

that it is not used as commonly as its effectiveness dictates it should be. We studied referrals to our Cardiology Unit from our linked national psychiatric unit to characterise the cardiac status in these individuals and determine outcome.

**Methods** Retrospective review of all adult patients on clozapine referred to our cardiology unit over 9 years. Electrocardiograms and echocardiographic data was collected as was mortality data. Students t-test, Mann-Whitney U and linear regression were used to analyse results.

**Results** 29 patients were seen by cardiology in the outpatient setting, all had a diagnosis of schizophrenia. Mean age 43 ±12 years; 34% female (n=10; table 1). 59 others underwent diagnostic testing but were never referred for review.

Median left ventricular ejection fraction (LVEF) was 52 (IQR44%-55%), median HR 98 (IQR85-114bpm). No significant difference in age at review and LVEF, QRS duration, heart rate or QTc interval. There was no significant relationship between duration on clozapine and LVEF (p=0.77) at review. There was a significant difference in length of time on clozapine and QTc interval (p=0.02; r=0.46; figure 1).

Follow-up time was 4.4 years(IQR 2-6) from initial review; 3 patients died (subarachnoid haemorrhage, aortic aneurysm rupture, suicide). 14 of the surviving 26 (54%) remain on clozapine at most recent follow-up and demonstrated no significant change in the LVEF during this time (p=0.66).

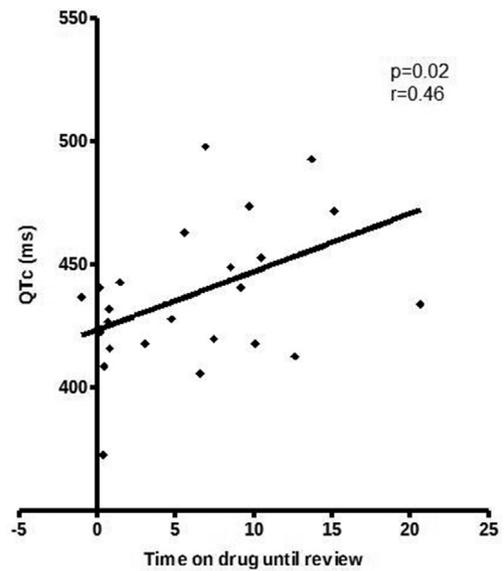
Those with LV impairment tended to be seen earlier than those who developed tachycardia without LV impairment (4.7 vs 7.2 years; p=0.27). Patients in the former group were far more likely to have clozapine discontinued compared to those in the tachycardia group (p=0.03).

**Discussion** Clozapine use is associated with persistent tachycardia, myocarditis and cardiomyopathy; the latter carry significant morbidity and mortality. Despite being very effective, many will have clozapine stopped if there is evidence of

cardiac toxicity. Our experience suggests that the short-medium term outcome in these patients is acceptable with very low cardiac risk and no observable deterioration in LVEF.

Our findings support two distinct modes of cardiac toxicity. The underlying pathophysiology is not yet well defined and is likely to include patient-specific factors. Our data supports the feeling that tachycardia represents a benign process as all our patients had normal LVEF at the time of review.

Prospective studies are required in this complex group; the significance of QTc prolongation with clozapine use remains unclear and further study is warranted. We suggest that these patients should be managed in an interdisciplinary manner with close liaison between cardiologists and psychiatrists.



Abstract 12 Figure 1 QTc interval would appear to prolong in relation to time on Clozapine.

Abstract 12 Table 1 Demographics and p values.

	Cardiomyopathy group (n=11)	Tachycardia group (n=18)	Total group (n=29)	p value
Age, years (%)	41±15	44±11	43±12	0.51
Gender (female)	4 (36%)	5 (28%)	9 (31%)	-/-
Length of time on clozapine at review, median (range) years	4.9 (0.4-13)	7.2 (1.3-10)	6.6 (0.8-10)	0.51
Heart rate Median (range) bpm	83 (76-90)	103 (97-115)	98 (85-114)	0.0006*
QRS duration Median (range) ms	90 (88-98)	90 (85-95)	90 (86-95)	0.66
QTc interval Median (range) ms	431 (416-440)	441 (418-468)	434 (418-458)	0.47
LVEF Median (range)%	45 (37-50)	55 (52-55)	52 (44-55)	<0.001*
Clozapine discontinued	8 (73%)	5 (28%)	13 (45%)	0.027*

13 **KEEPING UP THE BEAT: A QUALITY IMPROVEMENT PROJECT ON HEART FAILURE MONITORING & MANAGEMENT**

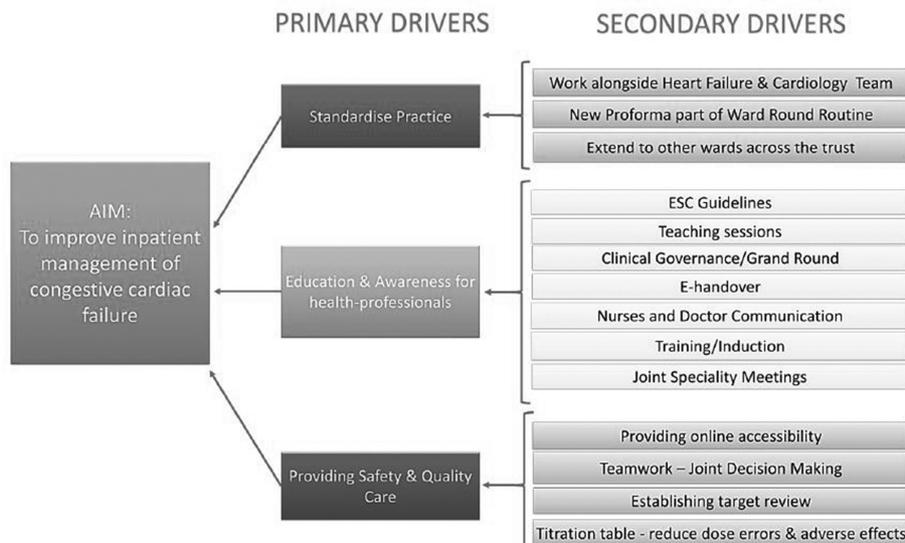
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**Introduction** There have been many cases observed in our cardiology wards and non-cardiology wards where heart failure patients are not having optimum fluid management and up-titrating therapy. One case led a patient to have prolonged hospital stay and outcome was death. Therefore, this project is important to raise awareness.

Currently, we only have a basic daily weight chart, which the nurses record the patients weight every morning. The heart failure medications are separately found on the drug chart, and bloods are found on Cyberlab. Plus the heart

Driver's Diagram:



Abstract 13 Figure 1

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**HEART FAILURE MONITORING & MANAGEMENT PATHWAY**

This proforma needs to be completed on admission to the ward and monitored regularly for patients with heart failure. It should be kept in front of the bedside folder, as it contains a section for recording and monitoring daily weights.

PLEASE SEE OVERLEAF FOR THE HEART FAILURE MANAGEMENT FLOWCHART

PATIENT DETAILS	
First Name	Surname
Date of Birth	Gender
Hospital ID	Ward

SECTION 1: ON WARD ADMISSION	Admission Date:
Patient's Weight on Admission	kg
Patient's Baseline Range for Renal Function (U&Es)	Na <sup>+</sup> :    K <sup>+</sup> : Urea:    Creatinine:

SECTION 2: PROGRESS	
Type & Severity of heart failure for this patient?	%
Does the patient have an abnormal echo and/or LVEF <40%? If yes, have you sent a referral to Cardiology & Heart Failure Specialist Nurse (see overleaf)?	Yes <input type="checkbox"/> No <input type="checkbox"/>

SECTION 3: DAILY WEIGHT CHART			Week Number:
Date	Current Dose & Changes of Heart Failure Medications <small>(Please see overleaf for flowchart)</small>	U&Es & Vitals within baseline? <small>(If worsening, please seek advice)</small>	Today's Weight <small>(Please weigh at 8am)</small>
		Na <sup>+</sup> : K <sup>+</sup> : Urea: Creatinine:	Amount of Weight Gain/Loss <small>(If &gt;2kg gain/loss in 5 days please inform doctor immediately)</small>
Day 1: / /			
Day 2: / /			
Day 3: / /			
Day 4: / /			
Day 5: / /			
Day 6: / /			
Day 7: / /			

Please note: It is essential this chart is reviewed daily for effective HF treatment - see overleaf for guidelines & dose titration

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University Hospitals NHS Trust

**HEART FAILURE MANAGEMENT FLOWCHART (ESC & NICE Guidelines)**

BELOW THIS SEE CARDIOLOGY & HF NURSE INPUT

HEART FAILURE DOSE TITRATION TABLE					
Medication	Routes	Starting Dose	Increments	Target Dose	Check common potential effects
<b>Diuretic</b> • Furosemide <small>(There are maximum doses. Reagents must be added for reduced patients to safely achieve doses)</small>	Oral IV	40mg Once or Twice Daily	40mg → 80mg → 120mg Twice Daily	120mg in total over 24 hours <small>(Consider titration)</small>	AKI Hypotension Hypokalaemia
<b>ACE-inhibitor</b> • Ramipril	Oral	1.25mg Once Daily	2.5mg → 5mg → 7.5mg → 10mg	10mg Once Daily	AKI Hypotension Hypokalaemia
<b>Beta-Blocker</b> • Bisoprolol <small>(Do not start or increase dose if significant acute pulmonary congestion)</small>	Oral	1.25mg Once Daily	2.5mg → 5mg → 7.5mg → 10mg	10mg Once Daily	Hypotension Bradycardia

If weight loss not achieved despite titrating furosemide, ACE or persistent hypotension, please call Cardiology for advice.

For further advice, please send a blue referral form to Cardiology Fax 3142 QH or 8492 KGH  
Also call Cardiology Registrar & Heart Failure Nurse (Dept 8492 QH/8393 KGH)

Abstract 13 Figure 2

failure guidelines are found on the European Society of Cardiology website. This shows that it takes a lot of effort and can cause time-consuming problems when accumulating this information together for heart failure monitoring; especially during busy ward round.

For effective heart failure monitoring, a new proforma was devised, which consists of daily weight, medications, U and Es

with electrolytes, patient heart failure details and admission weight. The guidelines (flow chart) are found at the back. This will be filled in by both nurses and doctors. This effectively improves titrating heart failure medications for better weight loss secondary to fluid overload.

**Aims and Outcomes** To improve inpatient management of Congestive Cardiac Failure. By July 2016 at Queen’s Hospital and King George Hospital, we should obtain 50% in:

1. Optimising fluid management (weight loss)
2. Up-titrating therapy to maximum prognostic benefit

**Methods** 2 PDSA (Plan-Do-Study-Act) cycles were completed trust-wide project at BHR Hospitals “Queens” and KGH Coronary Care Units (Figure 1):

In PDSA Cycle 1, a two-week based proforma was trialled and compatibility was checked with daily ward round and CCF management. In PDSA Cycle 2, it was changed to a 7 days based proforma with an additional aspect on renal function (figure 2 see below). The new proforma was used, analysed and edited for each PDSA cycle. Patient parameters were derived and confirmed from Solus and Cyberlab.

**Results** There was a significant improvement from the new proforma in heart failure monitoring and management. The results are shown in table 1:

**Abstract 13 Table 1** PDSA cycle results

	PDSA Cycle 1	PDSA Cycle 2
Number of Patients	n=9	n=10
Mean Age	73.9	70.2
Average Hospital Stay	18.5 days	17.5 days
Optimum Fluid Management (Percentage of Patient's losing weight by Day 7)	60%	100%
Developed Worsening AKI	22%	30%
Developed significant electrolyte imbalance	22%	0%
Up-titrating therapy	22%	100%
Quality of documentation: Daily weights recorded	89%	100%
Quality of documentation: U&Es recorded	55%	100%

There were limitations with unwell patients, especially those who developed AKI secondary to heart failure treatment. With reducing hospital stay, helps reduce costs to the NHS.

**Conclusions** Heart Failure monitoring and management is important to help reduce morbidity and mortality. There was

success from QI project by using the new proforma by improving patient care and co-ordinated care. It will be implemented in the trust on other medical wards like Acute Medicine.

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**ST2 LEVELS ARE ELEVATED IN PATIENTS WITH ADVANCED HEART FAILURE BUT ARE NOT CONSISTENTLY ASSOCIATED WITH OTHER MARKERS OF ADVERSE PROGNOSIS**

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**Introduction** Heart transplantation and mechanical circulatory support (MCS) improve survival in selected patients with advanced heart failure and an adverse prognosis. Soluble ST2 is a protein belonging to the interleukin-1 receptor family. ST2 is released in response to cardiomyocyte stress and thought to be a marker of adverse prognosis. We examined the association between ST2 levels and currently accepted markers of adverse prognosis in patients with advanced heart failure.

**Methods** We included 20 consecutive outpatients who were assessed for heart transplantation at Papworth Hospital over ten weeks. All patients underwent echocardiography, six minute walk testing, cardiopulmonary exercise testing and right heart catheterisation, in addition to blood tests including serum ST2 measurement using a commercial assay. Prognosis was estimated using the Seattle Heart Failure Model (SHFM) and the Meta-Analysis Global Group in Chronic Heart Failure (MAGGIC) scores. A multi-disciplinary team decided whether listing for heart transplantation was indicated. We examined the association between ST2 levels and other markers of adverse prognosis.

**Results** Ten patients were too well to be listed for heart transplantation (group A) and ten patients were sufficiently unwell to be listed for heart transplantation (group B). There was no difference in age, gender or body mass index. Key prognostic variables are presented in table 1. Compared with patients in group A, patients in group B had higher ST2 levels, lower peak VO<sub>2</sub>, shorter six minute walk distance and higher SHFM

**Abstract 14 Table 1**

	Total	Group A (well, n=10)	Group B (unwell, n=10)	p value
LVEF (%)	17.5 (12.5-52.5)	17.5 (13-35)	20 (12.5-52.5)	0.1700
Serum Creatinine (umol/L)	120 (53-653)	110 (53-175)	140.5 (89-653)	0.6560
NT-proBNP (pg/ml)	2499 (474-34491)	2126.5 (702-4594)	5043.5 (474-34491)	0.6560
ST2 (ng/ml)	54.17±42.91	30.94±22.19	77.19±46.86	0.0110*
Peak VO <sub>2</sub> (ml/kg/min)	15.1±4.7	18.3±3.6	11.8±3.1	0.0004*
6MWT (m)	346.1±103.1	400.2±102.8	292±73.2	0.0143*
Cardiac Index (L/min/m <sup>2</sup> )	1.97±0.37	2.01±0.31	1.94±0.44	0.7053
MAGGIC 1-year mortality (%)	14.42±5.73	12.80±5.34	16.04±5.91	0.2130
SHFM 1-year mortality (%)	8.30±5.28	4.90±2.85	11.70±5.01	0.0015*