of value in medical education. The family
doctor is as old as medicine itself, and a
study of the varying standards of his work,
and of the conditions in which he has car-
ried it out, has much to teach us. The very
status of the doctor has changed with the
centuries. When the physician was more
than somewhat of a magician, he could be
regarded as semi-divine. The practical
Romans of the time of Julius Caesar, ac-
cording to Suetonius, gave all their phy-
sicians the great benefits of Roman citi-
zenship. Almost within living memory, the
doctor's frock-coat and goldheaded cane
were symbols not merely of affluence but
of the respect in which he was held in the
repute of his fellowmen. Ups and downs
are man's natural lot, but it is a chastening
thought that the recent trend of change
in the status of the medical profession
may have been gradually and subtly for
the worse! It is supremely ironical that
today, when the doctor can be something
more than a pompous and ignorant hum-
bug, when he has at his command an im-
pressive diagnostic and therapeutic arma-
mentarium, respect for the doctor may
have lessened. Why should this be? Is it
just a matter of familiarity breeding con-
tempt by way of a certain loss of mys-
tique, resulting from the modern diffusion
of medical knowledge among laymen? If
it were so we would count it no very great
loss. But can we be sure that there is not
some much more serious reason, such as
a decay and decline in the personal human
relationship between the doctor and his
patients? And will not this decline reach
its nadir when the doctor can no longer
say “Everyman, I will go with thee and
be thy guide, in thy most need to go by
thy side”, because he has become just
another State functionary with an alle-
giance other than that of the interests of
his patients as his supreme consideration?

It is in the mutual Love of Doctor and
Patient that the medical relationship can
find and keep its soul. In the Epistle to
the Colossians, St. Paul crowned St. Luke
with the beautiful title of THE BELOVED
PHYSICIAN. Would that we could all
attain to its deserving.

A CASE OF BERI-BERI
HEART DISEASE
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A thirty year old male was referred
for progressive heart failure of unknown
etiology. His personal history was as fol-
lows: At 15 he had joined the Royal Navy,
playing Rugby football and taking part in
athletics. There was not, therefore, at that
time anything to suggest either congenital
or rheumatic cardiac lesions. Nine years
ago, he passed his “medical”, and joined
the Fleet Air Arm. Three years later he
began to complain of palpitations which
were attributed to emotional problems,
because exhaustive investigations in a
naval hospital had failed to show any
abnormality. Since one year he has found
himself progressively getting out of
breath on exertion — a symptom which he
attributed to heavy smoking (80 cigarettes
per day). Since two months his exertional
dyspnoea had become worse and he no-
ticed swelling of the ankles. No history of
chest pain could be obtained. After close
questioning he admitted that for the last
eight years he had been a heavy
drinker of whisky (up to one bottle a day) and of
beer (up to 20 bottles daily).

Physical examination showed a thin
man of medium height, who got easily out
of breath while undressing. The usual
signs and symptoms of congestive heart
failure were present, that is, exertional
dyspnoea, mild cyanosis, anorexia, raised
jugular venous pressure, basal rales,
hepatomegaly. There was pitting oedema
of the lower limbs. Palpation revealed a
biventricular thrust. There was a regular
tachycardia of 130/m and grade 2 systolic
(ejection type) murmurs over the apex and
base of the heart. The blood pressure was
140/0. Urinalysis showed a trace of albu-
men but no sugar. The E.C.G. showed low-
voltage of the QRS complex and flat or
slightly inverted T-waves. The X-rays con-
firmed the clinical findings of an enlarged
heart.

We were dealing, therefore, with a
case of congestive heart failure with a blood pressure of 140/0 and an abnormal electrocardiogram showing no specific pattern. Since aortic incompetence was not present the diagnosis was one of high-output failure with a collapsing pulse which was not due to an aortic lesion. Clinical and laboratory findings ruled out hyperthyroidism and anaemia (radio-iodine uptake and metabolic rate were normal, there were 4,800,000 r.b.cs. per ml and 92% Hgb) and we were therefore left with the following possibilities of high-output failure:

1. A—V fistula; 2. Severe liver failure; 3. Beri-beri heart disease. At this stage the following investigations were carried out: skeletal X-ray survey, radio-iodine uptake, metabolic rate estimation, liver function tests, electrophoretic pattern study, serum cholesterol, serum electrolytes, serum calcium and phosphorous estimation, alkaline phosphatase, transaminase and haemochromatosis tests all being normal. Wassermann test was negative. Pyruvic acid estimation showed an abnormal rise after glucose: 25 mgm, normal being 1 mgm. Blood urea was 10 mgm. per 100 ml.

A diagnosis of beri-beri heart disease was made and the patient was treated accordingly. He was put on vitamin B\textsubscript{1} 100 mgm by intramuscular injection three times daily for one week, followed by 150 mgm. orally daily. He was also given Digoxin 0.25 mgm. twice daily and 100 mgm. hydrochlorticoside weekly.

After one month's treatment the patient felt much better, was able to return to work, did not get out of breath, the murmurs disappeared and his blood pressure was 130/85. His blood urea had risen to 30 mgm. per 100 ml. The electrocardiogram now showed a normal T-wave and higher voltage of the QRS complex.

Mechanism of heart failure in Vit. B\textsubscript{1} deficiency: Since B\textsubscript{1} is the co-enzyme of carboxylase and is required for normal carbohydrate metabolism and utilisation, its absence renders the heart muscle unable to utilise lactate pyruvate normally. This leads to diminution of myocardial O\textsubscript{2} extraction, insufficient energy production and functional failure. In addition there is an accumulation of the vasodilating intermediate catabolites of glucose (pyruvic, lactic and other keto-acids) which cause widespread peripheral arterial dilatation. This acts like a large arterio-venous fistula augmenting the venous return to the right ventricle and making demands upon the left ventricle for increased output, thus putting a burden on a heart already working at a disadvantage (Konstam G. & Sinclair H. M. 1940). Cardiac catheterisation in a patient with beri-beri heart shows an average cardiac output of 16 litres per minute (normal 4—7 litres) and an O\textsubscript{2} consumption of 355 ml/m (normal average 760 ml/m). Alcoholics who take more of their calories in the form of alcohol (which requires B\textsubscript{1} for its metabolism) and do not eat enough B\textsubscript{1} containing foods, eventually develop heart failure through the mechanism described above.

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Reference

STAPEDECTOMY WITH A SPRING PROSTHESIS
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M.D., B.Sc., D.L.O., F.I.C.S.

In deafness due to otosclerosis the footplate of the stapes becomes ankylosed to the niche of the oval window in such a way that the stapedovestibular joint progressively disappears.

The treatment for this form of deafness is surgical. The stapes is removed and the gap between the incus and the oval window is bridged by means of a prosthesis. The operation is called a stapedectomy and the prosthesis may be either a polythene tube or Teflon. The oval window is usually closed by means of a vein graft but Prof. H. Schuknecht uses as his prosthesis a plug of fat taken from the lobe of the ear (which occludes the oval window) and stainless steel wire which is attached to the incus. Another