The relationship between sleep, nutrition and mood: a study of patients with anorexia nervosa

A. H. CRISP

EDWARD STONEHILL

*Academic Department of Psychiatry,
St George's Hospital, Tooting, London, S.W.17*

GEORGE W. FENTON

*Institute of Psychiatry,
De Crespigny Park, London, S.E.5*

Summary

An initial observation that insomnia, especially early morning waking, is a feature of anorexia nervosa has been confirmed. A further proposition that the insomnia is more closely related to the nutritional disturbance than to the mood state has also been confirmed using standardized measures of weight, sleep and mood. These findings are discussed in terms of the possible relevance to other illnesses involving weight change in which there is an alteration of sleep patterns.

Introduction

Insomnia is a common complaint in patients with psychiatric illness. Such sleep difficulty is often thought to be mainly and directly related to the associated mental state, such as anxiety, and depression (Mayer-Gross, Slater & Roth, 1960). Early morning waking in particular has been described as a feature of 'endogenous' depression and is regarded by some psychiatrists as a cardinal symptom differentiating this disorder from 'reactive' depression (Carney, Roth & Garside, 1965). However, sleep disturbance, including early morning waking, has been described by others (McGhie, 1966; Willis, 1965) as a more general phenomenon in psychiatric illness, although severely depressed patients often complain of it more than others (Samuel, 1964). Severely depressed patients are also sometimes agitated and restless and have lost considerable weight (Hinton, 1962; Kiloh & Garside, 1963). Clinically it has been noted that patients with primary anorexia nervosa frequently sleep badly, especially in terms of interrupted sleep and early morning waking, although they rarely complain of it (Crisp, 1967). Such patients suffer a severe disorder of weight loss associated with carbohydrate

starvation, probably as a constitutionally and experientially derived neurotic psychosomatic response to adolescent emotional difficulties. In a systematic study of sixty patients with anorexia nervosa, Crisp (1967) reported that sleep disturbance together with other manifestations of increased arousal such as diffuse restlessness, tended to be a feature of more severe cases. It was not clearly related to factors such as age, duration of illness, feeding pattern or type of diet and could not be explained as a result of affective disturbance. Only ten patients of the whole series showed evidence of clinical depression. It was concluded that the sleep disturbance was directly related to the malnutrition, and it was suggested that nutritional factors may play a significant role in the genesis of insomnia in other more common psychiatric conditions, especially depressive illness.

The present investigations were undertaken in order to examine in greater detail these clinical observations by a longitudinal study of two groups of anorexia nervosa patients, each patient acting as her own control. The first group was examined by means of sleep self-reports before treatment and after restoration of body weight, and the second group by continuous all-night EEG recordings before and after treatment.

1. Sleep self-reports

Method

Ten female inpatients of mean age 22 ± 4 years suffering from anorexia nervosa slept for five nights each in a single quiet room before treatment and again after their weight had been restored to normal levels. Treatment consisted of bedrest, chlorpromazine, re-feeding on a 3000 calorie 'normal' diet and weekly psychotherapy according to the regime

described by Crisp (1965). Nine of the patients received no night sedation and the tenth patient received the same hypnotic after treatment as before. The post-treatment investigation was carried out after patients had been taken off phenothiazines at least 2 weeks previously. All patients achieved their matched population mean weight during treatment.

Each patient retired to bed between 10.00 p.m. and 10.30 p.m. and was left to wake spontaneously in the morning. In the morning the patient was required to complete a questionnaire (Crisp & Stonehill, 1970) enquiring about the previous night's sleep. During the investigation periods, the patient completed two questionnaires measuring psychoneurotic status and personality, namely the Middlesex Hospital Questionnaire (MHQ) and the Eysenck Personality Inventory (EPI). The MHQ (Crown & Crisp, 1966) is a brief standardized psychoneurotic index. It comprises a printed questionnaire of forty-eight questions completed by the patient. Responses provide scores (0-16) on six scales of psychoneurotic status, namely an anxiety (anx:) scale, a phobic (pho:) scale, an obsessional (obs:) scale, a scale measuring somatic complaints (som:), a depression (dep:) scale and an hysteria (hys:) scale. The EPI (Eysenck & Eysenck, 1964) measures personality along the dimensions of neuroticism (N) and introversion-extroversion (E). In addition each patient was weighed on accurate weighing scales during the two periods of investigation. In order to allow for adjustment to the experimental conditions, the sleep questionnaires on the first two nights of each five-night period were discarded.

Results

Mean values of the various sleep items were calculated for the last three nights of each five-night period before and after treatment for the ten patients. Table 1 shows the mean pre-treatment and post-treatment values for weight, sleep items, MHQ and EPI scores on the ten patients. The mean group weight increased from 39.9 kg to 54.4 kg, a highly significant increase in weight ($P < 0.001$). The group mean total duration of sleep per night increased from just over 6½ hr to just over 7½ hr and this is significant at the 1% level. It is apparent that the mean group total change in sleep reflects changes occurring throughout the night; the pre-treatment delay in falling asleep of over 1 hr falling to just over ½ hr in the post-treatment period; the mean pre-treatment reported duration of interrupted sleep falling from 16 min to 7 min post-treatment; similarly final waking in the morning changing from just after 6.00 a.m. pre-treatment to after 6.30 a.m. post-treatment. These differences failed to reach statistical significance with the numbers involved ($P < 0.10$). On the MHQ all scales except the pho: and hys: revealed a decrease in mean score in the post-treatment period but only the som: and dep: scores reached significance ($P < 0.05$). However, these latter changes were contributed to by changes in scores on questions in both of the scales relating specifically to sleep. Moreover, none of the patients was judged to be suffering from severe depression on clinical grounds during the pre-treatment period. Thus, when the pre-treatment MHQ scores of this group of patients are compared with the mean

TABLE 1. Mean weight, sleep patterns, psychiatric status personality and significance of difference in ten patients with anorexia nervosa before and after treatment

	Pre-treatment	Post-treatment	Significance (P)
Weight	39.9 kg	54.4 kg	<0.001
Total sleep	6 hr 32 min	7 hr 35 min	<0.01
Time to fall asleep	67.7 min	38.5 min	<0.10
Broken sleep	15.8 min	6.8 min	<0.10
No. sleep interruptions	1.27	1.06	NS
Time of final waking	6.10 a.m.	6.38 a.m.	<0.10
Total time in bed	8 hr 29 min	8 hr 54 min	NS
Total time awake	127.5 min	77.5 min	<0.02
MHQ {	Anxiety	7.1	<0.10
	Phobic	2.5	NS
	Obsessional	9.6	NS
	Somatic	4.6	<0.05
	Depression	7.5	<0.05
	Hysteria	6.1	NS
	Total	37.8	<0.05
EPI {	N	12.1	NS
	E	6.3	<0.001

N, neuroticism; E, introversion-extroversion.

TABLE 2. Mean MHQ scores, standard deviations and significance of differences between ten inpatients with anorexia nervosa before treatment and twenty-four non-depressed psychoneurotic inpatients

	A	P	O	S	D	H
Ten inpatients with anorexia nervosa before treatment aged 22 ± 4 years	7.5 ± 3.5	2.5 ± 1.9	9.6 ± 3.1	4.6 ± 3.1	7.5 ± 2.7	6.1 ± 2.7
Twenty-four non-depressed psychoneurotic inpatients aged 30 ± 9 years	12.5 ± 1.8	9.0 ± 4.0	9.3 ± 3.0	9.3 ± 4.2	8.2 ± 2.7	7.5 ± 3.3
Probability (<i>P</i>)	<0.001	<0.001	NS	<0.001	NS	NS

A, anxiety; P, phobic; O, obsessional; S, somatic; D, depression; H, hysteria.

MHQ scores of twenty-four psychoneurotic inpatients who were not diagnosed as suffering from depressive illness (Table 2), there is no difference in the dep: scores between the two groups. On the EPI there was a mean increase in the E score after treatment which is significant at the 0.1% level.

During treatment all patients achieved their matched population mean weight but the weight increase necessary for this varied between patients. Table 3 is a chart displaying the changes between pre-treatment and post-treatment weights, sleep patterns, MHQ and EPI scores in the ten patients. When the amount of weight increase is correlated with each of the changes in sleep patterns (Table 4),

TABLE 4. Coefficients of correlation and significances between increase in weight and aspects of improved sleep in ten patients with anorexia nervosa during treatment

Sleep item	<i>r</i>	<i>P</i>
Total sleep	0.56	<0.1
Time to fall asleep	0.06	NS
Broken sleep (min)	0.16	NS
No. sleep interruptions	0.33	NS
Time of waking	0.89	<0.001

the main and significant correlation (with the small numbers involved) is between amount of weight increase and later time of waking up in the morning ($r = 0.89$; $P < 0.001$). Changes in weight, total sleep, time to fall asleep, interrupted sleep and time of final waking failed to correlate at a significant level with any of the MHQ and EPI score changes. When the correlation coefficient between increase in weight and later time of waking up in the morning is calculated with the effect of MHQ dep: score partialled out, it remains significant at the 0.1% level ($r = 0.88$).

2. Continuous EEG recordings

Five patients (four female and one male) admitted to a psychiatric metabolic unit for treatment of primary anorexia nervosa were examined. Their ages were 14, 16, 17 (male), 18 and 36 years. Each was seen 10–14 days after admission. At bedtime chlorided silver 'stick-on' electrodes were applied to

the scalp in the right frontal, central, parietal and occipital areas as well as the superior and lateral aspects of both orbits. These were connected to an eight-channel Offner type T electroencephalograph in an adjacent room.

The patient then retired to bed and bipolar recordings were performed continuously throughout the night, for at least 7 hr or longer until the patient woke spontaneously. This procedure was repeated during the following three nights, recordings being carried out on each patient during four consecutive nights. When the patients had gained weight, but not necessarily a weight amounting to their matched population weight, and prior to discharge from hospital, this procedure was repeated, each patient having a further four consecutive all-night sleep recordings performed. The interval between the two series of tests varied from 6 weeks to 3 months. Treatment consisted of re-feeding with a high protein and high carbohydrate diet. No daytime psychotropic drugs were prescribed. One patient received a hypnotic, the dose being maintained unchanged throughout the study. Four of the patients responded well to this regime, gaining at least 10 kg. One failed to show any appreciable weight gain. Daily ratings of mood and behaviour were carried out using a modification of the Phipps behaviour chart (Russell, 1967) and each patient's mood was also assessed independently by a psychiatrist unaware of the EEG findings, during an interview conducted on the day of the first of each series of recordings.

All sleep records were examined by a single investigator, who rated the depth of sleep for each 20-sec period throughout the night using the Loomis, Harvey & Hobart (1937) classification. Data obtained from the first night of each four-night series were discarded in order to allow for the so-called 'first night effect', ratings from subsequent nights being used.

Results

Results obtained from the four patients who gained weight were examined separately, the fifth patient who gained no weight being treated as a

TABLE 3. Changes in weight, sleep patterns, psychoneurotic status and personality in ten patients with anorexia nervosa before and after treatment

No.	Weight (kg)	Time to			No. of sleep interrupt- tions	Time of waking (min)	MHQ							EPI	
		Total sleep (min)	fall asleep (min)	Broken sleep (min)			A	P	O	S	D	H	Total	N	E
1	+20.9	+140	-75	0	+1.7	+87	-5	+1	+1	-9(-7)	-7(-5)	+4	-15	-10	+6
2	+14.5	+123	-100	+4	+0.3	+20	-6	+1	0	-3(-1)	0(0)	0	-8	-3	+3
3	+14.1	+11	+10	-2	-2.3	+22	-3	-3	-5	-9(-8)	-1(-1)	-3	-24	-1	+11
4	+6.3	-40	+6	0	-0.3	-28	+1	-1	+2	-2(+2)	-1(-1)	0	+3	+5	+6
5	+20.9	+70	+1	-4	-1.0	+68	+4	0	+4	-1(-1)	+1(+1)	-2	+6	0	+2
6	+17.2	+46	+4	-38	+0.3	+12	-6	0	-6	-3(-3)	-4(-2)	+2	-17	-6	+4
7	+13.1	+18	-3	-3	-0.3	+7	0	0	-2	0(0)	0(0)	0	-2	+1	+3
8	+7.3	+80	-87	-15	-0.7	-20	-1	+3	+3	-4(-4)	0(0)	-2	-1	-3	+7
9	+17.2	+133	-30	-27	+1.0	+76	-3	0	-5	-1(-1)	-6(-4)	+3	-11	-1	+6
10	+14.5	+48	-18	-5	-0.7	+23	-4	0	-8	-3(-3)	-5(-3)	0	-20	-7	+10

Score with sleep questions removed; A, anxiety; P, phobic; O, obsessional; S, somatic; D, depression; H, hysteria; N, neuroticism; E, introversion-extroversion.

control subject. In the light of the initial observation and the results of the sleep self-report study, these large amounts of EEG data were treated so as to provide twelve separate items of information both before and after treatment on the four patients, the corresponding nights of investigation before and after treatment being compared on each occasion.

The total time asleep per night, defined as the duration of time spent in stages B (light sleep), C (medium sleep), D (deep sleep), E (very deep sleep) and REM (rapid eye movement sleep) was increased following treatment, the mean for the initial twelve nights being 6.3 hr compared with the post-treatment value of 7.0 hr. This difference is significant at the 5% level.

Figure 1 displays in histogram form the amount of time spent in the various stages of sleep throughout the night, and Table 5 shows this data in tabular form with probability levels concerning the significance of the differences. Before treatment signi-

TABLE 5. Time in each stage of sleep in minutes. Mean values for twelve nights in four subjects with anorexia nervosa

Stage	Before treatment	After treatment	Significance (P)
Awake	28.5	5.9	<0.01
A	27.9	3.1	<0.005
B	88.8	93.4	NS
C	170.9	156.4	NS
D	65.2	89.1	<0.05
E	33.5	73.4	<0.005
REM	63.1	78.1	<0.05

ficantly more time was spent awake ($P < 0.01$) and in stage A (drowsy) ($P < 0.005$), whilst on retest an increase in duration of time spent in stages D ($P < 0.05$) and E ($P < 0.005$) was apparent. The amount of REM sleep was also greater after re-feeding ($P < 0.05$). The latency of onset of both slow wave and REM sleep was altered following weight gain. The mean length of time from the onset of the recording to the first 60 sec of C stage sleep was significantly reduced from 27.4 min before treatment to 7.6 min after treatment ($P < 0.025$). The mean latency of onset of REM appeared to be delayed pretreatment and occurred after 147.5 min but was reduced to 77.6 min post-treatment ($P > 0.02$). The mean number of shifts of stage of sleep per hour was also reduced from 43.1 before treatment to 36.1 after treatment but this trend did not reach statistical significance. The amount of time spent awake during the night was greatest during the first and last hours, especially the latter (Fig. 2). Before treatment the mean total amount of time spent awake during the seventh hour of recording was 16.0 min and this was reduced to 8.2 min after treatment, but this trend did not reach statistical significance.

The patient whose weight remained essentially unchanged can be regarded as a control subject and Fig. 3 reveals that there was no major change in the sleep pattern.

Ratings of the patients' mood states carried out by the nursing staff and assessment of the mental state carried out by the psychiatrist before and after treatment revealed no consistent mood changes.

FIG. 1. Mean duration of each stage of sleep in anorexia patients (four subjects), twelve nights before treatment and twelve nights after treatment. Closed columns, before treatment; stippled columns, after treatment.

FIG. 2. Pattern of wakefulness throughout the night: the total number of minutes spent awake during each hour in twelve nights of all-night recording in four patients with anorexia nervosa. Closed columns, before treatment; stippled columns, after treatment.

One patient, initially depressed, showed improvement, while the other three were unchanged. The control patient also showed no mood change.

FIG. 3. Control subject: mean of three nights before and three nights after treatment. Closed columns, before treatment; stippled columns, after treatment.

Discussion

These findings are taken to confirm the original observations concerning insomnia in patients with primary anorexia nervosa. They also lend support to the proposition that, in this group of patients, disturbances and changes in sleep are more closely associated with disturbances and changes in nutritional status than with disturbances and changes in emotional state. This finding may relate to the results reported in another study (Crisp & Stonehill, 1970) in which obese patients losing weight showed evidence of increasing insomnia whilst at the same time displaying improvement or no change in their mental state.

Such findings are taken to lend further support to the original proposition that such relationships might also hold for a variety of psychiatric and other disorders in which there is impaired nutritional status and weight loss.

Meanwhile, since there is a suggestion that patients who develop anorexia nervosa whilst still undergoing pubertal change are shorter than a control population by the time they present to doctors with the illness later in adolescence (Crisp, 1969), it is of interest that it has been shown (Sassin *et al.*, 1969) that release of human growth hormone during sleep is significantly directly related to the presence of the slow synchronized EEG stages of sleep, which it has now been shown are reduced in amount in anorexia nervosa patients.

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