# Observational case series of postural tachycardia syndrome (PoTS) in post-COVID-19 patients

Nicholas P Gall, Stephen James, Lesley Kavi

## Authors

**Nicholas Gall**
Consultant Cardiologist, King's College Hospital

**Stephen James**
Consultant in Intensive Care and Exercise Capacity, King's College Hospital

**Department of Cardiology, London Bridge Hospital, London, SE1 2PR**

**Lesley Kavi**
General Practitioner, Solihull, Visiting Professor, Birmingham City University, and Trustee and Chair of PoTS UK

**Seacole Building, Westbourne Road, Edgbaston, Birmingham, B15 3TN**

**Correspondence to:**
Dr N Gall
**(nickgall@doctors.org.uk)**

## Key words

autonomic, case series, COVID-19, long-COVID-19, postural tachycardia syndrome (PoTS), syncope, tachycardia

First published online January 26th 2022

doi: 10.5837/bjc.2022.003

*Br J Cardiol* 2022;**29**:16–20

**There is emerging evidence that a proportion of patients who develop long (post)-COVID-19 have abnormalities in the regulation of their autonomic nervous system manifesting as postural tachycardia syndrome (PoTS). We report a series of 14 patients who developed symptoms and signs compatible with PoTS following clinically diagnosed COVID-19 infection. Their symptoms and clinical findings were consistent with those of patients with non-COVID-related PoTS. The authors recommend an active stand test for patients who present after COVID-19 infection with cardiovascular symptoms including chest pain, palpitations, light-headedness and breathlessness that are worse with the upright posture. They further recommend training of clinicians and investment in health services to provide for the anticipated significant increase in patients presenting with PoTS and other forms of autonomic dysfunction due to the COVID-19 pandemic.**

## Introduction

Postural tachycardia syndrome (PoTS) was first described in American Civil War soldiers,[1] and subsequently in First World War soldiers.[2] These early descriptions were published by some of the most eminent cardiologists of the time. It was defined formally as a syndrome in 1993,[3] leading onto guidelines, more recently published by the Heart Rhythm Society 2015,[4] and the Canadian Cardiovascular Society 2020.[5] While we remain uncertain in detail about its underlying cause, it seems to be an abnormality in the regulation of the cardiovascular system causing excessive tachycardia on standing, with cardiovascular symptoms including chest pain, breathlessness, palpitation, faintness (pre-syncope) and loss of consciousness. It is common in younger women,


with possible hormonal and autoimmune causes.[6] There seems to be an association with other conditions including hypermobility,[7] although the exact reasons are uncertain. Small fibre neuropathy associates in some.[8] It can associate with more widespread abnormalities in other organ systems,[9] including bowel dysfunction, bladder dysfunction, migraines and, in some, inappropriate histamine release.[10] In many cases, it is precipitated by another illness, and, until the COVID-19 pandemic, infections,[11] e.g. Epstein-Barr virus, have been a particularly common precipitant.

The current SARS-CoV-2 (COVID-19) pandemic has led to severe illness in some, with a proportion dying. Other patients are asymptomatic or have milder symptoms. It is increasingly recognised that there are long-term effects regardless of the infection severity, known as long-COVID-19.[12,13] The National Institute for Health and Care Excellence (NICE) has produced guidance,[14] in terms of definitions and management, although data remain limited; they refer to the condition as post-COVID-19.

PoTS is mentioned in the NICE guidance as a potential cause of some post-COVID-19 symptoms. There have been a number of descriptions of autonomic disturbance associated with COVID-19,[15,16] initially in intensive care patients, but more recently single case reports of PoTS in COVID-19 patients not requiring critical care,[17-19] a series with suggestive symptoms from a syncope

unit,[20] and a small series of diagnosed patients.[21,22] The American Autonomic Society noted a recent increase in referrals to US autonomic clinics, and anticipate that numbers will increase further as the pandemic progresses.[23]

This patient series is a description of 14 patients with a history consistent with COVID-19, referred to a single centre with a possible PoTS diagnosis, and subsequent orthostatic measurements consistent with post-COVID-19 PoTS. This offers insights into post-COVID-19 PoTS that might be useful for clinicians caring for long-COVID-19 patients.

## Method

This is a retrospective analysis of the medical records of patients referred to a single clinic at London Bridge Hospital from soon after the start of the pandemic for the assessment for PoTS. No patient had prior PoTS symptoms. Since testing in the UK during the early months of the pandemic was not universally available, the COVID-19 diagnosis was made clinically by the referring physician based on a history consistent with preceding COVID-19 infection. All patients referred for the consideration of PoTS were assessed clinically with a full, structured medical history, including enquiry about cardiovascular and extra-cardiac symptoms and clinical examination. Hypermobility was assessed using the Beighton score.[24] As the patients were assessed during routine clinical practice, the assessments show some variation from patient to patient as not every aspect was indicated in each case. The retrospective study was approved by the Research and Ethics Committee, London Bridge Hospital, with a waiver of individual consent.

All patients completed a postural assessment of heart rate and blood pressure and fulfilled criteria for PoTS as defined in recent guidance:[4,5] in short, typical symptoms and a sustained heart rate (HR) increase of >30 beats per minute (bpm) within 10 minutes of standing, and without orthostatic hypotension (blood pressure [BP] fall >20/10 mmHg). For patients aged 12–20 years, an increase of >40 bpm was required. Some patients undertook a home active-stand test using commercially available HR and BP monitoring equipment. In this situation, patients were asked to lie flat for 10 minutes documenting HR and BP every minute with their symptoms. Subsequently, they were asked to stand unaided, measuring HR and BP once every minute for 10 minutes, again noting their symptoms.

In some cases, a formal tilt-table test was undertaken. This involved 12-lead electrocardiogram (ECG) monitoring, as well as the use of a continuous beat-to-beat HR and BP assessment system (Finapres Medical Systems, Netherlands). Patients were laid flat for 10 minutes in a quiet, warm, well-ventilated room, to allow their HR and BP to stabilise, and were subsequently tilted head-up for up to 15 minutes, with ongoing monitoring of HR, BP and symptoms.

Seven patients also underwent cardiopulmonary exercise testing (CPET) as part of a more detailed assessment. CPET (or $VO_{2max}$ test) is a non-invasive assessment of aerobic fitness. Our methods for this form of testing have been published elsewhere.[25]

After determining that patients met the criteria for PoTS, a management plan was offered following recent guidelines.[5] Self-management recommendations included a fluid intake of three litres per day in an adult and increased salt intake, lower body compression garments and non-upright exercise.

Descriptive data are reported as proportions and mean ± standard deviations (Excel, Microsoft Corporation, Redmond, WA, USA).

## Results

### Demographics and orthostatic vital signs

Of the 14 total patients, the mean age was 35 ± 10 years (range 17–48 years), and 12 patients were female (**table 1**). All but one patient developed their COVID-19 infections during the first UK wave between February and April 2020, while the last patient developed their infection in September 2020 (beginning of the second wave). All patients fulfilled haemodynamic criteria for PoTS when assessed (**table 1**). Home stand tests were done 8 ± 3 months after symptom onset, while tilt-table tests were done 5 ± 2 months after symptom onset. Three patients had both a home stand test and a tilt-table test with similar clinical findings. To meet the criteria for PoTS, patients should have experienced symptoms for a minimum duration of three months. One patient with a positive tilt-table test at two months still had excessive orthostatic tachycardia with a stand test at nine months.

### Illness progression and symptoms

Eleven patients reported that they remained symptomatic throughout their illness; three showed some initial clinical improvement after their acute COVID-19 infection with subsequent deterioration and the development of symptoms of PoTS (**table 2**, available online). None of these patients were hospitalised due to COVID-19 infection.

Eleven (79%) patients described chest discomfort with varying characteristics, often with musculoskeletal features or discomfort consistent with dysfunctional breathing (generalised chest tightness and the sense of an inability to fill the lungs). Twelve (86%) patients described exertional breathlessness with dysfunctional breathing characteristics, as we have previously described.[26] Thirteen (93%) patients described palpitation, in some cases consistent with ectopic beats. Most had an awareness of tachycardia, which would often take longer than expected to settle after activity. All patients described cardiovascular symptoms that were prominent while standing and improved with recumbence, with 12 (86%) patients describing postural faintness. The symptoms are very similar to those described by other, non-COVID-19 related PoTS patients.[4]

Many patients also complained of extra-cardiac symptoms. Seven (50%) patients exhibited clinical signs of joint hypermobility based on the Beighton score.[24] Eleven (79%) patients described migraine headaches; 10 (71%) patients noted gastrointestinal manifestations including nausea, bloating, constipation/diarrhoea; five (36%) suffered brain fog (an inability to think or concentrate); two (14%) patients suffered fatigue; two (14%) patients had prominent nerve symptoms (paraesthesiae, tingling, numbness and shooting pains); two (14%) patients described urinary dysfunction (increased urinary frequency, nocturia, an inability to pass urine easily); three (21%) patients developed joint pains; and one (7%) patient noted urticaria.

**Table 1. Demographics and orthostatic vital signs**

| Demographics | | | Home active-stand test | | | | | | Laboratory tilt-table test | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Patient number | Age | Sex | Supine HR | Supine BP | Standing HR at 10 minutes | Standing BP at 10 minutes | Delta HR | Months after onset of COVID-19 | Supine HR | Supine BP | Standing HR at 10 minutes | Standing BP at 10 minutes | Delta HR | Months after onset of COVID-19 |
| 1 | 48 | F | 54 | 100/60 | 100 | 98/74 | 46 | 11 | 68 | 112/80 | 107 | 99/80 | 39 | 2 |
| 2 | 27 | M | – | – | – | – | – | – | 75 | 125/72 | 106 | 113/76 | 31 | 6 |
| 3 | 39 | F | 59 | 105/59 | 107 | 97/75 | 48 | 5.5 | 57 | 107/62 | 87 | 135/88 | 30 | 6 |
| 4 | 23 | F | – | – | – | – | – | – | 88 | 136/79 | 120 | 128/81 | 32 | 5 |
| 5 | 37 | F | – | – | – | – | – | – | 70 | 99/55 | 115 | 109/83 | 35 | 6 |
| 6 | 34 | F | – | – | – | – | – | – | 71 | 133/76 | 131 | 127/80 | 52 | 9 |
| 7 | 23 | M | 52 | 118/57 | 100 | 110/82 | 40 | 7 | 51 | 121/65 | 90 | 116/75 | 39 | 5 |
| 8 | 45 | F | – | – | – | – | – | – | 78 | 138/72 | 110 | 155/86 | 32 | 4 |
| 9 | 27 | F | 60 | 117/63 | 108 | 107/81 | 48 | 4 | – | – | – | – | – | – |
| 10 | 44 | F | 72 | 88/54 | 110 | 90/62 | 38 | 5 | – | – | – | – | – | – |
| 11 | 17 | F | 79 | 91/63 | 124 | 96/63 | 45 | 11 | – | – | – | – | – | – |
| 12 | 43 | F | 60 | Awaited | 94 | Awaited | 34 | 9 | – | – | – | – | – | – |
| 13 | 48 | F | 64 | 123/80 | 101 | 116/93 | 37 | 9 | – | – | – | – | – | – |
| 14 | 31 | F | 67 | 106/76 | 100 | 100/85 | 33 | 10 | – | – | – | – | – | – |

**Key:** BP = blood pressure; F = female; HR = heart rate; M = male

Data are shown for their resting, supine heart rate (HR), their standing HR and the change in HR on standing (delta HR). No orthostatic hypotension was observed. The number of months between the onset of the COVID-19 infection and the test is also detailed.

### Other cardiovascular investigations

Seven patients underwent echocardiography, all showing no significant signs of structural heart disease. There was one case of mild-to-moderate tricuspid regurgitation with borderline pulmonary hypertension, but further assessment revealed normal ventilatory equivalents on CPET, demonstrating no V/Q mismatch. Three patients underwent cardiac magnetic resonance imaging (MRI) with no abnormalities found. Eight patients underwent 24-hour Holter monitoring with no significant arrhythmias documented.

Seven patients underwent CPET and pulmonary function tests. Five patients had an exercise tolerance within the normal range and two were deconditioned. All but one had normal lung function tests. Four patients developed signs of dysfunctional breathing.

## Discussion

This series represents a group of patients referred with symptoms and clinical signs of PoTS following recent COVID-19 infection. None of the patients included in this series had previous symptoms consistent with PoTS and, therefore, it is likely that these PoTS diagnoses represent the induction of PoTS by the COVID-19 infection rather than representing an exacerbation of a pre-existing condition.

It is well-recognised in the literature that PoTS can be precipitated by many different clinical events, perhaps most commonly a viral illness, with some suggestion, therefore, of an autoimmune driver.[6] It is unsurprising, therefore, that COVID-19 could precipitate a PoTS pattern in susceptible individuals. The difference, clearly in this situation, is of a novel infection affecting a very large number of patients over a very short period of time, potentially inducing a large number of cases, which has meant that a much larger number of patients with this constellation of symptoms are presenting at the same time.

A potential alternative explanation might be that COVID-19 has a particular propensity to induce PoTS; it is known to produce neurological effects and may be, therefore, more likely to induce this pattern, perhaps through a neuro-pathological mechanism.

The patients described in this series of post-COVID-19 PoTS are majority female and of a premenopausal age, which is very common in other non-COVID-19 PoTS series.[9] The explanation for this remains uncertain but autoimmune drivers may be relevant. The patients in this series present with classical cardiovascular symptoms, although general investigations have not highlighted structural cardiovascular or arrhythmic problems, as is the case in non-COVID-19 PoTS patients whose clinical signs also represent dysregulation in the cardiovascular system with postural tachycardia.

Dysfunctional breathing was seen in many of these patients in our series. This term describes a chronic change in breathing

pattern, usually faster and more shallow but also relating to far greater variance in the volume and rate instead of a predictable pattern of change during exercise; this leads to breathlessness and other symptoms in the absence of another significant disease. We have previously published our findings of breathing dysregulation in PoTS patients when assessed with CPET.[25] This inefficient breathing pattern can cause exercise limitation, and therapy can provide symptomatic benefit.[26] Interestingly, there are data suggesting that dysfunctional breathing is also notable in COVID-19 patients.[27] Half of the patients described in this series tested by CPET also showed breathing dysregulation.

It is well-recognised in the literature that a diagnosis of PoTS is often overlooked because it does not associate with structural or arrhythmic heart disease. Patients can spend many years searching for a diagnosis, can be given multiple alternative diagnoses during their journey and often see a mean of seven physicians prior to receiving the correct diagnostic label,[11] with obvious economic health service implications.

Recent publications highlight the significant burden of disease associated with PoTS,[11] the significant burden of financial effects produced by the condition,[28] and more recent data suggest that there are therapies that can be used to assist patients' symptoms, including exercise,[29] compression,[30] and ivabradine.[31]

Those expert in the management of PoTS recognise the dysregulatory processes on the basis of clinical history and relatively simple investigation, but it can be easily missed. It might be important for clinics and health systems to recognise that a dysregulatory process may be present in patients affected so that these symptoms are not ignored, ascribed to deconditioning or to a putative psychological or non-specific fatigue process.

In our experience prior to this pandemic, people with PoTS often experience an exacerbation of symptoms during and following a viral infection. We have also seen PoTS patients whose symptoms have deteriorated with COVID-19 infection; however, these patients were not included in this analysis.

NHS England have agreed that awareness of PoTS by clinicians is low and that services for affected patients were inadequate prior to the pandemic.[32] Waiting times for appointments in secondary and tertiary care are already very long (up to one year). The American Autonomic Society anticipates an increase in referrals to these already overwhelmed services, and recommends investment in infrastructure, staffing and testing capacity of autonomic centres and training of clinicians in non-specialist centres.[23] PoTS UK, the national charity that represents patients, is seeing a reduction in specialist services throughout the UK (personal communications) at a time when it is anticipated that there will be a significant increase in affected patients and referrals, and this healthcare deficit needs to be urgently addressed.

### Recommendations

Patients who present with post-COVID-19 syndrome should be asked if they have developed symptoms of PoTS, which include, but are not limited to, light-headedness or faintness (pre-syncope), palpitations, fatigue, breathlessness, chest pain and exercise intolerance. The minimal evaluation should include an active-stand test for 10 minutes with intermittent HR and BP recordings to identify PoTS[14] and orthostatic hypotension.

The clinical need is already here, and it is likely to increase. We recommend investment by NHS England in secondary and tertiary autonomic services and training of healthcare providers in primary and secondary care (including long-COVID-19 clinics) to recognise and manage patients with PoTS.

### Limitations

Testing for COVID-19 was not readily available in the first wave of the pandemic in the UK and, therefore, infections are defined on clinical grounds, as recommended by NICE.[14] This series suffers from a referral bias, as patients with suspected symptoms presented to a physician expert in PoTS for the requisite investigations. The use of the home HR and BP assessment systems is not clinically validated, although this is becoming increasingly common with ambulatory diagnostic tools.

Finally, we have not yet had the opportunity to follow-up the patients in this series to determine whether the usual treatments for PoTS are as effective in this cohort of patients.

## Key messages

- A proportion of post-COVID-19 patients present with autonomic symptoms including chest pain, palpitations, breathlessness and pre-syncope/syncope
- Postural tachycardia syndrome (PoTS) may be a cause for these symptoms and this needs to be investigated with an active-stand test as a minimum
- These patients should be treated as other PoTS patients are, pending developing evidence

## Conclusion

This is the largest case series from the UK, and among the largest in the world, detailing the clinical characteristics of patients affected with PoTS developing after a COVID-19 infection. Clinical characteristics appear identical to other PoTS patients. The potential for PoTS as a cause for long-COVID-19 symptoms will require more detailed and organised epidemiological study, and the recognition of this particular condition as a potential cause of symptoms needs to be recognised in long-COVID-19 clinics so that patient symptoms are investigated and managed appropriately.

Detailed prospective research in long-COVID-19 clinics will be required in the longer term to define the prevalence of autonomic dysregulation in long-COVID-19, and which of the putative therapies used in routine PoTS practice may provide most benefit to this cohort of patients

### Conflicts of interest

None declared.

### Funding

None.

### Study approval

This retrospective study was approved by the Research and Ethics Committee, London Bridge Hospital, with a waiver of individual consent.

## References


**1.** DaCosta J. On irritable heart; a clinical study of functional cardiac disorder and its consequences. *Am J Med Sci* 1871;**61**:17.

**2.** Mackenzie J. The soldier's heart. *BMJ* 1916;**1**:117–19. https://doi.org/10.1136/bmj.1.2873.117

**3.** Schondorf R, Low PA. Idiopathic postural orthostatic tachycardia syndrome: an attenuated form of acute pandysautonomia? *Neurology* 1993;**43**:132–7. https://doi.org/10.1212/WNL.43.1_Part_1.132

**4.** Sheldon RS, Grubb BP II, Olshansky B *et al*. 2015 Heart Rhythm Society Expert Consensus Statement on the diagnosis and treatment of postural tachycardia syndrome, inappropriate sinus tachycardia, and vasovagal syncope. *Heart Rhythm* 2015;**12**:e41–e63. https://doi.org/10.1016/j.hrthm.2015.03.029

**5.** Raj SR, Guzman JC, Harvey P *et al*. Canadian Cardiovascular Society Position Statement on postural orthostatic tachycardia syndrome (POTS) and related disorders of chronic orthostatic intolerance. *Can J Cardiol* 2020;**36**:357–72. https://doi.org/10.1016/j.cjca.2019.12.024

**6.** Vernino S, Stiles LE. Autoimmunity in postural orthostatic tachycardia syndrome: current understanding. *Auton Neurosci* 2018;**215**:78–82. https://doi.org/10.1016/j.autneu.2018.04.005

**7.** Roma M, Marden CL, De Wandele I, Francomano CA, Rowe PC. Postural tachycardia syndrome and other forms of orthostatic intolerance in Ehlers-Danlos syndrome. *Auton Neurosci* 2018;**215**:89–96. https://doi.org/10.1016/j.autneu.2018.02.006

**8.** Billig SCI, Schauermann JC, Rolke R, Katona I, Schulz JB, Maier A. Quantitative sensory testing predicts histological small fiber neuropathy in postural tachycardia syndrome. *Neurol Clin Pract* 2020;**10**:428–34. https://doi.org/10.1212/CPJ.0000000000000770

**9.** Thieben MJ, Sandroni P, Sletten DM *et al*. Postural orthostatic tachycardia syndrome: the Mayo Clinic experience. *Mayo Clin Proc* 2007;**82**:308–13. https://doi.org/10.4065/82.3.308

**10.** Doherty TA, White AA. Postural orthostatic tachycardia syndrome and the potential role of mast cell activation. *Auton Neurosci* 2018;**215**:83–8. https://doi.org/10.1016/j.autneu.2018.05.001

**11.** Shaw BH, Stiles LE, Bourne K *et al*. The face of postural tachycardia syndrome – insights from a large cross-sectional online community-based survey. *J Intern Med* 2019;**286**:438–48. https://doi.org/10.1111/joim.12895

**12.** The Lancet. Facing up to long COVID. *Lancet* 2020;**396**:1861. https://doi.org/10.1016/S0140-6736(20)32662-3

**13.** Gorna R, MacDermott N, Rayner C *et al*. Long COVID guidelines need to reflect lived experience. *Lancet* 2021;**397**:455–7. https://doi.org/10.1016/S0140-6736(20)32705-7

**14.** National Institute for Health and Care Excellence. COVID-19 rapid guideline: managing the long-term effects of COVID-19. London: NICE, 2020. Available from: https://www.nice.org.uk/guidance/ng188

**15.** Eshak N, Abdelnabi M, Ball S *et al*. Dysautonomia: an overlooked neurological manifestation in a critically ill COVID-19 patient. *Am J Med Sci* 2020;**360**:427–9. https://doi.org/10.1016/j.amjms.2020.07.022

**16.** Su XW, Palka SV, Rao RR, Chen FS, Brackney CR, Cambi F. SARS-CoV-2-associated Guillain-Barré syndrome with dysautonomia. *Muscle Nerve* 2020;**62**:E48–E49. https://doi.org/10.1002/mus.26988

**17.** Umapathi T, Poh MQW, Fan BE, Li KFC, George J, Tan JYL. Acute hyperhidrosis and postural tachycardia in a COVID-19 patient. *Clin Auton Res* 2020;**30**:571–3. https://doi.org/10.1007/s10286-020-00733-x

**18.** Kanjwal K, Jamal S, Kichloo A, Grubb B. New-onset postural orthostatic tachycardia syndrome following coronavirus disease 2019 infection. *J Innov Card Rhythm Manag* 2020;**11**:4302–04. https://doi.org/10.19102/icrm.2020.111102

**19.** Miglis MG, Prieto T, Shaik R, Muppidi S, Sinn DI, Jaradeh S. A case report of postural tachycardia syndrome after COVID-19. *Clin Auton Res* 2020;**30**:449–51. https://doi.org/10.1007/s10286-020-00727-9

**20.** Dani M, Dirksen A, Taraborrelli P *et al*. Autonomic dysfunction in "long COVID": rationale, physiology and management strategies. *Clin Med* 2021;**21**:e63–e67. https://doi.org/10.7861/clinmed.2020-0896

**21.** Johansson M, Ståhlberg M, Runold M *et al*. Long-haul post-COVID-19 symptoms presenting as a variant of postural orthostatic tachycardia syndrome: the Swedish experience. *JACC Case Rep* 2021;**3**:573–80. https://doi.org/10.1016/j.jaccas.2021.01.009

**22.** Blitshteyn S, Whitelaw S. Postural orthostatic tachycardia syndrome (POTS) and other autonomic disorders after COVID-19 infection: a case series of 20 patients. *Immunol Res* 2021;**69**:205–11. https://doi.org/10.1007/s12026-021-09185-5

**23.** Raj SR, Arnold AC, Barboi A *et al*. Long-COVID postural tachycardia syndrome: an American Autonomic Society statement. *Clin Auton Res* 2021;**31**:365–8. https://doi.org/10.1007/s10286-021-00798-2

**24.** Malfait F, Francomano C, Byers P *et al*. The 2017 international classification of the Ehlers-Danlos syndromes. *Am J Med Genet Part C Semin Med Genet* 2017;**175**:8–26. https://doi.org/10.1002/ajmg.c.31552

**25.** Loughnan A, Gall N, James S. Observational case series describing features of cardiopulmonary exercise testing in postural tachycardia syndrome (PoTS). *Auton Neurosci* 2021;**231**:102762. https://doi.org/10.1016/j.autneu.2020.102762

**26.** Reilly CC, Floyd SV, Lee K *et al*. Breathlessness and dysfunctional breathing in patients with postural orthostatic tachycardia syndrome (POTS): the impact of a physiotherapy intervention. *Auton Neurosci* 2020;**223**:102601. https://doi.org/10.1016/j.autneu.2019.102601

**27.** Motiejunaite J, Balagny P, Arnoult F *et al*. Hyperventilation: a possible explanation for long-lasting exercise intolerance in mild COVID-19 survivors? *Front Physiol* 2021;**11**:614590. https://doi.org/10.3389/fphys.2020.614590

**28.** Bourne K, Chew D, Stiles L *et al*. Postural tachycardia syndrome is associated with significant employment and economic loss. *J Intern Med* 2021;**290**:203–12. https://doi.org/10.1111/joim.13245

**29.** Fu Q, Levine BD. Exercise and non-pharmacological treatment of POTS. *Auton Neurosci* 2018;**215**:20–7. https://doi.org/10.1016/j.autneu.2018.07.001

**30.** Bourne KM, Sheldon RS, Hall J *et al*. Compression garment reduces orthostatic tachycardia and symptoms in patients with postural orthostatic tachycardia syndrome. *J Am Coll Cardiol* 2021;**77**:285–96. https://doi.org/10.1016/j.jacc.2020.11.040

**31.** Taub PR, Zadourian A, Lo HC, Ormiston CK, Golshan S, Hsu JC. Randomized trial of ivabradine in patients with hyperadrenergic postural orthostatic tachycardia syndrome. *J Am Coll Cardiol* 2021;**77**:861–71. https://doi.org/10.1016/j.jacc.2020.12.029

**32.** Samuel S, King T. Prescribed Specialised Services Advisory Group. Recommendations to ministers. London: PSSAG, 2017. Available from: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/809225/pssag-report-2017.pdf