SECTION VII

APPENDICES

APPENDIX 1

SUBMISSIONS SINCE INTERIM REPORT

SUBMISSIONS SINCE INTERIM REPORT

The following individuals and organisations made written submissions to the Committee after the tabling of the Interim Report.

Submission No.

64. The Hon. M.J. Ahern, Premier of Queensland, Brisbane, QLD

65. Queensland Gymnasium Owners Association, Brisbane, QLD

66. Australian Athletic Union, Moonee Ponds, VIC

67. Dr M. Sheehan, Department of Social and Preventive Medicine, University of Queensland, Herston, QLD

68. Queensland Rugby Football League Ltd., Milton, QLD

69. Mr L. Azar, Carrindale, QLD

70. Mr S. Shortis, Chatswood, NSW

71. Mr S. Zammataro, Miriwinni, QLD

72. Mr C. Dumke, Trinder Park Rest Home, Woodridge, QLD

73. National Basketball League, South Yarra, VIC

74. Victorian Football League, Melbourne, VIC

75. The Hon. N. Greiner, Premier of NSW, Sydney, NSW

76. Ms D.L. Jensen, Combined Regional Bodybuilding Association, Newcastle West, NSW

77. Mr J.G.H. Refshauge, Carlton, VIC

78. Ms Debbie Flintoff-King, Moorooduc, VIC

79. Mr Robert Wilks, Australian Powerlifting Inc., South Yarra, VIC

80. Mr G. Ellison, Kaleen, ACT

81. Mr S. Haynes, Australian Sports Drug Agency, Curtin, ACT

82. Mr G. Jones, Australian Drug Free Powerlifting Federation, Queanbeyan, NSW

83. Mr H. Cerncic and Ms J. Dobson, PO Box 3, Rushcutters Bay, NSW

84. Mr J. Czapla and Mr S. Ma, 5 Casino Avenue, Greystanes, NSW

85. Mr R. Rigby, PO Gordon, VIC

APPENDIX 2

SCHEDULE OF PUBLIC HEARINGS

SCHEDULE OF PUBLIC HEARINGS

Date of Hearing	Individuals/ Organisations	Represented By
11 September 1989 (Brisbane)	Mr N.B. Jones	
12 September 1989 (Brisbane)	Mr K.A. Wilson	
	Mr P. Kabakoff	
	Australian Drug Free Powerlifting Federation	Mr C.J. Turner, Secretary
	Department of Social and Preventive Medicine, The Medical School, University of Queensland	Dr M. Sheehan, Senior Lecturer
		Mr R. Henderson, Student Researcher
		Mr B.B. Kelly, Student Researcher
	Brisbane Broncos Rugby League Club	Mr B.W. Canavan, Development Officer
	Mr C.R. Dumke	
13 September 1989 (Brisbane)	Queensland Powersports Association	Mr D.D. Toci, Coaching Co-ordinator
	Mr W.A. Scarffe	
	Dr B.T. Ross	
	Mr L.A. Azar	
	Dr S. Hinchy	

Date of Hearing	Individuals/ Organisations	Represented By
14 September 1989 (Brisbane)	Australian Sports Drug Agency	Mr S. Haynes, Chief Executive
	Mr W.J. Lewis	
	Mr G.L. Olling	
	Dr K.T. Hobbs	
	Queensland Rugby Football League	Mr R.A. Livermore, Managing Director
15 September 1989 (Brisbane)	Mr G.S. Jensen	
	Mr P.J. McCarthy	
	Mr M. Jardine	
	Australian Sports Drug Agency	Mr S. Haynes, Chief Executive
25 October 1989 (Canberra)	NSW Rugby League Inc.	Mr J.R. Quayle, General Manager
1 November 1989 (Canberra)	Australian Soccer Federation	Mr I. Brusasco, Chairman
		Dr A.B. Corrigan, Director, Medical Commission
	National Basketball League	Mr W.A. Palmer, Junior, General Manager
13 November 1989 (Sydney)	Australian Drug Free Powerlifting Federation	Mr G.A. Jones, National Testing Officer
	Chief Inspector L.G. Topping	
	Mr J.C. Brent	

Date of Hearing	Individuals/ Organisations	Represented By
	Australian Sports Drug Agency	Mr S. Haynes, Chief Executive
	Australian Government Analytical Laboratories	Dr R. Kazlauskas, Principal Chemist
	Mr G. Zeltzer	
	Mr C.J. Bova	
14 November 1989 (Sydney)	Mr B. Stellios	
	Mr R. Caine	
	Australian Weightlifting Federation	Mr B.B. Walsh, Executive Director
15 November 1989 (Melbourne)	Mr M.K. Brittain	
	Mr R. Kabbas	
	Dr R.A. Ward	
16 November 1989 (Melbourne)	Dr N.A. Keks	
	Victorian Weightlifting Association	Mr P. Coffa, Executive Director
	Australian Weightlifting Federation	Mr S. Coffa, President
		Mr B. Kayser, Secretary
		Dr D.R. Kennedy, Chairman of Medical Committee
	Australian Medical Association	Dr R.J. Whiting, President Elect
		Dr P.A. Larkins, Member

Date of Hearing	Individuals/ Organisations	Represented By
	Australian Amateur Powerlifting Federation	Mr R.E. Rigby, Member
17 November 1989 (Melbourne)	Dr P. Brukner	
	Victorian Football League Players Association	Mr S.J. Madden, President
	Australian Football League	Dr A.G. Capes, Chairman of Drug Subcommittee
	Australian Football League Commission	Mr R. Graham, Chairman
20 November 1989 (Canberra)	Australian Powerlifting Federation Inc.	Mr R.J. Orr, President
		Mr R. Lewis, Secretary
6 December 1989 (Canberra)	Australian Powerlifting	Mr G.D. Ellison, Vice-President and Coaching Co-ordinator
7 December 1989 (Canberra)	Federal Government of Canada	Mr L.M. Maskosky, Assistant Deputy Minister, Ministry of Fitness and Amateur Sport

APPENDIX 3

SCHEDULE OF COMMITTEE CONTACT WITH PERSONS ADVERSELY MENTIONED IN THE SECOND DRUGS IN SPORT REPORT

SCHEDULE OF COMMITTEE CONTACT WITH PERSONS ADVERSELY MENTIONED IN THE SECOND DRUGS IN SPORT REPORT

CHAPTER	NAME	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
One	No adverse mentions			
Two	No adverse mentions			
Three	Mr Sam Coffa	16.11.89	23.01.90	19.03.90
Four	Mr Paul Coffa	16.11.89		
	Mr Barry Parnell		07.03.90	16.03.90
	Mr Bruce Walsh	14.11.89		
Five	Mr Alistair Edwards		Overseas	
	[The AIS provided the Committee with a set of documents relating to this incident, which included a letter from Mr Edwards detailing his knowledge of the incident.]			
	Mr Darren McCarthy		25.01.90* 27.02.90 29.03.90	

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Six	No adverse mentions			
Seven	Mr Michael Brittain	15.11.89	06.02.90	18.02.90
	Mr Phillip Christou (per Michael Noonan coach)		25.01.90	02.02.90
	Mr Paul Coffa	16.11.89	25.01.90*	07.02.90
	Mr Sam Coffa	16.11.89	15.12.89 03.01.90 23.01.90 31.01.90 19.02.90 08.03.90 17.04.90	18.12.89 08.01.90 19.03.90 19.03.90 27.02.90 and 19.03.90 19.03.90 21.04.90
	Mr Neville Cornelius		22.03.90*	
	Mr Jan Czapla	Informal discussion with Chair-man on 14.02.90	07.12.89	05.02.90 14.02.90
	Mr Frank Falcone		14.03.90	09.11.89
	Mr Robert Kabbas	15.11.89		
	Mr Boris Kayser	16.11.89	06.02.90	15.02.90

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Seven cont.	Dr David Kennedy	16.11.89	02.01.90 01.02.90 19.02.90	10.01.90 07.02.90 22.02.90
	Mr Phillip Kerr		28.03.90*	
	Mr David Lowenstein		14.03.90	06.11.89
	Mr Satry Ma	Informal Discussion with Chairman on 14.02.90	25.01.90	05.02.90 14.02.90
	Mr Jason Roberts		29.11.89 02.03.90 08.03.90	
	Mr Wayne Scarffe	13.9.89	28.03.90*	
	Mr Bill Stellios	14.11.89		
	Mr Christopher Stewart		(address unknown)	
	Mr George Stylianou		28.03.90*	
	Dr Alex Tahmindjis		25.01.90* 08.02.90	31.01.90 14.02.90 07.03.90
	Mr Ian Traill		11.12.89	14.12.89

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Seven cont.	Mr Nick Voukelatos	Informal Discussion with Chairman on 14.02.90	06.02.90 29.03.90	
	Mr Darren Walker		09.03.90	17.03.90
	Mr Bruce Walsh	14.11.89		
Eight	Mr Scott Boyd		22.03.90*	
	Mr Charlie Coleiro		(address unknown)	
	Mr Mason Jardine	15.09.89		
	Ms Rosita Kruhse		25.01.90*	
	Mr Terry Lonsdale		13.03.90*	
	Mrs Gael Martin	30.11.88		
	Mr Ray Rigby	16.11.89		
	Mr Wayne Scarffe	13.09.89		
	Mr Yuris Sterns		13.02.90*	
	Mr Dino Toci	13.09.89		
	Mr Larry Wallen		19.07.89	(Overseas)
	Mr Glenn Waszkiel		19.07.89	04.09.89

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Eight cont.	Mr Robert Wilks	20.11.89		
Nine	Mr Leon Azar	13.09.89	10.11.89 29.11.89	22.11.89 18.01.90
	Dr Breitkreutz		06.02.90*	
	Mr David Burgess		01.02.90	
	Mr Ross Everett		10.11.89	24.11.89
	Ms Bev Francis		21.02.90	25.02.90
	Dr Hinchy	13.09.89		
	Dr Jeremijenko		25.01.90*	
	Mr Michael John		07.02.90*	
	Dr Martin		06.02.90*	
	Dr Millar	21.11.88		
	Dr Mitchelson		Sept. 89 [Phoned on 12.12.89] 13.12.89	
	Dr Mullett		(address unknown)	
	Mr Michael Rothnie		10.11.89	14.11.89
	Dr Roudenko		06.02.90*	

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Nine cont.	Dr Ryan		20.09.89	04.10.89
	Mr Donald Steedman		16.02.90	
	Dr Alex Tahmindjis		25.01.90* 08.02.90	31.01.90 14.02.90 07.03.90
	Dr Trevor		06.02.90*	
	Mr Bruce Walsh	14.11.89		
	Dr Ward	15.11.89		07.12.88
Ten	Mr Leon Azar	13.09.89		
	Mr Scott Brodie		25.01.90*	
	Mr Paul Coffa	16.11.89	See Chapter 7 above	
	Mr Sam Coffa	16.11.89	See Chapter 7 above	
	Mr George Farquhar		25.01.90*	
	Mr Robert Huber		25.01.90*	
	Dr Jeremijenko		25.01.90*	
	Mr Michael John		07.02.90*	
	Mr Paul Jordan		25.01.90*	

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Ten cont.	Ms Sue-Ellen Law		02.04.90*	[Returned]
	Mr Joe Lopez	16.11.89		
	Mr Danny Mackay		01.02.90*	
	Mr Peter McCarthy	15.09.89	29.11.89	06.12.89
	Dr Mitchelson		Sept. 89 [Phoned on 12.12.89] 13.12.89	
	Mr Bill Moore		25.01.90*	
	Mr Serge Nubret		Overseas	
	Mr Andreas Olbrich		25.01.90*	
	Mr Doug Powell		25.01.90*	
	Mr Bruce Rigby		11.01.90	
	Mr Ray Rigby	16.11.89	11.01.90	22.1.90
	Mr Leslie Rudolf		25.01.90*	
	Mr Donald Steedman		16.02.90	
	Mr Tony Strutt		25.01.90*	
	Mr Rod Sylvia		25.01.90*	

CHAPTER CONTACT	NAME RESPONSE	HEARING	LETTER FROM COMMITTEE	LETTER TO COMMITTEE
Ten cont.	Mr Ken Ware		07.02.90*	
	Mr Leo Wimmera		25.01.90*	[Returned]
Eleven	Mr Scott Brodie		25.01.90*	
	Mr Michael Lawrence		22.01.90	12.02.90
	Mr Bruce Walsh	14.11.89		
Twelve	Dr Jeremijenko		25.1.90*	
Thirteen	No adverse mentions			

* pro forma letter

APPENDIX 4

PARLIAMENTARY PRIVILEGE

Procedures to be observed by Senate committees for the protection of witnesses

That, in their dealings with witnesses, all committees of the Senate shall observe the following procedures:

(1) A witness shall be invited to attend a committee meeting to give evidence. A witness shall be summoned to appear (whether or not the witness was previously invited to appear) only where the committee has made a decision that the circumstances warrant the issue of a summons.

(2) Where a committee desires that a witness produce documents relevant to the committee's inquiry, the witness shall be invited to do so, and an order that documents be produced shall be made (whether or not an invitation to produce documents has previously been made) only where the committee has made a decision that the circumstances warrant such an order.

(3) A witness shall be given reasonable notice of a meeting at which the witness is to appear, and shall be supplied with a copy of the committee's order of reference, a statement of the matters expected to be dealt with during the witness's appearance, and a copy of these procedures. Where appropriate a witness shall be supplied with a transcript of relevant evidence already taken.

(4) A witness shall be given opportunity to make a submission in writing before appearing to give oral evidence.

(5) Where appropriate, reasonable opportunity shall be given for a witness to raise any matters of concern to the witness relating to the witness's submission or the evidence the witness is to give before the witness appears at a meeting.

(6) A witness shall be given reasonable access to any documents that the witness has produced to a committee.

(7) A witness shall be offered, before giving evidence, the opportunity to make application, before or during the hearing of the witness's evidence, for any or all of the witness's evidence to be heard in private session, and shall be invited to give reasons for any such application. If the application is not granted, the witness shall be notified of reasons for that decision.

(8) Before giving any evidence in private session a witness shall be informed whether it is the intention of the committee to publish or present to the Senate all or part

of that evidence, that it is within the power of the committee to do so, and that the Senate has the authority to order the production and publication of undisclosed evidence.

(9) A chairman of a committee shall take care to ensure that all questions put to witnesses are relevant to the committee's inquiry and that the information sought by those questions is necessary for the purpose of that inquiry. Where a member of a committee requests discussion of a ruling of the chairman on this matter, the committee shall deliberate in private session and determine whether any question which is the subject of the ruling is to be permitted.

(10) Where a witness objects to answering any question put to the witness on any ground, including the ground that the question is not relevant or that the answer may incriminate the witness, the witness shall be invited to state the ground upon which objection to answering the question is taken. Unless the committee determines immediately that the question should not be pressed, the committee shall then consider in private session whether it will insist upon an answer to the question, having regard to the relevance of the question to the committee's inquiry and the importance to the inquiry of the information sought by the question. If the committee determines that it requires an answer to the question, the witness shall be informed of that determination and the reasons for the determination, and shall be required to answer the question only in private session unless the committee determines that it is essential to the committee's inquiry that the question be answered in public session. Where a witness declines to answer a question to which a committee has required an answer, the committee shall report the facts to the Senate.

(11) Where a committee has reason to believe that evidence about to be given may reflect adversely on a person, the committee shall give consideration to hearing that evidence in private session.

(12) Where a witness gives evidence reflecting adversely on a person and the committee is not satisfied that that evidence is relevant to the committee's inquiry, the committee shall give consideration to expunging that evidence from the transcript of evidence, and to forbidding the publication of that evidence.

(13) Where evidence is given which reflects adversely on a person and action of the kind referred to in paragraph (12) is not taken in respect of the evidence, the committee shall provide reasonable opportunity for that person to have access to that evidence and to respond to that

evidence by written submission and appearance before the committee.

(14) A witness may make application to be accompanied by counsel and to consult counsel in the course of a meeting at which the witness appears. In considering such an application, a committee shall have regard to the need for the witness to be accompanied by counsel to ensure the proper protection of the witness. If an application is not granted, the witness shall be notified of reasons for that decision.

(15) A witness accompanied by counsel shall be given reasonable opportunity to consult counsel during a meeting at which the witness appears.

(16) An officer of a department of the Commonwealth or of a State shall not be asked to give opinions on matters of policy, and shall be given reasonable opportunity to refer questions asked of the officer to superior officers or to a Minister.

(17) Reasonable opportunity shall be afforded to witnesses to make corrections of errors of transcription in the transcript of their evidence and to put before a committee additional material supplementary to their evidence.

(18) Where a committee has any reason to believe that any person has been improperly influenced in respect of evidence which may be given before the committee, or has been subjected to or threatened with any penalty or injury in respect of any evidence given, the committee shall take all reasonable steps to ascertain the facts of the matter. Where the committee considers that the facts disclose that a person may have been improperly influenced or subjected to or threatened with penalty or injury in respect of evidence which may be or has been given before the committee, the committee shall report the facts and its conclusions to the Senate.

Resolutions of the Senate - 25 February 1988

APPENDIX 5

IN CAMERA EVIDENCE

IN CAMERA EVIDENCE

A Senate Committee may agree to take evidence in camera. This means that the evidence will be taken in private, with the public and press excluded. In agreeing to take evidence in camera the Committee will inform a witness whether it is the intention of the committee to publish or present to the Senate all or part of the evidence. For example, where a matter is either before a court of law or pending legal proceedings (sub judice), the Committee might wish to hear evidence in camera in order to avoid influencing or prejudicing the outcome of court proceedings. In these circumstances the Committee may indicate that it will authorise the publication of the in camera evidence once the legal proceedings have been completed.

When receiving in camera evidence for other than sub judice reasons it will generally be the intention of the Committee that the evidence will not be published. However, it should be noted that the Committee is unable to give a binding assurance that evidence taken in camera will not be disclosed. This is because disclosure can be authorised by three mechanisms:

- a resolution of the Committee concerned can result in the publication or the presentation to the Senate of evidence taken in camera;
- the production and publication of undisclosed evidence can be authorised by the Senate;
- an individual member of the Committee preparing a dissenting report may, without reference to the Committee or the witness, disclose in camera evidence which the member claims is clearly relevant to the matter on which the Senator dissents and which forms a necessary part of the reasoning of the dissent.

Clearly, the first of these mechanisms is under the control of the Committee and is unlikely to be applied if the Committee has indicated it does not intend to disclose in camera evidence. However, the membership of the Committee may change or the Committee may decide at some later stage that the reasons for confidentiality may no longer exist. In this case the Committee would normally notify the witness and seek his or her up-to-date preference about the matter. The other two mechanisms through which disclosure can be authorised are outside the direct control of the Committee. However, it should be noted their use has been rare.

In giving in camera evidence it should be noted that the resolutions adopted by the Senate on 25 February 1988 concerning procedures to be observed by Senate committees for the protection of witnesses state that:

> [w]here evidence is given which reflects adversely on a person ... the committee shall provide reasonable opportunity for that person to have access to that evidence and to respond to that evidence by written submission and appearance before the committee. (paragraph 13)

When a Committee has taken evidence in camera involving allegations made against an individual, the Committee will normally try to raise these allegations with the individual concerned in such a way that the identity of the witness making the allegations is not disclosed. This would be done during the course of an in camera hearing.

Distribution of the Hansard transcript of in camera evidence is limited to the witness, the Committee members, the Committee secretariat and to Hansard. Extra security, such as double enveloping is used in the distribution of such evidence.

Unauthorised disclosure of in camera evidence is both a contempt of the Senate and a criminal offence. The Parliamentary Privileges Act 1987 sets out the penalties for unauthorised disclosure of in camera evidence as:

- in the case of a natural person, $5 000 or imprisonment for 6 months;
- in the case of a corporation $25 000

It should be noted that disclosure can be authorised only by the three methods described. Disclosure cannot be authorised by the witness providing the evidence. If a witness later changes his or her mind about the need for secrecy, the Committee should be advised as, in this case, the Committee might wish to consider the possibility of disclosure.

If a witness wishes to keep confidential the fact that he or she has appeared to give evidence before the Committee, as well as the evidence given, this should be made clear to the Committee secretary as soon as possible.

Note:

Where there is an absolute need to ensure confidentiality a Committee may agree to hold private discussions with a prospective witness rather than take formal evidence.

APPENDIX 6

INTERNATIONAL OLYMPIC COMMITTEE LIST OF DOPING CLASSES AND METHODS 1989

INTERNATIONAL OLYMPIC COMMITTEE
LIST OF DOPING CLASSES AND METHODS 1989

I. DOPING CLASSES

A. Stimulants
B. Narcotics
C. Anabolic Steroids
D. Beta-blockers
E. Diuretics

II. DOPING METHODS

A. Blood doping
B. Pharmacological, chemical and physical manipulation

III. CLASSES OF DRUGS SUBJECT TO CERTAIN RESTRICTIONS

A. Alcohol
B. Local anaesthetics
C. Corticosteroids

NOTE:

The doping definition of the IOD Medical Commission is based on the banning of pharmacological classes of agents.

The definition has the advantage that also new drugs, some of which may be especially designed for doping purposes, are banned.

The following list represents examples of the different dope classes to illustrate the doping definition. Unless indicated all substances belonging to the banned classes may not be used for medical treatment, even if they are not listed as examples. If substances of the banned classes are detected in the laboratory the IOC Medical Commission will act. It should be noted that the presence of the drug in the urine constitutes an offence, irrespective of the route of administration.

EXAMPLES AND EXPLANATIONS

I. DOPING CLASSES

A. Stimulants e.g.

amfepramone
amfetaminil
amiphenazole
amphetamine
benzphetamine
caffeine*
cathine
chlorphentermine

clobenzerox
cloprenaline
cocaine
cropropamide (component of 'micoren')
crothetamide (component of 'micoren')
dimetamfetamine
ephedrine
etafedrine
ethamivan
etilamfetamine
fencamfemin
fenetylline
fenproporex
furfenorex
mefenorex
methamphetamine
methoxyphenamine
methylephedrine
methylphenidate
morazone
nikethamide
pemoline
pentetrazol
phendimetrazine
phenmetrazine
phentermine
phenylpropanolamine
pipradol
prolintane
propylhexedrine
pyrovalerone
strychnine and related compounds

* For caffeine the definition of a positive depends upon the following: - if the concentration in urine exceeds 12 micrograms/ml.

Stimulants comprise various types of drugs which increase alertness, reduce fatigue and may increase competitiveness and hostility. Their use can also produce loss of judgement, which may lead to accidents to others in some sports. Amphetamine and related compounds have the most notorious reputation in producing problems in sport. Some deaths of sportsmen have resulted even when normal doses have been used under conditions of maximum physical activity. There is no medical justification for the use of 'amphetamines' in sport.

One group of stimulants is the sympathomimetic amines of which ephedrine is an example. In high doses, this type of compound produces mental stimulation and increased blood flow. Adverse effects include elevated blood pressure and headache, increased and irregular heart beat, anxiety and tremor. In lower doses, they e.g. ephedrine, pseudoephedrine, phenylpropanolamine, norpseudoephedrine, are often present in cold and hay fever

preparations which can be purchased in pharmacies and sometimes from other retail outlets without the need of a medical prescription.

THUS NO PRODUCE FOR USE IN COLDS, FLU OR HAY FEVER PURCHASED BY A COMPETITOR OR GIVEN TO HIM SHOULD BE USED WITHOUT FIRST CHECKING WITH A DOCTOR OR PHARMACIST THAT THE PRODUCT DOES NOT CONTAIN A DRUG OF THE BANNED STIMULANTS CLASS.

-Beta2 agonists

The choice of medication in the treatment of asthma and respiratory ailments has posed many problems. Some years ago, ephedrine land related substances were administered quite frequently. However, these substances are prohibited because they are classed in the category of 'sympathomimetic amines' and therefore considered as stimulants.

The use of only the following beta2 agonists is permitted in the aerosol form:

bitolterol
orciprenaline
rimiterol
salbutamol
terbutaline

B. Narcotic analgesics e.g.

alphaprodine
anileridine
buprenorphine
codeine
dextromoramide
dextropropoxyphen
diamorphine (heroin)
dihydrocodeine
dipipanone
ethoheptazine
ethylmorphine
levorphanol
methadone
morphine
nalbuphine
pentazocine
pethidine
phenazocine
trimeperidine and related compounds

The drugs belonging to this class, which are represented by morphine and its chemical and pharmacological analogs, act fairly specifically as analgesics for the management of moderate to severe pain. This description however by no means implies that their clinical effect is limited to the relief of trivial

disabilities. Most of these drugs have major side effects, including dose-related respiratory depression, and carry a high risk of physical and psychological dependence. There exists evidence indicating that narcotic analgesics have been and are abused in sports, and therefore the IOC Medical Commission has issued and maintained a ban on their use during the Olympic Games. The ban is also justified by international restrictions affecting the movement of these compounds and is in line with the regulations and recommendations of the World Health Organisation regarding narcotics.

Furthermore, it is felt that the treatment of slight to moderate pain can be effective using drugs - other than the narcotics - which have analgesic, anti-inflammatory and antipyretic actions. Such alternatives, which have been successfully used for the treatment of sports injuries, include Anthranilic acid derivatives (such as Mefenamic acid, Floctafenine, Glafenine, etc.), Phenylalkanoic acid derivatives (such as Diclofenac, Ibuprofen, Ketoprofen, Naproxen, etc.) and compounds such as Indomethacin and Sulindac. The Medical Commission also reminds athletes and team doctors that Aspirin and its newer derivatives (such as Diflunisal) are not banned but cautions against some pharmaceutical preparations where Aspirin is often associated to a banned drug such as Codeine. The same precautions hold for cough and cold preparations which often contain drugs of the banned classes.

NOTE: DEXTROMETHORPHAN IS NOT BANNED AND MAY BE USED AS AN ANTI-TUSSIVE. DIPHENOXYLATE IS ALSO PERMITTED.

C. Anabolic steroids e.g.

bolasterone
boldenone
clostebol
dehydrochlormethyltestosterone
fluoxymesterone
mesterolone
metandienone
metenolone
methyltestosterone
nandrolone
norethandrolone
oxandrolone
oxymesterone
oxymetholone
stanozolol
testosterone** and related compounds

** Testosterone: the definition of a positive depends upon the following - the administration of testosterone or the use of any other manipulation having the result of increasing the ratio in urine of testosterone/epitestosterone to above 6.

It is well known that the administration to males of Human Chorionic Gonadotrophin (HCG) and other compounds with related activity leads to an increased rate of production of androgenic steroids. The use of these substances is therefore banned.

This class of drugs includes chemicals which are related in structure and activity to the male hormone testosterone, which is also included in this banned class. They have been misused in sport, not only to attempt to increase muscle bulk, strength and power when used with increased food intake, but also in lower doses and normal food intake to attempt to improve competitiveness.

Their use in teenagers who have not fully developed can result in stunting growth by affecting growth at the ends of the long bones. Their use can produce psychological changes, liver damage and adversely affect the cardio-vascular system. In males, their use can reduce testicular size and sperm production; in females, their use can produce masculinisation, acne, development of male pattern hair growth and suppression of ovarian function and menstruation.

D. Beta-blockers e.g.

acebutolol
alprenolol
atenolol
labetalol
metoprolol
nadolol
oxprenolol
propranolol
sotalol and related compounds

The IOC Medical Commission has reviewed the therapeutic indications for the use of beta-blocking drugs and noted that there is now a wide range of effective alternative preparations available in order to control hypertension, cardiac, arrythmias, angina pectoris and migraine. Due to the continued misuse of beta-blockers in some sports where physical activity is of no or little importance, the IOC Medical Commission reserves the right to test those sports which it deems appropriate. These are unlikely to include endurance events which necessitate prolonged periods of high cardiac output and large stores of metabolic substrates in which beta-blockers would severely decrease performance capacity.

E. Diuretics e.g.

acetazolamide
amiloride

bendroflumethiazide
benzthiazide
bumetanide
canrenone
chlormerodrin
chlortalidone
diclofenamide
ethacrynic acid
furosemide
hydrochlorothiazide
mersalyl
spironolactone
triamterene and related compounds

Diuretics have important therapeutic indications for the elimination of fluids from the tissues in certain pathological conditions. However, strict medical control is required.

Diuretics are sometimes misused by competitors for two main reasons, namely: to reduce weight quickly in sports where weight categories are involved and to reduce the concentration of drugs in urine by producing a more rapid excretion of urine to attempt to minimise detection of drug misuse. Rapid reduction of weight in sport cannot be justified medically. Health risks are involved in such misuse because of serious side-effects which might occur.

Furthermore, deliberate attempts to reduce weight artificially in order to compete in lower weight classes or to dilute urine constitute clear manipulations which are unacceptable on ethical grounds. Therefore, the IOC Medical Commission has decided to include diuretics on its list of banned classes of drugs.

N.B. For sports involving weight classes, the IOC Medical Commission reserves the right to obtain urine samples from the competitor at the time of the weigh-in.

II. METHODS

A. Blood doping

Blood transfusion is the intravenous administration of red blood cells or related blood products that contain red blood cells. Such products can be obtained from blood drawn from the same (autologous) or from a different (non-autologous) individual. The most common indications for red blood transfusion in conventional medical practice are acute blood loss and severe anaemia.

Blood doping is the administration of blood or related blood products to an athlete other than for legitimate medical treatment. This procedure may be preceded by withdrawal of blood from the athlete who continues to train in this blood depleted state.

These procedures contravene the ethics of medicine and of sport. There are also risks involved in the transfusion of blood and related blood products. These include the development of allergic reactions (rash, fever etc.) and acute haemolytic reaction with kidney damage if incorrectly typed blood is used as well as delayed transfusion reaction resulting in fever and jaundice, transmission of infectious diseases (viral hepatitis and AIDS), overload of the circulation and metabolic shock.

Therefore the practice of blood doping in sport is banned by the IOC Medical Commission.

B. Pharmacological, chemical and physical manipulation

The IOC Medical Commission bans the use of substances and of methods which alter the integrity and validity of urine samples used in doping controls. Examples of banned methods are catheterisation, urine substitution and/or tampering, inhibition of renal excretion, e.g. by probenecid and related compounds.

III. CLASSES OF DRUGS SUBJECT TO CERTAIN RESTRICTIONS

A. Alcohol

Alcohol is not prohibited. However breath or blood alcohol levels may be determined at the request of an International Federation.

B. Local anaesthetics

Injectable local anaesthetics are permitted under the following conditions:

a) that procaine, xylocaine, etc. are used but not cocaine;
b) only local or intra-articular injections may be administered;
c) only when medically justified (i.e. the details including diagnosis; dose and route of administration must be submitted immediately in writing to the IOC Medical Commission).

C. Corticosteroids

The naturally occurring and synthetic corticosteroids are mainly used as anti-inflammatory drugs which also relieve pain. They influence circulating concentrations of natural corticosteroids in the body. They produce euphoria and side-effects such that their medical use, except when used topically, require medical control.

Since 1975, the IOC Medical Commission has attempted to restrict their use during the Olympic Games by requiring a declaration by the team doctors, because it was known that corticosteroids were being used non-therapeutically by the oral, intramuscular and even the intravenous route in some sports. However, the problem was not solved by these restrictions and therefore stronger

measures designed not to interfere with the appropriate medical use of these compounds became necessary.

The use of corticosteroids is banned except for topical use (aural, opthalmological and dermatological), inhalational therapy (asthma, allergic rhinitis) and local or intra-articular injections.

ANY TEAM DOCTOR WISHING TO ADMINISTER CORTICOSTEROIDS INTRA-ARTICULARLY OR LOCALLY TO A COMPETITOR MUST GIVE WRITTEN NOTIFICATION TO THE IOC MEDICAL COMMISSION.

APPENDIX 7

JOURNAL ARTICLE ON THROMBOGENIC EFFECTS OF ANABOLIC STEROIDS

ARE ANDROGENIC STEROIDS THROMBOGENIC?

To the Editor: The abuse of androgenic steroids persists in spite of their known toxic effects. The reported risks of such abuse have not been deemed serious enough by steroid-using athletes to diminish their use. Indeed, the credibility of official warnings about androgen toxicity have been questioned by many users. However, only recently has acute thrombosis been temporally linked to androgenic-steroid abuse.

Such a link has been proposed by reports of the development of nonfatal myocardial infarction and stroke in several athletes using androgens.[1-4] An additional unreported case has also recently come to light: a 22-year-old college athlete who was using androgenic steroids died suddenly. Postmortem examination revealed acute thrombotic occlusion of the left main and left anterior descending coronary arteries (Simson LR: personal communication). Other reports in nonathletes have linked medically administered androgens to thrombotic complications.[5,6] The clinical circumstances of these reports suggest that a causal relation between androgens and thrombosis should be investigated.

There is no direct experimental evidence that androgens are thrombogenic in humans. Nevertheless, acute thrombotic events and sudden death may represent an underappreciated, and therefore underreported, risk of androgen abuse. This is suggested in part by experimental data in which animals pretreated with an androgen had higher mortality rates, a greater clot size, and lower vessel-occlusion times in response to thrombotic stimuli than untreated controls.[7-9] These effects may be mediated through platelet aggregation.

Androgens potentiate platelet aggregation both in vitro and in vivo.[10-12] Platelet sensitization correlates directly with the concentration and potency of the specific androgen used.[11,12] Androgens may potentiate platelet aggregation through increased production of thromboxane A_2 (a potent platelet aggregator) or, in aortic smooth muscle, a decreased production of prostacyclin (prostaglandin I_2, an inhibitor of platelet aggregation).[12-14]

Alterations in coagulation or fibrinolytic proteins may theoretically predispose patients to thrombosis. Several 17-alpha-alkylated androgens have been used therapeutically to influence both systems; however, no association with thrombosis has been reported.[15,16]

Androgens may also predispose patients to thrombosis by increasing collagen and other fibrous proteins in arterial vascular tissue and skin.[17,18] Functionally, androgens have been linked to an enhancement of vascular reactivity.[19] Interestingly, specific androgen receptors have been identified in vascular tissues and the myocardium of several species of animals.[20] The function of these receptors is unknown.

Existing evidence is consistent with but does not establish the thrombogenicity of androgenic steroids. Although caution is needed in extrapolating conclusions from indirect data to normal subjects (i.e., athletes using androgens), these findings do provide some insight into possible mechanisms of androgen-associated thrombosis. Further studies are needed to assess the influence of androgenic steroids on thrombotic risk among athletes and the hemostatic mechanisms. The abuse of androgens may diminish if acute thrombotic complications become clearly associated with their uncontrolled use among athletes.

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APPENDIX 8

JOURNAL ARTICLE ON ANABOLIC STEROID DEPENDENCE

Anabolic Androgenic Steroid Dependence

Kirk J. Brower, M.D., Frederic C. Blow, Ph.D., Thomas P. Beresford, M.D., and Craig Fuelling, M.D.

The authors believe that this is the first published case report of a patient whose dependence on a combination of anabolic and androgenic steroids meets the DSM-III-R criteria for psychoactive substance dependence. Tolerance, withdrawal symptoms, and the use of steroids to alleviate withdrawal symptoms occurred. An uncontrolled pattern of steroid use continued, despite adverse consequences, such as severe mood disturbance, marital conflict, and deterioration of the patient's usual values. Clinicians should be alerted to the possibility of dependence when asked to prescribe anabolic or androgenic steroids and should suspect steroid use among athlete patients who have mood or psychosocial disturbances.
(J Clin Psychiatry 50:31-33, 1989)

Androgenic steroids, which include testosterone, function primarily to develop and maintain male sex characteristics. Anabolic steroids are synthetic derivatives of testosterone that were developed to minimize testosterone's androgenic or masculinizing effects while promoting its effects on protein synthesis and muscular growth.[1] Both anabolic and androgenic steroids are increasingly being used by athletes to add muscle bulk and to enhance athletic performance.[2-4] (Although athletes and others commonly refer to the androgenic and anabolic steroids together as "anabolic steroids," we use the technically more correct designation "anabolic-androgenic steroids."[4] That designation is preferred because none of the compounds are purely anabolic or purely androgenic in their effects and because athletes usually use the steroids in combination.)

The long-term health consequences of chronic anabolic-androgenic steroid use are largely unknown, and that ignorance raises concerns, because athletes often use steroids in doses and for durations that exceed the regimens described in existing controlled studies.[5,6] The reported psychiatric effects of anabolic-androgenic steroids include euphoria, aggression, irritability, nervous tension, changes in libido, hypomania, mania, and psychosis.[1,7-10] Some reviews[3,5] of the subject suggest that anabolic-androgenic steroids may even have an addictive potential similar to other drugs of abuse, although we are unaware of any case reports, let alone systematic studies, of the phenomenon.

In this case report, we describe a patient who met the diagnostic criteria for psychoactive substance dependence on anabolic-androgenic steroids as defined in DSM-III-R.[11] As anabolic-androgenic steroids are not specifically mentioned by DSM-III-R as a class of substances associated with dependence, the diagnosis was coded as psychoactive substance dependence not otherwise specified (304.90).

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CASE REPORT

A 24-year-old man, a noncompetitive weight lifter, came to the psychiatric emergency room. He complained chiefly of depression and increased outbursts of anger, which he associated with his use of anabolic-androgenic steroids. He requested professional help to discontinue the steroid use because he felt controlled by the steroids and was unable to stop on his own. On the night before he came to the emergency room, he had fleeting suicidal thoughts of crashing his car. He was admitted to a psychiatric inpatient unit, where further assessment could be quickly provided. He had no prior psychiatric history or treatment for chemical dependency. He had a family history of drug abuse but not of mood disorders.

The patient began using steroids 1 year earlier because of his dissatisfaction with his body image in comparison with others. For his first 3 months of use, he obtained the drugs by prescription, and he cycled on and off them, as directed by his physician. The directions were to take the drugs for 4 weeks and then discontinue them for the next 4 weeks. After the third month, however, the patient wanted to continue taking the steroids. He was satisfied with the effects on his weight-lifting performance and his muscle mass; when he discontinued using the steroids, he felt that his motivation for training and his endurance declined. He quit seeing his physician and discovered an illicit source of supply.

For the next 9 months, he used the drugs nearly every day and trained at the gym 6 days a week. He gradually increased the dosage and added other hormonal preparations. By the time of his admission, he was taking approximately the following: (1) testosterone cypionate (Depo-Testosterone and others) 200 mg i.m. q. 3 days, (2) nandrolone decanoate (Deca-Durabolin and others) 100 mg i.m. q. 3 days, (3) oxandrolone (Anavar) 25 mg p.o. q. d., (4) methandrostenolone (Dianabol) 40 mg p.o. q. d., (5) bolasterone (Finaject) 30–45 mg s.q. q. 2–3 days, and (6) human chorionic gonadotropin 1000–2000 U.S.P. Units i.m. q. 2–3 days. His last use of steroids was 3 days before his admission. He denied using any other drug, including alcohol and tobacco, except for trying marijuana several times at age 16. His use of caffeine was limited to three or four beverages a day.

On that regimen of hormones, the patient experienced a lability of mood, with alternating elation and irritability. As a result, he tried to titrate his doses against his mood states and performance. When he tried to stop or cut down the steroid use, he felt depressed, low in energy, fatigued, and weak, and he suffered from headaches. Without the drugs, he missed the high he felt from them. With the drugs, he felt energetic, he required only 4 to 5 hours of sleep each night, and he thought he looked and performed better; but he was disturbed by his temper outbursts, over which he lacked control. Two weeks before his admission, he and his wife separated after 5 years of marriage because of his temper outbursts associated with his steroid use.

Despite a stable work history, the patient began to deal steroids illicitly to other weight lifters at his gym in order to cover the costs of his own use. The patient had no prior arrest record and no childhood history of antisocial behavior.

At admission, his mental status examination revealed a well-dressed and well-groomed muscular man who was fully alert and oriented. His muscles were markedly hypertrophied. His mood

was predominantly depressed, with some anxiety. His affect was appropriate, constricted to dysphoria, but not labile. Irritability was not noted, and the patient was pleasant and cooperative with the examiner. Mild psychomotor retardation was present. The patient's thought processes were intact and associated with normal speech. No delusions or hallucinations were manifested, nor were any paranoid ideas present. He was no longer suicidal, despite the thoughts noted above, and homocidal ideation was likewise absent. Cognitive testing revealed five digits forward and four backward, one mistake out of five calculations of serial sevens, and three out of three words remembered in 5 minutes.

The results of physical and laboratory examinations, including thyroid-function tests, were essentially normal except for mild elevations in creatinine, SGOT, and SGPT. (Weight lifters can be expected to have elevations in creatinine because of increased muscle mass and elevations in nonspecific liver tests on the basis of intensive training alone, even without steroid use.[1]) The results of the admission urine-drug screen were negative for amphetamines, barbiturates, benzodiazepines, other sedatives, cannabinoids, cocaine, opiates, and phencyclidine. Urine testing for steroid use is not routinely available at our institution and, [illegible]s, was not performed.

By the second hospital day, the patient's mood had improved, the full range of affect was apparent, and his psychomotor behavior was normal. He was optimistic about treatment and participated fully in a chemical dependency program until the fifth hospital day, when he signed out against medical advice for no clear reason.

DISCUSSION

Anabolic-androgenic steroids are psychoactive compounds, as evidenced by their well-documented effects on behavior and psychological functioning.[1] Neuronal androgen receptors have been identified in the brain,[2] suggesting the neurochemical basis for their psychoactive effects. Not all psychoactive substances, however, have the potential to produce dependence.

According to DSM-III-R,[3] at least three out of nine criteria must be met for a period of at least 1 month for a diagnosis of psychoactive substance dependence to be made. Our patient met at least six of those criteria: (1) the substances were taken over a longer time period than was intended initially, when the patient was cycling on and off the substances; (2) he was unsuccessful in his efforts to cut down on use; (3) he continued to use the steroids despite his knowledge that he was having emotional and marital problems related to their use; (4) he had tolerance, as shown by the supratherapeutic doses taken at the time of his admission; (5) he had withdrawal symptoms of depression, fatigue, psychomotor retardation, and headaches; and (6) he regularly took the substances to avoid those withdrawal symptoms. In short, the patient exhibited the core signs and symptoms that are characteristic of most definitions of dependency: an uncontrolled pattern of use, persistent use despite adverse consequences, tolerance, and withdrawal.

The course and the consequences of his drug use were strikingly similar to those observed with the use of alcohol, cocaine, and the opioids. With those other addictive substances, the initiation of use is often related to peer influences. Similarly, our patient initiated steroid use because he wanted to be comparable to and competitive with his peers, some of whom he knew were using steroids to enhance their body images. Another similarity to the users of cocaine and opioids was the deterioration of the patient's usual values as he began to deal drugs illicitly to support his own habit. In addition, his abuse of multiple substances (five different steroids and human chorionic gonadotropin) is a pattern commonly found among those dependent on alcohol, cocaine, and opioids. Among athletes, the pattern of using multiple steroid substances is called "stacking."[4] The patient used human chorionic gonadotropin to stimulate his testes to produce endogenous androgens, thereby augmenting the exogenous steroids that he was self-administering. Although the patient became dependent by using high doses of several anabolic and androgenic steroids in addition to human chorionic gonadotropin, the differential addictive potential of any one of those substances is unknown. It is also unknown whether dependence occurs at smaller doses than those reported here.

Many of the patient's symptoms were suggestive of a mood disorder. He reported a history of irritability, euphoria, decreased need for sleep, increased activity, and increased self-esteem when taking the drugs. During the first 24 hours of his evaluation, we observed suicidal ideation, depressed mood, low energy, psychomotor retardation, and trouble in concentrating on cognitive tasks. We attributed those symptoms and signs to drug use and drug withdrawal, respectively. The lack of any reported affective symptoms preceding his first use of steroids, the disappearance of those symptoms after 4 days of abstinence, and the lack of any family history of mood disorders argued against a diagnosis of mood disorder in our patient. However, only a longer period of observation of the patient when he was not taking drugs could have determined with certainty if he had an independent bipolar disorder that may have first become apparent during, if not triggered by, his steroid use.

Similarly, Pope and Katz[4] recently reported that 9 (22%) of 41 steroid-using athletes in their sample developed either a full manic or a depressive episode during periods of steroid use or withdrawal, as determined by a structured diagnostic interview. Thus, we recommend that athletic patients with affective symptoms be closely questioned about their possible use of steroids. Such patients may not be aware of the connection themselves or may fear criticism of their use and, therefore, may not report it.

The prevalence of anabolic-androgenic steroid dependence is not known. With our patient, there was a time lag of 1 year between the onset of use and the troublesome effects that precipitated treatment-seeking. Therefore, as the prevalence and the awareness of steroid use increases, clinicians may begin to see increasing numbers of steroid addicts within 1 or 2 years. The trend will be particularly troublesome among adolescent athletes,[2] because their developing nervous and skeletal systems may be more vulnerable to adverse effects and because they lack the psychological maturity to cope with the powerful mood changes produced.

CONCLUSION

Our patient developed a dependence on a combination of anabolic and androgenic steroids that was strikingly similar to dependencies seen with other substances. Clinicians should be alerted to the possibility of dependence when asked to prescribe anabolic or androgenic steroids and should suspect steroid use among athletes with mood disturbances, psychoses, or psychosocial disturbances. Further study of the prevalence, the course, and the optimal treatment of those syndromes is warranted.

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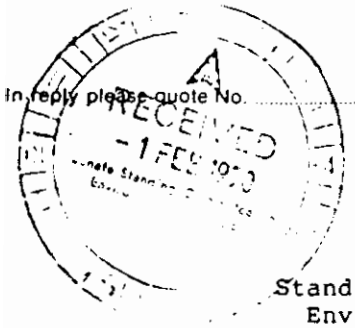
APPENDIX 9

POST-MORTEM REPORT ON BODYBUILDER



In reply please quote No. .....

CORONER'S COURT
40-46 Parramatta Road
Glebe, N.S.W. 2037
Telephone: 660 5977

24 January 1990

Standing Committee on
Environment, Recreation
and the Arts
Australian Senate
Parliament House
CANBERRA A.C.T. 2600

Attention: Mr P C Grundy

Dear Sir/Madam

Death of Maurice FERRANTI
Your Ref: letter8/11/89

Please find enclosed a copy of the postmortem report relating to the abovenamed deceased.

Yours faithfully,

Clerk of the Local Court. jd

enc.

CORONERS ACT. 1980

Medical report upon the examination of the dead body of:-

Name: Maurice FERRANTI PM Number: 89/1912

I Johan Duflou a legally qualified medical practitioner, carrying on my profession at the Division of Forensic Medicine, in the State of New South Wales, do hereby certify as follows:

At 8.00 in the fore noon, on the 26 day of October, 1989 at Sydney in the said State, I made an internal examination of the dead body of a male identified to me by Dr. Hollinger of Division of Forensic Medicine in the State aforesaid, as that of Maurice FERRANTI aged about 23 years.

I opened the three cavities of the body.

Upon such examination I found:

The body was that of very well-built, heavily muscled adult male whose appearances were consistent with the stated age.
There was minimal subcutaneous body fat.
Body weight 79 kg. Body length 1.75 m.
The body was cold to touch and there was faint dorsal postmortem lividity.
Early decompositional change was evident in the form of softening of organs and green discolouration of the anterior abdominal wall.

External examination of the body:

1. There was an endotracheal tube in situ.

2. There were three E.C.G. dots on the anterior trunk.

3. Intravenous cannulae were in situ in the right antecubital fossa as well as on the anterior surface of the left lower arm.

4. No ante- or peri-mortem injury was identified on the surface of the body.

Head and neck:

The scalp and skull were normal.
Specifically there were no skull fractures.
The meninges were similarly normal and there was no extradural, subdural or subarachnoid haemorrhage.
The brain weighed 1680 g and was placed in formalin of later detailed examination once fixed.
The eyes, ears, nose and mouth were normal.
The neck was similarly of normal appearances and there were no cervical spine fractures.

Cardio-vascular system:

The pericardium was healthy.
The heart weighed 360 g and showed mild biventricular dilatation.
The atria and valves of the heart were within normal limits.
The free wall thickness of the right ventricle was 3 mm and that of the left ventricle was 15 mm.
There were areas of alternating pallor and congestion of the myocardium of the left ventricle.
There were no mural thrombi, nor were there areas of old fibrosis within the myocardium.
The coronary arteries were involved by very early atherosclerotic disease, but there was no obvious narrowing of their lumina.
The aorta, proximal carotid arteries, renal arteries and iliac arteries all showed moderately advanced fatty streaking of the intima.
The venous system was normal.
There were no pulmonary emboli.

Respiratory system:

The pharynx, larynx and trachea contained grey charcoal-like material.
The bronchi contained blood-stained fluid.
The left weighed 740 g and the right lung weighed 860 g.
Both lungs were markedly congested throughout.
No focal pulmonary lesions were identified.
The chest wall and diaphragm were normal.
There were no rib fractures.

Gastro-intestinal system:

The tongue, oesophagus, and stomach mucosae were coated by charcoal-like material.
The stomach contained approximately 300 ml black fluid.
The duodenum was normal.
The remainder of the bowel on external examination appeared normal and was not opened further.

Hepato-biliary system:

The liver weighed 2420 g and was markedly congested and fatty.
No focal hepatic lesions were identified macroscopically.
Bile from a normal gallbladder could be expressed with ease through the extrahepatic biliary system into the duodenal cavity.
The pancreas was autolytic.

Haemopoietic system:

The spleen weighed 200 g and was uniformly congested.
There was no lymphadenopathy.

Genito-urinary system:

The left kidney weighed 180 g and the right kidney weighed 150 g.
The capsules of both kidneys stripped with ease to reveal normal renal parenchyma throughout.
Both ureters were patent throughout their lengths, ending in a normal urinary bladder containing approximately 50 ml cloudy urine.
The prostate gland was of normal appearances.

The right testis weighed 15 g and the left testis weighed 12 g. Both testes were markedly atrophic.

Endocrine system:

The pituitary gland and thyroid gland appeared normal.
The left adrenal weighed 9 g and the right adrenal weighed 8 g. The adrenal glands were of normal macroscopic appearances.

Histology being performed. (Brain)

Blood was sent for the estimation of alcohol, and blood, liver, stomach and contents, urine and bile for chemical analysis.

The body was identified to Dr. Hollinger by Const. D. Kneipp of No. 24 Division.

MICROSCOPIC EXAMINATION:

Heart:

Sections of right and left ventricles, interventricular septum and cardiac conductive system show no abnormalities apart from occasional agonal subendocardial contraction bands.

Lungs:

Show fairly extensive intra-alveolar haemorrhage and oedema in all sections. Bronchial basement membranes are thickened.

Liver:

Shows centrilobular congestion only.

Spleen:

No abnormality detected.

Pancreas:

There is advanced autolysis.

Kidneys:

No abnormality detected.

Adrenals:

Numerous eosinophilic intracytoplasmic inclusion bodies are noted in the zona glomerulosa of the adrenal cortex, highly suggestive of "Aldactone bodies". There is cortical lipid depletion.

Pituitary:

Shows no histological abnormalities.

Thyroid:

Normal.

Testes:

There is some fibrosis of the seminiferous tubules and partial spermatocytic arrest is identified.

Skeletal Muscle:

Sections stained with H & E, and frozen fat stains show no histologic abnormalities.

MACROSCOPIC REPORT OF THE BRAIN:

The leptomeninges are thin and transparent. The vessels at the base of the brain have a normal architectural pattern with no atheroma. The external surface of the cerebrum, cerebellum and brain stem appears normal.

The cerebrum is sectioned in the coronal plane in 1 cm slices. No abnormalities are seen on the cut surfaces of the cerebral cortex, white matter, basal ganglia, hippocampi or diencephalon.

The cerebellum is sectioned in the sagittal and parasagittal planes. No abnormalities are seen on the cut surfaces of the cerebral cortex, white matter or dentate nuclei.

The brain stem is sectioned in the transverse plane in 0.5 cm slices. No abnormalities are seen on the cut surfaces of the midbrain, pons or medulla.

MACROSCOPIC DIAGNOSIS

Normal brain.

MICROSCOPIC REPORT OF THE BRAIN:

Normal.

In my opinion death had taken place about 5 days previously and the cause of death was:

1. **DIRECT CAUSE:**
   Disease or condition directly leading to death:
   (a) CARDIAC ARREST (due to)

ANTECEDENT CAUSES:
Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last:

(b) HYPERKALAEMIA (following)

(c) COMBINED SPIRONOLACTONE AND POTASSIUM INGESTION.

2. Other significant conditions contributing to the death but not relating to the disease or condition causing it:

ANDROGENIC STEROID INGESTION.

TO THE STATE CORONER, (Signature).....

SYDNEY (Date) 23rd January, 1990.

ANALYST BEFORE SEEN

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Address all correspondence to the Director

CERTIFICATE OF ANALYSIS

CLIENT (0350) Department of Health
Division of Forensic Medicine
42-50 Parramatta Road
GLEBE NSW 2037

SAMPLE DESCRIPTION : URINE SAMPLE T890757 FERRANTI

Laboratory Report Number - NS9/049711
Client Reference Number -

DAL-95 AEH.ML

A sealed urine sample was given to me for the analysis of anabolic steroids and diuretics. The sample was that of Maurice Ferranti and was labelled with the code T890757

The pH was measured at 7.0 and the specific gravity was 1.000. Analysis of the sample using our standard screening procedure using gas chromatography/mass spectrometry gave three detectable anabolic steroids.

METHENOLONE (Primobolan)
METHYLTESTOSTERONE (Testomet)
STANOZOLOL (Winstrol)

This procedure also confirmed the presence of canrenone which is a metabolite of SPIRONOLACTONE (Aldactone) .This method does not quantitate each substance

The natural androgenic steroids were greatly suppressed indicating the possibility of long term steroid use. The epitestosterone was not detectable while testosterone was easily seen. This could indicate the use of testosterone as an anabolic steroid as well.

Further analysis using High Pressure Liquid Chromatography gave Hydrochlorothiazide (23.5ug/l) (Moduretic) and Canrenone (11ug/l)

Signed R. Gill
(For Regional Director)
Date 18/12/89