concentration need to be established. These cut-off points have been provided in this study. Although cut-off points are arbitrary, they provide a necessary basis for defining normal or abnormal lipid concentrations. The role of lipids and lipoproteins in the aetiology of coronary heart disease is incompletely defined. There are numerous risk factors for coronary heart disease. Abnormal lipid transport is, at any rate, closely associated with coronary heart disease. Abnormality in transport of cholesterol is particularly associated with ischaemic heart disease[10,12].

The present study therefore provides a basis for the definition of hyperlipidaemia of a hypercholesterolaemic nature in this Nigerian population. Although total serum cholesterol determination alone does not indicate whether there is increased low density lipoprotein cholesterol concentration, it is the index that determines whether lipoprotein phenotyping is necessary or not. In children high density lipoprotein cholesterol concentration is high[21]. In children, and in adults also, increased serum cholesterol may be due to increased cholesterol concentration in the high density lipoprotein particles. For such situations, therefore, total serum cholesterol determination is followed by lipoprotein phenotyping.

This low cholesterol level found in the Nigerian population may contribute to the rare incidence of ischaemic heart disease in Nigeria.

It has been shown that high density lipoprotein can act as an antiatherogenic agent[22]. In another study it has been reported that there is high level of high density lipoprotein cholesterol in the Nigerian population.

1 Acknowledgment. I am grateful to Mr S.O. Oguike and Mr O. Uzoechi for technical assistance. I also thank Dr E.O. Anosike for his help in arranging for the subjects of study.
2 K. Suzuki, H. Watanabe, N. Takahashi, S. Tanaka and S. Koyama, Jap. Heart J. *4*, 323 (1963).
3 A.G. Shaper, J. Jones and J. Kyobe, Lancet *2*, 1324 (1961).
4 M.C. Nath and I. Vaishwanar, Indian J. Physiol. allied Sci. *16*, 93 (1963).
5 A.O. Williams, Br. Heart J. *33*, 95 (1971).
6 D.S. Fredrickson, R.I. Levy and R.A. Lees, N. Engl. J. Med. *276*, 32 (1967).
7 L.A. Carlson, Acta med. scand. *167*, 377 (1960).
8 P.J. Scott, Clin. chim. Acta *15*, 449 (1967).
9 B. Lewis, I.D.P. Wootton, D.M. Krikler, A. February, A. Chait, C.M. Oakley, G. Sigurdsson and B. Maurer Lancet *1*, 141 (1974).
10 D. Aldersberg, Am. J. Med. *11*, 60 (1951).
11 L.A. Carlson and L.E. Bottiger, Lancet *1*, 865 (1972).
12 W.B. Kannel, W.P. Castelli, T. Gordon and P.M. McNamara, Annls int. Med. *74*, 1 (1971).
13 A. Keys, Atherosclerosis *22*, 149 (1975).
14 A.G. Shaper and K.W. Jones, Lancet *2*, 1305 (1962).
15 A.G. Shaper, Br. Med. J. *4*, 32 (1972).
16 J. Higginson and W.J. Pepler, J. clin. Invest *33*, 1366 (1954).
17 B.J.P. Becker, S. Afr. J. med. Sci. *11*, 97 (1946).
18 J.C. Edozien, J.W. Afr. Sci. Ass. *10*, 3 (1965).
19 G.O. Taylor, Trop. geogr. Med. *23*, 158 (1971).
20 J.T. Anderson and A. Keys, Clin. Chem. *2*, 145 (1956).
21 I.C. Ononogbu, Thesis. London University 1976.
22 G.J. Miller and N.E. Miller, Lancet *1*, 16 (1975).

Level of marker enzymes in spermatogenesis on administration of $PGF_{2\alpha}$ in rats

Hari Pal Singh, S.S. Kakar and N.C. Ganguli[1]

National Dairy Research Institute, Karnal 132001, Haryana (India), 14 February 1979

Summary. Intraperitoneal administration of $PGF_{2\alpha}$ in rats significantly increased testicular acid phosphatase ($p < 0.05$), decreased hyaluronidase ($p < 0.05$), whereas the activities of 5′-nucleotidase, N-acetyl-B-glucosaminidase, B-galactosidase and uridine diphosphatase remained unaffected.

Available histological and histochemical data suggest that prostaglandins decreased spermatogenesis, weight of testis and accessory sex gland and the level of plasma testosterone in rats[2,3]. PGE_1 and PGE_2 decreased spermatogenesis with a reduction in the spermatid formation[4,5]. The activities of certain 'marker' enzymes viz. acid phosphatase, hyaluronidase, 5′-nucleotidase, N-acetyl-B-glucosaminidase, B-galactosidase and uridine diphosphatase (UDPase) in the testis have been correlated with the cell differentiation in the germinal epithelium during spermatogenesis[6-11]. The effect of $PGF_{2\alpha}$ on the activities of these enzymes has been investigated and this communication presents the results obtained.

Materials and methods. p-Nitrophenylphosphate was obtained from Patel Chest Institute, Delhi. Substrates for all other enzymes studied were from Sigma Chemical Company, USA. $PGF_{2\alpha}$ (tromethamine salt) was a gift sample from Upjohn Company, USA. Other chemicals used were of analytical grade. Male albino rats, weighing 110–150 g, were from the Institute's Small Animal House.

The rats were randomly allotted to 2 groups and housed individually in separate cages. Prior to treatment they were under uniform feeding for about 15 days on cow milk followed by rat feed and water ad libitum. 1 group received $PGF_{2\alpha}$ (tromethamine salt), 3 mg/kg b.wt in saline, i.p., once daily for 15 days, whereas the other group, serving as

Effect of $PGF_{2\alpha}$ on the activity of marker enzymes in rat testis

Treatment	No. of rats	Specific activity** Acid Phosphatase	Hyaluronidase	5′-nucleotidase	N-acetyl-B-glucosaminidase	B-galactosidase	UDPase
Control	4	971.35 ± 52.8	231.35 ± 11.75	1067.95 ± 54.75	204.57 ± 14.43	81.3 ± 2.56	1169.0 ± 121.0
$PGF_{2\alpha}$	3	1084.7* ± 63.17	210.0 ± 10.82	1062.93 ± 93.24	209.43 ± 024.68	84.63 ± 4.66	1225.0 ± 95.8

* Significant at $p < 0.05$. ** Specific activity was defined as the μmoles product released per μg protein/30 min (15 min for 5′-nucleotidase) at 37 °C.

control, received only normal saline i.p. The animals were weighed on the 16th day of administration and sacrificed by decapitation. The testes were excised, stripped of the tunica albuginea, weighed, chilled and processed according to Majumdar et al[12]. The supernatant of the testicular homogenate was then used for enzyme assay. The epididymis was also removed, freed from the adipose tissue, and weighed.

The 'marker' enzymes in cell differentiation during spermatogenesis, namely, acid phosphatase, hyaluronidase, 5'-nucleotidase, N-acetyl-B-glucosaminidase, B-galactosidase and UDPase were assayed according to the methods of Bergmeyer[13], Rhodes et al.[14], Huang and Keenan[15], Conchie[16], Lederberg[17], and Xuma and Turkington[10], respectively. Protein was estimated by the method of Lowry et al.[18].

Results and discussion. The rate of increase of body weight, the weight of the testes and epididymis were less in rats treated with $PGF_{2\alpha}$ than in the control. The differences were, however, insignificant. The table shows the specific activities of the 'marker' enzymes in testes in treated and control rats. The activity of acid phosphatase was higher ($p < 0.05$) in treated than in the control rats. Such increase in activity would mean an increase in the proacrosomal bodies on the administration of $PGF_{2\alpha}$[19]. The level of hyaluronidase in treated rats was significantly less ($p < 0.05$) than in the control. Hyaluronidase in testis is localized in acrosome of developing spermatids and mature spermatozoa[6]. A decrease in the level of hyaluronidase indicates suppression of the formation of acrosome in the newly formed spermatids. The activities of 5'-nucleotidase, N-acetyl-B-glucosaminidase, B-galactosidase and UDPase in the treated animals were not significantly different from that in the controls.

1 Acknowledgment. We are grateful to Dr D. Sundaresan, for his interest in the study and to Mr B.M. Sharma for technical assistance.
2 G.N. Memon, Contraception *8*, 361 (1973).
3 G.N. Memon, J. Life Sci. *4*, 31 (1974).
4 E.R. Abbatiellow, M. Kaminsky and S. Weisbroth, Int. J. Fert. *20*, 177 (1975).
5 E.R. Abbatiellow, M. Kaminsky and S. Weisbroth, Int. J. Fert. *21*, 82 (1976).
6 J.L. Males and R.W. Turkington, J. biol. Chem. *245*, 6329 (1970).
7 J.L. Males and R.W. Turkington, Endocrinology *88*, 579 (1971).
8 M. Parvinen and T. Vanha-Pertulla, Anat. Rec. *174*, 435 (1972).
9 T. Vanha-Pertulla and V. Nikkanen, Acta endocr. *72*, 376 (1973).
10 M. Xuma and R.W. Turkington, Endocrinology *91*, 415 (1972).
11 G.C. Majumdar and R.W. Turkington, Biochemistry *13*, 2857 (1974).
12 G.C. Majumdar, S. Lessin and R.W. Turkington, Endocrinology *96*, 890 (1975).
13 H.U. Bergmeyer, ed., Methods of Enzymatic Analysis. Academic Press, London 1963.
14 C. Rhodes, K.S. Dudyason, A.H. Olavesen and B. Hogberg, Biochem. J. *122*, 575 (1971).
15 C.M. Huang and T.W. Keenan, Biochim. biophys. Acta *274*, 246 (1972).
16 J. Conchie, Biochem. J. *58*, 552 (1954).
17 J. Lederberg, J. Bact. *60*, 381 (1950).
18 O.H. Lowry, N.J. Rosebrough, A.L. Farr and R.J. Randall, J. biol. Chem. *193*, 265 (1951).
19 R.W. Turkington and G.C. Majumdar, J. Cell Physiol. *85*, 495 (1975).

Effects of several preoperative medications on fat cell lipolysis, and activity of adipose tissue cyclic AMP phosphodiesterase

P.B. Curtis-Prior[1,2], M. Jenner, Y-H. Chan and I. McColl

Research Department, The Marie Curie Memorial Foundation, Oxted (Surrey), and Department of Surgery, Guy's Hospital, London (England), 19 February 1979

Summary. Effects were examined of atropine, diazepam, pethidene, promethazine, scopolamine, omnopon and papaverine on basal and noradrenaline-stimulated lipolysis in rat isolated fat cells and on rat adipose tissue cyclic AMP phosphodiesterase activity. Papaverine at high concentration (1 mM) inhibited both basal and hormone-stimulated lipolysis, whereas diazepam enhanced basal lipolysis. At a 'clinical dose', omnopon increased both basal and noradrenaline-stimulated lipolysis. Adipose tissue cAMP phosphodiesterase activity was strongly inhibited by 1 mM diazepam, papaverine, promethazine and omnopon (280 μg ml^{-1}). Lack of enhancement of lipolysis by the established cAMP phosphodiesterase antagonist papaverine, is compatible with simultaneous inhibition also of adipose adenyl cyclase. Diazepam-stimulated lipolysis is compatible with its phosphodiesterase inhibitory activity. It is proposed that papaverine-containing omnopon may offer some survival advantages during surgical stress by facilitating a caloric supply.

Human adipose tissue for study, in vitro, may be obtained by fat biopsy[3] or by harvesting available tissue from patients undergoing major surgery. This latter method provides large samples and is widely-used; yet little information is available regarding the effects of medications received by patients on the subsequent behaviour of their adipose tissue, when incubated in vitro.

The aim of this study was to investigate the effects of several commonly-used preoperative medications on adipose tissue lipolysis, using a rat isolated fat cell as a model; and to compare observed lipolytic activity with cyclic AMP (cAMP) phosphodiesterase activity, in view of the involvement of the cAMP-system in the lipolytic process.

Methods. Male Sprague-Dawley rats weighing 190–210 g were fasted overnight but allowed water ad libitum. They were sacrificed by a blow on the head and epididymal adipose tissue extirpated and used to prepare suspensions of isolated fat cells in Krebs-Ringer bicarbonate buffer solution (containing glucose 45 mg 100 ml^{-1} and bovine serum albumin 3.5 g 100 ml^{-1}) at pH 7.4 using a technique[4] modified from Rodbell[5].

The compounds examined were atropine (as the sulphate), diazepam, pethidine and promethazine, hydrochlorides, scopolamine hydrobromide and omnopon. Omnopon is a preparation of the hydrochlorides of the opium alkaloids, morphine representing about 50% of the total (and respon-