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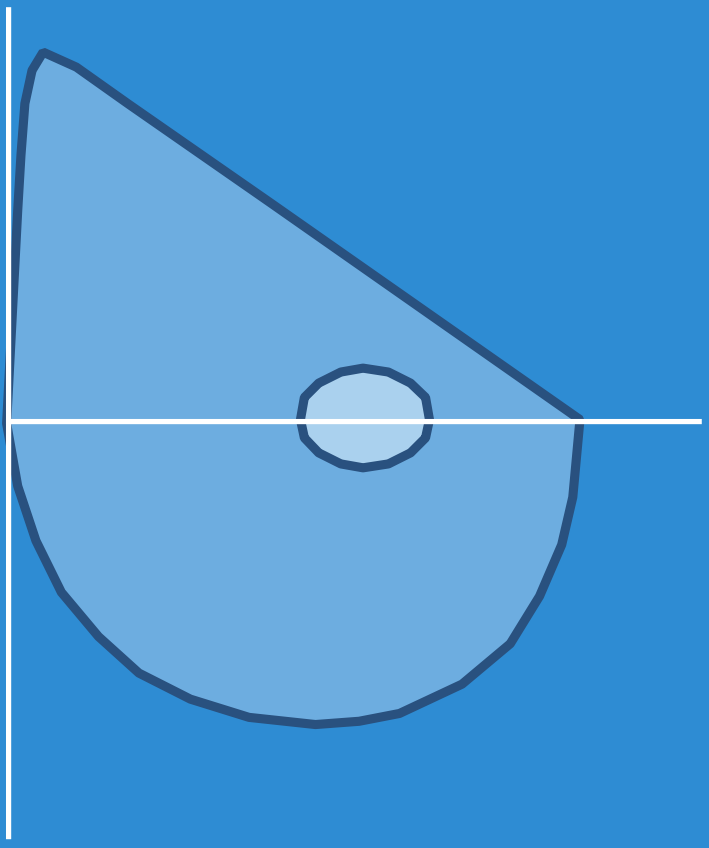
Inspire



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The Official Journal of the Association for Respiratory Technology & Physiology

Inspire

The Official Journal of The Association for Respiratory Technology and Physiology

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BURSARY INFORMATION

Bursaries are available to ARTP members, which can be used to support attendance at National ARTP, BTS or STS meetings. Other relevant respiratory meetings or approved training courses will also be considered. Bursaries are available to student, associate and full ARTP members of any grade. They can be used for partial or total funding of registration, travel and accommodation costs for the whole or part of the meeting/course. All bursaries are considered by the ARTP Executive Committee on the reason for the request and the commitment to an article for *Inspire*.
For further details or an application form please contact: **Gill Butcher (Bursary Secretary), Cardiorespiratory Unit, Queen's Hospital Burton, Belvedere Road, Burton on Trent, DE13 0RB. Tel: 01283 566333 Ext 5334 or via e-mail: bursary@artp.org.uk**

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ARTP MERCHANDISE

ARTP HANDBOOKS

SPIROMETRY HANDBOOK:	£ 25 (members)	£ 35 (non-members)
RESPIRATORY FUNCTION TESTING:	£ 40 (members)	£ 55 (non-members)

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For current prices and an order form please contact the ARTP Administrator

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Discounts available for multiple issues

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Supplied by advertiser (< 200 gm)	£150
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MAILSHOTS

Monthly (last Friday in the month) £600 Urgent (within 5 days) £750

Flyers and mailshots will also be distributed via the ARTP e-mail forum

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WEB SITE ADDRESS: www.artp.org.uk

E-MAIL FORUM: forum@artp.org.uk

CORRESPONDENCE: admin@artp.org.uk

FIRST WORD

Wishing all ARTP members a Happy Christmas and New Year.

I have just realised that "First Word" is no longer the first word in the new style *Inspire*, but I will persevere with the name until "inspired" with a better one!

Hopefully everyone is looking forward to the ARTP Winter Conference, which looks like being another record year for attendance of both delegates and manufacturers. From the sound of Jackie Hutchinson's report it looks like the Blackpool hotels will be overrun with us all. My only hope is that the De Vere (where I'm staying) is within staggering distance of the Hilton ..... after a heavy day of lectures of course!! Four ARTP bursaries have been granted for the 2002 Conference, which is good news for both the applicants and future editions of Inspire. The number of bursary enquiries has significantly increased this year and this hopefully means, that after receiving an application form, ARTP members are managing to secure alternative funding from their Trusts. CPD requirements can only highlight further funding issues for training and development.

With the recent national headlines about the high incidence of lung disease maybe we will see the next Government National Service Framework coming our way! The NSF in Coronary Heart Disease has created significant pressures on Cardiac Departments but has also served the purpose of raising the profile of the work and technical staff involved and has highlighted staffing problems common to us all.

We are still desperately looking for volunteers to help with regular or ad. hoc. features in *Inspire*, in particular the E-mail Forum news which Keith Butterfield no longer has time to compile with his many other commitments.

As a final word I would like to thank the manufacturers for using Inspire for advertising their products. As well as the very welcome revenue it does make for a more colourful journal.

Gill Butcher, Cardiorespiratory Unit, Queen's Hospital Burton, Belvedere Road, Burton on Trent, Staffs DE13 0RB
e-mail: inspire@artp.org.uk

DATES FOR YOUR DIARY

DON'T MISS IT !!

ARTP WINTER CONFERENCE 2002

January 17th to 19th Hilton Hotel Blackpool

For last minute bookings contact: Jackie Hutchinson 0121 241 1611

ARTP SHORT COURSE IN ADVANCED RESPIRATORY PHYSIOLOGY

15th to 19th April 2002 at Coventry University

This course is a development in the area of post-basic training and education and has been designed to reflect current and future practice. The cost of the week long course will be £300 including tea, coffee and literature but excluding accommodation and subsistence.

For details and application form see the Education section on the website www.artp.org.uk

CONTENTS

EMAIL FORUM DIGEST

I have to apologise for not being able to produce the usual précis of the many messages that have passed through the Forum since the last issue of *'Inspire'*. On top of my usual workload I am working on several other projects for my Trust, ARTP & RCCP and quite frankly something had to give! This is why Gill asked for a volunteer to take over writing this column in the Editorial in the last issue. I don't believe anyone has come forward yet. Please contact either Gill of myself if you are interested.

I do hope someone is able to take up the mantle because there are many interesting topics discussed which should to be brought to the attention of those members who do not access the Forum via e-mail. Not that reading the *'Forum Digest'* column is any substitute for getting involved in the discussion as it is an interesting learning experience following the course of debates on many and varied topics.

There are currently about 180 members subscribed to the Forum (whereas there are now over 500 ARTP members). Of course, the column would be unnecessary if everyone subscribed via e-mail!

It would also be nice to hear more from the *'lurkers'*. *'Lurkers'* are those subscribers who just read what is posted and don't post anything themselves. Considering that there are 180 subscribers and the same 20-30 names seem to crop up all the time there are quite a few of you out there. Feel free to *'de-lurk'* at any time, don't be afraid that your opinion will be shot down in flames; any opinion is valid and worthy of discussion.

A couple of new uses for the Forum were pioneered in the last few months... Firstly the discussion of a set of patient results. A couple of points if you do decide to put out a set of results for discussion (of course, make sure they are anonymised) if you scan a trace try to make it a JPG format and as small a file size as possible (though many members subscribe via the NHSnet where file size doesn't matter it can be annoying for people who subscribe via modem on internet accounts that cost according to access/download time) and it would be convenient if you could also type the basic results in the text of the e-mail as many hospital e-mail systems do very strange things to attachments making them virtually unreadable unless you really know what you are doing. Another new use was the announcement of births so congratulations to Sean Tilbrook at Kings Mill Hospital on the birth of a baby girl in November.

Keith Butterfield (e-mail: webmaster@artp.org.uk)
ARTP Website – http://www.artp.org.uk
Forum e-mail Address – forum@artp.org.uk

ARTP ADMINISTRATOR'S UPDATE

With still a couple of weeks to go to the biggest and best ARTP Conference yet, previous records have been well and truly smashed. Delegate numbers have broken through the 300 mark, 26 exhibition stands have been booked and paid for and the lunchtime workshops oversubscribed 3 times over. We ran out of Hilton bedrooms back in October, the Imperial by Mid November and our 30 rooms at the De Vere might not be enough to satisfy demand.

The Head of Department meeting and manufacturers workshops scheduled to take place during the day on Thursday have attracted more than 100 attendees extending the conference by a full day. All conference delegates are invited to attend the Thursday afternoon workshops (but please let me know if you are coming!).

This year's keynote address is by Dr Brendan Madden, Consultant Cardiothoracic Physician based at St George's London and other speakers include Mr Marzouk from Birmingham Heartlands, Melissa Hack from Newport and Andy Davies from Manchester to name but a few. The after dinner speech at the gala dinner is not to be missed, and sessions including an exercise clinic and panel discussions on current professional issues will bring a new dimension to the conference content. *If you have been on another planet for the last few months and haven't registered for the conference yet, please contact me immediately.*

Putting on my next hat..... The Voluntary Register for Clinical Physiologists has now received more than 900 applications with over 600 active members. Unfortunately respiratory applications are still only trickling in with less than 70 active respiratory members. As most of you are aware, the "proposers" section on page 1 can currently be left blank and is completed for you by Council members, but this luxury will be coming to a close in the near future so please complete your forms and get them submitted asap.

On a final note I would like to thank all the labs who have been so helpful when contacted regarding the staffing level survey that we have been conducting. The results will enable the ARTP to provide up to date information to help towards workforce planning as well as enable us to prepare a national picture of respiratory labs and the work being carried out within them. If there are any labs that have not been contacted could you please contact me immediately so that you can be included. Anyone who has not returned their completed list of procedures should also fax them back asap.

Thank you for all your help throughout the year and I look forward to seeing you all at the Blackpool Conference.

JACKIE HUTCHINSON, ARTP ADMINISTRATOR
TEL: 0121 241 1611 e-mail: jackiehutchinson@aol.com

EXECUTIVE NEWS

CHANGES TO THE ARTP EXECUTIVE COMMITTEE

Due to pressures of her new post at Gloucester, Melanie Marshall has had to step down from the role of Education Secretary for the ARTP. We would like to acknowledge our gratitude as an Executive Committee and extend our thanks for all her hard work. She will, however, continue to work in the Strategic Group for Education.

Clare Newall from the Queen Elizabeth Hospital in Birmingham has agreed to take on the role of Education Secretary. Clare has been involved with the Strategic Group for Education, the B.Sc. Syllabus for Respiratory Physiology, and made a major contribution to ARTP handbooks, exams and lecturing in Respiratory Physiology.

Sue Revill's new post as Clinical Scientist at Sherwood Hospital, means she has insufficient time to give to the Executive Committee, and so she has also resigned her Committee post. She has been a stalwart member of the ARTP Exec for over ten years and we are sure she will still be making valuable contributions to the ARTP in different ways.

Steve Scholey is involved in a major PFI project within his Trust and is stepping down from the Committee to work on this for the next twelve months or so. We hope to see him back on the Executive when the project is complete.

ARTP/BTS SPIROMETRY CENTRES

Approved Training Centres for the ARTP/BTS Certificate in Spirometry

NUMBER: 001
Address: Department of Respiratory Medicine
North Staffs Hospital Trust
City General Hospital
Newcastle Road
Stoke-on-Trent
ST4 6QG
Lead trainer: Angela Evans 01782552362

NUMBER: 002
Address: Northern General Hospital Sheffield
Herries Road
Sheffield
S5 7AU
Lead trainer: Cheryl Roberts 0114 2714784

NUMBER: 003
Address: Lung Investigation Unit
Nuffield House
QE Hospital
Edgbaston
Birmingham
Lead trainer: Sue Hill/Joanna Harrison 01216272087

NUMBER: 004
Address: Respiratory Function Laboratory
2nd Floor OPD
Western General Hospital
Crewe Rd South
Edinburgh
EH4 2XU
Lead trainer: Jill Lenney 01315371984

NUMBER: 005
Address: Lung Function Unit
Royal Brompton & Harefield NHS Trust
Fulham Road
London
SW3 6HP
Lead trainer: Derek Cramer 020 7351 8057

NUMBER: 006
Address: Lung Function Dept
Nottingham City Hospital
Hucknall Road
Nottingham
NG5 1PB
Lead trainer: Brendan Cooper 01158402615

NUMBER: 007
Address: Clinical Measurement Unit
Walsall Manor Hospital
Moat Road
Walsall
WS2 9PS
Lead trainer: Claire Thomas 01922656583

NUMBER: 008
Address: Cardiothoracic Measurement
Derbyshire Royal Infirmary
London Road
Derby
DE1 2QY
Lead trainer: Dena Muirhead 01332254885

NUMBER: 009
Address: Lung Function Laboratory
Good Hope Hospital
Rectory Road
Sutton Coldfield
B75 7RR
Lead trainer: Julie Lloyd 01213782211 X2390

NUMBER: 011
Address: Pontefract General Infirmary
Friarswood lane
Ponterfract
West Yorkshire
WF8 1PL
Lead trainer: Stephen Scholey 01977606634

NUMBER: 012
Address: Lung Function Laboratory
Queens Medical Centre
University Hospital NHS Trust
Nottingham
NG7 2UH
Lead trainer: Laura Watson 01159249924 X 44470

NUMBER: 013
Address: Cardio-Respiratory Department
Medical Physics
A level
Rotherham Hospitals NHS Trust
Moorgate Road
Rotherham
S60 2UD
Lead trainer: Jane Caldwell 01709304572

NUMBER: 014
Address: Respiratory Investigation Unit
Monklands Hospital
Monklands Court
Airdrie
ML6 0JS
Lead trainer: Christine Downie 01236748 X 2215

NUMBER: 015
Address: Dep. of Cardiology and Respiratory Medicine
North Tyneside Hospital
Rake Lane
North Shields
NE29 8NH
Lead trainer: Nigel Wardrobe-Wong 0191 293 2720

NUMBER: 016
Address: Respiratory Function Unit
Royal Hallamshire Hospital
Glossop Road
Sheffield
S10 2JF
Lead trainer: Judith Waterhouse 0114 271 2734

NUMBER: 017
Address: Respiratory Dept
Outpatients Dept
Falkirk Royal Infirmary
Majors Loan
Falkirk FK1 5QE
Lead trainer: Barbara Oatway/Anne Douglas 01324 616098

NUMBER: 018
Address: Dept of Respiratory Medicine
Queen Elizabeth Building
Royal Infirmary
Alexandra Parade
Glasgow
G31 2ES
Lead trainer: Roger Carter 0141 211 5462

NUMBER: 019
Address: Medical Physics Dept
Diana Princess of Wales Hospital
Scartho Road
Grimsby
DN33 2BA
Lead trainer: Tracey Broom 01472874111 7627

PRIVATE
NUMBER: 20
Address: Respiratory Physiology Unit
Glenfield Hospital NHS Trust
Groby Road
Leicester
LE3 9QP
Lead trainer: Mr DD Vara 0116 256 3544

NUMBER: 21
Address: The South West Centre
Details to come
Lead trainer: Dr A Kendrick + M Marshall
0117 928 6442

ARTP REPORT TO THE BRITISH THORACIC SOCIETY

(November 2001)

PREPARED BY DR BRENDAN COOPER – HONORARY CHAIRMAN ARTP

LINKS BETWEEN BTS AND ARTP

The BTS/ARTP Liaison Committee has been replaced by new methods of reporting, which include (i) ARTP Chairman having direct access to BTS Chief Executive throughout the year, (ii) ARTP Chairman attending or reporting to the BTS Executive once a year and (iii) ARTP representation on Standards of Care Committee projects that include any aspect of lung function/clinical measurement. There will continue to be a member of ARTP on the BTS Education Committee. The ARTP Executive will have reciprocal links with the BTS. In the light of national scientific and technical service developments in the NHS, the ARTP is forming a new "college-type" structure for education and training and will liaise with the BTS as required.

VOLUNTARY REGISTRATION FOR MTOs IN RESPIRATORY PHYSIOLOGY

The Voluntary Register through the Registration Council for Clinical Physiology (RCCP) has been established since May 2001. To date there are currently 50 lung function staff on the register, with numbers growing all the time. It is essential that BTS physicians should be informed of the importance of getting all their eligible staff on the register as soon as possible. This forerunner to State Registration will enable competent practitioners, who provide excellent lung function services, to be registered under voluntary arrangements and eventually onto the State Register of the Health Professions Council. It also makes it imperative that BTS physicians encourage and support all lung function staff to attend regular and relevant training courses as part of their continued professional development (CPD) throughout the year. This will be a central requirement for State Registration of staff.

Clinical Scientists are already state registered to be able to practice and to maintain annual registration they must undertake CPD. ARTP Executive look to the BTS to support their staff in this initiative.

HSC 2000/026

This Department of Health Directive has threatened the use of ANY drugs by lung function staff including the administration of bronchodilators in reversibility tests, bronchial challenge testing and even administration of 'Algipan' cream for capillary blood gases. The initial government body that devised the legislation was advised to include lung function staff on the list of *"approved professionals"* to administer drugs but "forgot" the advice. Currently the ARTP, in conjunction with the RCCP, are working with the Department of Health to get the legislation changed. All practitioners who are State Registered are covered by the legislation. In the interim, advice has been offered to ARTP members to get written confirmation from their clinical leads and hospital management that they are allowed to administer designated drugs within the protocol/directive of the hospital policy.

ARTP DEPARTMENT SURVEY 2000

The departmental survey conducted in 2000, which is a follow up to the 1995 survey has been completed and will be presented at the ARTP Annual Conference. It contains important insights into the number, grade, qualifications and profile of lung function staff in the UK. The number and type of tests and services offered throughout the UK is covered and changing patterns are available in the report. ARTP Executive feels that these finding will have important implications to BTS members, and particularly to the Peer Review process of BTS. ARTP intend to move towards accrediting lung function departments, and lay down criteria for this, but will consult with the BTS during this process. ARTP Executive are seeking the support of BTS Executive for the minimum establishment of at least one fully trained and state registered lung function practitioner in every unit, and the establishment of training programmes for Clinical Scientists.

PROPOSED ARTP TRAINING COURSES

The ARTP will be running the following training courses in the future which will be very useful for SpRs in training and for continued professional development:

ARTP Sleep Study Course (Basic)
Bristol 8th & 9th November
Bristol 22nd & 23rd November

ARTP Sleep Study Course (Advanced)
Bristol
To be announced – Spring 2002

ARTP Short Course in Respiratory Physiology
Birmingham and Edinburgh
To be announced – Spring 2002

ARTP/BTS Lung Function Interpretation Course
Birmingham
To be announced – Summer 2002

ARTP/BTS Non-Invasive Ventilation Course
Nottingham
To be announced – Spring 2002

ARTP/BTS Exercise Physiology Course
Glasgow
To be announced – 2002

ARTP ANNUAL CONFERENCE 2002

After last years success at the Hilton Hotel, Blackpool, where over 350 attended the meeting, this years Conference will be held at the same venue on the 17th to 19th January 2002. The conference is the largest physiology meeting in the UK and offers an excellent opportunity for Specialist Registrars to understand clinical lung function testing, the equipment used and services available.

ARTP WEBSITE

The website (www.artp.org.uk) has been available for over 18 months and still requires completion of links to the BTS website. It receives 50 - 70 hits per month and has a very active Forum on respiratory issues and information for ARTP members. ARTP would like BTS Executive to help SpRs become aware of this excellent resource for information and training. The ventilator equipment list to support the BTS Guidelines for NIV will be available on the website soon.

NATIONAL ISSUES

Registration Council for Clinical Physiologists – Cost effective processing of applications

The Council have been receiving applications for almost 6 months and several hundred Practitioners are now on the voluntary register. Council members have been very impressed with the effort made and the quality of applications we are receiving. As I am sure we all appreciate early success in our endeavour requires us to continue momentum of the voluntary register.

It is also important from both the applicant and council's perspective to ensure the process is both efficient and successful on first attempt. Omissions, errors and repeated applications are proving to be extremely expensive to administrate and leading to a good deal of exasperation for potential applicants.

We have therefore decided to produce the following as advice for completion of the form and as a checklist prior to sending the form: -

1. Applications on official RCCP forms will be accepted and can be written or typed [individuals having problems with complying with this are recommended to contact their discipline representative for advice/help]
2. Maiden or previous surnames should appear in brackets after the surname
3. Do not put your home address on the form unless you want this information in the public domain on publication of the register
4. The report (see page 5 of form) should be split into sections using the headings supplied on the form. [This will ensure that **all** the information required is present]
5. Provide sufficient information for each section/under each heading e.g. for investigations or treatments include all the process' you are currently and have previously been involved with, including the calibration and quality control of equipment if appropriate.
6. All sections of the form must be completed but the information only needs to appear once, repetition is not necessary, cross-reference where applicable.
7. Your reference must be from your current place of employment, on official [e.g. NHS] headed paper and be signed.
8. Your referee should be your current clinical lead/head of department, preferable medical, other statutory regulated Practitioners or someone currently on the RCCP register.
9. Check that all additional information is present when sending the form: reference, report, cheque and copies of your certifications
10. If you do have any queries contact your discipline representative on RCCP before sending the form or the RCCP Administrator on 0121 241 9699.
11. If you require further application forms please send a SAE to the RCCP Administrator, 202 Maney Hill Road, Sutton Coldfield, West Midlands. B72 1JX

NB Unofficial forms [e.g. examples downloaded from professional web sites and altered] will be returned to applicants.

We are working towards introducing a computer-generated form sometime in the future.

Because of soaring administration costs if applications / reports are incomplete and have to be reassessed it will be necessary to reapply and pay an additional fee of £12.

"ON THE BLOWER" - Manufacturers' News

1. Manufacturers' Liaison

Well, *On the Blower* moves into a new era, as Nigel Clayton (Wythenshawe) takes over as being responsible for putting it together. (That's not to say that I will not have a input into the articles!!). We are moving over to a team effort with help from Alan Moore (City Hospital, Birmingham), and anybody else who wishes to contribute. When we started the feature in 1996, little did we know how much influence, usefulness and benefit to the profession it would have. In some respects it has been partly replaced by the ARTP Forum on the website, but I believe there is room for both approaches in the future.

The first article covered the Sensor Medics launch of VMAX Lung Function equipment, the news that P. K. Morgan "joined the Medical Division of Ferraris Group plc" and that Nellcor and Puritan Bennett have joined/merged, etc. Some things never change really - the mergers continue and the Morgan name limps on as part of the Ferraris Group today.

What were the highlights for me? Well comparing companies with cars is often quoted back to me, especially the Medic Aid Reliant Robin (.... they thought I would forget!!). Getting Morgan to improve (but not yet perfect!!) their customer services has been one definite achievement. But perhaps most importantly, winning the confidence of all the manufacturers who now so generously support and enjoy attending the ARTP Annual Conference can in part be attributed to this feature. Long may it continue!

2. Trade Stand

Lung function equipment

I received a deputation from **Cranlea & Co.** from Bournville, near Birmingham (Yes Bournville, home of chocolate!!...80% of ARTP membership suddenly have an interest in the article!!!) last week.. They have been suppliers of physiological measurement equipment for years and are sole agents for Hans Rudolf (the world-beating mouthpiece manufacturer!), Lode ergometers, and lots of useful respiratory equipment. They have recently taken on a popular colleague Mr Tim Allen (erstwhile of Jaeger UK) and wish to support ARTP membership who require respiratory equipment including Douglas bags!!! (For newer members, Douglas Bags are what Heads of Department do a "Yorkshire Man" sketch of reminiscence about at Christmas parties. Many a trainee was told they were actually "flatus collection" bags.....well mine were!!) Cranlea & Co, Tel: *0121 472 0361*, Web site: www.cranlea.co.uk

I was interested to see Flexible Spirometry Software from **Medikro Oy** (They're Finish,.... so they've started!), which is sold as the Spiro 2000, and offers a platform for several spirometers to operate with the same database. This may have a use with COPD outreach teams, research studies, or just running the lab. Contact www.medikro.com

MicroMedical have sent me brochures on their MicroNEP (negative expiratory pressure) system which offers a different way to look at airway function especially in severe COPD. See them at the Annual Conference in Blackpool.

Finally I received some literature from "Air Liquide" regarding their products, but the representatives were so uninspiring and rude, I couldn't be bothered to write them up here! It's a tough life.

Sleep study equipment

I have had a short preview of the sleep apnoea screening system from **Breas**, the Breas SC20. It can measure up to 10 channels (SpO2, airflow, pulse, breathing effort, snoring, limb movement, body position, light and "other"), has some tasty software and sells for about £2750 excl VAT. If you need a step up from oximetry, this

is a very attractive deal indeed. Another exciting development in CPAP is due from Breas in late January 2002. Watch this space. Contact Kath Johnson at Breas on *Tel:* 01252 731660. *Web site:* www.breas.com

Mallinkrodt have sent me information on their range of oximeters (Nellcor NPB-290, NPB-295, NPB-395) which use Oxismart advanced signal processing. The NPB-295 and NPB-395 have storage facilities and are suitable for sleep apnoea screening. They are also compatible with their SCORE analysis software that produces pretty reports. I suspect they won't be compatible with **SSI** Download programs for the moment- but who knows what 2002 will bring! Remember that Nellcor oximeters measure "functional saturation" and not "fractional saturation" so they read about 2% higher than most other oximeters.

I have also received information about the Masimo Radical pulse oximeter available through **Artemis Medical Ltd**, Dartford, Kent. I know nothing about them apart from the fact that they are grey with green screen.... the oximeters not the company!! Contact Jo Simmonite at Artemis on 01322 628877 or look at their website: www.artemismedical.co.uk

Nasal assisted ventilation (CPAP, NIPPV, BiLevel, etc)
The new **DeVilbiss** CPAP devices, the Vega 9000 and 9001 have hit the market. The basic 9000 is a sleek design, easy to use, lightweight (1.7kg) with lock function, breath activated compliance, variable ramp rates and a universal power supply. For £300 (excl VAT), you get machine, lead, tubing and bag. Obviously, local wheeler dealing applies as ever! The 9001 is basically the same device but with data storage space for upto 2 years and compatability with their LT Software (very pretty) and even a download facility to a Palm device (£100 at Dixons!!). This lists at £380 and I feel is expensive for what you get. Other manufacturers provide download facility a lot cheaper. I think DeVilbiss should bite the bullet and only offer the 9001 in the UK for £300. Mind you, I'm not making a living out of CPAPs!!

Medic-Aid are still selling Respironics REMstar Plus and REMstar ProCPAP devices. The ProCPAP uses their compatible Encore software that uses Smartcards, which can be posted back to the department to check patient compliance without visits. No prices were supplied, but I am aware they are available from NHS Supplies RDCs.

Miscellaneous
Finally, as a result of European legislation (in vitro Diagnostic Medical device Directive (98/79/EC)) from 2003 the safety, quality and performance of all diagnostic (in vitro) products sold in the UK will have to conform to a number of essential requirements and standards. This will particularly apply to blood gas machines and will be covered by CE marking on new equipment. It should not make much difference to most of us, but it further signifies the importance of standard operating procedures, accredited laboratories and state registered departments.

3. Complaints Database and WatchDog.
I have received a complaint about De Vilbiss Serenity masks that apparently have had a weakness in the forehead adjuster part. These have apparently been breaking very easily. Fair play to DeVilbiss they have replaced them rapidly, but I am not sure that all ARTP members are aware of the problem.

When writing to the Complaints Database and WatchDog, please state (i) exact dates, (ii) names of people you dealt with and (iii) state clearly your grievance. Also, give a summary account of the history of your complaint (a maximum of one page of A4). There is no need to send photocopies of correspondence at this stage.
Mr Nigel Clayton, (ARTP Manufacturer's Liaison Officer) North West Lung Centre, Wythenshawe Hospital, Manchester, M23 9LT. Tel: 0161 291 2406 *Email:* admin@artp.org.uk

BTS/ARTP NATIONAL ASSESSMENT IN RESPIRATORY PHYSIOLOGY 2001

Congratulations to the following candidates who have passed the National Assessment this year:

Peter Smith	Kingsmill Hospital, Mansfield	Merit	
Amina Mohamed	City Hospital, Birmingham	Pass	
Suzanne Howell	City Hospital, Birmingham	Merit	
Lorraine Smith	Heartlands Hospital, Birmingham	Merit	
Shirley Yarde	Royal United Hospital, Bath	Award	
John Wilson	West Cumberland Hospital	Pass	
Franek Leszkowski	Kings Cross Hospital, Dundee	Merit	
Amanda Peace	Doncaster Royal Infirmary	Pass	
Claire Desforges	Wordsley Hospital, Stourbridge	Merit	
Glynn Parkinson	Wordsley Hospital, Stourbridge	Pass	
Kevin Hay	Falkirk Royal Infirmary	Merit	(21 passes)
Bernadette Forde	Cragboy Hospital, Galway	Pass	
Mr R Patel	Glenfield Hospital	Merit	
Victoria Green	Wythenshawe Hospital	Merit	
Andrew Morley	Strathclyde Hospital	Pass	
William Cargill	Monklands Hospital	Pass	
Linda Topley	Queens Medical Centre, Nottingham	Merit	
Samantha Webb	Pontefract General Infirmary	Merit	
Sandra Davies	Prince Charles Hospital, Cardiff	Merit	
Andrew Skeggs	Addenbrookes, Cambridge	Merit	
Kim Lord	Basildon & Thurrock Hospital	Merit	

These results have been sent to the BTS Education Committee for ratification prior to the presentation of certificates at the ARTP Annual Conference in January.

Brendan reported to the BTS that the standard has been good again this year, with increasing numbers of technical staff opting to take the Assessment. When state registration is established it will be mandatory for all new entrants to the profession to complete the Assessment. It would still be a great advantage and proof of competence if many current technical staff registered for the National Assessment in the next few years.

ARTP NATIONAL ASSESSMENT PART 1 2002

Registration is now taking place. For further details and an application form please contact:
Jackie Hutchinson, Maney Hill Road, Sutton Coldfield, B72 1JX Tel: 0121 241 1611 e-mail: admin@artp.org.uk

CLOSING DATE FOR RECEIPT OF APPLICATIONS IS FRIDAY 8th FEBRUARY 2001

NEWS FROM THE SCOTTISH FORUM
ARTP Scottish Forum Autumn Meeting

The Autumn meeting of the ARTP Scottish Forum was held at Falkirk Royal Infirmary on Friday 26th October. Thirty-four members were present, representing sixteen different labs. The meeting was chaired by Dr Andy Robson (Western General, Edinburgh; Forum Co-ordinator) who also gave the first presentation: "Assessment of fitness to fly: an update" where he compared the results he has accumulated over the last three years with the draft British Thoracic Society guidelines. In general the BTS guidelines agreed very well with his results from 67 patients.

The second presentation was a development of an idea first used at last year's Autumn meeting and consisted of a three-way presentation from representatives of Micro Medical, Vitalograph and H.A. West (representing Ferraris Medical) who were promoting the benefits of turbine, wedge bellows and pneumotachograph spirometers respectively. With a tight rein kept on proceedings to make sure that things didn't turn into a prolonged advert, each representative was given 20 minutes to discuss the benefits of the particular type of spirometer their company produced. This generated a lot of useful discussion, and no punches were being pulled!

Following a break for lunch, the afternoon session started with the Forum Annual General meeting (which will be reported separately), which was chaired by Dr Pat Warren (University of Edinburgh Medical School) who was drafted in as a "neutral observer". After her stint chairing the AGM Pat gave a presentation on the effects of anaesthetics and tranquillisers on respiratory drive. Pat was able to show that strong sedatives such as temazepam are capable of altering a patient's respiratory drive (especially their sensitivity to increased levels of CO_2) during the postoperative period.

Roger Carter (Glasgow Royal Infirmary) gave an interesting presentation on the use of transcutaneous O_2 and CO_2 monitoring, especially during cardiopulmonary exercise. He showed how accurate monitoring of arterial levels of O_2 and CO_2 could be achieved providing proper calibration of the monitors was performed first.

To close the meeting, Dr Patricia Tweeddale (Royal Infirmary, Edinburgh) gave a fascinating presentation on the detection and correction of hyperventilation syndrome, or behavioural breathing disorder as she prefers to call it. This is not an easy condition to either diagnose or treat effectively and requires a significant investment of time, both by the patient and the practitioner.

Andy would like to thank Micro Medical, Vitalograph, Morgan Medical and H. A. West for sponsorship of the meeting. He would also like to thank Barbara Oatway for her hard work as the local liaison for the meeting and finally the members for turning up and continuing to support the Forum.

The next meeting of the Forum will probably be held in April 2002 at Perth Royal Infirmary.
For further details about events further north please contact:
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**** BUS TRIP TO BLACKPOOL ****

The Scottish Forum will be running a bus to Blackpool for the ARTP Winter Conference 2002. The format will be the same as last year with pick-ups in Edinburgh and Glasgow with the aim of arriving mid afternoon. Costs will be approximately £35 each but will depend on numbers travelling. If you are interested please contact Andy Robson on Tel: 0131 537 2351 or e-mail him at AROBSON235@AOL.COM

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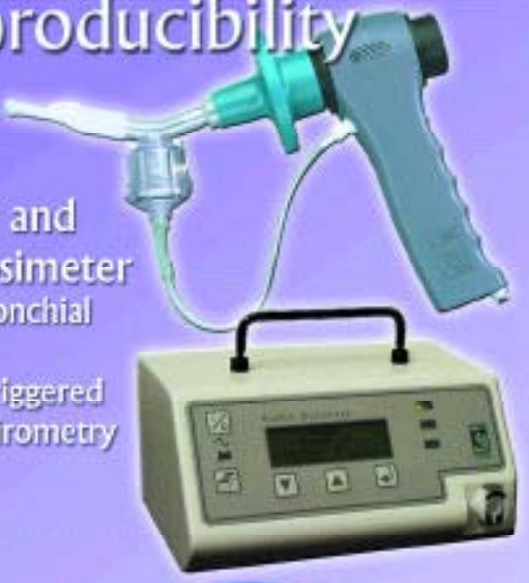

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EXERCISE LIMITATION IN CARDIAC FAILURE

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INTRODUCTION

Cardiac failure is a condition in which the cardiac output fails to meet the metabolic demands of the tissues. The clinical manifestations are loss of cardiovascular-pulmonary reserve capacity, with the initial complaint of patients being the inability to perform desired activities due to dyspnoea or fatigue.

Exercise intolerance is therefore a major feature of symptomatic cardiac failure and has thus been extensively studied (1-4). Early studies attempted to link resting ventricular function (ejection fraction, cardiac index and pulmonary capillary wedge pressure) to exercise capacity, but found poor correlation between resting measurements of cardiac function and exercise capacity. Franciosa and colleagues (5,6) demonstrated that resting heart rate, blood pressure, cardiac index, pulmonary capillary wedge pressure, left ventricular end-diastolic diameter, and ejection fraction did not correlate with bicycle exercise performance in ambulatory cardiac failure patients New York Heart Association (NYHA) class II to IV. In addition, treadmill exercise duration and maximum oxygen uptake ($VO_{2,max}$) show poor correlation with resting ejection fraction, cardiac index, and resting pulmonary capillary wedge pressure (7,8). A full cardiopulmonary exercise test therefore 1) provides a mechanism for assessing the functional capacity of patients with cardiac failure, 2) allows the determination of the sources of limitation and symptoms induced by activity and 3) is useful in the prognostic assessment of patients with cardiac failure (3,9,10).

The exercise response in cardiac failure is determined by a complex interaction of the central nervous system, central haemodynamics, skeletal muscle, peripheral factors, and the pulmonary system (2,4). None of these factors alone has been implicated as the single source of exercise limitation or the degree of breathlessness observed in these patients. The central haemodynamic and peripheral responses to maximal upright bicycle testing has been characterised by Sullivan et al (11) in a group of thirty patients with chronic cardiac failure and a mean ejection fraction of 24% when compared with a group of normal controls. The patients with cardiac failure had lower work rates, decreased $VO_{2,max}$, with a higher minute ventilation (VE) for the work rate achieved, while performing at maximal or near maximal effort. Although the cardiac failure patients had a higher heart rate at rest, the heart rate reserve was limited, so that patients reached a lower heart rate at maximum exercise when compared with the normal controls. The patients achieved a significantly lower cardiac output and stroke volume while having a higher than normal central arterio-venous oxygen difference at rest but not at maximal exercise. Maximum mean arterial pressure was lower in the cardiac failure patients, indicative of a lower workload achieved while pulmonary capillary wedge pressure and systemic and pulmonary vascular resistance were higher at rest and at each stage of exercise compared to the normal controls. These investigators found a correlation between peak cardiac output and $VO_{2,max}$ on exercise. Weber *et al* (12) allocated groups of patients with cardiac failure according to the severity of VO_2 limitation into groups A to E. The classification relates to the level of the VO_2 achieved and to the anaerobic threshold. In these patients, there was a strong relationship between the peak VO_2 achieved and the maximal cardiac index on exercise testing. This classification is commonly used today to categorise the level of impairment of cardiac failure patients.

As well as central haemodynamic abnormalities, patients with cardiac failure demonstrate peripheral blood flow abnormalities. Leg blood flow is reduced with a concomitant increase in leg vascular resistance both at rest and on exercise (11). Nakamura et al (13) found an abnormal endothelial response to acetylcholine and an abnormal vasodilatory response to nitroprusside in a group of cardiac failure patients, suggesting that endothelial dysfunction may account for an abnormality that decreases peripheral blood flow. In addition, flow mediated vasodilation is impaired in cardiac failure, with an inability to release nitric oxide (14). The impaired peripheral blood flow, by reducing oxygen delivery to the muscles, may contribute to the impaired skeletal muscle performance observed in these patients and itself, may contribute to exercise limitation. Several studies have reported decreased muscular strength and endurance in the skeletal muscle of cardiac failure patients. Although patients with chronic cardiac failure have lower muscle strength than healthy individuals, the difference is proportional to the degree of atrophy. The force generated per cross-sectional area is unchanged (15). There is, however, a marked reduction in muscular endurance, disproportionate to the degree of atrophy (15,16). Evidence of intrinsic muscle dysfunction has been provided by studies showing abnormal endurance as measured by the decline in force during consecutive isokinetic knee extensions. The loss of endurance may be related to the change in muscle fibre type leading to a decrease in oxidative capacity (17). The degree of muscle abnormality in cardiac failure is related to the degree of exercise intolerance, in contrast to resting indices of cardiac function. Lipkin et al (18) observed that peripheral muscle strength was related to exercise performance in chronic cardiac failure but not in normal subjects. It may be that the observed muscle abnormalities are simply due to inactivity in patients with chronic cardiac failure and hence de-conditioning. The patterns of abnormality seen in patients with cardiac failure with a change in fibre type and mitochondrial abnormalities are, however, unlike those seen in deconditioning (19). It is postulated that the excessive sympathetic stimulation, decreases in peripheral blood flow and cytokine activity may be responsible for some of the muscle derangement seen in patients with cardiac failure (20,21).

The search for the causes of dyspnoea on exertion has led investigators to examine the ventilatory response of cardiac failure patients to exercise. Hyperventilation, or an inappropriate increase in the VE, has been described in cardiac failure but has not been consistently linked to pulmonary pressure or to symptoms of dyspnoea (22-26). A tight relation between VE and carbon dioxide output (VCO_2) has also been described with the slope of the VE/VCO_2 being significantly steeper than in normal subjects. Myers *et al* (23) reported a maximum ventilatory response that is 25-35% higher in cardiac failure patients compared to normal subjects with an increase in ventilatory dead space attributed to ventilation-perfusion mismatch, which has been confirmed by subsequent studies (22,24-27). It has been suggested that decreased pulmonary perfusion may play a role in

excessive hyperventilation due to the decreased cardiac output in patients with cardiac failure and that exercise hyperventilation in cardiac failure contributed to the impairment of functional capacity. This could be considered as a compensatory response to abnormal haemodynamics and lung blood distribution in order to keep blood gas concentrations normal.

It has also been suggested that respiratory muscle deoxygenation may contribute to exertional dyspnoea in patients with cardiac failure and that this may parallel decreases in skeletal muscle flow (28). Selective respiratory training has been shown to significantly improve submaximal and maximal exercise capacity as well as subjective dyspnoea with activities of daily living (29). Other authors have suggested that the abnormal ventilatory response may not be due to an increase in dead space ventilation but rather to an abnormal signal originating in skeletal muscle (30) or to an increased responsiveness of the respiratory centres (23,30-32).

Breathlessness is, therefore, a common disabling symptom in patients with chronic cardiac failure (33). The mechanisms of breathlessness in these patients are complex and not related to the acute rise in pulmonary capillary wedge pressure during exercise (34-37). Respiratory muscle weakness and abnormal ventilatory responses to exercise have been proposed as possible causes of breathlessness and exercise intolerance (28, 38-40). As already mentioned, patients with chronic cardiac failure are characterised by excessive ventilatory responses to exercise (22,23, 39-45). The role of this excessive ventilatory response in the sensation of dyspnoea and exercise intolerance is controversial (46-53). In addition, the cause of this abnormality is not clear (54). The early lactic acidosis, which is commonly observed in these patients, plays an important role (34,55-57) but other factors independent of VCO_2 are also believed to contribute to the excessive ventilatory response in patients with chronic cardiac failure (25,45).

The aim of this study was to investigate the effects of ventilatory and gas exchange abnormalities on exercise capacity in patients complaining of breathlessness due to chronic cardiac failure, using continuous transcutaneous blood gas monitoring.

METHODS

Study population

Heart Transplant Candidates: Fifty heart transplant candidates who had had full pulmonary function and cardiopulmonary exercise testing during assessment for possible heart transplantation due to cardiac failure were included. They all complained of breathlessness on exertion due to left ventricular dysfunction. Anti-failure medication consisted of diuretics (all patients), digoxin (32 patients), angiotensin-converting enzyme inhibitors (41 patients), and other vasodilators (18 patients). All patients were stable at the time of assessment and none had a history of primary lung disease.

Normal subjects: 30 normal subjects recruited from the general population in whom there was no evidence of cardiopulmonary disease.

Cardio-Pulmonary Exercise Test

The formal exercise test protocol was carried out using an electrically braked bicycle ergometer (Seca Cardiotest 100) with the patient breathing through a low dead space, low resistance valve box (Hans Rudolph). The valve box incorporates a flexible pneumotachograph on the inspired limb for the measurement of tidal volume and respiratory frequency for calculation of inspired minute ventilation (Flexiflow, Morgan Medical, Kent, UK). The expired limb of the valve box was connected to a mixing chamber from which mixed expired gas could be analysed for the fractional concentration of O_2 (Fuel cell and zirconium analyser, PK Morgan Ltd) and CO_2 (infra-red spectrometer, PK Morgan Ltd). Gas analysers were calibrated with certified gas mixtures, and the pneumotachograph system was calibrated and verified using a 3-litre calibration syringe before each exercise test. Arterial oxygen and carbon dioxide ($tcPO_2$ and $tcPCO_2$) were monitored by a previously validated technique using a combined heated oxygen and carbon dioxide transcutaneous electrode (45ºC) and monitoring system (TCM3, Radiometer Ltd) following an *invivo* calibration routine (58-61) using an arterialised ear lobe capillary sample. Electrocardiographic monitoring was carried out throughout the exercise test.

Before each test, subjects were seated in a comfortable chair and a brief history was taken aimed at identifying any recent respiratory illness or cardiac decompensation and estimating the functional status at the time of the assessment. The current drug regimen of the patient was recorded. After explaining the procedure, a transcutaneous electrode was attached to the upper left quadrant of the chest and a standard 12-lead electrocardiogram was started. Following an *invivo* calibration of the transcutaneous electrode system, the subjects were initially monitored for 2 minutes whilst seated on the bicycle ergometer with a nose clip in place and breathing through the valve box system. After this initial period, the patients were instructed to cycle with no additional load for a further two minutes. Thereafter the workload was increased by variable increments (10-25 watts) every two minutes until symptoms prevented further exercise and the primary symptom limiting exercise was recorded. Blood pressure was measured using standard cuff sphygmomanometer at the end of each stage. The criteria for terminating the exercise before patients reached a maximum symptom-limited point were: ischaemic changes on ECG, ventricular arrhythmia, systemic hypotension (resting systolic BP < 90 mmHg or falling BP during exercise), or severe systemic hypertension (systolic BP > 220 mmHg).

From expired gas analysis, minute ventilation, transcutaneous gas tensions and cardiac monitoring the following values were calculated (62) and compared with predicted normal values (63) - VO_2, VCO_2, V_E, ventilatory equivalent of CO_2 (V_E/VCO_2), alveolar-arterial O_2 gradient ($A\text{-}aO_2$), dead space/tidal volume ratio (V_D/V_T), breathing reserve (predicted maximum voluntary ventilation-V_E at maximum exercise), O_2 pulse (VO_2/heart rate) and heart rate reserve. The anaerobic threshold (VO_2 AT) was estimated by the curve fitting method (64). An algorithm based on Wasserman *et al* was used to define whether limitation of exercise capacity was the result of cardiac, ventilatory, circulatory or other factors (65)

PULMONARY FUNCTION TESTING

Equipment and procedures

Standard spirometry and lung volumes were measured using a constant volume body plethysmograph (V6200 Autobox, SensorMedics Corporation, California USA). Measured variables included vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC ratio, residual volume (RV), total lung capacity (TLC), and specific airways conductance (SG_{aw}). The single breath transfer factor for carbon monoxide (TL_{CO}) transfer coefficient (K_{CO}) and the subdivisions of TL_{CO} – diffusing membrane capacity (D_M) and pulmonary capillary blood flow (V_C) were measured using the Transflow System (Model 540, Morgan Medical, Kent, England) (66–68). Quality control and procedures of testing were performed according to formal guidelines established by the European

Respiratory Society (ERS) and recommended by the British Thoracic Society (BTS) and the Association for Respiratory Technology and Physiology (ARTP) (69). Results of at least 3 satisfactory manoeuvres were analysed and the reported values were the highest value for FEV_1 and FVC and the mean of the 3 results for each of the remaining indices.

Normal Values

Results of the pulmonary function tests were expressed in absolute values and as percentage of predicted. Predicted normal values were determined using the European Community for Steel and Coal equations (ECCS), which are recommended by the European Respiratory Society and endorsed by the British Thoracic Society (70).

The severity of cardiac failure was assessed by left ventricular ejection fraction (LVEF), duration of exertional dyspnoea and New York Heart Association (NYHA) functional status. LVEF was measured by radionuclide imaging at the time of lung function assessment. Pulmonary haemodynamics were measured by the referring cardiologists at various centres as part of routine assessment for possible heart transplantation. They were all performed within 3 months (range 1-13 weeks) of pre-transplant lung function assessment and included mean pulmonary artery pressure (mPAP), mean pulmonary capillary wedge pressure (mPCWP) and the transpulmonary gradient (TPG).

Data presentation and analysis

Unless stated otherwise, values are expressed as mean ± one standard error of the mean. Lung function and cardio-pulmonary exercise data in heart transplant candidates were compared to those of normal subjects using the one-way analysis of variance (ANOVA). The relationship, between 2 or more variables was assessed using the Pearson's correlation coefficient and stepwise multiple linear regression analysis.

Results

Subject characteristics: Subject characteristics are summarised in Table 1.

Resting Pulmonary Function: Table 2 compares the resting pulmonary function test results. In the cardiac failure patients the FEV_1, K_{CO}, D_M and V_C were significantly lower compared to normal subjects.

Cardio-Pulmonary Responses to Exercise: Table 3 shows the results of resting and maximal cardio-respiratory data in the heart transplant patients and normal subjects.

At rest, the resting heart rate and V_D/V_T were significantly higher in transplant patients than in the normal subjects. There were no significant differences in the A-aO_2 or VO_2 between the two groups.

Maximum symptom limited oxygen uptake (VO_2), as a percentage of predicted was significantly lower in cardiac failure patients than in normal subjects. The ventilatory anaerobic threshold (VO_2 AT %predicted $VO_{2,max}$) was significantly reduced in candidates compared to normal controls. The ventilatory and gas exchange responses to exercise (V_E/VCO_2, V_D/V_T and A-aO_2) were all significantly higher in the cardiac failure patients than in normal controls. The heart rate response on exercise was markedly elevated in the cardiac failure patients compared with normal controls, whilst the O_2 pulse was significantly lower in the cardiac failure patients compared with normal controls.

Figure 1 shows the regression plot of the ventilatory response on exertion (V_E/VCO_2) against % predicted VO_2 at maximum exercise in cardiac failure patients. There was a significant negative correlation between the ventilatory response on exertion and the maximal symptom limited oxygen uptake as a percentage of predicted ($r = -0.67$; $p<0.001$). In contrast, there was no correlation between these two variables in normal controls ($r = -0.24$; $p = 0.06$).

Dyspnoea was the primary symptom-limiting exercise in 40 patients and in 5 normal controls. In contrast, 5 patients with cardiac failure and 25 controls had leg fatigue as the primary symptom, and the remaining 5 patients reported dyspnoea and fatigue as equally limiting symptoms. The dyspnoea index (V_E/MVV) at maximum symptom limited exercise was similar in the two groups but this level of ventilation was achieved at the significantly reduced $VO_{2,max}$ in the cardiac failure patients compared to controls. Figure 6 shows the regression plot of dyspnoea index (V_E/MVV) against % predicted VO_2 at maximum exercise. There was a significant negative correlation between the V_E/MVV at maximum exercise and the maximum VO_2 achieved in cardiac failure patients ($r = -0.61$; $p<0.001$).

Table 3 shows that the increased ventilatory response in patients with cardiac failure was associated with a raised degree of "wasted ventilation" (V_D/V_T). At rest, the degree of "wasted ventilation" as assessed by the V_D/V_T was significantly increased in cardiac failure patients compared to controls. At maximum symptom-limited exercise, V_D/V_T decreased normally in controls, but persisted in patients making the difference between the two groups greater than at rest. Although the A-aO_2 gradient increased slightly on exertion in cardiac failure patients compared to controls it remained within normal limits and did not reach statistical significance.

Figure 3 shows the regression plot of V_D/V_T at maximum exercise against V_E/VCO_2 in cardiac failure patients. There was a significant positive correlation between the degree of "wasted ventilation" at maximum exercise and the ventilatory response on exertion ($r = 0.79$;$p<0.001$).

At maximum symptom limited exercise the tidal volume was significantly reduced in the cardiac failure patients compared to normals, and although the breathing frequency was reduced in patients with cardiac failure compared to normals, it did not reach significance (Table 3).

The Relationship Between Pulmonary Function And Exercise Parameters: Since measured transfer factor (TL_{CO}) is directly influenced by haemoglobin concentration, analysis of the relationship between TL_{CO} and exercise parameters was made after correction for haemoglobin. There was a significant positive correlation between TL_{CO} and VO_2 in the cardiac failure patients ($r = 0.42$, $p<0.01$). In contrast, there was no correlation between these variables in normal controls. Similarly, within the cardiac failure patients there were significant correlations between VO_2 and D_M ($r = 0.42$, $p<0.001$) and VO_2 and V_C ($r = 0.35$, $p<0.05$).

There was no relationship between % predicted VO_2 and any other indices of lung function in either cardiac failure patients or normal controls. TL_{CO} was also positively correlated with the anaerobic threshold in the cardiac failure patients ($r = 0.54$, $p < 0.001$) and inversely correlated with the non-invasive indices of gas exchange (V_D/V_T, $r = -0.29$, $p < 0.05$; A-aO_2, $r = -0.38$, $p < 0.01$; V_E/VCO_2, $r = -0.43$, $p < 0.001$). There was no relationship between TL_{CO} and any of these parameters in the normal controls.

DISCUSSION

Resting pulmonary function: Ischaemic heart disease is the most common cause of congestive cardiac failure. Other causes include

valvular heart disease, systemic hypertension and primary myocardial disease. Lung function studies indicate that lung function abnormalities are common in congestive cardiac failure. Severe lung dysfunction is uncommon, but mild to moderate abnormalities of various lung function indices have been reported (71,72). A restrictive ventilatory defect is the most common abnormality with reduced lung volumes and reduced pulmonary compliance (73,74). The results of resting pulmonary function in this study are in agreement with these previous reports. Studies in non-heart transplant recipients (i.e. patients with less severe cardiac failure) showed that the transfer factor is usually normal or only slightly reduced even during episodes of acute cardiac failure (75,76). However, in patients with moderate to moderately severe chronic cardiac failure, Siegel et al (77) showed that the mean TL_{CO} in the entire group was 73% of predicted. The TL_{CO} in this group was significantly lower in patients with evidence of pulmonary oedema (rales on auscultation) compared to those without evidence of pulmonary oedema (53% vs. 83% predicted). In addition, in the patients with pulmonary oedema, TL_{CO} correlated with the left ventricular ejection fraction ($r = 0.81$; $p<0.001$). It was concluded that the TL_{CO} was a useful predictor of clinically evident congestive cardiac failure. The present study would support these findings with a mean TL_{CO} of 62% of predicted in a group of patients with moderately severe to severe cardiac failure.

Analysis of the components of the TL_{CO} suggests that this reduction occurs because of a proportionate reduction in both D_M and V_C. A reduction in the membrane component (D_M) has been reported in stable patients with mild to moderately severe congestive cardiac failure (NYHA functional classes II and III) (78,79). In 1995, the same investigators (80) showed that the reduction in D_M in patients with congestive cardiac failure was significantly correlated with maximal exercise capacity and was inversely correlated with the pulmonary vascular resistance. It was concluded that the reduction in D_M was the major cause of TL_{CO} impairment in patients with cardiac failure and it appeared to contribute to exercise intolerance. It was also suggested that D_M might be a useful marker for the alveolar-capillary membrane damage caused by pulmonary hypertension.

In patients with mitral stenosis, D_M has been shown to decline progressively with increasing severity of disease (81). In contrast, V_C has a biphasic relation with disease severity. In mild to moderately severe mitral stenosis, V_C is usually normal and may even be increased whereas in severe cases it is reduced (82,83). This suggests that if pulmonary arterial hypertension or pulmonary vascular resistance is severe then both components of the transfer factor are reduced (84). A reduced V_C would therefore suggest a significant derangement of the pulmonary vascular bed. The V_C in patients with severe chronic cardiac failure is determined by two factors acting in opposite directions, the increased pulmonary venous pressure tending to increase it and the pulmonary oedema and pulmonary hypertension and fibrosis tending to decrease it. Pulmonary hypertension, oedema and fibrosis will also lead to a progressive decline in D_M. The findings of equally reduced D_M and V_C in our transplant patients are similar to the findings in patients with severe mitral stenosis (85), suggesting that these patients have significant pulmonary parenchymal and vascular abnormalities (86). That chronic cardiac failure disturbs the alveolar capillary interface and increases the resistance to gas transfer by affecting both D_M and V_C have been confirmed in a recent review by Guazzi (87).

Cardio-Pulmonary Responses To Exercise: Exercise intolerance is a major cause of morbidity in patients with chronic cardiac failure, even when they are on optimal medical treatment and free of symptoms at rest (88). Fatigue, secondary to muscle underperfusion and deconditioning, is characteristic of chronic cardiac failure and is generally accepted as the primary cause of exercise limitation in this condition (32). Breathlessness is a common disabling symptom in these patients (33) and frequently coexists with fatigue, although in some patients it is the primary symptom (88).

Patients with chronic cardiac failure exhibit an excessive ventilatory response to exercise (22,23,39-45). The role of this excessive ventilatory response in the sensation of exertional breathlessness and exercise intolerance is controversial. Some investigators have reported a significant association between the ventilatory response and maximal oxygen consumption (39-42), with conflicting results in other studies (34,55). The variation of results in these studies may, in part, reflect the heterogeneous populations with chronic cardiac failure from various causes and of varying degrees of severity. Our results in stable patients with advanced chronic cardiac failure confirm that excessive ventilatory response to exercise is characteristic of chronic cardiac failure. In addition, the ventilatory response at maximum symptom limited exercise and the maximal symptom-limited oxygen uptake as a percentage of predicted were inversely related in cardiac failure patients. In contrast, there was no correlation between these two variables in controls. Patients with cardiac failure were more likely to stop exercising because of breathlessness than controls. In patients with cardiac failure, the dyspnoea index calculated as the V_E/MVV was also inversely related to the maximal rate of oxygen consumption. These findings support the concept that the excessive ventilatory response may contribute to exercise intolerance in patients with chronic cardiac failure (39-42).

Gas exchange measurements have been used to grade the severity of heart disease in the selection of patients for heart transplantation (9,90). These and other studies have shown that the greater the cardiac dysfunction the greater the increase in the ventilatory response on exertion (23,41,45,91). The cause of the increased ventilatory response to exercise in patients with chronic cardiac failure is uncertain. Classically it has been suggested that the acute rise in pulmonary venous pressure on exertion stimulates the juxtacapillary receptors, leading to rapid shallow breathing; thereby, increasing the ventilatory response and the work of breathing in these patients. There is, however, substantial evidence that exercise capacity is not related to the pulmonary capillary wedge pressure in these patients (25). Exercise training in patients with chronic cardiac failure reduces the ventilatory response, and it is possible that this abnormality may be partly due to physical deconditioning (25,92).

One major factor contributing to the excessive ventilatory response in cardiac failure is the metabolic changes in exercising muscles. Early lactic acidosis occurs in these patients resulting in increased carbon dioxide production, which stimulates ventilation to maintain normal blood pH (34,43,55,57). Recent studies, however, have suggested that additional factors independent of carbon dioxide production may be involved in driving the excessive ventilation (25,93). These include abnormal control of breathing with rapid shallow breathing (56) and increased degree of "wasted ventilation" on exertion (22,40,93).

The ventilatory response on exertion depends on how tightly pH and arterial CO_2 are regulated as work rate increases. There appears, however, to be nothing unusual about the control mechanisms that regulate pH and the transcutaneously monitored arterial carbon dioxide in the patients with cardiac failure. The end

exercise transcutaneous PCO_2 was only slightly reduced from that at rest in these patients, a finding supported by the study of Wasserman et al (92).

Another theory relates the elevated ventilatory requirement in patients with chronic cardiac failure to increased dead space ventilation and ventilation/perfusion abnormalities on exertion (22,43,94,95). Lewis et al (93) compared the ventilatory cost of exercise in chronic cardiac failure and chronic anaemia and demonstrated that exercise duration and maximum VO_2 were similarly reduced in the cardiac failure and anaemia groups compared to normal controls. The V_E/VCO_2 slope, however, was normal in the anaemic patients, but steeper in patients with chronic cardiac failure, suggesting ventilation/perfusion mismatch rather than early lactic acidosis as the primary cause of the excessive ventilatory response in chronic cardiac failure. Metra et al (40) and Sullivan et al (22) demonstrated a significant correlation between the slope of the V_E/VCO_2 and the V_D/V_T ratio. These studies, however, have been criticised because of the equation used to derive V_D/V_T (25,95). They used the modified alveolar gas equation (96) that relates V_E/VCO_2 and V_D/V_T in the

$$V_E = VCO_2 \times \frac{863}{P_aCO_2} \times \left[1 - \left(\frac{V_D}{V_T}\right)\right]$$

following manner:

$$\frac{V_I}{VCO_2} = \frac{863}{P_aCO_2} \times \left[1 - \left(\frac{V_D}{V_T}\right)\right]$$

And hence:

Where 863 is a constant to standardize gas measurements to body temperature, pressure and saturation. Because of their relationship in this equation, the two variables of interest (V_E/VCO_2 and V_D/V_T) would necessarily correlate with each other without evoking any causal relationship. The present study differs in that the V_D/V_T was derived independently of the V_E/VCO_2 relationship. We used the combined oxygen and carbon dioxide transcutaneous electrode, which had been previously validated (61), to monitor arterial blood gases throughout exercise testing non-invasively. This allowed the estimation of V_D/V_T using the

$$\frac{V_D}{V_T} = \frac{(P_aCO_2 - P_ECO_2)}{P_aCO_2}$$

Bohr equation (97):

Where P_ECO_2 is mixed expired carbon dioxide tension.

The results of this study show that patients with chronic cardiac failure have an increased degree of "wasted ventilation" as assessed by V_D/V_T at rest, and this persisted on exertion. The level of wasted ventilation at maximum symptom limited exercise was significantly correlated with the ventilatory response, confirming the findings of Metra et al (40) and Sullivan et al (22). The elevated resting V_D/V_T as estimated using continuous transcutaneous monitoring of arterial blood gases is in agreement with then findings of Rajfer at al (97) who used the same equation as in the present study but with direct arterial sampling in 10 patients with chronic cardiac failure. In this investigation, the resting V_D/V_T was elevated (0.42 ± 0.05) and declined at maximal exercise (0.33 ± 0.06). Unlike the present findings, this decline was statistically significant, but V_D/V_T was still higher than would be expected in normal controls at maximal exercise. This finding has been confirmed in subsequent studies (93,99). These results suggest that the failure to decrease the level of wasted ventilation on exercise may be partially responsible for the excessive ventilatory response observed in patients with chronic cardiac failure.

The finding that the V_D/V_T increases in proportion to the degree of exercise limitation without the development of arterial hypoxaemia or a significantly increased $A\text{-}aO_2$ gradient is an observation of great importance in understanding why patients with more severe cardiac dysfunction have a greater ventilatory requirement at any given work rate. These observations suggest that pathologically high ventilation/perfusion ratio mismatching occur in cardiac failure patients without significant low ventilation/perfusion mismatching. This suggests that the abnormality leading to the increased degree of "wasted ventilation" is on the pulmonary circulation rather than the airway side of the gas exchange unit i.e. perfusion is reduced in a well-ventilated lung.

The mechanism underlying the elevated V_D/V_T was not investigated in this study but several mechanisms have been suggested in previous studies. Tachypnoea with shallow breathing and increased ventilation/perfusion mismatch have been suggested as possible causes of this increased V_D/V_T (94,56). Increased pulmonary congestion on exercise would be expected to stiffen the lungs and stimulate the pulmonary juxtacapillary receptors leading to tachypnoea with shallow breathing, and thereby raising the V_D/V_T (56). In the present study, the tidal volume was significantly lower in patients compared to controls at maximum symptom limited exercise, but the respiratory frequency was not significantly different in the two groups. During exercise, the tidal volume values in cardiac failure patients were above resting levels, whereas V_D/V_T values were higher than normal resting values. The levels of the respiratory frequency and tidal volume reached in patients with cardiac failure suggest that tachypnoea with a shallow breathing pattern, which would produce a reduction in the alveolar ventilation compared to total ventilation, was not a major factor in the observed high V_D/V_T ratio on exertion in these patients. In addition, if pulmonary congestion developed during maximal exercise then arterial hypoxia and cough might be expected to develop. In this study, as mentioned, previously the alveolar arterial oxygen gradient was usually normal at maximal exercise, and a cough after the exercise test was uncommon in the cardiac failure patients studied.

A second mechanism suggested for the increase in V_D/V_T is uneven elevations in pulmonary venous pressure. This is postulated to result in greater reduction in perfusion to lung units with higher venous pressure (93). This would have the effect of decreasing pulmonary perfusion relative to alveolar ventilation and hence, causing ventilation/perfusion inequality.

A third mechanism accounting for the increase in V_D/V_T might be a change in paracrine secretion regulating pulmonary vasomotor tone. Relative low blood flow and decreased shear stress in the pulmonary blood vessels might reduce nitric oxide production and increase endothelin-1 production, both of which would promote pulmonary vasoconstriction (99). A reduction in nitric oxide production has been described in the artery of an animal model of chronic cardiac failure (100). The pulmonary vasoconstriction resulting from the reduction in vasodilator paracrines and the increase in vasoconstrictor paracrines in the pulmonary circulation would be expected to increase V_D/V_T. This vasoconstrictor mechanism would protect the lungs from the development of pulmonary oedema. This hypothesis is supported by the report of Bocchi et al (101) that showed that administration of exogenous nitric oxide to the lungs of cardiac patients promotes pulmonary oedema in cardiac failure patients but not other cardiac patients.

TL_{CO} and exercise capacity: It is tempting to link the decrease in pulmonary blood flow to the ventilated lung as is evident from the findings of an increased V_D/V_T but normal A-aO_2 gradient to the changes in lung mechanics observed in cardiac failure. In this study, we have shown that transfer factor impairment, which is common in patients with cardiac failure, was associated with exercise intolerance. The correlation between haemoglobin corrected TL_{CO} and exercise performance was independent of lung volumes and airway function both of which were normal in most patients and had no influence on exercise capacity. TL_{CO} impairment has been previously shown to predict pulmonary gas exchange abnormalities and exercise intolerance in patients with mild to moderately severe cardiac failure (80,102,103). Puri et al (80) reported a positive correlation between TL_{CO} and exercise capacity in patients with mild to moderately severe congestive cardiac failure (NYHA, grades II and III) and showed that it was primarily due to impairment of the diffusing capacity of the alveolar capillary membrane (D_M). Unlike these reports, there was no relationship between the maximal VO_2 and any of the diffusion parameters in heart transplant candidates. The difference between our findings and the others may be due to the severity of cardiac failure in the patients studied. A recent review by Guazzi (87) has confirmed the significant contribution of altered gas transfer, as assessed by the transfer factor and its components, to the pathogenesis of exercise limitation and ventilatory abnormalities in patients with cardiac failure.

The mechanism by which TL_{CO} impairment causes exercise intolerance is not clear. Gas exchange in the lungs depends on several interdependent processes. These include ventilation, perfusion, ventilation-perfusion matching and diffusion (104). The measured TL_{CO} is affected by disturbance in any of these processes and, therefore can be considered as a composite index of the integrity of the pulmonary gas exchanging unit, rather than being specifically determined by the process of diffusion. In addition, isolated impairment of diffusion is very rare and because of the large physiological reserve, diffusion impairment is not considered an important limiting factor in the transfer of O_2 to the arterial blood, even in patients with severe lung disease (105). The relationship between TL_{CO} and exercise performance in heart transplant recipients is therefore likely to represent a general dysfunction of pulmonary gas exchange rather than an isolated diffusion defect. This is confirmed by the inverse relationship found between TL_{CO} and other measurements of ventilation/perfusion inequality (V_D/V_T and A-aO_2 gradient) on exertion

Conclusion: In conclusion, using completely non-invasive cardiopulmonary exercise testing, we found that patients with chronic cardiac failure have a significant degree of "wasted ventilation", which is associated with an excessive ventilatory response on exertion. The increased ventilatory response on exertion appears to contribute to exercise limitation in these patients. The elevated V_D/V_T on exertion in patients with cardiac failure in the presence of a normal A-aO_2 gradient is consistent with areas of the lung being underperfused compared to their ventilation. In addition, TL_{CO} impairment in cardiac failure is associated with the abnormal ventilatory and pulmonary gas exchange responses to exercise. Pulmonary dysfunction as reflected by TL_{CO} impairment also appears to contribute to exercise intolerance in patients with cardiac failure.

It is likely that these ventilation/perfusion abnormalities reflect a limited capacity to increase cardiac output compared to ventilation. This mechanism is of increasing importance, as the cardiac failure patient becomes more dysfunctional. The lungs of the cardiac failure patient also show abnormalities of gas transfer. These changes may be linked to the high ventilation/perfusion mismatching that takes place in the lungs of cardiac failure patients.

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Legend to Figures

Figure 1. Regression plot of the ventilatory response equivalent for carbon dioxide output (V_E/VCO_2) against % predicted $VO_{2,max}$ corrected for body weight (mls.kg^{-1}.min^{-1}) in 50 patients with chronic cardiac failure.

Figure 2. Regression plot of V_E/MVV (Dyspnoea Index i.e. maximum minute ventilation/maximum voluntary ventilation) against % predicted maximal oxygen uptake corrected for body weight (VO_2 ml.kg^{-1}.min^{-1}) in 50 patients with chronic cardiac failure

Figure 3. Regression plot of dead space/tidal volume ratio (V_D/V_T) at maximum exercise against the ventilatory response equivalent for carbon dioxide output (V_E/VCO_2) in 50 patients with chronic cardiac failure.

Table 1 Characteristics of the study groups.

	Cardiac Failure	Normal
N	50	30
Age (yrs)	48.1 (19-66)	40.4 (19-61)
Male: Female	42: 8	23: 7
Current/Ex*-/Non-smokers	10: 40: 0	16: 11: 3
Diagnosis		
Ischaemic heart disease	31	-
Dilated cardiomyopathy	16	-
Others	3	-
Pre-transplant LVEF	13.4 ± 5.4	-
Pre-transplant transpulmonary gradient	9.2 ± 4.8	-
Haemoglobin g/dl Mean	12.2 ± 1.2	Assumed 14.6
Pre-Transplant Functional Class		
NYHA III	14	-
NYHA IV	36	-

> 6 months. Data are given as mean ± standard deviation or as mean (range)

Table 2: Resting Pulmonary function results (as percentages of predicted) in heart transplant candidates and normal controls (*p< 0.05).

	Cardiac Failure	Normals
FEV1 Litres	90.1 ± 2.3	107.2 ± 3.9*
FEV1/FVC	93.1 ± 1.4	98.6 ± 1.8
TLC Litres	96.3 ± 2.3	100.2 ± 3.3
TLCO (mmol.min-1.kPa-1)	57.5 ± 2.0	98.6 ± 1.3*
TLCO (Hb-corrected)	62.3 ± 2.1	-
KCO (mmol.min-1.kPa-1.l-1)	65.9 ± 2.4	105.3 ± 2.2*
KCO (Hb-corrected)	71.4 ± 2.5	-
D_M (mmol.min-1.kPa-1)	81.4 ± 5.4	100.0 ± 1.3*
V_C (mls)	80.2 ± 4.2	102.0 ± 2.8*

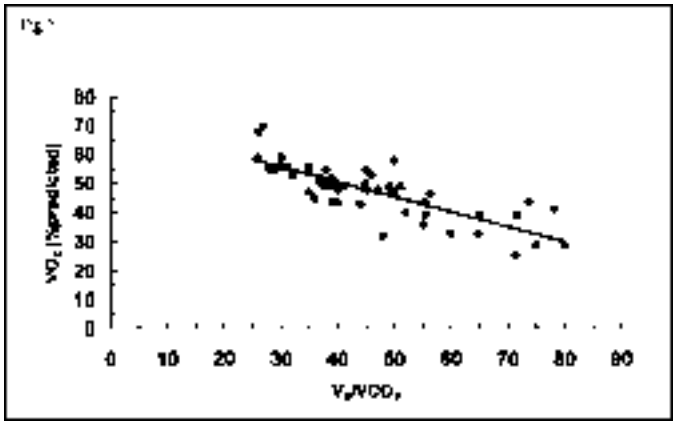
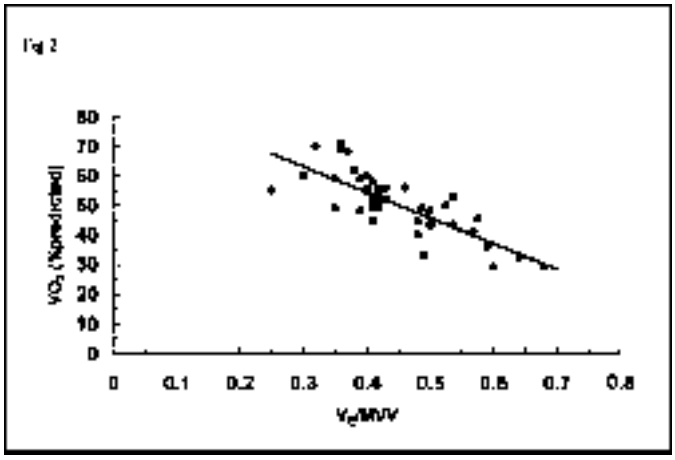
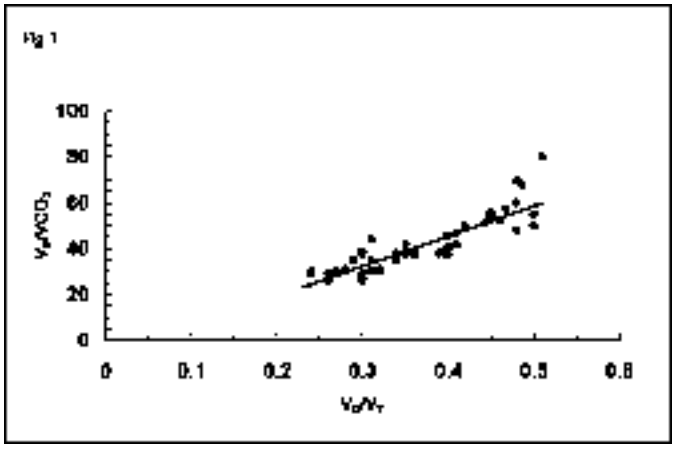


Table 3 Resting and maximal cardio-respiratory data in Cardiac Failure and normal controls (*p < 0.05).

	Cardiac Failure	Normals
Resting Data		
VO_2 (L.min $^{-1}$)	0.26 ± 0.25	0.25 ± 0.23
HR (beats.min $^{-1}$)	91.0 ± 2.0	79.0 ± 2.0*
V_D/V_T	0.40 ± 0.01	0.30 ± 0.01*
A-aO_2 (kPa)	1.9 ± 0.1	1.6 ± 0.1
Maximal Exercise Data		
VO_2 % pred.	45.3 ± 2.2	92.9 ± 2.5*
VO_2 AT %	31.5 ± 1.1	52.6 ± 1.9*
V_E L.min $^{-1}$	46.4 ± 1.6	72.4 ± 4.2*
V_E % pred.	45.6 ± 1.8	57.9 ± 2.1*
V_E /VCO_2	45.6 ± 2.5	24.3 ± 1.6*
V_E/MVV	0.44 ± 0.12	0.40 ± 0.07
V_D/V_T (max)	0.35 ± 0.02	0.19 ± 0.01*
Tidal Volume (litres)	1.69 ± 0.38	2.02 ± 0.43*
Breathing Frequency (.min^{-1})	27.7 ± 4.8	35.1 ± 6.9
A-a O_2 kPa	2.4 ± 0.3	1.8 ± 0.1
Heart Rate (HR) % pred.	82.2 ± 2.6	86.4 ± 1.9
HR response beats. L^{-1}	72.1 (6.5)	35.7 (2.0)*
O_2 pulse, ml.beats^{-1}	7.7 (0.4)	16.5 (0.6)*





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