hospital clinics, approximately 70 per cent were women (Lance & Anthony, 1967; Touraine & Draper, 1934; Knopf, 1952). This would seem to corroborate the reports by Godinova (1967) and Moench (1951) of greater incidence in women. However, this may reflect the sex balance of applicants to hospital clinics rather than migraine incidence in the total population. Men may be more hesitant to seek treatment due to feelings of unmanliness about 'sick' headaches. Moreover, men who seek treatment may utilize a family physician who offers a definite appointment time that does not conflict with business hours. On the other hand, since it interferes with his job performance, the family breadwinner is more likely to seek immediate help when onset of attacks occurs.

Even if incidence were equal between the sexes, women may seek treatment more often, since there is some indication that their attacks are more frequent and intense. One possible cause may be related to menstruation in women, and will be considered subsequently. Thus, it is not clear whether the above statistics accurately reflect a sex difference in migraine incidence.

Cyclical Hormonal Changes

The findings that migraine onset is most common during late adolescence and young adulthood and the suggestion of sex differences in migraine incidence led researchers to collect data on the relation of migraine to menstruation and pregnancy in women. Of the 22 women participating in Knopf's study (1952) six had onset more than one year before menstruation, nine had onset during the first year of menstruation, five had onset more than one year after the start of menstruation, and two could not remember.

Several factors argue against concluding that onset is related to hormonal changes in women. Thirty per cent of her sample failed to display this relationship. Moreover, because Knopf used the suggestion of a menstrual relationship to migraine attacks as one of several criteria in selecting her Ss, her finding that 70 per cent of Ss report a connection between menstrual cycle and attacks might not be true of a normal population. Instead, it is a greatly inflated estimate.

However, Knopf also pointed out that these particular women were characteristically emotionally unprepared to assume an adult female role. Thus, it could be argued that onset was not hormonally determined, but was related to events which threatened their opportunities for dependency by thrusting them into a more adult role.

Thus, the deprivation model could also explain these results. On the other hand, the fact that seven of 22 women did not report a menstral relationship to migraine indicates that neither a hormonal theory nor the maternal deprivation hypothesis are adequate explanations for the age of onset in all women.

The question of onset of chronic migraine is distinct from the question of whether specific attacks in later life occur as a function of cyclical hormonal changes. Thirteen of the Ss in Knopf's study had been pregnant. Only four of these women experienced relief from migraine during pregnancy. For 70 per cent of the women, the termination of monthly hormonal changes did not bring relief from migraine. Moreover, only one of the 22 women reported her attacks occurred solely in response to menstruation. Seven others reported that fatigue, emotional strain, and anger were required in addition to menstruation, and ten reported that strain, anger, and worries were sufficient cause in themselves.

One study did find that pregnancy brought migraine relief. Lance and Anthony (1967) found that in 252 pregnancies of 120 migrainous women, pregnancy brought a higher incidence of relief to women in whom there was a relationship between menstruation and attacks, than those in whom the relationship was absent. This difference was statistically significant.

However, there is other data which tends to refute the suggestion of hormonal changes being causal in migraine. In the study by Touraine and Draper (1934), 16 of the 37 women participating (43 per cent) reported no menstrual connection with attacks. The remaining women reported they never had their period without also having a migraine attack. However, all of these women reported attacks occurred at varying intervals during the month.

Of the 37 women in this study, 15 had been pregnant. Seven of these women, reporting menstrual relationships to attacks, experienced complete or partial relief during pregnancy. Of the eight remaining women who experienced no relief during pregnancy, six had reported a menstrual relationship to attacks. Thus, 50 per cent of women who reported a menstrual relationship experienced relief during pregnancy, while the other 50 per cent did not.

In conclusion, periodic hormonal changes appear related to the occurrence of attacks in only a portion of the female population. There are no apparent additional characteristics which identify or predict which women this occurs in.

It does seem possible that hormonal patterns may function as an additional trigger for migraine attacks in women that is not present for men. Relevant biorhythm information on men is not available at present to evaluate this suggestion. If it is true, it would be

congruent with those reports that find migraine more common in women than in men.

Strong emotional responses are indicated as co-contributors and sometimes as sufficient stimulus in themselves for an attack. A purely hormonal theory is not warranted, although hormones appear to function as one of a number of possible triggers for migraine in women. Many women suffer drastic water buildup at some point in their menstrual cycle. At this point they may be extremely irritable, and hence vulnerable to migraine attacks. One should also consider the woman's emotional reactions to her body functions. Extreme shame over menstruation could be the emotional trigger for an attack. This would be particularly relevant to older studies.

Migraine and Epilepsy

Early theories of migraine etiology posited a link between migraine and epilepsy. Collier (1928) suggested that both resulted from metabolic disturbances or kidney dysfunction. In his review of the relevant literature, Pearce (1969) cited the following studies:

TABLE 3

Incidence of Epilepsy in Migraine Patients[1]

Study	Sample Size	Number diagnosed epileptic	Per cent epilepsy
Selby and Lance (1960)	348	38	11
Lennox and Lennox (1960)	415	27	6.5
Lees and Watkins (1963)	354	9	3.9
Lance and Anthony (1966)	500	8	1.6
Lance and Anthony (1966)	100 tension headache controls	2	2.0
Frequency of epilepsy in general population			0.5

[1]Cited by Pearce (1969).

Pearce concluded that epilepsy occurs with increased frequency in migraine sufferers. He dismissed the results of the Lance and Anthony study (1966) which found no significant difference between migraine and non-migraine Ss in epilepsy incidence. Pearce did this because epilepsy incidence reported by Lance and Anthony was four times higher in their control group (2 per cent) than the reported incidence in the general population (.5 per cent).

However, Pearce's dismissal of Lance and Anthony's report (1966) seems premature. The fact that control group incidence was higher than expected may indicate that the diagnostic criteria Lance and Anthony used differed from previous investigations of incidence in the general population. Since Lance and Anthony used the same criteria for diagnosis of epilepsy in migrainous and non-migrainous Ss, their results still suggest no significant increase of epilepsy among migraine sufferers. Moreover, the other three studies cited by Pearce reported much higher epilepsy incidence in migraine patients than did Lance and Anthony. Since these earlier studies did not report control group norms, the possibility that the high epilepsy incidence found in migrainous Ss reflected extremely broad and inaccurate diagnostic criteria cannot be ruled out. At the present time, increased incidence of epilepsy among migraine sufferers has not been clearly established.

Electroencephalographic Abnormalities

Stimulated by early suggestions that migraine was related to epilepsy, researchers have examined the EEG recordings of migraine sufferers for indications that some type of brain dysfunction was responsible for migraine attacks. The EEG research is of two basic types: studies of migrainous people during attack-free periods, and studies of migrainous people following attacks. The results of the latter type studies will not be considered, because they are basically studies of the long-term symptomatology of hemiplegic and ophthalmoplegic migraine (see subclassification of migraine types, page 8). Thus, this research is not relevant to our consideration of classic and common migraine. A thorough review of this literature is provided by Wolff (1972).

In the studies of EEG during attack-free periods, the abnormality of interest has been the presence of increased "slow" brainwave activity in the 3 to 6 cycles per second range. This has been labeled "dysrhythmic" migraine (Cohn, 1949).

Selby and Lance (1960) reported 122 abnormal EEG records in their study of 459 migraine patients (26 per cent). Cohn (1949) reported that about half of his 83 migrainous Ss displayed excessive slow wave activity between attacks. Heych (1956) reported only

20 per cent of 62 Ss displayed slow wave dysrhythmias. Strauss and Selinsky (1941), Dow and Whitty (1947), and Panzani (1959) have reported from 60 to 75 per cent abnormal records from migraine patients. However, these studies involved the use of hyperventilation or visual stimulation by lights while the EEG recordings were made, and their criteria for abnormality was more liberal than that used by Selby and Lance (1960) or Heych (1956). It should be noted that EEG studies generally do not employ control groups in their design, and rely instead on reports of the incidence of slow wave abnormalities in the general population.

Sacks (1970) summarizes the relevant research as follows:

> It is evident that these studies have failed to uncover any clear and consistent EEG abnormality peculiar to migraine. Lennox and Lennox (1960), summarizing a 20 year experience of such recordings conclude that there is 'nothing distinctive' in the tracings of migraine patients; one cannot, for example, diagnose migraine on the basis of an EEG record.
>
> It has been impossible to define any EEG abnormality which bears a specific relation to migraine, as wave-and-spike patterns do to epilepsy. At most, there is a questionable statistical increase of slow wave dysrhythmias beyond the 15 to 20 per cent incidence of these in non-migrainous populations (1970, p. 206).

Barolin (1970) similarly concluded from his study of 450 migraine patients that there was no indication that abnormal EEG was causal of migraine attacks.

Personality and Case History Studies

In Chapters III, IV, and V, several theories of migraine causation were presented. The personality of the migrainous person was implicated in the migraine syndrome by each theory. In the present section, the data which spawned these theories will be presented. The focus here will be on examining the support for each theory that is available in the research data, rather than explicating the theory itself.

Fromm-Reichmann (1934) reported the personality dynamics of eight patients she had treated via psychoanalysis. The 'data' of her report consisted of her interpretations of behavior and free associations of two men and six women, during an unreported number of hourly sessions. She concluded that migraine was caused by repression of hostility directed at consciously beloved persons. However, she gathered no control group or inter-rater reliability data.

Furmanski (1952) studied the similarities between character patterns of 65 women and 35 men with migraine, according to the psychoanalytic principles and concepts of character formation. He noted that 10 per cent of Ss reported migraine attacks during sleep, and 19 per cent reported attacks upon awakening. He interpreted this as indicative of unconscious hostility, since hostility is thought to be less controlled during dream life and closer to

conscious experience. However, since sleep takes up 33 per cent of a person's daily life, it is not very surprising that 29 per cent of Furmanski's Ss report attacks during this time. Furmanski also noted strong indications of repressed hostility in the content of free associations during therapy sessions, but he did not utilize a control group for comparison purposes. Thus, these observations do not conclusively implicate either repression or hostility in migraine causation.

The fact that 35 per cent of his Ss reported initial onset of attacks in puberty was interpreted by Furmanski to coincide with the marked increase in self-assertiveness in adolescence, which intensifies the person's ambivalence. However, Furmanski (1952) did not account for onset in the remaining 65 per cent of his Ss who did not being during adolescence.

Furmanski (1952) said that Ss developed strong narcissistic traits in reaction to early deprivation of parental affection and attention. He reported that Ss displayed the ultra-moralistic traits, characteristic of a rigid superego. Furmanski stated that this latter trait resulted from overly strict child-rearing practices by the parents.

Examination of his conclusions brings out an important shortcoming of Furmanski's paper: his failure to separate his observations from his inferences. His primary data was his observations of

personality traits, such as 'narcissism.' He then used his psychoanalytic training to derive the conclusion that migrainous patients suffer from early deprivation of parental affection. In fact, his conclusions about parental treatment appear to be pure inference, not even substantiated by the recollections of his patients.

It should be pointed out that the actual behaviors that Furmanski cited as evidence of traits such as "narcissism" are relatively universal among migrainous and non-migrainous people, alike. For example, starting tasks right away, anxiety in anticipation of impending failure or criticism, resentment to being under pressure, sensitiveness to interference, and the desire to collect 'coproic symbols' such as money, coins, stamps, and art. While the author provides a rationale for finding these traits in migrainous individuals, he makes no attempt to show that these traits are found any more often in migrainous individuals than non-migrainous individuals. Thus, he offers no evidence that these traits play a causal role in the etiology of the migrainous person.

Another shortcoming in this study is the lack of theoretical consistency. The author claims that his patients were developmentally fixated at both the oral and anal stages of psychosexual development. Furthermore, he reports that almost all Ss had also developed mature adult sexual relationships that suggest a genital level of psychosexual functioning!

The studies by Fromm-Reichmann (1934) and Furmanski (1952) are similar in several respects. They both suggest the prominance of repressed hostility in the migrainous personality. Both authors suggest the migrainous person is strongly ambivalent in his feelings because of strong parental injunctions against expression of anger and the threat of loss of love if it occurs.

However, the studies are also similar in methodological problems which weaken their conclusions. In particular, no control group or inter-rater reliability data is available. Yet these facts are critical since all data reported is highly subjective and dependent upon the interpretations of the therapist. Furthermore, the strong theoretical commitments of the therapists make the likelihood of interpretation bias rather great.

In a similar style, Sperling (1952) reported conclusions based on the results of psychoanalytic treatment of 14 migrainous adults and nine migrainous children. Sperling does present fragmentary data in the form of transcripts of actual sessions. However her article is largely a reflection on past experiences in therapy, rather than a report of controlled observations. Most of the factors that led to her conclusions are not presented in the text of the article. Sperling concluded that migrainous individuals were orally fixated. This fixation was reflected in the failure of all 14 adults (nine women and five men) to attain a genital level of psychosexual

development. Particularly in male Ss, preoccupation and practice of oral sex was its main feature of expression. The author also said she found an extreme intolerance to denial of narcissistic needs and impulse gratification.

The lack of control group data by which to judge normal adult sexual behavior makes Sperling's conclusion about the restricted psychosexual development of migrainous people tenuous. It is likely that the author would identify a large portion of the general population as lacking a mature genital sexuality. Thus, she did not demonstrate that the failure to establish "mature" sexuality is a specific feature of migraine.

The studies by Touraine and Draper (1934) and Knopf (1952) differ from the previous studies. These reports derive from data specifically collected for the purpose of the study, rather than from retrospective interpretations of therapy sessions.

Touraine and Draper (1934) studied the personality features of 50 migrainous Ss (37 women and 13 men). Much of the data they collected, such as age of onset, duration of attacks, menstrual relationships in women, and family relationships, has been presented previously. Several more controversial aspects of their report will be presented here.

The authors reported that none of the 37 women experienced relaxation or satisfaction from intercourse, and frequently refused to partake. Fourteen women volunteered report of their sexual dissatisfactions. Although it is likely that many non-migrainous women in 1934 would have been judged to be sexually inhibited, it is doubtful that a control group would have unanimously reported sex unsatisfying. While the report that seven women possessed no sexual information before the age of 20, and three were unaware of the birth process until their own pregnancy suggests an avoidance of sexual curiosity in migrainous women, the lack of control group data prohibits accepting this finding as characteristic of migraine.

From their comparison of the 50 case histories, Touraine and Draper noticed a pattern of "unusually intense" attachment to the parents, especially to the mother. They report that the regularity and pre-eminence of this attachment overshadowed all other relationships and attracted attention at the outset of the study. Initial onset of migraine reportedly occurred in situations involving the loss of home protection, which required the individual to stand alone and take adult responsibilities. They concluded that the case histories portrayed a constant conflict between the wish to separate from the mother and a compulsion not to leave her. The mother, in turn, appeared to withhold recognition of the child's individuality. Invariably, histories related situations in which the adult was not

able to separate from the parents, often to the extreme detriment of peer relationships. Migrainous adults sometimes married, but they remained emotionally dependent on their parents. Often the initial onset of attacks occurred at the time of the parent's death.

Although it is probable that strong attachments are not limited to migrainous people, it is unlikely that the degree of parental symbiosis and dependency observed would be found in 50 people selected at random in the general population. However, the absence of a control group for comparison purposes weakens the strength of their findings.

Knopf (1952) conducted a study similar to the above one, which involved 30 Ss. Nine of the 22 women in the study were reportedly unprepared and uninformed as to adult sexuality. Four were unaware of menstruation at the time of onset. Knopf placed particular emphasis on this as evidence of restricted psychosexual development. She also found that 25 of 30 Ss perceived their childhood as unhappy, or were undecided about their childhood. Fourteen of the 30 Ss reported being very sickly or nervous as a child. However, the significance of this cannot be properly evaluated without control group data.

Wolff's (1937) examination of the personality functions of 46 migrainous Ss (25 women and 21 men) bears similarities with both groups of studies reported above. Although he did not use free-association or other therapeutic tools to collect data, he made no

attempt at statistical analysis. The personality tratis identified were subjectively found in most Ss to varying degrees, although no S demonstrated the entire range of traits. The traits Wolff identified represented group approximations and did not reflect a rigid profile attributable to all, or even solely to migrainous people.

Wolff reported that in recalling their childhood, his Ss prominently reported contrasting qualities of character. The same person reported being courteous and accommodating at times, while being openly defiant and rebellious at others. His Ss recalled being given responsibilities at an early age, and remembered themselves as being thoughtful and responsive to the wishes and needs of their elders. Strong attachment to mother was sometimes noted, but generally it was no deeper than the typical close childhood identification with the mother.

In adult life, character traits became more accentuated. Wolff (1937) judged 90 per cent of his Ss to be unusually ambitious and preoccupied with achievement and success. They attempted to dominate their environments by forceful demands, tyranical moody periods, or by acquisition of money, power, and distinction (judged to be the more successful method). Ss had difficulty allocating responsibility and often preferred to do tasks themselves rather than risk being disappointed by others. Wolff judged them to be quite inflexible, often becoming upset when forced to operate in disorderly

or hurried circumstances. Wolff judged two-thirds of his Ss to hold intense lasting resentments, finding it difficult to forgive or accept the shortcomings of other people. Moreover, when they deviated from their own rigid standards, they engaged in punitive self-accusations and self-punishment. They were unaccepting of their own physical limitations, foregoing sleep and relaxation in pursuit of work productivity that Wolff felt exceeded the limits of common sense.

In the area of sexual adjustment, Wolff judged more than 80 per cent of the women to be sexually dissatisfied and often sexually dysfunctional. Although he judged the sexual activity of his male Ss to be "adequate in nature and frequency," Wolff (1937) felt that more than a third of these men revealed incomplete sexual adjustment in their desire for "excessive dependency" when having a migraine attack.

Several remarks on Wolff's report (1937) are in order. Clearly, the childhood traits reported can be observed in all children at times. Moreover, the adult traits reported are evident in migrainous and non-migrainous people alike. Wolff recognized this and he differs from the psychoanalytically oriented writers in that he does not suggest that certain personality traits are causal in migraine. Rather, he assumed that these were common traits that operate in migrainous individuals to evoke strong emotional responses.

It is interesting to note that the traits identified by Wolff (1937) represented attempts by the individual to control the environment

and circumvent dependency feelings, rather than traits specifically related to latent hostility. Wolff's report (1937) seems more theoretically consistent with the maternal deprivation model than the psychoanalytic model.

In conclusion, the results reported in this section are most clearly consonant with the psychoanalytic model of migraine causation, or with the maternal deprivation model. In particular, Touraine and Draper's finding (1934) that migrainous people had very ambivalent feelings towards their mother is not surprising in light of the maternal deprivation hypothesis. The ambivalence may develop from the child's desire to be with his mother after she has been unavailable to him for a while, together with the child's anger at the memory of being abandoned by her. Moreover, the ambivalent relationship may be perpetuated in adult life by the person's fantasy that they might yet obtain the gratification from mother that she did not supply previously. In the same way, sexual maturity may be denied by the migrainous adult in the perpetual hope that the long sought-after parent-child relationship might yet occur.

However, these studies are not without their methodological problems. Although these are some consensual validation for personality traits common to migraine sufferers, there is no evidence for a causal link between personality and migraine causation. No study attempts to test the suggested links between affect, defense

mechanisms, and migraine attacks. Most of the support offered to the psychoanalytic formulations stems from the trait interpretations and trait-based inferences of the writers and not from primary observation itself. In no case does the nature of the data permit independent evaluation by the reader.

Judgments of restricted sexuality seem to be made with a theoretical model of mental health (i.e. 'complete' psychosexual development) rather than a group of randomly selected individuals, as a reference group. Even so, sexuality is the largest area of disagreement between reports! Sperling (1952) said that migrainous individuals were orally fixated, whereas Furmanski (1952) said they were both orally and anally fixated. Sperling reported that migrainous adults failed to achieve genital psychosexual development, whereas Furmanski reported his patients to have achieved adult sexual development. Knopf (1952) reported that 73 per cent of the women in her study were unprepared for adult sexuality, whereas Touraine and Draper (1934) found 100 per cent of their women to be unprepared.

Although there is a compelling quality to the case material collected in some of the studies, this highly select material may be very misleading. Most investigators used migraine as a vehicle to validate some of their firm beliefs about the nature of psychopathology. From the amorphous data of long case histories and free-associations in therapy sessions, they could always find some pieces that would

support their contentions. But this might have been equally true with any other hypothesis that they started with. Indeed, this may well explain many of the inconsistencies among them. Thus, these conclusions about personality structure will probably not be accepted by readers who are unsympathetic to psychoanalytic theory; all the studies lacked proper controls.

Stress Reactions

Marcussen and Wolff (1949) studied detailed histories of 20 migraine patients for the 24 hours preceding a migraine attack. They found in almost every case, an episode had occurred in which S reacted with rage and resentment to which he was unable to give full expression. Marcussen and Wolff presented several case histories to illustrate their findings.

In one case, a harried migrainous mother experienced relief from attacks when she confessed difficulties in rearing her child to a friendly sympathetic physician, but sustained an attack a week later when the same physician was stern and unsympathetic.

In the second case, an aspiring biologist was working hard to complete a long-term research project. The biologist sustained an attack when he was reproved by his superior for not completing his writeup earlier.

Marcussen and Wolff (1949) point out that both patients were under considerable stress for prolonged periods, during which time tension and fatigue accumulated. The headache was precipitated by an anger-provoking event to which the people felt helpless to do anything about. The authors suggest that this is the actual trigger behind the migraine attack.

However, there is nothing in their report that proves that it was the inability to express anger that produced the migraine. First there was no check to see if similar events did not produce migraine attacks at other times. Second, there was no check to see that anger was not expressed in the cases cited. It is possible that this aspect of the case histories happens to reflect how anger is handled in virtually all situations in our modern society.

Marcussen and Wolff (1949) cite two other cases which serve to illustrate their hypothesis in another way. One is the case of a woman who experienced attacks weekly. She remained attack-free for a four-month period after she received placebo pills with words of kindness and reassurance. The woman reported feeling more relaxed and secure during this time. In the case of the housewife and the biologist, the nature of the headaches was established as migraine by administration of ergotamine tartrate. However, in this case of the placebo cure, the tartrate test was not administered. Thus, the

alternative diagnosis of hysteria or tension headache cannot be ruled out.

They also present the case of a hard-working musician whose migraine attacks ceased when a 15-year-old case of syphilis destroyed his compulsive performance strivings. Marcussen and Wolff inferred from this that developments which reduce tension, anxiety, frustration, and resentment will reduce the likelihood of an attack. However, they failed to consider the possibility that the advanced physiological damage caused by syphilis was the primary cause for cessation of attacks, and that the personality changes were irrelevant.

Conditioning

Mitchell and Mitchell (1971) hypothesized that migraine was caused by physiological over-reactivity of the sympathetic nervous system to stressful events in the environment. They believed they could reduce the frequency of migraine attacks if they increased the patient's ability to control his emotional reactivity. They reported a number of previous studies in which behavior therapy techniques were successful in reducing the frequency and severity of migraine attacks.

Mitchell and Mitchell (1971) employed three different types of therapy. First they taught Ss a modified form of Jacobsen's

(1938) progressive relaxation technique. They also used Wolpe's (1958) method of systematic desensitization with topics chosen by each S. They also used what they called 'assertive' therapy. 'Assertive' therapy involved daily tasks designed to change S's behavior in particular 'minor' problem areas, such as sexuality or interpersonal relationships.

All Ss attended individual 30-minute sessions which presented Mitchell and Mitchell's model of migraine as a psychosomatic disease with learned psychological mechanisms. All treatment condition (non-control) Ss received 15, one-hour sessions, occurring twice a week. All sessions were conducted by the senior author. The pre-treatment measurements covered the eight weeks preceding the experiment. Post-treatment testing occurred twice, at intervals of eight and sixteen weeks.

All told four experimental groups were formed. Eight Ss were given no treatment (control group). Seven Ss received only extensive practice at progressive relaxation. Five Ss were given both progressive relaxation and systematic desensitization training (although how much time was spent in each activity was not reported). Twelve Ss received progressive relaxation, systematic desnsitization, and assertive therapy (with no indication of how much time was divided among them).

TABLE 4

THE RESULTS OF BEHAVIOR THERAPY TREATMENT OF MIGRAINE
(From Mitchell and Mitchell, 1971)

Treatment	N	Mean Decrease in Number of Attacks	Mean Decrease in Number of Hours Duration
No treatment (control)	8	-2.2	-0.4
Progressive relaxation	7	-2.0	0.0
Progressive relaxation plus systematic desensitization	5	-5.8	-3.0
Progressive relaxation, systematic desensitization, and assertive therapy	12	-8.9	-4.6

Table 4 presents their basic results. Progressive relaxation alone had no apparent effect. Relaxation plus desensitization had a marked effect in reducing migraine. Unfortunately since no group was run with desensitization alone, there is no way of knowing the extent to which the results for the two treatments together represent an interaction of the two. When assertive therapy is added to relaxation and desensitization therapy, the decrease in migraine is about half again as great. This suggests that assertive therapy is about half as effective as desensitization. However, assertive therapy was neither tried alone, nor given in combination with only desensitization. Thus, there is no way of knowing what portion of the increase due to combining all three methods was due to double or triple interactions among them. [Mitchell and Mitchell (1971) also report two other studies in which the triple combination treatment was superior to a no-treatment control group.]

Thus, Mitchell and Mitchell (1971) have definitely established that migraine attacks can be reduced by some form of psychotherapy. Furthermore, therapy of the group receiving relaxation plus desensitization was oriented toward reducing emotional reactivity. Thus, there is some support for Mitchell and Mitchell's argument that it is a strong emotional response which triggers the migraine attack. On the other hand, since no measure of physiological activity was made, the support for the SNSA hypothesis is at best indirect. Furthermore,

it is solely a matter of conjecture that the effect of assertive therapy was achieved by reducing the number of occasions which might evoke strong emotional reactions. Indeed, one might suppose that the 'real life practice' of assertive therapy produced strong emotional experiences.

Mitchell and Mitchell (1971) apparently gathered some data bearing directly on the issue of emotionality, but their report is sketchy and incomplete. Some Ss were given the Cattell Anxiety Scale, and showed no decrease in anxiety as a result of treatment. Yet, the authors report that some Ss showed 'considerable' decreases in anxiety in specific 'minor' areas such as sex, morals, academic achievement, and social interaction. If one were to take these inconsistent reports at face value, it would appear that anxiety played no major role in migraine. This would be very damaging to their theory as stated. However, they report no attempt to check other strong emotions such as anger, which would have the same effect on SNSA.

These results appear damaging to the psychoanalytic model. The classic assumption is that migraine is a deep-seated unconscious mode of punishing the self for hostile impulses. Thus successful treatment would seem to presuppose providing the patient not only with the insight that the underlying cause of his migraine is repressed hostility toward his parents, but also the opportunity for a full abreaction of that repressed hostility. Clearly Mitchell

and Mitchell (1971) did neither. Their therapy was geared to the 'here and now,' avoiding confrontation with 'repressed' memories of past events.

The results also appear damaging to the modified psychoanalytic theory of Marcussen and Wolff (1949). They postulated that migraine is an unconscious malingering for the purpose of relieving the patient of responsibility without guilt. That is, they postulated that migraine attacks are produced by high 'energy' use and 'fatigue.' Yet relaxation training failed to reduce migraine in the Mitchell and Mitchell study, and there was a positive effect from 'assertive' therapy, which asked the S to devote extra time (from an already presumably punishing schedule) to high risk and highly stressful change experience.

Summary

In this chapter, the results of research on migraine parameters were presented. By way of brief review:

1. Onset of migraine most often occurs during the years of late adolescence and young adulthood.

2. Migraine is reported to be about twice as common in women as in men. Moreover, attacks are reported to be more severe in women.

There is some indication that attacks in some women may be triggered by changing hormonal levels during the menstrual cycle.

3. The suggestion that migrainous people are more likely than non-migrainous people to have epilepsy has not been confirmed by research evidence.

4. The suggestion that migraine is caused by abnormal brain activity (slow-wave dysrhythmia) has not been confirmed by research evidence.

The implications of these results for the psychological, constitutional, and maternal deprivation theories were discussed. Moreover, the review also covered the actual research from which these theories of migraine causation developed. As it happens, even the research done by the theorists themselves is equivocal as regards the presence or absence of any connection between migraine and particular personality structure.

CHAPTER VII

DISCUSSION AND CONCLUSIONS

Discussion of Past Research

In the previous chapters, the theories of the migraine trigger, and the results of research on various migraine parameters have been presented. The present chapter will focus on the implications of the research and the common methodological problems that were encountered. Suggestions for future research topics and methodologies will also be presented.

Overall, the research on migraine was notable in its inability to critically evaluate any of the conceptual models of the trigger for the attack. This failure stems from three sources.

For one, the studies were prone to serious design flaws. The personality and case studies were inconclusive due to failure to include control groups in their design. This shortcoming was common to the reports from Touraine and Draper (1934), Wolff (1937), Weber (1932), Fromm-Reichmann (1939), Furmanski (1952), Knopf (1952), and Sperling (1952). The strength of the conclusions of these reports was also

diminished by the absence of multiple observers. This would have reduced the potential for rater biases effecting the outcome of the studies. Since the authors did not distinguish between the empirical observations and their theoretical conclusions, it is probable that their conceptual biases determined which portion of a case history would be considered "significant" and which portion would be ignored.

The foregoing discussion actually questions the utility of using ongoing therapy as an experimental design for exploring migraine parameters. Obviously, therapists do not undertake the treatment of 'normal' individuals in order to establish a control group personality profile. However, the problem created by the lack of control group data in the case studies was not an unavoidable one. Therapists do observe non-migrainous people in treatment. Although therapists report certain character traits and personality dynamics in their migrainous clients, they apparently never check to see if these same traits are also present in their non-migrainous clients. If migrainous and non-migrainous clients display different personality dynamics, and migrainous clients are homogeneous with respect to these traits, then at least one could say that that therapist found certain traits to be associated with migraine attacks. If the same dynamics were found in all his patients, one could conclude either that the syndrome found is what the therapist reads into all his clients, or that only patients with that syndrome stick with him as clients.

Another methodological problem remains. Since the therapist is not double-blind, the therapist's behavior may produce rather than 'uncover' certain personality traits in migraine patients. Similarly, even if the therapist's behavior could be proven to be uncorrelated with the client's migraine status, the therapist's interpretation of behavior and free-association of migrainous clients may be biased by his prior hypotheses about migraine per se. This problem could be ameliorated if there were cases in which the personality dynamics were uncovered (or produced for that matter) before migraine was found in the patient.

Since previous personality studies have been subject to considerable methodological difficulties, a word about the design for a new personality study is warranted. If videotape equipment is employed, S's behavior and verbal content can be independently rated by trained observers at a later date. Moreover, the inter-rater reliability of the interpreted behavior can be determined and the problems of rater biases are consequently diminished. Larger sample sizes can reduce the impact of S idiosyncrasies and establish a more realistic picture of the migraine population as a whole.

The problems of the interviewer acting as a causal agent in the interview process can also be controlled. First, the interviewer can be double-blind to the S's migrainous status. The data on attack parameters can be obtained by additional interviewers, or by

questionnaire. Second, the interviewer's gestures and speech can also be videotaped, and his impact on the interview can be assessed. Third, the use of several interviewers, randomly assigned to Ss, permits the effects of interviewer differences to be assessed and controlled.

From the pragmatic point of view, the use of a standardized questionnaire may be superior to the interview-rater method. A questionnaire is less time consuming to administer and score, it requires less man and machine power, and the resulting data is more readily quantifiable. However, a standardized and validated 'migraine questionaire' has yet to be constructed. In any event, the design proposed above for a new personality study has merit in merely demonstrating that the methodological problems of previous case studies can be overcome.

The inconclusiveness and outright conflicts between reported results was a second source of difficulty in evaluating the different conceptual models. The issue of sexual functioning of adult migrainous males is a case in point. Wolff (1937) judged the overt sexual activity of his migrainous male Ss to be adequate in nature and frequency, but felt that other personality characteristics indicated incomplete sexual adjustment in their basic personality structure. Furmanski (1952) also judged that his male patients achieved normal sexual functioning, but judged them fixated at the anal and oral stages of psychosexual development. However, Sperling (1952)

judged none of her male Ss to have developed normal adult sexuality. In contrast to Wolff (1937), Sperling (1952) felt her male patients were preoccupied with oral sexual contact. Sperling (1952) also failed to identify anal fixations in her Ss, in contrast to Furmanski (1952).

There is further confusion in this area. Wolff (1937) found no overt sexual dysfunction in males, but judged over 80 per cent of his female Ss to be dissatisfied and withdrawn from sexual activity. Furmanski (1952) did not find any difference between males and females in regard to sexual adjustment. Sperling (1952) found every one of her male and female Ss to lack normal adult sexual adjustment. To suggest these results are inconclusive seems to be indulging in understatement. Moreover, it seems ironic that the advocates of the psychological model of causation report results which seem to indicate that personality structure does not determine behavior. Ss who are pre-genitally fixated should not function on an adult sexual level. If personality does not determine behavior, can it determine physiology?

However, problematic results are not solely limited to the personality case studies. The data on sex differences in migraine incidence and the involvement of biorhythms as a migraine trigger is also inconclusive.

If it had been documented that migraine was more common in women than in men, it would not eliminate either the psychological or constitutional models. Constitutional theorists would argue that

the basis for the difference lies in differences in male and female physiology. Psychologically oriented theorists would suggest that women have more migraine because social expectations at that time (1930's) demanded that women repress their anger and sexual impulses more than men.

Ironically, a review of the differences in incidence in men and women in the 1970's could provide a test of the psychological models of causation. Now that women have more loosely defined sex-role expectations (at least among the young and middle and upper-classes) they may express their anger and sexuality more freely, i.e., in a style more closely approximating that enjoyed by men. If this is true, and if the psychoanalytic model is valid, then sex differences in migraine incidence should decrease.

Moreover, women are pursuing careers and positions of responsibility formerly held only by men. Thus, Marcussen and Wolff's (1949) theory would suggest that since they have more equivalent opportunities for fatigue and frustration, and the same restrictions on venting hostility on the job, the incidence of migraine in women should increase. Of course, this makes the male chauvinist assumption that working in the business world is more fatiguing or frustrating than managing a home and children.

The data on duration and frequency of attacks does not discriminate between theoretical models. Although there is a relatively

high degree of consistency in the research reports, none of the conceptual models is inconsistent with the data.

The overall incidence of migraine is not consistent with the psychoanalytic model. The personality characteristics that this model identifies as being causal elements in migraine can be identified in a wide range of the general population. Yet reports indicate that migraine occurs in only approximately eight per cent.

The available reports on frequency of migraine incidence, as well as the data on incidence along family heredity lines is supportive of a recessive genetic trait, that is the basis of the constitutional model.

The Mitchell and Mitchell (1971) migraine treatment study also presents difficulties for the psychological model. They found that changing particular feelings offered little benefit in reducing migraine. Instead, they found that modifying behavior patterns and reducing anxiety and tension were significantly more effective in reducing migraine frequency and severity. Fromm-Reichmann (1934) claimed that migraine attacks could be terminated by having the client elaborate and ventilate his hostile feelings. In light of Mitchell and Mitchell's results, Fromm-Reichmann may have actually been reporting the beneficial effects of implementing new behavior patterns for coping with hostility rather than the benefits of cartharsis and insight.

In conclusion, currently available migraine research does not permit evaluation of the various theoretical models of the trigger for migraine attacks with any degree of finality. In this light, several important questions which may serve to further classify this issue are offered.

Suggestions for Future Research.

Do migrainous individuals express less hostility than non-migrainous people? If migrainous Ss express the same amount, or more, hostility than non-migrainous Ss, it would tend to contradict the psychoanalytic model. Similarly, these results would be contrary to the Marcussen and Wolff (1949) hypothesis that migrainous Ss store their hostility rather than express it. The outcome of this question would not have conclusive bearing on the constitutional model. If more hostility was expressed by migrainous people, it would support the constitutional model, which requires heightened emotionality of some nature to occur. However, if less hostility were expressed, it would not conclusively rule out the constitutional model, since no hypothesis about specific emotions involved in the etiology of migraine attacks are offered.

Do migrainous Ss report seeking contact with their parents more often than non-migrainous Ss? Do migrainous Ss live physically

close to their parents? Or express warmer feelings for them? If this is not the case, it would tend to conflict with reports of unresolved parental attachments and incomplete individuation of migrainous adults.

What about the migrainous S's sense of frustration with his life, the degree and range of responsibilities he has undertaken, and his work/leisure balance? Marcussen and Wolff (1949) offer no data to support their assumption that migrainous people compulsively assume responsibility, overwork themselves, and become frustrated with their lives.

If the personality differences suggested by the maternal deprivation hypothesis do exist between migrainous and non-migrainous people, then they might be evident in a study of the children of non-migrainous parents who are adopted during infancy by migrainous women. If these children develop the migrainous personality, then these results could be explained by a modeling (learning) theory, as well as the maternal deprivation hypothesis. However, a second study should focus on the migrainous women whose own mother was not migrainous (i.e. women who probably inherited migraine from their father). These women would have attacks but not the migrainous personality, according to the maternal deprivation hypothesis. If the adopted children of these women had the migrainous personality but did not have attacks, this would be damaging to a modelling theory. The maternal deprivation hypothesis could readily account for these findings.

On the other hand, do these adopted children have the migrainous personality traits without having migraine as a clinical problem? If so, it would suggest that a genetic predisposition is a prerequisite for migraine attacks. This would be supportive of the constitutional model. Likewise, it would conflict with the psychological model since personality would not be sufficient in itself to create migraine attacks.

Are migrainous Ss more sensitive to changes in serotonin and MAO levels in the blood stream than non-migrainous Ss? If this is true, it would strongly support the constitutional model. If there were no difference between migrainous and non-migrainous Ss in the ease of precipitating migraine by injections of MAO and serotonin, the constitutional model would be suspect.

Finally, it would be productive to conduct a personality study of migrainous and non-migrainous adults. If the traits identified by previous personality studies were not confirmed, it would indicate against both the psychological and maternal deprivation models, which suggest a 'migrainous personality.' If all migrainous Ss have the suggested personality traits and non-migrainous Ss don't, this would support the psychological models which suggest that personality dynamics are causal of migraine. If these characteristics were found in both migrainous and non-migrainous Ss, it would argue against the psychological models and tend to support the maternal deprivation

model. If it were found that the characteristics appeared in the children of migrainous mothers, but were absent from the children of 'normal' mothers, the support for the maternal deprivation model would be highly convincing. However, if the 'migrainous' personality traits were found as predominantly in the children of migrainous fathers and 'normal' mothers, as in the children of migrainous mothers and 'normal' fathers, it would argue against the maternal deprivation model.

As the trigger for migraine attacks is more clearly defined, the plan of action for those individuals undertaking treatment of migraine patients, which has heretofore been quite indefinite, will become less so. Should we be involved in genetic counseling? Should we be taking special steps to teach migrainous mothers how to circumvent extraordinary deprivation? Should we be teaching migrainous mothers how to connect cognitive labels to their own emotions so they may pass this on to their children? Should we be intervening with the children of migrainous parents, to help them develop response styles that are less likely to upset their physiological predisposition? Should we suggest that migrainous people attempt to control their emotions? Should we point to particular feelings of hostility, or frustration, or strong feelings in general? Should we teach migrainous people not to hold back their feelings, and instead give full expression to them as they arise? Perhaps the future research suggested in this chapter will provide some of the much-needed answers.

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