International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	TAKOTSUBO CARDIOMYOPATHY IN DIVERS: A NARRATIVE REVIEW OF THE ASSOCIATION WITH IMMERSION PULMONARY OEDEMA
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5867
RECEIVED	26 March 2026
ACCEPTED	15 June 2026
PUBLISHED	22 June 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

TAKOTSUBO CARDIOMYOPATHY IN DIVERS: A NARRATIVE REVIEW OF THE ASSOCIATION WITH IMMERSION PULMONARY OEDEMA

Karolina Chudecka *(Corresponding Author, Email: karolachudecka@gmail.com)*
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0004-0262-5036

Michał Chylewski
University Clinical Hospital No. 1 PMU in Szczecin, Szczecin, Poland
ORCID ID: 0009-0007-4789-1903

Aleksandra Kędzierska
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0008-7050-4139

Jakub Lamorski
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0004-5456-7413

Izabela Szubert
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0006-7162-6651

Julia Suchecka
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0001-9975-355X

Wiktor Nesterak
Pomeranian Medical University, Szczecin, Poland
ORCID ID: 0009-0009-5322-903X

Katarzyna Józwa
Independent Public Provincial Integrated Hospital in Szczecin, Szczecin, Poland
ORCID ID: 0009-0003-5615-2388

Gabriela Stępień
University Clinical Hospital No. 2 PMU in Szczecin, Szczecin, Poland
ORCID ID: 0009-0006-4505-5162

Urszula Przewoźna
University Clinical Hospital No. 2 PMU in Szczecin, Szczecin, Poland
ORCID ID: 0009-0003-2186-6604

ABSTRACT

Background: Takotsubo cardiomyopathy (TTC), also known as stress cardiomyopathy or broken heart syndrome, is a transient form of left ventricular dysfunction classically triggered by acute physical or emotional stress. An emerging body of literature has documented TTC occurring in the context of underwater diving, frequently in association with immersion pulmonary oedema (IPO). The exact nature of this relationship — whether IPO precipitates TTC, TTC precipitates IPO, or both arise from a shared catecholaminergic surge — remains incompletely understood.
Objectives: To perform a systematic narrative review of all published cases describing TTC in association with diving or immersion, to characterise patient demographics, clinical presentations, diagnostic findings, management strategies, and outcomes, and to discuss proposed pathophysiological mechanisms and implications for diving medicine practice.
Methods: A systematic literature search was conducted in PubMed/MEDLINE, the Cochrane Library, and Google Scholar (January 2000 – December 2025) using pre-specified search terms combining TTC terminology with diving-related terms. Seven primary studies meeting inclusion criteria were identified and narratively synthesised. Quality appraisal of non-randomised evidence was guided by the Scale for the Assessment of Narrative Review Articles (SANRA).
Results: A total of approximately 30 cases of diving-associated TTC were identified across the included studies. Affected individuals were predominantly middle-aged to elderly women, though cases in men were also documented. Presentations consistently featured acute dyspnoea during or after diving, pulmonary oedema on chest imaging, troponin elevation, ECG changes including T-wave inversion and QTc prolongation, and reversible left ventricular wall motion abnormalities on echocardiography with preserved or normal coronary arteries on angiography. Cardiac magnetic resonance imaging (CMR), when performed, showed myocardial oedema and patchy late gadolinium enhancement in the mid-to-apical segments, consistent with TTC. All surviving patients demonstrated complete cardiac recovery within days to weeks. Mortality was confined to cases complicated by fatal arrhythmias or out-of-water cardiac arrest.
Conclusions: Diving represents a recognised and likely underappreciated trigger for TTC, typically in the context of IPO. The association is mediated by an intense catecholaminergic surge provoked by physiological diving stressors including cold-water immersion, increased preload, exertion, and psychological stress. Clinicians evaluating divers presenting with acute cardiorespiratory distress should consider TTC in the differential diagnosis and initiate prompt cardiac investigations. Guideline-based management and long-term restriction from diving are advisable following confirmed episodes.

KEYWORDS

Takotsubo Cardiomyopathy; Stress Cardiomyopathy; Apical Ballooning Syndrome; Immersion Pulmonary Oedema; Scuba Diving; Diving Medicine

CITATION

Karolina Chudecka, Michał Chylewski, Aleksandra Kędzierska, Jakub Lamorski, Izabela Szubert, Julia Suchecka, Wiktor Nesterak, Katarzyna Józwa, Gabriela Stępień, Urszula Przewoźna. (2026) Takotsubo Cardiomyopathy in Divers: a Narrative Review of the Association with Immersion Pulmonary Oedema. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5867

Introduction

Takotsubo cardiomyopathy (TTC) — also referred to as stress cardiomyopathy, apical ballooning syndrome, or broken heart syndrome — is an acute, reversible form of cardiac dysfunction characterised by transient left ventricular (LV) wall motion abnormalities occurring in the absence of obstructive coronary artery disease (Bybee & Prasad, 2008; Gianni et al., 2006). First described in Japan by Dote and colleagues in 1991, the name derives from the distinctive octopus-trap shape (takotsubo in Japanese) that the ballooned left ventricle assumes on ventriculography (Dote et al., 1991). Although originally considered a rare phenomenon, TTC is now recognised to account for approximately 1–2% of all presentations initially suspected to be acute coronary syndrome (ACS), and it has been formally incorporated into the American Heart Association classification of acquired cardiomyopathies (Parodi et al., 2007; Templin et al., 2015).

The hallmark of TTC is the presence of a precipitating physical or emotional stressor. Extreme physiological demands — including acute illness, surgery, neurological events, or strenuous physical exertion — activate the sympathetic nervous system and generate a massive surge of circulating catecholamines, which

in turn trigger myocardial stunning (Kato et al., 2017; Templin et al., 2015). The syndrome predominantly affects post-menopausal women, though it can occur in individuals of any age or sex, particularly when the precipitating stressor is physical rather than emotional (Templin et al., 2015).

Underwater diving constitutes a physiologically demanding activity that imposes multiple stressors on the cardiovascular system simultaneously: acute cold-water immersion causes peripheral vasoconstriction and increased afterload; the hydrostatic pressure of immersion increases central venous return and preload; negative-pressure breathing through a regulator may raise pulmonary capillary pressures; and the psychological stress inherent to an adverse diving event can further amplify sympathoadrenal activation (Stokes et al., 2022; Baber et al., 2016). This constellation of stressors creates a plausible physiological substrate for the development of TTC.

Immersion pulmonary oedema (IPO) — the sudden onset of pulmonary oedema in the setting of water immersion or diving — is the most common cardiac complication encountered in divers and swimmers (Edmonds et al., 2019). A growing body of case literature has described IPO and TTC co-occurring in the same patient, raising important questions about causal directionality, shared pathophysiology, and clinical implications for both acute management and return-to-diving decisions (Ng & Edmonds, 2015; Reed et al., 2016; Stokes et al., 2022; Demoulin et al., 2020).

Despite the accumulating evidence, no comprehensive narrative review has synthesised the published literature on diving-associated TTC against current SANRA quality standards or with a structured PRISMA-guided search methodology. This review therefore aims to: (1) systematically identify and describe all published cases of TTC occurring in the context of underwater diving or immersion; (2) characterise the clinical, diagnostic, and outcome profiles of affected individuals; (3) evaluate proposed pathophysiological mechanisms; and (4) formulate evidence-based recommendations for clinical practice and diving medicine.

Methods

Search Strategy

A systematic literature search was conducted in PubMed/MEDLINE, the Cochrane Library, and Google Scholar, covering the period January 2000 to December 2025. The search strategy (provided in Supplementary File 1) employed Boolean combinations of TTC-specific terms — including ‘takotsubo cardiomyopathy’, ‘stress cardiomyopathy’, ‘apical ballooning syndrome’, ‘broken heart syndrome’, and ‘transient left ventricular apical ballooning’ — with diving-related terms including ‘diving’, ‘scuba diving’, ‘underwater diving’ and ‘immersion’. Additional records were identified through manual reference checking of included articles and relevant review papers.

Inclusion and Exclusion Criteria

Studies were included if they: (1) described one or more cases of TTC (or equivalent diagnosis such as reversible myocardial dysfunction or stress cardiomyopathy) occurring during or immediately after underwater diving or water immersion; (2) provided sufficient clinical detail to assess the diagnosis; and (3) were published in peer-reviewed journals in the English language. Conference abstracts, editorials without original case data, and duplicate reports of the same case were excluded.

Data Extraction and Quality Appraisal

Two reviewers independently screened titles and abstracts, followed by full-text review of eligible records. Data were extracted regarding patient demographics, dive characteristics, clinical presentation, diagnostic investigations, management, and outcomes. Given the predominance of case reports and small case series in this literature, formal meta-analysis was not feasible. Quality of included studies was appraised using the Scale for the Assessment of Narrative Review Articles (SANRA), which evaluates six domains: justification of the review, literature search description, use of citations, reproducibility, scientific reasoning, and use of appropriate language (Baethge et al., 2019).

PRISMA Flow Diagram

The literature search and selection process is summarised in Figure 1 in accordance with PRISMA 2020 reporting guidelines.

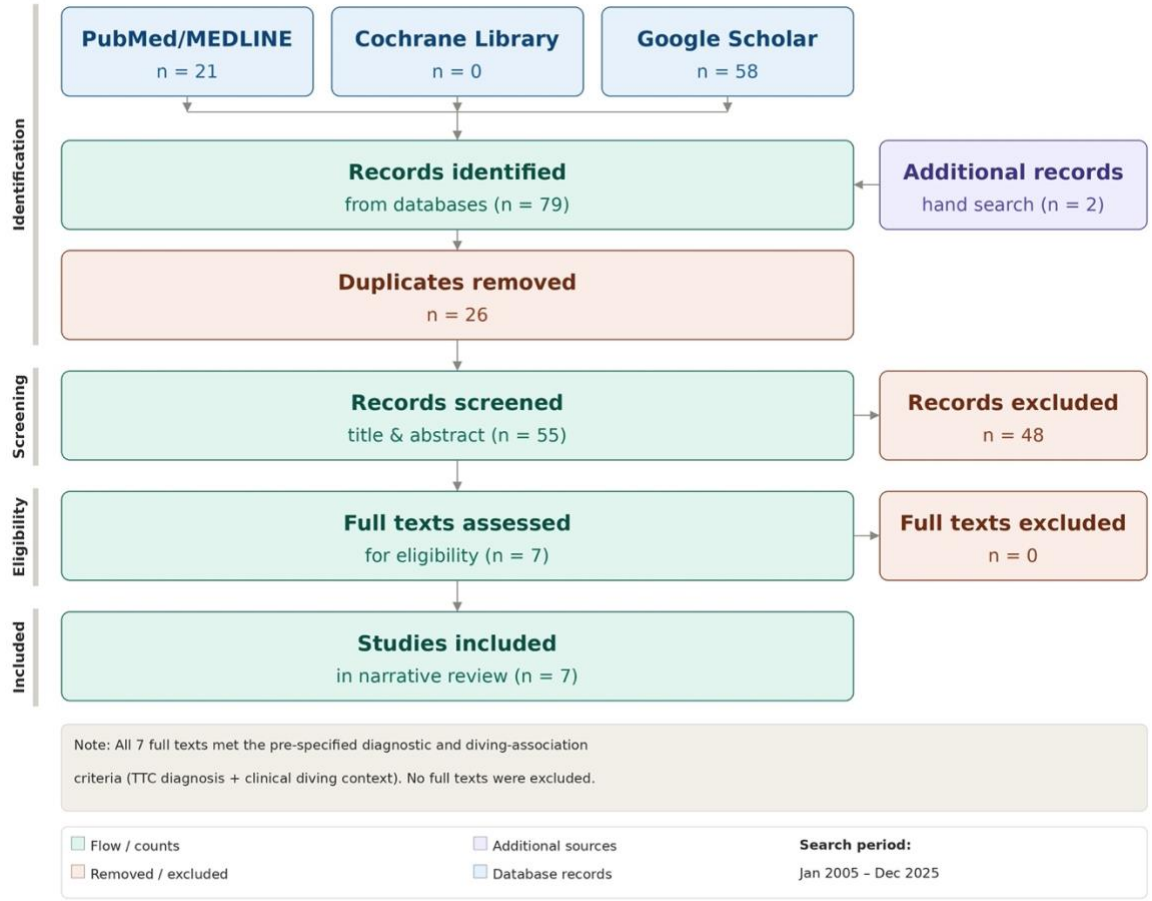

Fig. 1. *PRISMA 2020 flow diagram for the systematic literature search. PubMed/MEDLINE (n = 21), Cochrane Library (n = 0), Google Scholar (n = 58); additional records from hand searching (n = 2); after deduplication and screening, 7 studies were included in the narrative review.*

Background: Takotsubo Cardiomyopathy

Definition and Diagnosis

Takotsubo cardiomyopathy is defined by a cluster of features that distinguish it from acute myocardial infarction while sharing many of its presenting characteristics. The Mayo Clinic diagnostic criteria, first proposed in 2004 and subsequently adopted by the International Takotsubo Registry (InterTAK Registry), require: (1) transient LV wall motion abnormalities involving the apical and/or mid-ventricular segments, extending beyond the territory of a single epicardial coronary distribution; (2) absence of obstructive epicardial coronary artery disease or acute plaque rupture on angiography; (3) new ECG abnormalities, most commonly transient ST-segment elevation or diffuse T-wave inversion; and (4) absence of conditions that could account for the wall motion pattern, such as phaeochromocytoma or myocarditis (Hurst et al., 2010; Bybee & Prasad, 2008).

The syndrome is most commonly characterised by apical ballooning with hyperkinesia of the basal LV segments, producing the octopus-pot appearance after which it is named. However, morphological variants exist, including mid-ventricular, basal (inverted or reverse Takotsubo), and focal forms, accounting for approximately 15–25% of cases (Hurst et al., 2010). In the Oceania case series by Demoulin and colleagues (2020) — the largest published series of diving-associated cases — the echocardiographic localisation was notably focal and non-apical, with a mean LV ejection fraction (LVEF) of approximately 45%, which the authors interpreted as consistent with ‘Takotsubo-like’ atypical topography secondary to the specific physiological stressors of underwater diving.

Epidemiology

TTC accounts for 1–2% of all suspected ACS presentations and carries an overall in-hospital mortality of approximately 1.1%, though complication rates including cardiogenic shock, ventricular arrhythmias, and LV thrombus formation are non-trivial (Templin et al., 2015; Gianni et al., 2006). Recurrence at four years has been reported in up to 11.4% of patients (Elesber et al., 2007 cited in Ng & Edmonds, 2015). The syndrome demonstrates a striking demographic predilection: in general population series, over 90% of patients are women, and 80% are post-menopausal. However, in diving-associated TTC, this female predominance is less absolute, with cases reported in men, including a series of predominantly male French military divers (Demoulin et al., 2020; Gempp et al., 2013).

Pathophysiology

The central pathophysiological mechanism of TTC is catecholamine-mediated myocardial injury. Plasma catecholamine levels measured during acute TTC have been found to be 10–20 times above normal (Kato et al., 2017; Stokes et al., 2022), and the syndrome can be experimentally induced by infusion of sympathomimetic agents. The apical predominance of myocardial stunning in classical TTC has been attributed to the higher density of sympathetic nerve terminals and adrenoreceptors in the LV apex, rendering apical myocytes disproportionately vulnerable to catecholamine-mediated injury (Kato et al., 2017). Additional proposed mechanisms include epicardial and microvascular coronary vasospasm, direct catecholamine toxicity via oxidative stress, and oestrogen deficiency-related impaired sympatholysis in post-menopausal women.

Cardiac magnetic resonance imaging (CMR) has emerged as the gold-standard modality for tissue characterisation in suspected TTC. T2-weighted/short-tau inversion recovery (STIR) imaging demonstrates high-signal myocardial oedema in a non-ischaemic distribution, dissociated from individual coronary territories. Patchy, low-intensity late gadolinium enhancement (LGE) in the mid-to-apical segments is increasingly recognised, and critically, both oedema and LGE resolve completely on follow-up imaging, consistent with the reversible nature of the syndrome (Stokes et al., 2022; Kato et al., 2017). The absence of dense subendocardial or transmural LGE in a coronary distribution helps exclude ischaemic scar.

Background: Immersion Pulmonary Oedema

Immersion pulmonary oedema (IPO) — also referred to as scuba divers' pulmonary oedema (SDPE) or swimming-induced pulmonary oedema (SIPO) — is defined as the acute onset of pulmonary oedema during or immediately after water immersion, in the absence of aspiration or other identifiable causes such as pulmonary barotrauma (Wilmshurst, 2019; Edmonds et al., 2019). Clinical features include acute dyspnoea, cough, frothy or blood-tinged sputum, tachypnoea, hypoxaemia, and bilateral pulmonary crepitations. The condition was first characterised in the 1980s and has been increasingly reported in the subsequent decades, both in recreational and military divers (Wilmshurst et al., 1981 cited in Ng & Edmonds, 2015; Edmonds et al., 2019).

The pathophysiology of IPO is multifactorial. Immersion in water causes a redistribution of blood from the peripheral venous circulation to the thorax, increasing central blood volume by up to 700 mL and raising pulmonary capillary pressures (Stokes et al., 2022). This effect is compounded by cold water-mediated peripheral vasoconstriction (increasing afterload), increased inspiratory effort against a regulator (negative-pressure breathing), physical exertion, and, in predisposed individuals, underlying subclinical hypertension, valvular disease, or cardiac dysfunction. The net result is a rise in pulmonary capillary hydrostatic pressure sufficient to drive fluid into the alveolar space. Risk factors consistently identified in the literature include hypertension, female sex, older age, cold water, tight wetsuits, and the use of certain medications including beta-blockers (Stokes et al., 2022; Edmonds et al., 2019; Baber et al., 2016).

IPO can be fatal when it causes profound hypoxaemia and drowning, particularly in remote or underwater settings. A retrospective Australian series documented 31 IPO incidents over 16 years, identifying transient cardiomyopathies — consistent with TTC or stress cardiomyopathy — in 26% of cases (Edmonds et al., 2019). The risk of recurrence following an index IPO event is estimated at 30–40%, underscoring the importance of accurate diagnosis and appropriate advice regarding return to water activities.

Results: Summary of Included Studies

Overview of the Literature

Seven studies met inclusion criteria and were included in the narrative synthesis (Table 1). These comprised one prospective observational cohort study (Demoulin et al., 2020), two retrospective case series (Gempp et al., 2013; Edmonds et al., 2019), and four case reports or case series of 1–3 patients (Chenaitia et al., 2010; Ng & Edmonds, 2015; Reed et al., 2016; Stokes et al., 2022). One additional report by Baber et al. (2016) provided a case report with a contemporaneous literature review. In total, approximately 30 individual cases were documented across all included studies, spanning geographical settings including France, Australia, the United Kingdom, and Hawaii (USA).

Table 1. Summary of Included Studies

Study	Design	n (cases)	Setting / Country	Patient profile	Key findings	Outcome
Chenaitia et al. (2010)	Case report	1	France	51F, menopausal, no cardiac history	Apical ballooning; LVEF 35%; ECG T-wave inversions; normal coronaries	Full recovery
Ng & Edmonds (2015)	Case report	1	Australia	67F, experienced diver, no cardiac Hx	LVEF 38%; TTE hypokinesis; Troponin T 4,052 ng/L; recovery in 6 days	Full recovery; advised to cease diving
Reed et al. (2016)	Case series	3	Hawaii, USA	62F, 60M, 47F; no prior cardiac disease	Apical ballooning; LVEF 25–45%; ECG changes; normal angiography in all	All recovered; EF normalised at 1 month
Baber et al. (2016)	Case report + review	1	USA (Pennsylvania)	47F, scuba diving, IPO	Basal hypokinesis (reverse TTC); NIPPV; troponin elevated; stress echo normalised at 2 days	Full recovery
Gempp et al. (2013)	Retrospective cohort	~20 (RMD subset)	France (military)	Predominantly male military divers	28% of IPO patients had reversible myocardial dysfunction; troponin elevation, wall motion abnormalities	All survived; advised to cease diving
Demoulin et al. (2020)	Prospective observational	20	France (military hospitals)	Mean age 47; male predominance in military cohort	Focal, non-apical LV dysfunction; mean LVEF 45%; T-wave inversion + QTc prolongation; discordance between troponin and echo severity	All survived; TTC considered definitive diving contraindication
Stokes et al. (2022)	Case report	1	UK	~40F, PADI advanced diver; undiagnosed hypertension	CMR: mid-apical oedema + LGE; LVEF 46%; AF; full CMR resolution at 7 months	Full recovery; CMR resolved; loop recorder implanted
Edmonds et al. (2019)	Retrospective series	31 IPO (TTC/RMD in ~8)	Oceania (Australia, NZ, Asia-Pacific)	Mix of sexes and ages; some with comorbidities	TTC/SCM in 26% of IPO cases; contraction band necrosis at autopsy in fatal cases	4 fatalities (cardiac arrest); remainder recovered

Note. F = female; M = male; LVEF = left ventricular ejection fraction; TTC = Takotsubo cardiomyopathy; IPO = immersion pulmonary oedema; RMD = reversible myocardial dysfunction; CMR = cardiac magnetic resonance imaging; LGE = late gadolinium enhancement; AF = atrial fibrillation; NIPPV = non-invasive positive pressure ventilation; SCM = stress cardiomyopathy.

Patient Demographics

Across all identified cases, affected individuals ranged in age from the early forties to their late sixties. A female predominance was observed in recreational diving cases (Ng & Edmonds, 2015; Reed et al., 2016; Stokes et al., 2022), consistent with the known epidemiology of TTC in the general population. In contrast, the French military cohort reported by Demoulin et al. (2020) was predominantly male, reflecting the demographic composition of that particular diving population. Many individuals were experienced divers with no prior cardiac history at the time of their events, arguing against pre-existing structural heart disease as a prerequisite. Hypertension, either documented or detected after the event, was the most common cardiovascular risk factor identified (Stokes et al., 2022; Edmonds et al., 2019; Ng & Edmonds, 2015).

Dive Characteristics and Precipitating Circumstances

Events occurred across a range of diving conditions. Depths ranged from shallow recreational dives (8–20 metres of sea water, msw) to moderate depth dives (35 msw), and most incidents developed during or immediately after ascent to the surface (Ng & Edmonds, 2015; Reed et al., 2016; Chenaitia et al., 2010). Cold water (below 20°C) was a recurring feature (Ng & Edmonds, 2015; Stokes et al., 2022), as were reports of emotional stress in the period preceding the dive (Chenaitia et al., 2010). Technical difficulties during the dive — including regulator malfunction, buoyancy control problems, and out-of-air situations — were described in several cases and are likely to have heightened sympathoadrenal activation (Stokes et al., 2022). One fatal case documented in the Oceania series (Edmonds et al., 2019) demonstrated contraction band necrosis on autopsy myocardial histology, a pathological hallmark of catecholamine-mediated cardiac injury consistent with TTC.

Clinical Presentation

The presenting symptom in all cases was acute dyspnoea during or immediately after surfacing, accompanied by cough and frothy, blood-tinged, or pink sputum. Haemoptysis was documented in several cases (Stokes et al., 2022; Edmonds et al., 2019). Chest pain was variably reported — typically absent or minimal in isolated IPO, but present in some cases when TTC was the dominant presentation, mimicking ACS (Chenaitia et al., 2010; Baber et al., 2016). Oxygen saturations on initial assessment were severely reduced, ranging from 82–92% on room air across reported cases. Loss of consciousness or syncope was documented in cases where cardiovascular collapse occurred, including in the fatal cases reported by Edmonds et al. (2019). One patient required cardiopulmonary resuscitation, and another was retrieved from the water in a pulseless state.

Diagnostic Findings

Chest radiography uniformly demonstrated bilateral pulmonary oedema. The electrocardiographic pattern was remarkably consistent across cases: symmetric T-wave inversions in the precordial leads (most commonly V1–V6) were the dominant finding, with corrected QT interval (QTc) prolongation a frequent accompaniment (Demoulin et al., 2020; Stokes et al., 2022). The latter is clinically significant, as QTc prolongation in the setting of TTC is associated with a risk of torsades de pointes and potentially fatal ventricular arrhythmias (Madias et al., 2011 cited in Stokes et al., 2022). ST-segment changes, including transient ST elevation in V1–V3, were documented in some cases (Reed et al., 2016; Chenaitia et al., 2010), prompting initial consideration of STEMI.

Cardiac biomarkers were elevated in all cases where measured. Troponin elevations (both troponin I and T) were moderate and disproportionately low relative to the degree of ventricular dysfunction observed on imaging — a pattern recognised as characteristic of TTC and contrasting with the usually concordant relationship between biomarker peak and infarct size in ACS (Demoulin et al., 2020). Brain natriuretic peptide (BNP) and N-terminal pro-BNP were also elevated in most cases. A notable discordance between enzymatic peak and echocardiographic severity of wall motion abnormality was specifically highlighted by Demoulin et al. (2020) as a potential diagnostic clue.

Echocardiography demonstrated reversible LV systolic dysfunction in all cases. The classic pattern — apical ballooning with basal hyperkinesia — was documented in several recreational diving cases (Reed et al., 2016; Ng & Edmonds, 2015). However, the Demoulin et al. (2020) cohort of 20 military divers demonstrated a predominantly focal, non-apical pattern of LV dysfunction, distinct from classical Takotsubo topography and more consistent with the atypical mid-ventricular or regional variant. Mean LVEF was approximately 45% at

the time of the acute presentation, recovering to normal (typically >55%) within days to two weeks. Coronary angiography performed in the majority of cases confirmed normal or non-obstructing coronary arteries, satisfying a key diagnostic criterion for TTC.

CMR was reported in one case in detail (Stokes et al., 2022), representing the first published description of CMR findings in diving-associated TTC. On day six post-incident, CMR demonstrated circumferential akinesia in the mid-to-apical LV segments with diffuse myocardial oedema on T2/STIR imaging and patchy, low-intensity, non-ischaemic LGE in the mid-apical segments — a pattern congruent with that described in general TTC series. A follow-up CMR at seven months showed complete resolution of oedema, LGE, and wall motion abnormalities, confirming full myocardial recovery.

Management

Acute management across all cases followed general supportive principles for pulmonary oedema and cardiogenic stress response: supplemental high-flow oxygen, diuretics (intravenous furosemide in most cases), and in some cases non-invasive positive pressure ventilation (CPAP or BiPAP). Aspirin and/or clopidogrel were administered empirically in several cases prior to the diagnosis of TTC being established, and were subsequently discontinued once ACS was excluded. Anticoagulation was not routinely employed. Beta-blockers (bisoprolol) and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers were used in the subacute phase to support LV recovery and treat concomitant hypertension (Stokes et al., 2022; Ng & Edmonds, 2015). Demoulin et al. (2020) specifically recommended ACE inhibitor and beta-blocker initiation until normalisation of ventricular kinetics as the standard approach in their cohort. Implantable cardiac loop recorders were inserted in at least one patient for ongoing arrhythmia surveillance (Stokes et al., 2022).

The specific choice of antihypertensive therapy in Stokes et al. (2022) merits comment: an angiotensin receptor blocker (losartan) was preferred over an ACE inhibitor to avoid the potential cough side effect, which could confound the clinical picture of ongoing respiratory symptoms in a diver who had experienced IPO. Additionally, a calcium channel blocker (amlodipine) was added, reflecting anecdotal evidence from the IPO literature that calcium channel blockade may reduce recurrence of IPO by attenuating vasoconstriction responses to physiological stimuli (Stokes et al., 2022).

Outcomes and Return to Diving

The prognosis for surviving patients was excellent. Complete recovery of LV systolic function was documented in all cases where follow-up echocardiography was performed, typically within one to four weeks. Where measured, repeat TTE at six days (Ng & Edmonds, 2015) to six months showed normalisation of LVEF and resolution of wall motion abnormalities. This pattern is consistent with the general TTC literature, in which approximately 96% of patients recover full cardiac function (Sharkey et al., 2010 cited in Reed et al., 2016).

Mortality was reported in four of the 31 cases in the Oceania series by Edmonds et al. (2019), all associated with out-of-water cardiac arrest, profound hypoxaemia, or fatal arrhythmias — reflecting the severity of the acute cardiorespiratory presentation rather than a late complication of TTC per se.

Return-to-diving recommendations were consistently cautious across all studies. The majority of authors advised permanent cessation of scuba diving following an episode of TTC in the context of IPO, citing the risk of recurrence and the potentially fatal consequences in an underwater environment (Ng & Edmonds, 2015; Demoulin et al., 2020). Demoulin et al. (2020) explicitly characterised diving-associated TTC complicated by QTc prolongation and ventricular arrhythmia risk as a definitive contraindication to further diving. Where limited surface swimming or snorkelling was considered, authors recommended ensuring adequate blood pressure control, open-circuit equipment use, diving with an experienced buddy, and surface oxygen availability (Stokes et al., 2022).

Discussion

The Causal Relationship Between IPO and TTC in Divers

The key mechanistic question arising from the described cases is whether IPO causes TTC, TTC causes IPO, both arise simultaneously from a shared trigger, or the association is coincidental. Ng and Edmonds (2015) outlined four theoretical relationships: either condition may precipitate the other, both may arise from the immersion stress response independently, or the co-occurrence may be coincidental. The weight of available evidence supports a model in which the intense physiological stressors of diving — particularly cold immersion, increased preload, negative-pressure breathing, exertion, and acute psychological stress — simultaneously precipitate IPO through pulmonary capillary hypertension and TTC through catecholaminergic myocardial stunning. This parallel pathway is consistent with the temporal coincidence of both diagnoses at the time of presentation in most reported cases.

However, a bidirectional relationship is plausible: acute hypoxaemia from IPO may itself constitute a severe physiological stressor sufficient to trigger catecholamine release and precipitate TTC (Reed et al., 2016). Conversely, TTC-induced LV systolic dysfunction and elevated left atrial pressures could worsen pulmonary oedema already initiated by immersion physiology. This pathophysiological entanglement may explain the severity of cardiorespiratory compromise observed in combined IPO-TTC presentations, which can exceed that typically seen with either condition alone.

Pathophysiological Mechanisms in the Diving Context

The diving environment represents a uniquely dense constellation of TTC triggers. Cold water immersion stimulates peripheral vasoconstriction via alpha-adrenergic activation, raising systemic vascular resistance and LV afterload. Immersion itself increases central venous return, elevating preload. Strenuous exertion, whether from swimming against a current, ascending with buoyancy problems, or the physical demands of emergency manoeuvres, further increases cardiac oxygen demand. The psychological stress of an adverse diving event — loss of air supply, equipment malfunction, entrapment, or witnessing another diver in distress — can generate the type of acute emotional trigger classically associated with TTC in non-diving contexts (Kato et al., 2017; Chenaitia et al., 2010).

Negative-pressure breathing, intrinsic to open-circuit scuba equipment, may additionally contribute by raising transmural pulmonary pressures. Catecholamine levels measured in the acute phase of diving-associated TTC have not been directly reported in the included studies, but the postulated mechanism — a sympathoadrenal surge with circulating epinephrine and norepinephrine at 10–20 times normal — is well supported by the general TTC literature (Kato et al., 2017; Stokes et al., 2022). Hypertension, identified in several divers either preceding the event or discovered at hospital presentation, likely represents a vulnerability factor that lowers the threshold for both IPO and TTC by impairing LV diastolic function and increasing baseline pulmonary capillary pressures.

The atypical, non-apical distribution of LV dysfunction observed in the Demoulin et al. (2020) military cohort warrants comment. The authors hypothesised that the specific haemodynamic stressors of diving — particularly the pronounced increase in preload and afterload — may produce a pattern of catecholamine-mediated myocardial injury different from that seen in emotionally triggered TTC, perhaps preferentially affecting the mid-ventricular rather than apical myocardium. This may also reflect differences in the underlying autonomic receptor density and the speed of onset of the catecholaminergic surge in the submerged environment. Recognition of these atypical patterns is clinically important, as they may not satisfy classical TTC diagnostic criteria and could lead to diagnostic uncertainty or delay.

Cardiac Magnetic Resonance Imaging

CMR remains underutilised in the evaluation of diving-associated TTC. Only one case in the reviewed literature (Stokes et al., 2022) included prospective CMR with follow-up imaging. Yet CMR offers unique value in this context: its ability to characterise myocardial oedema (via T2/STIR mapping), detect LGE, assess biventricular function, and, crucially, demonstrate reversibility of all abnormalities at follow-up provides the most definitive evidence of TTC as opposed to ischaemic cardiomyopathy or myocarditis (Stokes et al., 2022). In divers presenting with acute LV dysfunction and no obstructive coronary disease, CMR should be considered part of the standard diagnostic workup — particularly given the therapeutic and occupational implications of the diagnosis. The temporal window for CMR acquisition is important: oedema resolves within days to weeks, and CMR performed beyond this window may be falsely normal.

Implications for Diving Medicine Practice

The published cases carry several important implications for clinicians evaluating acutely unwell divers. First, any diver presenting with acute dyspnoea, hypoxaemia, and frothy sputum during or after a dive should be assessed for both IPO and TTC as co-existing diagnoses, not mutually exclusive alternatives. Early cardiac investigations — ECG, troponin, echocardiography, and BNP — should be performed promptly, since the reversible nature of TTC means that normal investigations obtained days later do not exclude an acute event (Ng & Edmonds, 2015). The ECG finding of diffuse T-wave inversions with QTc prolongation should raise particular concern given the risk of potentially fatal ventricular arrhythmias (Demoulin et al., 2020; Madias et al., 2011 cited in Stokes et al., 2022).

Second, clinicians must resist the temptation to prematurely assume cardiac normality when routine investigations are performed too late. Ng and Edmonds (2015) specifically cautioned that investigations conducted days or weeks after the event, when wall motion abnormalities and ECG changes have spontaneously resolved, may create a false impression of a normal cardiac study. Serial investigations, beginning as soon as possible after the acute event, are mandatory.

Third, from a safety perspective, TTC in the diving context should be regarded as a serious adverse event with implications for fitness to dive. The combination of haemodynamic vulnerability, potential for arrhythmia during QTc prolongation, risk of recurrence of IPO, and the practical impossibility of providing safe emergency cardiac care at depth or underwater all argue strongly against return to diving. Demoulin et al. (2020) have made the most unequivocal recommendation in this regard, proposing that diving-associated TTC represents a definitive diving contraindication given the potential for sudden cardiac death during recurrence.

Fourth, diving medicine physicians conducting pre-dive fitness assessments should be alert to risk factors for both IPO and TTC, including hypertension, female sex, post-menopausal status, and a history of prior IPO episodes. Prospective counselling about the risk of IPO, recognition of early symptoms, the importance of surfacing promptly, and the availability of oxygen at the surface may reduce the likelihood of severe combined presentations.

Limitations of the Available Evidence

The body of literature on diving-associated TTC consists almost exclusively of case reports and small case series, with inherent limitations including selection bias (publication bias favouring dramatic or unusual cases), incomplete clinical data, variability in diagnostic criteria applied, and absence of control groups. The true incidence of TTC in divers with IPO is unknown and likely underestimated, as echocardiographic assessment is not routinely performed in all IPO cases, particularly those occurring in remote or resource-limited settings (Edmonds et al., 2019). The predominance of military cohorts in the French literature (Demoulin et al., 2020; Gempp et al., 2013) may not be generalisable to recreational diving populations. Standardised diagnostic criteria for both IPO and TTC were not uniformly applied across studies, and the nomenclature employed varied between authors.

Future research should prioritise prospective, multicentre registry studies of IPO incorporating systematic echocardiographic assessment and, where feasible, CMR, to accurately characterise the prevalence and prognostic significance of TTC in this setting. Standardised reporting criteria, agreed diagnostic case definitions, and consensus guidance on return-to-diving decisions following TTC are urgently needed.

SANRA Quality Assessment

This review was conducted and reported in accordance with the Scale for the Assessment of Narrative Review Articles (SANRA) (Baethge et al., 2019), which evaluates six key criteria: (1) justification of the review — the review addresses a clinically relevant and incompletely characterised topic (TTC in divers) with clear potential to improve practice; (2) literature search — a structured, reproducible search strategy was employed across multiple databases with pre-specified search terms (Supplementary File 1) and a defined date range; (3) use of citations — all major factual claims are supported by cited primary literature; (4) scientific reasoning — mechanistic conclusions are distinguished from speculative inference, and the limitations of the case-based evidence base are explicitly acknowledged; (5) appropriate language — findings are described without unwarranted causal claims in the absence of controlled data; (6) focus of the review — the review maintains a focused clinical and pathophysiological scope. These standards are intended to maximise transparency and reproducibility for a narrative review in a field where randomised controlled trial data are unlikely to emerge.

Conclusions

Takotsubo cardiomyopathy is an emerging and likely underrecognised complication of underwater diving, most commonly presenting in association with immersion pulmonary oedema. The physiological stressors intrinsic to the diving environment — including cold immersion, increased preload and afterload, negative-pressure breathing, physical exertion, and acute psychological stress — create a uniquely potent substrate for catecholaminergic myocardial stunning. Clinicians evaluating acutely unwell divers must maintain a high index of suspicion for TTC and institute prompt cardiac investigation, including ECG, troponin, echocardiography, and BNP measurement. CMR should be considered the gold standard for diagnosis and monitoring, particularly when performed within the first week of the acute event. Surviving patients demonstrate consistent and complete cardiac recovery, but the risk of recurrence, the potential for fatal arrhythmias during QTc prolongation, and the inability to provide emergency cardiac care underwater collectively support a policy of permanent diving restriction following a confirmed event. Prospective multicentre registry studies are required to determine the true prevalence of TTC in divers with IPO and to establish evidence-based guidelines for return-to-diving decisions.

Acknowledgements: No funding was received for this review. The authors confirm adherence to the requirements for ethical publishing as outlined in Shewan et al. (2015).

REFERENCES

1. Baethge, C., Goldbeck-Wood, S., & Mertens, S. (2019). SANRA—A scale for the quality assessment of narrative review articles. *Research Integrity and Peer Review, 4*, Article 5. https://doi.org/10.1186/s41073-019-0064-8
2. Baber, A., Nair, S. U., Duggal, S., Bhatti, S., & Sundlof, D. W. (2016). Stress cardiomyopathy caused by diving: Case report and review of the literature. *The Journal of Emergency Medicine, 50*(2), 277–280. https://doi.org/10.1016/j.jemermed.2015.09.045
3. Bybee, K. A., & Prasad, A. (2008). Stress-related cardiomyopathy syndromes. *Circulation, 118*(4), 397–409. https://doi.org/10.1161/CIRCULATIONAHA.106.677625
4. Chenaitia, H., Coullange, M., Benhamou, L., & Gerbeaux, P. (2010). Takotsubo cardiomyopathy associated with diving. *European Journal of Emergency Medicine, 17*(2), 103–106. https://doi.org/10.1097/MEJ.0b013e32832dd8ee
5. Demoulin, R., Poyet, R., Castagna, O., Gempp, E., Druelle, A., Schmitt, P., Capilla, E., Rohel, G., Pons, F., Jégo, C., Brocq, F.-X., & Cellarier, G. R. (2020). Epidemiological, clinical, and echocardiographic features of twenty "Takotsubo-like" reversible myocardial dysfunction cases with normal coronarography following immersion pulmonary oedema. *Acta Cardiologica*. https://doi.org/10.1080/00015385.2020.1726627
6. Dote, K., Sato, H., Tateishi, H., Uchida, T., & Ishihara, M. (1991). Myocardial stunning due to simultaneous multivessel coronary spasms: A review of 5 cases. *Journal of Cardiology, 21*(2), 203–214.
7. Edmonds, C., Lippmann, J., & Fock, A. (2019). Immersion pulmonary edema: Case reports from Oceania. *Undersea and Hyperbaric Medicine, 46*(5), 581–601.
8. Gempp, E., Louge, P., Henckes, A., Demaistre, S., Heno, P., & Blatteau, J.-E. (2013). Reversible myocardial dysfunction and clinical outcome in scuba divers with immersion pulmonary oedema. *The American Journal of Cardiology, 111*(11), 1655–1659. https://doi.org/10.1016/j.amjcard.2013.01.328
9. Gianni, M., Dentali, F., Grandi, A. M., Sumner, G., Hiralal, R., & Lonn, E. (2006). Apical ballooning syndrome or Takotsubo cardiomyopathy: A systematic review. *European Heart Journal, 27*(13), 1523–1529. https://doi.org/10.1093/eurheartj/ehl032
10. Hurst, R. T., Prasad, A., Askew, J. W., 3rd, Sengupta, P. P., & Tajik, A. J. (2010). Takotsubo cardiomyopathy: A unique cardiomyopathy with variable ventricular morphology. *JACC: Cardiovascular Imaging, 3*(6), 641–649. https://doi.org/10.1016/j.jcmg.2010.01.009
11. Kato, K., Lyon, A. R., Ghadri, J. R., & Templin, C. (2017). Takotsubo syndrome: Aetiology, presentation and treatment. *Heart, 103*(18), 1461–1469. https://doi.org/10.1136/heartjnl-2016-309783
12. Ng, A., & Edmonds, C. (2015). Immersion pulmonary oedema and Takotsubo cardiomyopathy. *Diving and Hyperbaric Medicine, 45*(4), 255–257.
13. Parodi, G., Del Pace, S., Carrabba, N., Salvadori, C., Memisha, G., Simonetti, I., Antoniucci, D., & Gensini, G. F. (2007). Incidence, clinical findings, and outcome of women with left ventricular apical ballooning syndrome. *The American Journal of Cardiology, 99*(2), 182–185. https://doi.org/10.1016/j.amjcard.2006.07.080
14. Reed, T., Sorrentino, D., & Azuma, S. (2016). Immersion pulmonary edema in the setting of Takotsubo cardiomyopathy. *International Cardiovascular Forum Journal, 5*, 62–63. https://doi.org/10.17987/icfj.v5i0.260
15. Sharkey, S. W., Windenburg, D. C., Lesser, J. R., Maron, M. S., Hauser, R. G., Lesser, J. N., Haas, T. S., Hodges, J. S., & Maron, B. J. (2010). Natural history and expansive clinical profile of stress (tako-tsubo) cardiomyopathy. *Journal of the American College of Cardiology, 55*(4), 333–341. https://doi.org/10.1016/j.jacc.2009.08.057
16. Shewan, L. G., Coats, A. J. S., & Henein, M. (2015). Requirements for ethical publishing in biomedical journals. *International Cardiovascular Forum Journal, 2*, Article 2. https://doi.org/10.17987/icfj.v2i1.4
17. Stokes, R. J., Sayers, R., Sieniewicz, B. J., & Kim, W. C. (2022). Takotsubo cardiomyopathy findings on cardiac magnetic resonance imaging following immersion pulmonary oedema. *Diving and Hyperbaric Medicine, 52*(3), 217–220. https://doi.org/10.28920/dhm52.3.217-220
18. Templin, C., Ghadri, J. R., Diekmann, J., Napp, L. C., Bataiosu, D. R., Jaguszewski, M., Cammann, V. L., Nienaber, C. A., & Lüscher, T. F. (2015). Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. *New England Journal of Medicine, 373*(10), 929–938. https://doi.org/10.1056/NEJMoa1406761
19. Wilmshurst, P. T. (2019). Immersion pulmonary oedema: A cardiological perspective. *Diving and Hyperbaric Medicine, 49*(1), 30–40. https://doi.org/10.28920/dhm49.1.30-40