The following information resources have been selected by the National Health Library and Knowledge Service Evidence Virtual Team in response to your question. The resources are listed in our estimated order of relevance to practicing healthcare professionals confronted with this scenario in an Irish context. In respect of the evolving global situation and rapidly changing evidence base, it is advised to use hyperlinked sources in this document to ensure that the information you are disseminating to the public or applying in clinical practice is the most current, valid and accurate. For further information on the methodology used in the compilation of this document—including a complete list of sources consulted—please see our National Health Library and Knowledge Service Summary of Evidence Protocol.

YOUR QUESTION

What is the evidence on additional risk for people >65 with cardiovascular disease?

IN A NUTSHELL

COVID-19 is an infectious disease caused by SARS-CoV-2 that has significant implications for the cardiovascular care of patients[12]. First, those with COVID-19 and pre-existing cardiovascular disease have an increased risk of severe disease and death. Second, infection has been associated with multiple direct and indirect cardiovascular complications including acute myocardial injury, myocarditis, arrhythmias and venous thromboembolism. Third, therapies under investigation for COVID-19 may have cardiovascular side effects. Fourth, the response to COVID-19 can compromise the rapid triage of non-COVID-19 patients with cardiovascular conditions. Finally, the provision of cardiovascular care may place health care workers in a position of vulnerability as they become hosts or vectors of virus transmission.
COVID-19 may preferentially infect individuals with cardiovascular conditions; is more severe in subjects with cardiovascular comorbidities; may directly or indirectly affect the heart; and may interact with cardiovascular medications[6]. In addition, the widespread effects of the pandemic on the global healthcare system affects the routine and emergency cardiac care for patients who are, may be or are not infected with COVID-19.
Potential mechanisms of cardiovascular injury have been identified and include direct myocardial injury from hemodynamic derangement or hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction or thrombosis due to hypercoagulability, or systemic inflammation [cytokine storm], which may also destabilize coronary artery plaques[10]. Pneumonia and influenza infections have been associated with six-fold increased risk of acute myocardial infarction (MI).

Until more data with larger numbers of patients are available, it seems reasonable to consider all patients with history of CVD, hypertension or diabetes at higher risk. The risk may be highest for patients with these risk factors, older age, known history of heart failure or clinically significant valvular disease[10].

During the period of the pandemic, older people living with frailty and long-term conditions will continue to experience episodes of ill-health, falls or other unforeseen events, and health professionals will need to continue to respond to provide high-quality, person-centred care[4]. During national emergencies and pandemics, health care services and supply chains may be disrupted. Health care resources may be limited and a focus on controlling a pandemic will deflect focus from other areas. These may include routine care of those with LTCs such as asthma, diabetes and hypertension[11].

IRISH AND INTERNATIONAL GUIDANCE

What does the World Health Organization say?

World Health Organization (2020) Q and A: Older People and COVID-19[1]

Older people and people of all ages with pre-existing medical conditions such as diabetes, high blood pressure, heart disease, lung disease or cancer appear to develop serious illness more often than others.

What does the European Centre for Disease Prevention and Control say?

European Centre for Disease Prevention and Control (23 April 2020) Rapid Risk Assessment. Coronavirus disease 2019 (COVID-19) in the EU/EEA and the UK. Ninth update[2]

What is the risk of severe disease associated with SARS-CoV-2 infection in populations with defined factors associated with elevated risk for COVID-19?

The analysis of data shows that persons over 65 years of age and/or people with underlying health conditions, are at increased risk of severe illness and death compared with younger individuals. These vulnerable populations account for the majority of severe disease and fatalities to date.

In summary, the impact of COVID-19 is assessed as very high for elderly and individuals with defined risk factors.

What do the Centers for Disease Control and Prevention (United States) say?

Centers for Disease Control and Prevention (2020) Groups at Higher Risk for Severe Illness[3]

Based on currently available information and clinical expertise, older adults and people of any age who have serious underlying medical conditions might be at higher risk for severe illness from COVID-19.

Conditions and Other Risk Factors

- asthma
- chronic lung disease
- diabetes
- serious heart conditions
- chronic kidney disease being treated with dialysis
- severe obesity
- people aged 65 years and older
- people in nursing homes or long-term care facilities
- immunocompromised
- liver disease

British Geriatrics Society (2020) BGS statement on the COVID-19 pandemic[4]

During the period of this pandemic, older people living with frailty and long-term conditions will continue to experience episodes of ill-health, falls or other unforeseen events and health professionals will need to continue to respond to provide high-quality, person-centred care. While COVID-19 will be the main concern for the health system as a whole, much of the care provided during this time may be routine care. As far as possible, efforts will need to be made to provide such care at home or in community settings, keeping older people out of hospital unless strictly necessary.

Irish Heart Foundation (2020) FAQs. The Coronavirus, Heart Disease and Stroke[5]

Question: The HSE has issued a list of people who are said to be medically vulnerable and these people must cocoon. These groups include: anyone with a heart transplant; pregnant women with heart disease; people with

severe heart failure or severe vascular disease. Why are these people considered to be medically vulnerable?
Answer: Patients in the group above are considered to be medically vulnerable because they are immunocompromised. Being immunocompromised means that your immune system is weaker than those without such conditions and you are therefore less likely to be able to fight off infections including those caused by the coronavirus.

Gupta et al (2020) Current perspectives on Coronavirus 2019 (COVID- 19) and cardiovascular disease: a white paper by the JAHA editors[6]
COVID- 19 may preferentially infect individuals with cardiovascular conditions; is more severe in subjects with cardiovascular comorbidities; may directly or indirectly affect the heart; and may interact with cardiovascular medications. In addition, the widespread effects of the pandemic on the global healthcare system affects routine and emergency cardiac care for patients who are, may be or are not infected with COVID-19.

American Heart Association (2020) Coronavirus precautions for patients and others facing higher risks[7]
Who is at risk of infection or complications?
It continues to seem that older people with coronary heart disease or high blood pressure are more likely to develop more severe symptoms. Stroke survivors and those with heart disease, including high blood pressure and congenital heart defects, may face an increased risk for complications if they become infected with the COVID-19 virus.

POINT-OF-CARE TOOLS

What does BMJ Best Practice say?

BMJ Best Practice (2020) Coronavirus disease 2019 (COVID-19)[8]
Cardiovascular Complications
COVID-19 is associated with a high inflammatory burden that can result in cardiovascular complications with a variety of clinical presentations. Inflammation in the vascular system can result in diffuse microangiopathy with thrombosis. Inflammation in the myocardium can result in myocarditis,

heart failure, arrhythmias, acute coronary syndrome, rapid deterioration and sudden death.
Prevalence of cardiac disease is high among patients who are severely or critically ill, and these patients usually require intensive care and have a poor prognosis and higher rate of in-hospital mortality. Patients with cardiac injury were more likely to require non-invasive or invasive ventilation compared with patients without cardiac injury. Patients with cardiac disease also have a higher risk of thromboembolic events and septic shock compared with patients without a history of cardiac disease. Patients with underlying cardiovascular disease but without myocardial injury have a relatively favourable prognosis.
Predictors for myocardial injury include older age, presence of cardiovascular-related comorbidities and elevated C-reactive protein. Elevated myocardial markers predict risk for in-hospital mortality.

BMJ Best Practice (2020) Management of coexisting conditions in the context of COVID-19[9]
Use of ACE inhibitors and angiotensin-II receptor antagonists
People with cardiovascular disease are at higher risk of severe complications and death from COVID-19; however, there is currently no evidence that use of ACE inhibitors or angiotensin-II receptor antagonists should be discontinued in these patients. Any change in medication should be based on individual patient risk assessment.

What does UpToDate say?

UpToDate (2020) Coronavirus disease 2019 (COVID-19): Myocardial infarction and other coronary artery disease issues[10]
Patients with cardiovascular disease, hypertension, obesity and diabetes are at increased risk of a poor prognosis. In addition, patients with myocardial injury, irrespective of cause, have a poorer prognosis.

IMPACT ON THE CARDIOVASCULAR SYSTEM
It is likely that COVID-19 directly and indirectly affects the cardiovascular system and the heart in particular. Potential mechanisms of cardiovascular injury have been identified and include direct myocardial injury from hemodynamic derangement or hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction or thrombosis due to hypercoagulability, or systemic inflammation [cytokine storm], which may

also destabilize coronary artery plaques. Pneumonia and influenza infections have been associated with six-fold increased risk of acute myocardial infarction. Patients with severe COVID-19 disease, such as those with high fever or hypoxia due to lung disease, may need a significant increase in cardiac output. Type II myocardial ischemia, therefore, may result in patients with obstructive CAD.
The clinical impact of SARS-CoV-2 infection will, across a population, be greater in those with prior disease and increasing age. In one study, patients with prior cardiovascular disease made up 22.7 percent of all fatal cases, and the case fatality rate was 10.5 percent.

ASSOCIATION BETWEEN BASELINE CVD AND COVID-19

There is substantial evidence of an association between cardiovascular disease risk factors of hypertension, diabetes, prior CAD, and the risk and severity of COVID-19 infection.
Until more data with larger numbers of patients are available, it seems reasonable to consider all patients with history of CVD, hypertension or diabetes at higher risk. We consider this risk likely to be highest for patients with these risk factors, older age, known history of heart failure, or clinically significant valvular disease.
Furthermore, given the association with more severe disease and increased risk for acute myocardial injury, we advise early clinical evaluation for any suspect symptoms.

INTERNATIONAL LITERATURE

What does the international literature say?

Oxford University Centre for Evidence-Based Medicine (2020) Supporting people with long-term conditions [LTCs] during national emergencies[11]
Disruption of care, diversion of healthcare resources and interruptions to medical supplies can all impact patients with long-term conditions during national emergencies. Some LTCs may be further exacerbated by increased stress and changes in diet and activity patterns. The data does not rule out any LTCs as not being at risk of neglect, but particularly highlights cardiovascular disease, diabetes, older people and people in deprived areas as being at increased risk.

During national emergencies and pandemics, health care services and supply chains may be disrupted. Health care resources may be limited and a focus on controlling a pandemic will deflect focus from other areas. These may include routine care of those with LTCs such as asthma, diabetes and hypertension.
Many common LTCs may put people at higher risk of COVID-19 severity and complications. Knowledge of other infections suggests that it is not just presence of these conditions but instead how well they are controlled that will contribute to different outcomes from infection: eg better glucose control is likely to be a protective factor in people living with diabetes. Stress can exacerbate some LTCs as can inactivity, changes to diet and issues with accessing healthcare. All of these factors may arise as supply chains are disrupted, food stock is temporarily depleted and people living with LTCs are asked to self-isolate.

Driggin et al (2020) Cardiovascular Considerations for Patients, Health Care Workers, and Health Systems During the COVID-19 Pandemic[12]
Patients with pre-existing CVD appear to have worse outcomes with COVID-19. CV complications include biomarker elevations, myocarditis, heart failure and venous thromboembolism which may be exacerbated by delays in care. Therapies under investigation for COVID-19 may have significant drug-drug interactions with CV medications.
COVID-19 is an infectious disease caused by SARS-CoV-2 that has significant implications for the cardiovascular care of patients. First, those with COVID-19 and pre-existing cardiovascular disease have an increased risk of severe disease and death. Second, infection has been associated with multiple direct and indirect cardiovascular complications including acute myocardial injury, myocarditis, arrhythmias and venous thromboembolism. Third, therapies under investigation for COVID-19 may have cardiovascular side effects. Fourth, the response to COVID-19 can compromise the rapid triage of non-COVID-19 patients with cardiovascular conditions. Finally, the provision of cardiovascular care may place health care workers in a position of vulnerability as they become hosts or vectors of virus transmission.

Gross et al (2020) SARS-CoV-2 receptor ACE2-dependent implications on the cardiovascular system: From basic science to clinical implications[13]
COVID-19 requires the collaboration of nearly 200 countries to curb the spread of SARS-CoV-2 while gaining time to explore and improve treatment

options especially for cardiovascular disease and immunocompromised patients who appear to be at high risk to die from cardiopulmonary failure. Currently unanswered questions are why elderly people, particularly those with pre-existing comorbidities, seem to exhibit higher mortality rates after SARS-CoV-2 infection; and whether intensive care becomes indispensable for these patients to prevent multi-organ failure and sudden death. To address these challenges, we here summarize the molecular insights into viral infection mechanisms and implications for cardiovascular disease. The fact that SARS-CoV-2 hijacks ACE2 for cell-entry has spurred controversial discussions on the role of ACE2 in COVID-19 patients. In this review, we highlight the state-of-the-art knowledge on SARS-CoV-2-dependent mechanisms and the potential interaction with ACE2 expression and cell surface localization.

We aim to provide a list of potential treatment options and a better understanding of why CVD is a high-risk factor for COVID-19 susceptibility and further discuss the acute as well as long-term cardiac consequences.

Interestingly, the drastically increased mortality rate in patients with pre-existing or new cardiac injuries is associated with elevated serum levels of troponin T (TnT), a biomarker for cardiac damage. Indeed, a previous study[14] points out that CVD patients without significantly raised TnT serum levels only represent a slightly increased mortality compared to patients without CVD. Therefore, in the case of expected cardiac damage due to distinct comorbidities or severe disease progression, TnT levels should always be strictly monitored, but also critically interpreted since some medications and interventions themselves can cause an upregulation of TnT.

Guo et al (2020) Cardiovascular Implications of Fatal Outcomes of Patients with Coronavirus Disease 2019 (COVID-19)[14]

Information regarding the impact of cardiovascular complication on fatal outcome is scarce. Aim: to evaluate the association of underlying cardiovascular disease and myocardial injury with fatal outcomes in patients with COVID-19. Among 187 patients with confirmed COVID-19, 144 patients (77%) were discharged and 43 patients (23%) died. The mean (SD) age was 58.50 (14.66) years. Overall, 66 (35.3%) had underlying CVD including hypertension, coronary heart disease and cardiomyopathy, and 52 (27.8%) exhibited myocardial injury as indicated by elevated TnT levels. The mortality during hospitalization was 7.62% (8 of 105) for patients without underlying CVD and normal TnT levels, 13.33% (4 of 30) for those with underlying CVD and normal TnT levels, 37.50% (6 of 16) for those without underlying CVD but

elevated TnT levels, and 69.44% (25 of 36) for those with underlying CVD and elevated TnTs. Patients with underlying CVD were more likely to exhibit elevation of TnT levels compared with the patients without CVD (36 [54.5%] vs 16 [13.2%]). Myocardial injury is significantly associated with fatal outcome of COVID-19, while the prognosis of patients with underlying CVD but without myocardial injury is relatively favorable. Myocardial injury is associated with cardiac dysfunction and arrhythmias. Inflammation may be a potential mechanism for myocardial injury. Aggressive treatment may be considered for patients at high risk of myocardial injury.

Kang et al (2020) Cardiovascular manifestations and treatment considerations in COVID-19[15]

Pre-existing comorbidities such as hypertension, diabetes and cardiovascular disease are associated with a greater severity and higher fatality rate of COVID-19. Furthermore, COVID-19 contributes to cardiovascular complications including acute myocardial injury as a result of acute coronary syndrome, myocarditis, stress-cardiomyopathy, arrhythmias, cardiogenic shock and cardiac arrest. The cardiovascular interactions of COVID-19 have similarities to that of severe acute respiratory syndrome, Middle East respiratory syndrome and influenza. Specific cardiovascular considerations are also necessary in supportive treatment with anticoagulation, the continued use of renin-angiotensin-aldosterone system inhibitors, arrhythmia monitoring, immunosuppression or modulation and mechanical circulatory support.

Guzik et al (2020) COVID-19 and the cardiovascular system: implications for risk assessment, diagnosis, and treatment options[16]

Mortality is increased by comorbidities: cardiovascular disease, hypertension, diabetes, chronic pulmonary disease and cancer. The most common complications include arrhythmia [atrial fibrillation, ventricular tachyarrhythmia, and ventricular fibrillation], cardiac injury [elevated highly sensitive troponin I (hs-cTnI) and creatine kinase (CK) levels], fulminant myocarditis, heart failure, pulmonary embolism and disseminated intravascular coagulation (DIC).
Mechanistically, SARS-CoV-2, following proteolytic cleavage of its S protein by a serine protease, binds to the transmembrane angiotensin-converting enzyme 2 (ACE2) — a homologue of ACE-to enter type 2 pneumocytes, macrophages, perivascular pericytes and cardiomyocytes. This may lead to myocardial dysfunction and damage, endothelial dysfunction, microvascular

dysfunction, plaque instability and myocardial infarction (MI). While ACE2 is essential for viral invasion, there is no evidence that ACE inhibitors or angiotensin receptor blockers (ARBs) worsen prognosis. Hence, patients should not discontinue their use. Moreover, renin-angiotensin-aldosterone system (RAAS) inhibitors might be beneficial in COVID-19. Initial immune and inflammatory responses induce a severe cytokine storm [interleukin (IL)-6, IL-7, IL-22, IL-17, etc.] during the rapid progression phase of COVID-19. Early evaluation and continued monitoring of cardiac damage (cTnI and NT-proBNP) and coagulation (D-dimer) after hospitalization may identify patients with cardiac injury and predict COVID-19 complications. Preventive measures such as social distancing and social isolation also increase cardiovascular risk. Cardiovascular considerations of therapies currently used, including remdesivir, chloroquine, hydroxychloroquine, tocilizumab, ribavirin, interferons and lopinavir/ritonavir as well as experimental therapies such as human recombinant ACE2 (rhACE2) are discussed.

Sankrityayan et al (2020) Evidence for Use or Disuse of Renin-Angiotensin System Modulators in Patients Having COVID-19 With an Underlying Cardiorenal Disorder[17]

Cardiovascular diseases and diabetes are important risk factors for a lethal outcome of COVID-19. Extensive research ensuing the outbreak of coronavirus-related severe acute respiratory syndrome in the year 2003 and more recently COVID-19 revealed a role of renin-angiotensin system (RAS) components in the entry of coronavirus wherein angiotensin-converting enzyme 2 (ACE2) had garnered significant attention. This raises the question whether the use of RAS inhibitors, the backbone of treatment of cardiovascular, neurovascular and kidney diseases could increase the susceptibility for coronavirus infection or unfortunate outcomes of COVID-19. Thus, currently, there is a lack of consensus regarding the effects of RAS inhibitors in such patients. Moreover, expert bodies such as the American Heart Association and American College of Cardiology have now released official statements that RAS inhibitors must be continued, unless suggested otherwise by a physician. In this brief review, we will elaborate on the role of RAS and ACE2 in pathogenesis of COVID-19. Moreover, we will discuss the potential effect of the use and disuse of RAS inhibitors in patients having COVID-19 with cardiometabolic comorbidities.

Cheng et al (2020) Cardiovascular Risks in Patients with COVID-19: Potential Mechanisms and Areas of Uncertainty[18]

We aim to review available clinical and biomedical literature on cardiovascular risks of COVID-19. SARS-CoV2, the virus responsible for COVID-19, enters the cell via ACE2 expressed in select organs. Emerging epidemiological evidence suggests that cardiovascular risk factors are associated with increased disease severity and mortality in COVID-19 patients. Patients with a more severe form of COVID-19 are also more likely to develop cardiac complications such as myocardial injury and arrhythmia. The true incidence of and mechanism underlying these events remain elusive. Cardiovascular diseases appear intricately linked with COVID-19. Robust epidemiologic and biologic studies are urgently needed to better understand the mechanism underlying these associations to develop better therapies.

Shi et al (2020) Characteristics and clinical significance of myocardial injury in patients with severe coronavirus disease 2019[19]

To investigate the characteristics and clinical significance of myocardial injury in patients with severe coronavirus disease 2019 (COVID-19), we enrolled 671 eligible hospitalized patients with severe COVID-19 from 1 January to 23 February 2020 with a median age of 63 years. Clinical, laboratory and treatment data were collected and compared between patients who died and survivors. Risk factors of death and myocardial injury were analysed using multivariable regression models. A total of 62 patients (9.2%) died, who more often had myocardial injury (75.8% vs. 9.7%; $P< 0.001$) than survivors. The area under the receiver operating characteristic curve of initial cardiac troponin I (cTnI) for predicting in-hospital mortality was 0.92 [95% confidence interval (CI), 0.87–0.96; sensitivity, 0.86; specificity, 0.86; $P< 0.001$]. The single cut-off point and high level of cTnI predicted risk of in-hospital death, hazard ratio (HR) was 4.56 (95% CI, 1.28–16.28; $P= 0.019$) and 1.25 (95% CI, 1.07–1.46; $P= 0.004$) respectively. In multivariable logistic regression, senior age, comorbidities such as hypertension, coronary heart disease, chronic renal failure and chronic obstructive pulmonary disease and high level of C-reactive protein were predictors of myocardial injury. Conclusion: The risk of in-hospital death among patients with severe COVID-19 can be predicted by markers of myocardial injury and was significantly associated with senior age, inflammatory response and cardiovascular comorbidities.

Wei et al (2020) Acute myocardial injury is common in patients with COVID-19 and impairs their prognosis[20]

We sought to explore the prevalence and immediate clinical implications of acute myocardial injury in a cohort of patients with COVID-19 in a region of China where medical resources are less stressed than in Wuhan, the epicentre of the pandemic.

We prospectively assessed the medical records, laboratory results, chest CT images and use of medication in a cohort of patients presenting to two designated COVID-19 treatment centres in Sichuan, China. Outcomes of interest included death, admission to an ICU, need for mechanical ventilation, treatment with vasoactive agents and classification of disease severity. Acute myocardial injury was defined by a value of high-sensitivity troponin T (hs-TnT) greater than the normal upper limit.

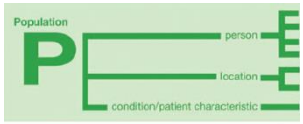
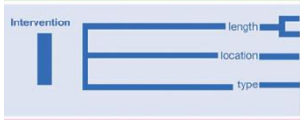
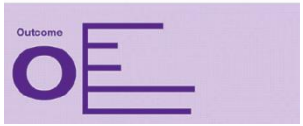
A total of 101 cases were enrolled from January to 10 March 2020 with an average age 49 years, IQR 34–62 years. Acute myocardial injury was present in 15.8% of patients, nearly half of whom had a hs-TnT value fivefold greater than the normal upper limit. Patients with acute myocardial injury were older, had a higher prevalence of pre-existing cardiovascular disease and more were likely to require ICU admission (62.5% vs 24.7%, p=0.003), mechanical ventilation (43.5% vs 4.7%, p<0.001) and treatment with vasoactive agents (31.2% vs 0%, p<0.001). Log hs-TnT was associated with disease severity (OR 6.63, 95% CI 2.24 to 19.65), and all of the three deaths occurred in patients with acute myocardial injury. Conclusion: Acute myocardial injury is common in patients with COVID-19 and is associated with adverse prognosis.

Barison et al (2020) Cardiovascular disease and COVID-19: les liaisons dangereuses[21]

Patients with cardiovascular risk factors or established cardiovascular disease have an increased risk of developing COVID-19 and have a worse outcome when infected, but translating this notion into effective action is challenging. At present it is unclear whether cardiovascular therapies may reduce the probability of infection or improve the survival of infected patients. Given the crucial importance of this issue for clinical cardiologists and all specialists dealing with COVID-19, we tried to recapitulate the current evidence and provide some practical recommendations.

Produced by the members of the National Health Library and Knowledge Service Evidence Team†. Current as at 26 May 2020. This evidence summary collates the best available evidence at the time of writing and does not replace clinical judgement or guidance. Emerging literature or subsequent developments in respect of COVID-19 may require amendment to the information or sources listed in the document. Although all reasonable care has been taken in the compilation of content, the National Health Library and Knowledge Service Evidence Team makes no representations or warranties expressed or implied as to the accuracy or suitability of the information or sources listed in the document. This evidence summary is the property of the National Health Library and Knowledge Service and subsequent re-use or distribution in whole or in part should include acknowledgement of the service.

The following PICO(T) was used as a basis for the evidence summary:

	COVID-19 PATIENTS >65 WITH CARDIOVASCULAR DISEASE
	
	
	WHAT IS THE EVIDENCE ON ADDITIONAL RISK?

The following search strategy was used:

[ABBREVIATED] COVID-19 OR CORONAVIRUS OR "CORONA VIRUS" OR (WUHAN N3 VIRUS) OR (("2019-NCOV" OR "2019 NCOV")) OR "SEVERE RESPIRATORY SYNDROME CORONAVIRUS2" OR (("2019" AND (NEW OR NOVEL) AND CORONAVIRUS)) (HCOV-19) AND (CARDIO*) AND (65 + YEARS)

† Emma Quinn, Librarian, St. Luke's General Hospital, Kilkenny [Author]; Brendan Leen, Area Library Manager, HSE South [Editor]

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