

The Maudsley®

Prescribing
Guidelines
for Mental Health
Conditions in
Physical Illness

Siobhan Gee
David Taylor

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**The Maudsley® Prescribing
Guidelines for Mental
Health Conditions in
Physical Illness**

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Preface

More than 40% of people diagnosed with a serious mental illness have at least one concurrent physical illness. Prescribing medicines to treat the psychiatric condition can be complex in these circumstances. The prescriber will need to consider both the potential for prescribed medication to adversely affect any physical health condition and the possibility of altered response because of the presence of physical illness. Choice of medication in the physically unwell patient can be further complicated by a host of other factors. Some patients, for example, cannot take medicines orally, some are at the end of life, some may be on renal replacement therapy, and so on.

The challenges of managing mental health medicines in patients with complex physical comorbidity are faced daily in liaison psychiatry (also known as consultative psychiatry or consultation-liaison psychiatry). As the global population ages and the prevalence of psychiatric conditions continues to increase, these challenges are also increasingly faced by psychiatrists working in all specialisms, both acute and chronic, inpatient and outpatient. This book aims to help the clinician navigate these prescribing scenarios and to be of use not only to psychiatrists but general physicians too.

The importance of treating psychiatric conditions in people with physical illness cannot be overstated. There is no doubt that for every physical illness, the presence of mental ill health adversely affects both morbidity and mortality. We hope this book will not only enable the choice of safe and effective medication in physically unwell patients, but also engender confident prescribing for mental illness, even in the face of severe physical illness.

*Siobhan Gee
David M. Taylor
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Abbreviations

ACE	angiotensin-converting enzyme
ADHD	attention deficit hyperactivity disorder
APD	automated peritoneal dialysis
ARF	acute respiratory failure
ATP	adenosine triphosphate
BD	bis die (twice a day)
BMI	body mass index
BNP	B-type natriuretic peptide
BP	blood pressure
cAMP	cyclic adenosine monophosphate
CAPD	continuous ambulatory peritoneal dialysis
CCV	central compartment volume
CHD	coronary heart disease
CNS	central nervous system
COPD	chronic obstructive pulmonary disorder
CRP	C-reactive protein
CYP	cytochrome P
DSM-5	<i>Diagnostic and Statistical Manual 5</i>
ECG	electrocardiogram
ECHO	echocardiogram
ECT	electroconvulsive therapy
ENRICHD	enhancing recovery in coronary heart disease
EPSE	extrapyramidal side effect
FBC	full blood count
FDA	Food and Drug Administration
GABA	gamma-aminobutyric acid
GI	gastrointestinal
HDAC9	histone deacetylase 9
IBD	inflammatory bowel disease
ICS	inhaled corticosteroid
ICU	intensive care unit
IL	interleukin
IM	intramuscular

INR	international normalised ratio
IV	intravenous
LABA	long-acting beta-2 agonist
LAMA	long-acting muscarinic antagonist
MAO	monoamine oxidase
MAOI	monoamine oxidase inhibitor
MDMA	3,4-methylenedioxymethamphetamine
MI	myocardial infarction
MIND-IT	myocardial infarction and depression intervention trial
MOOD-HF	mood and mortality in depressed heart failure patients
NaCl	sodium chloride
NaSSA	noradrenaline and specific serotonergic antidepressant
NG	nasogastric
NHS	National Health Service
NICE	National Institute for Health and Care Excellence
NJ	nasojejunal
NMDA	N-methyl-d-aspartate
NNT	number needed to treat
NOAC	non-vitamin K antagonist oral anticoagulant
NRT	nicotine replacement therapy
NSAID	non-steroidal anti-inflammatory drug
NT-pro BNP	N-terminal pro B-type natriuretic peptide
OD	omni die (once a day)
PEG	percutaneous endoscopic gastrostomy
QTc	QT interval adjusted for heart rate
RASS	Richmond Agitation Sedation Scale
RCT	randomised controlled trial
SABA	short-acting beta-2 agonist
SADHART-CHF	sertraline against depression and heart disease in chronic heart failure
SAMA	short-acting muscarinic antagonist
SC	subcutaneous
SIADH	syndrome of inappropriate antidiuretic hormone secretion
SL	sublingual
SMI	serious mental illness
SNRI	serotonin and noradrenaline reuptake inhibitor
SSRI	selective serotonin reuptake inhibitor
STAR*D	Sequenced Treatment Alternatives to Relieve Depression programme
TCA	tricyclic antidepressant
TDS	ter die sumendum (three times a day)
TNF	tumour necrosis factor
UC	ulcerative colitis
UK	United Kingdom
USA	United States of America
Vd	volume of distribution

Chapter 1

Cardiac Disease

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INTRODUCTION

The influence of psychiatric symptoms on the functioning of the heart was first described in 1628 by Sir William Harvey, the English physician who discovered the cardiac circulatory system. Since then, numerous studies have proven him correct, finding mental illness to be a significant predictor of cardiac mortality across the spectrum of cardiac diseases. Treatment of the mental illness is therefore vital not only for relief of psychiatric symptoms, but also for optimal treatment of the cardiac disease.

Few data are available to compare efficacy of drugs for mental illness within individual physical illnesses, such as heart failure or coronary heart disease, and even fewer for patients who have more than one concurrent physical illness. When extrapolating data from studies in patients without cardiac disease, it should be noted that populations studied in these trials are different (e.g. cardiac disease patients tend to be older than populations with general depression). Perhaps more importantly, the biological symptoms of mental illnesses that are measured by standard rating scales may not appear to improve on addition of psychiatric drugs because of the overlap of these physical symptoms with ongoing symptoms of the heart disease (e.g. fatigue, insomnia). Failure to demonstrate response to a drug on a rating scale is of little importance in clinical practice (symptoms are the target) but is relevant if trial data are used to make decisions about drug choice. Conversely, it is also possible that illnesses such as depression or anxiety – specifically in the context of heart disease – are biologically distinct from general depression. Consequently, drug treatments may not be effective for this reason.

HEART FAILURE

Depression and anxiety in heart failure

As many as one in five patients with heart failure suffer from depression, more than doubling the mortality risk and trebling the risk of non-compliance with medical treatment recommendations¹. Clinically significant symptoms of anxiety are also commonly reported in patients with heart failure (30%)². Symptoms of heart failure and those of anxiety may overlap, increasing the apparent prevalence. A clear link between anxiety and mortality in heart failure has not been fully established, but an increased risk is evident for patients with other cardiac disorders such as coronary artery disease³. Of course, depression and anxiety may co-exist, and together they increase the risk of both cardiac rehospitalisation and mortality in patients with heart failure⁴.

There are several factors that may give rise to the link between depression and anxiety, and poor cardiac outcomes in heart failure. These include biological changes that occur in association with the mental health condition (inflammation, autonomic dysfunction, alterations in the ability of platelets to aggregate, and endothelial dysfunction²). Adherence to medicines for the treatment of the heart failure or comorbidities may be affected, as may maintenance of a healthy lifestyle (smoking cessation, diet, exercise).

There are few data relating to the efficacy of pharmacotherapy in depression specifically with comorbid heart failure. The most well-known studies are SADHART-CHF (Sertraline Against Depression and Heart Disease in Chronic Heart Failure) and MOOD-HF (Mood and Mortality in Depressed Heart Failure patients). SADHART-CHF demonstrated safety (although not efficacy) of sertraline⁵, and MOOD-HF⁶ the same for escitalopram. There are no randomised trials examining the pharmacological treatment of anxiety in heart failure patients.

Psychosis and bipolar disorder in heart failure

Patients with serious mental illness (SMI – schizophrenia, bipolar disorder, and severe depression) have a reduced life expectancy compared with the non-SMI population⁷. Cardiovascular disease is a significant contributor to this⁸. Lifestyle interventions are as important in this population as they are in the general population⁹. Antipsychotics and mood stabilisers (lithium and mood-stabilising antiepileptics) commonly cause weight gain, hyperglycaemia, and hyperlipidaemia. Despite this, patients who take them have an overall reduction in cardiac (and all-cause) mortality¹⁰. This may be a direct beneficial effect of reduction in psychiatric symptoms, improved adherence to healthy lifestyle choices, and/or better compliance with physical health treatments. Heart failure outcomes in patients who have SMI are therefore strongly linked to the outcome of their mental illness, making effective treatment of the psychiatric symptoms a priority. This is an important factor when weighing the risks and benefits of individual psychiatric medication choice. Medication that is perceived as safer in heart failure but less effective for the mental disorder may not actually be the optimal choice for overall cardiac outcomes¹¹.

Antidepressants

In general, SSRIs are considered first-line antidepressants, and this is also true for patients with heart failure. Of the SSRIs, sertraline^{12,13} is generally well tolerated and efficacious in non-heart failure populations¹⁴. It has few drug interactions, less propensity than citalopram to prolong the QTc, and has been studied in patients with heart failure (it is safe, but efficacy is unproven)⁵. Escitalopram has also demonstrated safety (although not efficacy) in patients with heart failure⁶, but is more often associated with QT prolongation¹⁵ than sertraline (although this association is disputed¹⁶).

Other options carry some cautions. Mirtazapine is consistently shown to promote appetite, probably due to α_2 receptor blockade and affinity for H₁, D₁, and D₂ receptors¹⁷, and is therefore less desirable in conditions such as heart failure where excess weight can be detrimental to clinical outcomes. Citalopram may be more likely than other antidepressants to prolong the QT interval and is not recommended for use in uncompensated heart failure¹⁸. SNRIs (venlafaxine and duloxetine) are associated with dose-dependent increases in blood pressure¹⁹ (see section on hypertension), and venlafaxine and fluoxetine may also cause prolonged QT, particularly in combination with ivabradine²⁰. Tricyclic antidepressants (TCAs) are generally avoided in patients with cardiac disease due to their effects on cardiac contractility, their proarrhythmic effects (due to blockade of cardiac sodium and potassium channels), and their potential to worsen ischaemic heart disease.

Hyponatraemia is a risk with all antidepressants in the first month of treatment. Depending on the patient's risk profile, the diuretic dose may need to be adjusted. If hyponatraemia persists, the dose of sacubitril may need to be reduced or stopped. Mirtazapine and agomelatine may be less commonly associated with hyponatraemia (but are not completely without risk). Close monitoring of sodium levels is recommended, especially in the first few weeks of treatment²¹ and if patients have additional risk factors for developing hyponatraemia.

Recommendation: sertraline.