Serum cholesterol levels in anorexia nervosa

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In contrast to earlier investigators (Pardee, 1941; Stephens, 1941; Emmanuel, 1956) more recent workers (Crisp, 1965a, 1967; Oberdisse, Solbach & Zimmerman, 1965; Klinefelter, 1965; Crisp & Stonehill, 1967) have reported that some of their patients with anorexia nervosa have high serum cholesterol levels.

Crisp (1965a, b, 1967) in a study of eighty patients (Nos. 1–80) has also emphasized that anorexia nervosa is predominantly a state of severe chronic carbohydrate starvation having its origins in the patient’s phobia of and consequent need to avoid normal adolescent weight. Such patients who will have initially turned to eating supposedly ‘non-fattening’ foods such as cheese, fruit, meat and salad, may later surrender to their increasing impulse to eat carbohydrate by having binges. They may start to vomit in relation to their binges and conceal and deny one or both these aspects. Usually such binges also alternate with phases when the patient stops eating altogether. Such cycles may recur every 24 hr or may span several weeks. These complex and often interrelating patterns of feeding make it more than usually difficult to assess the past dietary intake.

Meanwhile, a further study by Crisp et al. (1967) has confirmed that thirty-seven female patients aged 14–33 with anorexia nervosa had significantly higher serum cholesterol levels \( P < 0.0002 \) than thirty-seven normal women with whom they were matched individually for age (Fig. 1). The serum cholesterol levels in the control group are the mean of two results from the two halves of the menstrual cycle. However, there was a wide variation of serum cholesterol levels amongst the anorexia nervosa group. It was confirmed that such levels bore no relationship to thyroid function or reduced plasma volume and were unlikely to be due to diminished oestrogen activity, a universal characteristic in this condition. From a clinical standpoint high levels were not found to be related to age, duration of illness or prognosis. However, there appeared to be a relationship between particularly high serum cholesterol levels and the feeding pattern of intermittent bulimia involving ingestion of large quantities of carbohydrate and fat interspersed with phases of abstinence. A further investigation was therefore aimed at the study of serum cholesterol levels in relation to controlled dietary intake. The results of this study are reported below.

Patients and method

The last eight consecutive patients with anorexia nervosa admitted to this hospital were studied. Five patients were characterized by intermittent bulimia preceding their admission...
(Group 1) although one of these (No. 76) in addition had long periods of total abstinence. The other three patients had persistently severely restricted their carbohydrate intake over a period of many months prior to admission. All of the patients were strikingly underweight, except one (No. 38) and all were suffering from amenorrhoea. Serial serum cholesterol levels were examined in these patients, by the method of Zurbowski (1964), in relation to their treatment programme. This involved the restoration of their weight to within normal limits by means of a balanced diet of the order of 2000–3000 calories daily and chlorpromazine in a dose of 500–800 mg daily as previously described by Crisp (1966).

**Results**

Table 1 shows the serum cholesterol levels before and after refeeding in the two groups. Fig. 2 provides an example of the pattern of change in weight and serum cholesterol level seen in four of the five patterns in Group 1. On her two admissions to hospital with severe weight loss the patient had serum cholesterol levels of over 300 mg/100 ml which fell to within normal limits on refeeding. During two periods of relapse with weight loss the serum cholesterol steadily rose. This illustrates a 'reciprocal' relationship.

Fig. 3 provides an example of patients in Group 2 in which serum cholesterol levels gradually rise to above normal levels during the refeeding period and therefore show a 'parallel' relationship to changes in body weight. Fig. 4 shows the relationship of serum cholesterol levels to increase in body weight in patient No. 76, the patient with a mixed feeding pattern prior

**TABLE 1**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Serum cholesterol (mg/100 ml)</th>
<th>Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Before treatment</td>
<td>After treatment</td>
</tr>
<tr>
<td><strong>GROUP 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>19</td>
<td>350</td>
<td>220</td>
</tr>
<tr>
<td>27</td>
<td>19</td>
<td>480</td>
<td>280</td>
</tr>
<tr>
<td>38</td>
<td>23</td>
<td>430</td>
<td>350</td>
</tr>
<tr>
<td>6</td>
<td>13</td>
<td>260</td>
<td>160</td>
</tr>
<tr>
<td>76</td>
<td>19</td>
<td>290</td>
<td>290</td>
</tr>
<tr>
<td><strong>GROUP 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>70</td>
<td>27</td>
<td>220</td>
<td>340</td>
</tr>
<tr>
<td>75</td>
<td>22</td>
<td>230</td>
<td>350</td>
</tr>
<tr>
<td>77</td>
<td>19</td>
<td>220</td>
<td>280</td>
</tr>
</tbody>
</table>

**FIG. 2.** The relationship of serum cholesterol to body weight (O): an example of the 'reciprocal' relationship. •••••, Serum cholesterol; •••••••••, serum cholesterol without serial observations. Patient No. 23.

**FIG. 3.** The relationship of serum cholesterol (O) to body weight (O): an example of the 'parallel' relationship. Patient No. 70.

**FIG. 4.** The relationship of serum cholesterol levels to increase in body weight in patient No. 76, the patient with a mixed feeding pattern prior
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to admission. It would seem that she shows an
'intermediate' pattern.

![Graph](image)

**FIG. 4.** The relationship of serum cholesterol (●) to
body weight (○): an example of an 'intermediate'
relationship. Patient No. 76.

**Discussion**

Evidence so far suggests that anorexia nervosa
patients with high serum cholesterol levels may
owe this, at least in part, to their feeding pattern
of intermittent bulimia with or without vomiting.
When these patients are treated by refeeding the
serum cholesterol levels tend to fall. In contrast,
patients with low or normal serum cholesterol
levels tend to be characterized by eating small,
regular, low carbohydrate meals and their serum
cholesterol levels tend to rise during refeeding.
In addition there would appear to be an interme-
tiate group who have a mixed feeding pattern
with periods of bulimia interspersed amongst
long periods of abstinence as characterized by
patient No. 76. It may be that when a larger
number of patients with this condition are
studied this intermediate group will prove to be
more common.

The above findings are not dissimilar to those
of Fabry et al. (1964) who showed a similar
relationship between serum cholesterol levels
and frequency of eating in a male obese population,
although the feeding patterns were not so ex-
treme or bizarre as in our group of patients.
Our results are also in agreement with other
authors who have shown that animals forcibly
fed with large occasional meals have raised lipid
levels as compared to animals who nibble nor-

1961; Hollifield & Parson, 1962; Tepperman &
Tepperman, 1964). Gwinup et al. (1963) and
Cohn (1964), comparing frequent and infrequent
experimental dietary patterns in humans, have
produced similar results.

Chlorpromazine has been shown to have a
variable effect on cholesterol and lipid meta-
bolism. Kai (1961) found a decrease in choles-
sterol levels in patients suffering from schizo-
phrenia treated with chlorpromazine whilst
Hollister & Kanter (1955) did not find any con-
sistent change in elderly people. Since all eight
patients received this drug in comparable
amounts it is unlikely to have produced the
differences in cholesterol levels described here.

The significance of the different patterns of
change in serum cholesterol levels during refeed-
ing remains a problem. Further investigation into
dietary content and patterns of eating in anor-
exia nervosa and their effect on cholesterol and
other parameters of fat metabolism both before
and during treatment is now in progress.

**Acknowledgments**

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allowing us to study patients under their care.

**References**

Cohn, C. (1961) Meal eating, nibbling and body meta-

Cohn, C. (1964) Feeding patterns and some aspects of

Crisp, A. H. (1965a) Clinical and therapeutic aspects of
anorexia nervosa—A study of 30 cases. J. psychosom. Res.
9, 67.

Crisp, A. H. (1965b) Some aspect of the evolution, presenta-
tion and follow up of anorexia nervosa. Proc. roy. Soc.
Med. 58, 814.


Crisp, A. H., Blendis, L. M., Pawan, G. L. S. & Lrace, A.

Crisp, A. H. & Stonehill, E. (1967) Hypercarotenaemia as

Emmanuel, R. W. (1956) Endocrine activity in anorexia

Fabry, P., Fodor, J., Heil, Z., Braun, T. & Zvolánková,
K. (1964) The frequency of meals; its relation to overweight
hypercholesterolaemia and decreased glucose tolerance.
Lancet, ii, 614.

Gwinup, G., Byron, R., Rouw, W., Weisler, A., Kruger,

to a 'stuff and starve' feeding program. J. clin. Invest.
41, 250.

Hollister, L. E. & Kanter, S. L. (1955) Essential hyper-
lipaeemia treated with heparin and with chlorpromazine.
Gastroenterology, 29, 1069.


