A Diabetic Clinic in a Peripheral Hospital

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SUMMARY

The establishment and operation over two-and-a-half years of a diabetic clinic in a 500-bed peripheral hospital are described.

Fifty-eight White patients, 37 females and 21 males, received treatment: 18 with insulin, 28 with oral hypoglycaemic drugs, and 12 by diet alone. Control was considered good in 31 (53%), fair in 15 (26%) and poor in 12 (21%).

The drugs used are reviewed, and mortality and complications briefly described. There were 11 patients with heart disease, mostly ischaemic, and all deaths in the group were cardiac. Some important therapeutic points and some evidence for and against an association of sulphonylureas and deaths from coronary heart disease, are discussed.


Before the establishment of a diabetic clinic in February 1969, all newly diagnosed diabetics, or patients with problems of stabilization, had to be either referred to various heavily booked medical outpatient clinics, or admitted to a medical ward. There was no special provision for their regular supervision, other than continued attendance at these clinics, or reporting to the general outpatient medical officers for more medicine. At this time, the Department of Medicine already had one subspeciality clinic, with others in view; and this led to the establishment of a diabetic clinic. Patients and doctors were not compelled to use the facilities, and so it made a slow start, but, although the numbers are small, now that the advantages of the clinic are becoming apparent, patients are being referred in increasing numbers. This is a report of our methods and results, and a discussion of some points raised in the routine care of diabetics.

MATERIALS AND METHODS

The patients, comprising White adults eligible for hospital, as distinct from private, care, were referred initially by the general outpatient medical staff, but later referrals came from other medical outpatient clinics and local general practitioners, as inpatients eligible for hospital care. The management of diabetes in pregnancy, and for surgery, and details of inpatient stabilization of insulin-dependent diabetics, are not included in this report.

Almost all cases had been diagnosed, some of them many years before; most had been confirmed by an oral glucose tolerance test (GTT) at some stage, using a 50 g load. The evidence was reviewed and the diagnosis confirmed if in the presence of compatible symptoms the fasting blood sugar was 140 mg/100 ml or more, thus making the presence of a true blood glucose of at least 120 mg/100 ml virtually certain, or if the GTT showed a diabetic curve with a 2-hour level above 120 mg/100 ml.

With the exception of the Pima Indians of Arizona, the response: to a GTT in diabetics and nondiabetics are not bimodal, but form a single curve, so the dividing line is necessarily rather arbitrary. With 1 or 2 exceptions, the diagnosis of cases has been no problem; they included cases with significant symptoms, very abnormal curves or fasting blood sugars, or known ketosis-prone, insulin-dependent patients of long standing.

All newly referred patients, after a full medical history and examination, are weighed and their chests X-rayed. Only investigations suggested by clinical findings are done, as we have had to focus our attention on diabetes because of increasing numbers of patients and pressure of work. We feel that the comprehensive care of the patients rests with their usual doctor, to whom our findings are available. We hoped that the history, clinical examination, and strictly relevant tests demanded by our findings, would show up any underlying cause for the diabetes, such as haemochromatosis, or pancreatitis. Patients undergoing outpatient stabilization were seen weekly, often after initial inpatient treatment, or at fortnightly or monthly intervals if newly stabilized or hard to control; and quarterly or twice a year if stable patients on active treatment or well controlled on diet alone. Between visits they were seen by their own doctors. A number of these well-controlled patients have been referred back for further care to their personal doctors.

New insulin-dependent cases received instruction for their individual management in the wards during stabilization, and the technique of newly referred insulin-dependent patients was reviewed. Some mistakes by those claiming to be knowledgeable induced us to do this.

All patients test their urine in the early morning and before supper with Clinistest tablets and, latterly, with Acetest, and record the results properly. The degree of glycosuria between visits is noted and the patients' urine charts are filed when complete. At each visit the patients are weighed, their urine tested and blood sugar estimated with Dextrostix by the sister-in-charge, interpolating the strip if necessary. The tests are carried out between 1000 and 1030, when the clinic opens. When levels above 175 mg/100 ml are found, a specimen of venous blood is sent...
Occasional findings of 4+ without ketones disregarded as inevitable.

Because insulin assays are not available as a routine the diagnosis of the type of diabetes for the initiation of therapy has been made on clinical grounds.

Urinary sugars:
- Nil or trace at least once a day, usually not more than 2+ at other times: Good
- 2-3+ fairly often, 1+ at other times, occasionally nil: Fair
- A greater degree of glycosuria with or without ketones: Poor

When there was a low blood sugar reading with considerable glycosuria, venous blood was used to confirm the Dextrostix result. The most doubtful, low Dextrostix readings and those above 175 mg/100 ml were confirmed by the laboratory, but the regular testing of urine between visits formed the only guide available to the patient to determine control and because of the limitations of Dextrostix this was a great help to us.

Table I shows the blood sugar and urinary sugar criteria for assessing the degree of control. The blood sugar levels used to determine this were somewhat limited by the Dextrostix estimations, because we did not wish to use interpolated readings to differentiate between good, fair, and poor.

Control may not be fair in those with blood sugar levels above 150 mg/100 ml. Results recorded on the 175 mg/100 ml colour are indicated in Table II and may be added to the number whose control was poor, to give, perhaps, a better reflection of the number indifferently controlled.

Because insulin assays are not available as a routine the diagnosis of the type of diabetes for the initiation of therapy has been made on clinical grounds.

### Table I. Criteria for Control

<table>
<thead>
<tr>
<th>Blood Sugar</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>130 mg/100 ml or less</td>
<td>Good</td>
</tr>
<tr>
<td>130 - 175 mg/100 ml range, including 175 mg/100 ml</td>
<td>Fair</td>
</tr>
<tr>
<td>&gt;175 mg/100 ml</td>
<td>Poor</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Urinary Sugars*</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil or trace at least once a day, usually not more than 2+ at other times</td>
<td>Good</td>
</tr>
<tr>
<td>2-3+ fairly often, 1+ at other times, occasionally nil</td>
<td>Fair</td>
</tr>
<tr>
<td>A greater degree of glycosuria with or without ketones</td>
<td>Poor</td>
</tr>
</tbody>
</table>

* Occasional findings of 4+, without ketones, disregarded as inevitable.

### Table II. Degree of Control

| Nonobese | Insulin | Diet alone | On oral drugs | Obese on oral drugs | Total | % (%)
<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>18</td>
<td>12</td>
<td>17</td>
<td>11</td>
<td>58</td>
<td>(50)</td>
</tr>
<tr>
<td>Good control</td>
<td>8</td>
<td>11</td>
<td>7</td>
<td>5</td>
<td>31</td>
<td>53 (53)</td>
</tr>
<tr>
<td>Fair control</td>
<td>7</td>
<td>1</td>
<td>6</td>
<td>1</td>
<td>15</td>
<td>26 (14)</td>
</tr>
<tr>
<td>Poor control</td>
<td>3</td>
<td>0</td>
<td>4</td>
<td>5</td>
<td>12</td>
<td>21 (33)</td>
</tr>
<tr>
<td>'Fair', with recorded blood sugar of 175 mg/100 ml</td>
<td>(4)</td>
<td>(3)</td>
<td>(7)</td>
<td>(12)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Numbers in brackets represent patients whose control was considered fair by criteria given, whose recorded blood sugars were 175 mg/100 ml.

Numbers in brackets in right-hand end column are corrected percentages if these patients are considered to be poorly controlled.
RESULTS

Fifty-eight patients, comprising 37 women and 21 men, were treated. Fig. 1 shows the age distribution, the shaded portion representing those who received insulin. Table III shows the 4 kinds of treatment. All those treated by diet alone or with oral hypoglycaemic drugs were of late onset. In the absence of exact criteria, such as skin-fold thickness, some of the so-called 'nonobese' must fall into the 'obese' group on drugs. The heavier of those treated by diet alone, form a bridge.

In the overweight group, there were 5 who had not first been tried on diet alone before resorting to drugs. In other obese patients on drugs, diet had failed owing to poor cooperation. A number of the heavier patients in our nonobese group had also unsuccessfully tried diet alone. The degree of control is shown in Table II.

Table IV shows the details of insulin used, though it reflects only the degree of control found at the time of review of the records. Many patients had lapsed from optimal control from time to time for the usual reasons, including infection, other illness, dietary indiscretion, insulin mistakes, and in one case, an emotional upset.

**TABLE III. CATEGORIES OF TREATMENT SHOWING HEAVIEST, LIGHTEST AND AVERAGE WEIGHTS IN KILOGRAMS**

<table>
<thead>
<tr>
<th></th>
<th>Nonobese</th>
<th>Obese on oral drugs</th>
<th>Obese on oral drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin alone</td>
<td>18</td>
<td>12</td>
<td>17</td>
</tr>
<tr>
<td>Average weight</td>
<td>63</td>
<td>76</td>
<td>64</td>
</tr>
<tr>
<td>Heaviest in group</td>
<td>81</td>
<td>100</td>
<td>74</td>
</tr>
<tr>
<td>Lightest in group</td>
<td>36</td>
<td>60</td>
<td>48</td>
</tr>
</tbody>
</table>

These patients are in the obese group, whose dietary co-operation needs no drugs. The first hump of the bimodal distribution in Fig. 1 is largely made up of insulin-dependent juvenile patients. There are 4 in the 31 - 40-year group presumed to be late-onset types, and the increase in numbers of those on insulin in the 51 - 80-year group is accounted for by those in whom oral drugs have failed, and adult-onset insulin-dependent cases (probably the so-called 'J type', in the Lal et al. classification). In only 1 patient could we be certain of an identifiable underlying cause, for his symptoms followed an attack of pancreatitis. The weights used for Table III are the last recorded; since referral several patients have lost, and a few have gained some weight.

**TABLE IV. COMBINATIONS, TYPES AND DOSES OF INSULIN, WITH 3 DEGREES OF CONTROL**

<table>
<thead>
<tr>
<th>Control</th>
<th>Details of insulin given</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>1. Lente insulin 20/U before breakfast (on insulin for years, oral drugs tried briefly, unsuccessfully because of ketosis)</td>
</tr>
<tr>
<td></td>
<td>2. Lente insulin 60/U before breakfast</td>
</tr>
<tr>
<td></td>
<td>3. Lente insulin 40/U before breakfast</td>
</tr>
<tr>
<td></td>
<td>4. Lente insulin 20/U, soluble insulin 30/U in separate syringes, before breakfast</td>
</tr>
<tr>
<td></td>
<td>7. PZ insulin 44/U + 12/U soluble, mixed, before breakfast</td>
</tr>
<tr>
<td></td>
<td>8. Lente insulin 40/U before breakfast, soluble occasionally if needed in evening</td>
</tr>
<tr>
<td></td>
<td>9. Lente insulin 8U + semilente 32/U mixed, before breakfast</td>
</tr>
<tr>
<td></td>
<td>10. Lente insulin 15/U, soluble 25/U, separate syringes, before breakfast</td>
</tr>
<tr>
<td></td>
<td>11. Lente insulin 30/U before breakfast</td>
</tr>
<tr>
<td></td>
<td>12. Lente insulin + semilente 35/U, mixed, before breakfast</td>
</tr>
<tr>
<td></td>
<td>13. Lente insulin 36/U daily (previous oral drug failure despite proper diet)</td>
</tr>
<tr>
<td></td>
<td>14. Lente insulin 40/U before breakfast, soluble 10 - 15/U, evening</td>
</tr>
<tr>
<td></td>
<td>15. Soluble insulin 20/U before breakfast, soluble 16/U, evening</td>
</tr>
<tr>
<td></td>
<td>16. Lente insulin 80/U + phenformin 50 mg, morning</td>
</tr>
<tr>
<td></td>
<td>17. Lente insulin 30 - 40/U daily (poor cooperation with diet and dose)</td>
</tr>
<tr>
<td></td>
<td>18. Semilente 30/U daily. (Patient requires different regimen but hard to convince)</td>
</tr>
</tbody>
</table>
Table V shows some details of oral hypoglycaemic drugs used. In one case of supposed tolbutamide failure, review has shown an inadequate dose. In 2 other failures, control had been good until they suffered myocardial infarcts, from which they died; leaving 6, in one of whom poor control was largely due to poor co-operation with diet. To the 5 remaining failures must be added 2, successfully switched to insulin, making a total of 7 (14%).

**TABLE V. ORAL HYPOGLYCAEMIC DRUGS USED, SHOWING COMBINATIONS AND NUMBERS POORLY CONTROLLED**

<table>
<thead>
<tr>
<th>Drugs used</th>
<th>No. of patients</th>
<th>No. poorly controlled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpropamide alone</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Chlorpropamide and phenformin</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Tolbutamide alone</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Tolbutamide and phenformin</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Phenformin alone</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Glibenclamide alone</td>
<td>2</td>
<td>0</td>
</tr>
</tbody>
</table>

Maximal doses used (the usual doses given are shown in brackets) have been chlorpropamide, 500 mg daily (250 mg daily), tolbutamide 500 mg t.d.s. (500 mg b.d. or t.d.s.), phenformin, 100 mg b.d. (50 mg daily), and glibenclamide, 10 mg daily in one patient, and 5 mg in the other.

**MORTALITY**

There were 6 deaths, 3 men and 3 women, the average age at death being 73 years. In 4 there was evidence at autopsy or on the electrocardiograph that death was due to ischaemic heart disease, while left ventricular failure from severe hypertensive heart disease accounted for another. The sixth was an 82-year-old woman who died at home from congestive cardiac failure and general debility, the presumed aetiology being ischaemic heart disease. Five had had sulphonylureas (tolbutamide, 3; chlorpropamide, 2) for at least 12 years, and 3 of these patients had ECG evidence of ischaemic heart disease.

The fourth patient with ECG evidence of ischaemic heart disease had had a previous myocardial infarction but had been well controlled on insulin for years. He developed gangrene of the leg, necessitating amputation. Postoperatively he developed intestinal obstruction from old abdominal adhesions, necessitating further surgery, after which he died from recurrent myocardial infarction; the diabetes remaining well controlled despite suction and parenteral fluid.

**COMPLICATIONS AND OTHER DISEASES**

**Heart Disease**

Table I shows the number with heart disease and associated findings. The 3 not classified as definitely ischaemic, comprise 2 with hypertensive heart disease, and the 82-year-old woman previously mentioned with presumed ischaemic heart disease.

**Diabetic Retinopathy**

There were 8 patients (14%) with diabetic retinopathy, comprising 5 males and 3 females, with an average age of 63 years.

This was confirmed at the ophthalmic clinic. Six patients had stages 1 and 2, and 2 had severe proliferative diabetic retinopathy. One of these was a man aged 26 years, with a 17-year history of diabetes, who now has moderate visual impairment, and for whom pituitary ablation had received serious consideration at a teaching hospital. Most patients have had diabetes for many years but in one it was diagnosed 4 years ago, and in another, last year.

**Peripheral Neuritis**

The full-blown picture was seen in only 3 cases. A number of older patients have disturbances of bowel and bladder function, or have had episodes of ulceration of the feet thought to be a combination of the 'very peripheral vascular disease' of diabetes and trophic changes of peripheral neuritis.

**Other Complications**

These include intercurrent infections, boils, carbuncles, lapses of control, hypoglycaemic attacks, and albuminuria, though no patient had classical functional evidence of severe intracapillary glomerulosclerosis. One young insulin-dependent female had evidence of renal disease compatible with a degree of papillary damage, but did not have a history of ingestion of large quantities of analgesics. No patient died as a direct result of poor control, and no-one from the clinic was admitted in diabetic coma, although

**TABLE VI. MORTALITY, COMPLICATIONS, AND OTHER DISEASES**

<table>
<thead>
<tr>
<th>No. with heart disease</th>
<th>Definite IHD in</th>
<th>IHD deaths</th>
<th>Diet alone deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Definite on SU</td>
<td>on SU</td>
<td>group</td>
</tr>
<tr>
<td></td>
<td>No. deaths from</td>
<td>(including</td>
<td></td>
</tr>
<tr>
<td></td>
<td>cardiac</td>
<td>deaths</td>
<td></td>
</tr>
<tr>
<td></td>
<td>IHD disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>8 (4)*</td>
<td>6 (3)*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6 (1)*</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5 (2)*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

IHD = ischaemic heart disease. SU = sulphonylurea drugs. * Number of males shown in brackets after total in each group.
2 new patients were referred after control of such epi-
sodes in hospital. We have had to withdraw chlorpropamide
only 4 times, due to unwanted side-effects, twice for
rashes and twice for hypoglycaemia.
Phenformin has in the main been well tolerated with
minimal side-effects, but has had to be withdrawn in 1
case because of severe gastro-intestinal symptoms.

**DISCUSSION**

The age incidence of our series illustrates particularly well
the bimodal distribution of juvenile-onset insulin-depen-
dent, and late-onset diabetes and emphasizes that these
could be looked upon as different diseases with a number
of common features.

A number of our insulin-dependent patients received
more than one type of insulin. Oakley1 wrote: 'In the severe,
ketosis-prone or growth-onset diabetic, no single
injection regimen is as effective as the use of 2 injections
of soluble insulin,' but he qualified this by drawing
attention to the wide fluctuations in blood glucose, and
advocated some form of compromise between long-acting
and soluble insulins in difficult cases. This is in fact how
our practice has evolved. The simpler, more stable cases
are often not referred to us, as we are, not unnaturally,
beginning to be looked upon as a repository for problems.

Our numbers are too small to draw valid conclusions
regarding deaths from ischaemic heart disease associated
with the use of sulphonylureas, particularly since this has
been studied retrospectively without control of the other
variables such as age, weight, sex, and class of diabetes
inherent in a random allocation to various treatment
groups. Moreover, a number of our group treated by
diet alone, are mild cases. Since, however, an association
between deaths from myocardial infarction and sulphonylu-
reas has been claimed, it is necessary to examine the
evidence. In a 12-centre prospective study,4 there was
greater mortality from myocardial infarction in mild, non-
etoketic, stable diabetics who had been allocated at random
to the tolbutamide group, than in groups on a fixed dose
of insulin, a variable dose of insulin, and placebo (lactose).
However, it has been pointed out in an editorial com-
ment,4 that the conclusions are inconsistent because 2
centres contributed less than a quarter of the cases, yet
half the number of deaths, in the tolbutamide and placebo
groups.

Moreover, since autopsies were said to have been per-
formed in only one-third of cases, the accuracy of the
diagnosis of some of the cases was questioned.

In a previous prospective study to ascertain whether
treatment influences prognosis, Keen et al.5 actually
showed that patients on tolbutamide had fewer cardiovas-
cular episodes than those in 2 other groups consisting of
patients on different dietary schedules.

Balodimos et al.6 have shown a slight, but statistically
significant, increase in mortality due to coronary heart
disease in sulphonylurea-treated males, compared with
those treated with insulin, and a slight difference, not sta-
tistically significant, between male groups on sulphonylurea
alone and those on diet alone. These findings are contra-
dictory and do not support a wholesale retreat from the
sulphonylureas.

However, it seems wise to follow the standard teaching,
that every effort should be made to control symptoms in
late-onset, stable, nonketotic, overweight diabetics, with
diet alone, before prescribing oral hypoglycaemic agents.

One association suggested by our findings was the higher
female incidence of ischaemic heart disease and death
in those taking sulphonylureas. A higher female incidence
of ischaemic heart disease in diabetes is, however, known
to occur for reasons still unknown.5

Tolbutamide has a half-life of 5 hours in the body, being
oxidized and excreted as butyl-P-carboxyphenylsulphonyl-
urea, so that 2 or 3 doses are needed, while chlorpropa-
mide, with a half-life of 36 hours, is slowly excreted,
unchanged, and must be given once only in the morning.

Hypoglycaemia resulting from chlorpropamide tends to
recur after initial treatment, and this is a potentially
dangerous drug. A daily dose of 500 mg should not be
exceeded, and the usual effective dose is 250 mg.

It has recently been shown by Tomkin et al.6 that
metformin caused vitamin B6 malabsorption in about a
third of cases, which raises the possibility that the bigu-
anides, of which phenformin is the other drug in clinical
use, may owe part of their hypoglycaemic action to a
degree of malabsorption. This is in accordance with the
high incidence of gastro-intestinal side-effects of phen-
formin, which remains, nevertheless, a useful drug. Bloom7
points to its value in reducing the high insulin require-
ments in brittle diabetics.

Adverse reactions to oral anti-diabetic drugs are the
subject of a recent review by the Committee on Safety of
Drugs in London.8 Sulphonylureas may cause rashes,
blood dyscrasias, and liver damage, and while chlorpropa-
mide is the more powerful agent, the incidence of side-
effects is lower with tolbutamide, though the over-all
incidence is extremely low with the sulphonylureas.

Glibenclamide, with its powerful hypoglycaemic and
yet relatively short action, is a recent addition to our
armamentarium, and promises to be very useful. It is
very much in the news with a quite voluminous literature
in several symposia, conferences, and papers.9-11 Although
there are a number of hypoglycaemic agents available,
seems wise to keep to 2 or 3 whose properties can
become thoroughly known by experience.

In conclusion, we believe that, even in peripheral hos-
pitals, the establishment of the management of some
major disease groups in special clinics has much to offer
in the way of increased efficiency and better results. By
concentrating the care of patients in a single clinic de-
ved to their particular problem, a consistency of
documentation and experience, relevant to the condition,
is introduced. Any inadequacies of management very soon
become apparent and can be corrected.

Our results to date are not particularly outstanding, but
the experience gained in a controlled and organized way
should result in improved care.
We wish to thank Dr L. Fernley, Medical Superintendent of Grey's Hospital, for permission to publish; the house physicians who have assisted at the clinic; Sister J. M. Pocock and her assistants, Sisters A. J. Jaffray, M. Roughhead and P. Fortgens, without whose help this service could not have been run. We should also like to thank our Secretary, Miss J. M. Ayliffe, for her help in preparing this article for publication.

REFERENCES


Oxprenolol in Angina Pectoris*


SUMMARY

The clinical response and the ability to perform a measured exercise load were assessed in 7 patients with angina pectoris, who were given oral oxprenolol in weekly-increasing doses of 240 mg, 320 mg and 480 mg per day.

Oxprenolol relieved the angina and improved the patients' exercise capacity and angina threshold. The working capacity at the angina threshold was doubled in 6 of the 7 patients. Oxprenolol was effective by reducing heart rate, systemic arterial pressure, and pressure time developed per minute. Optimal results were achieved with a dose of 320 mg per day.


Beta-adrenergic blockade ameliorates angina pectoris, but the negative inotropic effect may precipitate cardiac failure. The newer beta-blocking agents (oxprenolol, alprenolol and practolol) have less negative inotropic effect, but also relieve angina pectoris.*

We have made a study of the effect of oral oxprenolol (Trasicor) in a group of 7 patients with angina pectoris, and the results will be reported.

PATIENTS AND METHODS

Seven patients with severe angina pectoris were selected for study. The clinical and electrocardiographic findings were confirmed by cardiac catheterization and selective coronary cine-angiography in 6 patients. The clinical, haemodynamic, and angiographic features of the patients are summarized in Table I. Three patients had severe, and 3 moderate, angina pectoris. In 1 the angina was mild, although he had severe coronary artery disease at angiography. One of the patients with severe angina was also hypertensive.

The study was undertaken without altering the patient's way of life or his medical therapy. Four patients continued to take digitalis; another received bethanidine and alpha-methyl-dopa throughout the study.

*Date received: 14 February 1972.

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