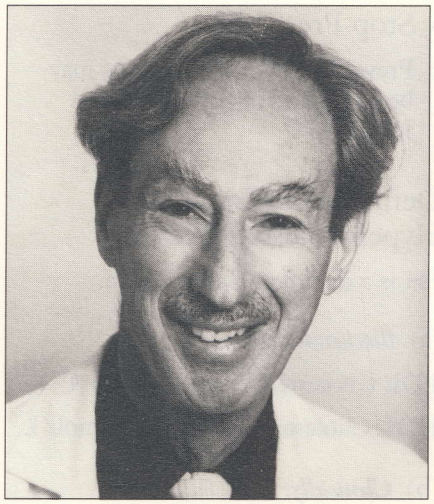
Hyperlipidaemia – Prof HC Seftel



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Curriculum vitae

Prof Seftel was born, lived, educated and worked in Johannesburg. He calls himself a medical evangelist, and serves on a long list of all kinds of committees and organizations. He is currently Professor of African Diseases, University of the Witwatersrand, and Professor of Medicine at the Hillbrow Hospital, as well as Head of the Diabetic Clinic, Johannesburg Hospital. He is a founding member of Hypertension Society of Southern Africa and is actively involved in studies on smoking, alcoholism and drug dependence. Prof Harry is author and co-author of about 200 publications in the fields of endocrinology, metabolism, diabetes, cardiology, haematology, nephrology, nutrition, pharmacology, community medicine and infective diseases. And his hobbies are running, weeding and house cleaning!.

Summary

Hyperlipidaemia is dangerously common in the RSA but the contribution of the front-line doctor can make a massive improvement to both the population strategy as well as to the care of the at-risk individual. Very basic and practical action principles and plans are given in his article.

S Afr Fam Pract 1990; 11: 233-6

KEYWORDS: Hyperlipidemia; Arteriosclerosis; Coronary Disease; Cholesterol; Diet Therapy; Life Style; Physicians, Family.

Hyperlipidaemia is important as it is a *major risk factor* and probably a *fundamental cause* of atheroma. The risk of CHD (coronary heart disease) is X2 with a moderate increase and X4 associated with a marked increase in cholesterol.

It is very common in westernized populations, especially those in RSA: 80% of adults have raised levels, 60% being moderate and 20% markedly raised. Furthermore lowering cholesterol reduces CHD. A decrease in cholesterol of 10% lowers the incidence of CHD by 20-30%. And what's more, lowering cholesterol is effective in both primary and secondary prevention of CHD.

Action Plan of SA Heart Foundation

1. Measure TC

(total cholesterol) as part of clinical assessment.

2. Rank CHD risk

according to TC Action Graph and other risk factors:

- *Desirable risk*

TC in desirable range
no other risk factors

- *Moderate risk*

moderate↑ TC
no other risk factors

- *High risk*

a) Moderate↑ TC plus other risk factors

b) Marked↑ TC with or without other risk factors.

Note: A full lipogram (TC, TG, HDL and HDL-C) is not usually necessary, exept in high risk patients.

3. Treatment

Correct risk factors or cause of TC↑

Control chol < "Drive for five" / "Skuif na vyf"

Always Diet first

Desireable risk

Congratulate and continue FFF (Fish Foul Fibre) or VVV (Vis Voël Vesel).

Moderate risk

Step 1 diet: Total fat <30% of energy intake; saturated <10% of energy intake; cholesterol 300mg/d.

High risk

Step 1 diet - If fails to go

Step 2 diet - Total fat 25% of energy intake; saturated 7% of energy intake; cholesterol 200mg/d.

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If fails - medicines:

(Questran)
Cholestyramine leads to TC↓

(Lurselle)
Probucol leads to TC↓ but also HDL-C↓

(Bezalip)
Bezafibrate leads to TC↓, TG↓,HDL-c↑

(Zocor)
Simvastatin still undergoing trial

- most effective agent for TC↓
- also TG↓ and maybe HDL↑

Medicines may be used alone or together.

Always use medicines together with diet.

4. Monitor Lipid Levels

3 to 6 to 12 monthly.

Examination

* Mass and height
* Eyes - arcus and xanthelasma
* Hands, elbows, knees, HEELS - xanthoma
* Heart and neck - bruits
* Feel pulses (together with xanthomas)
* Urinalysis - glucose, protein, bile pigments.

Check 1° Relatives

- TC, TG
- Examine as above.

No need for fancy and expensive laboratory tests.

You, yourself, can do TC and TG by reflotron.

Principles of Therapy also simple and same for

Most Patients ← Monogenic, Polygenic, Environment

1. Correct risk factors
2. Prudent diet:

   Fish, fowl, fibre
   Vis, voël, vesel
3. Medicines - Few, safe, standard dose:
   - Cholestyramine LDL↓ 8g BD
   - Probucol LDL↓ HDL↓ 500mg BD
   - Bezafibrate LDL↓ TG↓HDL↑ 200mg BD or TDS
4. Monitor effects : 1-4 times yearly.

Patients presenting with Raised TC or TG levels are easily diagnosed by Primary Carers History

* Have you had chest pain, leg pain or stroke (big or small)?
* What do you eat, drink, drug?
* Tell me about your family:
  - Afrikaner or Jew?
  - Anybody else have "cholesterol"?
  - What did they die of and when?
* Has your "cholesterol" been treated?

  If so, *with what and with what result*?

* Stop Press:

  Prevalence of FH in Indians may be as high as in Afrikaners and Jews.

Definition of Familial Hypercholesterolaemia

FH is a disorder characterized:

(a) *Biochemically*

LDL Cholesterol↑ 7-14 mmol/ℓ

HDL Cholesterol N or ↓< 1 mmol/ℓ.

(b) *Clinically:*

Premature atheroma
Tendon Xanthoma
Corneal arcus, Xanthelasma

(c) *Genetically:*

Autosomal Monogenic.

Dominant Trait

* *FH Prevalence in Afrikaners, Jews and ? Indians is ± 1:75. This is about 7 times higher than in any other world population.*

More Important - is Specialist referral necessary?

Whatever the Increase in TC Level, the causes are few:

* Genetic - FH; other, eg FC
* Diet - animal fat intoxication!
* Drugs - eg thiazides (mild↑ TC)
* Some diseases: hypothydroidism, nephrosis, cholestasis.

Cause of ↑ TG are also few:

* Obesity
* CHO excess

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* Alcohol excess
* Diabetes
* Drugs eg beta-blockers.

Is Referral to Lipid Clinics or Specialists Feasible?

Few specialists and clinics in South Africa

BUT

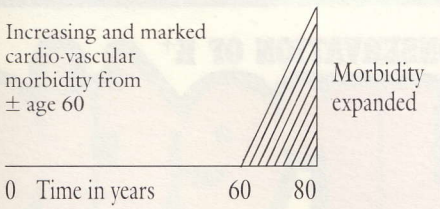
Many people with marked hypercholesterolaemia:

- 50 000 + FH heterozygotes
- Many more thousands with environmental/polygenic hypercholesterolaemia

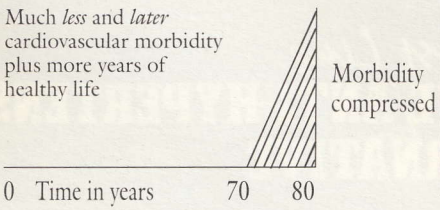
And spread all over the land. Generalist doctors are also spread over the same space!

So consider 2 scenarios in westernized populations whose life expectancy has been extended to 80 years by general environmental uplift:

I. *Without specific effort to lower TC*



II. *With TC Lowering*



We must not only treat those worst affected specially but take the whole population to lower TC levels. See Fig 1.

Last Point for Prudence

If this is what a *single measure,* TC lowering achieves, *multiple measures* (TC↓, smoking O, exercise↑ etc) will *add years to life plus life to years.*

Countering the Cynics

Compressing Morbidity or "Adding Life to Years"

(Fries et al. Lancet 1989, i, 481)

Cynics say lowering cholesterol has little or no effect on *mortality.*

This is largely true, but ignores the *considerable* effect on *morbidity.*

The LRC double-blind randomized primary prevention trial of cholestyramine vs placebo in 3806 hypercholesterolaemic men lasting 7-10 years showed:

(a) 12 fewer CHD deaths in cholestyramine group

But

(b) 206 fewer CV events (eg MI) in the group.

In Conclusion

A dual strategy to control the cholesterol epidemic.

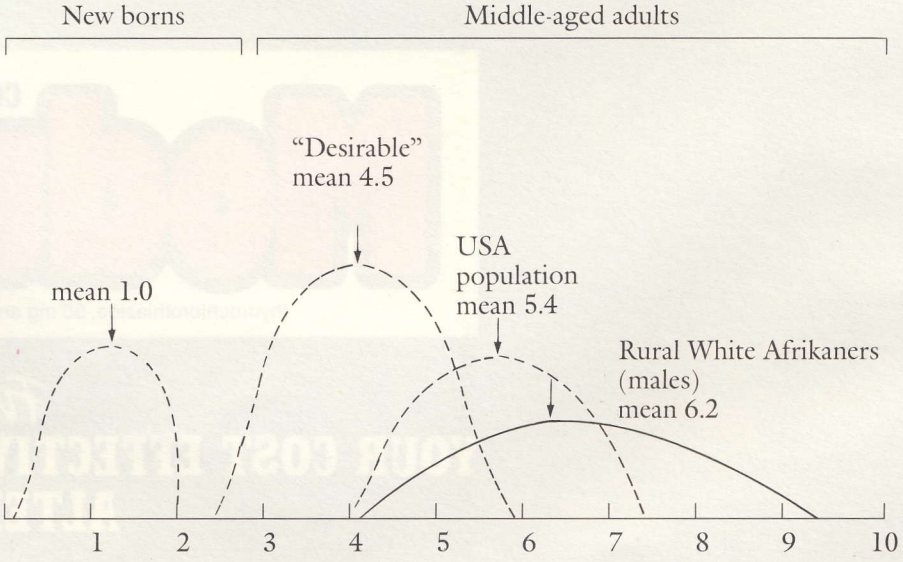
A. *Lowering Cholesterol in Populations*

* Long-term public health programme involving lifestyle changes, especially eat-style.
* Mass TC screening not feasible.

Total Serum Cholesterol Concentration (mMol/ℓ)

Frequency distribution of total serum cholesterol concentrations in Rural White Afrikaners.

Figure I



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B. *Lowering Cholesterol in Individuals*

* Do selective screening or case-finding by measuring TC like BP, at:

  -All First Visits, especially in High Risk Patients:

  Early CVD
  Bad Family History
  Hypertensive
  Smoker
  Diabetes

- Executive or other routine exam
- Life insurance examination
- Employment examination
- Army induction.

In both the population strategy and the care of at-risk individuals the *front-line* doctor can make a major contribution. By regular encouragement of the prudent diet and a healthy life style by all, and selective screening, a massive improvement can be achieved.