PREHOSPITAL MEDICAL ADVISORY COMMITTEE MEETING AGENDA (PMAC)

PMAC MEMBERS PER POLICY 8202:

Air Transport Provider Representative
11-Kent McCurdy

American Medical Response
5-Douglas Key

BLS Ambulance Service Representative
12-Lori Lopez

Cathedral City Fire Department
5-Justin Vondriska

Corona Regional Medical Center
1-Robert Steele, MD
4-Tamera Roy

County Fire Chiefs' Non-Transport ALS Provid
10-Vacant

County Fire Chiefs' Non-Transport BLS Provid
9-Phil Rawlings (Vice Chair)

Desert Regional Medical Center
1-Joel Stillings, D.O
4-G. Stanley Hall

Eisenhower Health
1-Mandeep Daliwhal, MD
4-Tasha Anderson

EMT / EMT-P Training Programs
6-Maggie Robles

EMT-at-Large
13 David Olivas

Paramedic-at-Large
14-Sarah Coonan

Hemet Valley Medal Center
1-Todd Hanna, MD
4-Victoria Moor

Idyllwild Fire Protection District
5-Patrick Reitz

Inland Valley Regional Medical Center
1-Zeke Foster MD
4-Daniel Sitar

JFK Memorial Hospital
1-Troy Cashatt, MD
4- Evelin Millsap

Kaiser Permanente Riverside
1-Jonathan Dyreyes, MD
4-Carol Fuste

This Meeting of PMAC is on:
Monday, April 22, 2019
9:00 AM to 11:00 AM
The Towers of Riverwalk
4210 Riverwalk Parkway, Riverside
First Floor Conference Rooms – Lemon and Orange

1. **CALL TO ORDER & HOUSEKEEPING (3 Minutes)**
   Misty Plumley

2. **PLEDGE OF ALLEGIANCE (1 Minute)**
   Zeke Foster, MD (Chair)

3. **ROUNDTABLE INTRODUCTIONS (5 Minutes)**
   Zeke Foster, MD (Chair)

4. **APPROVAL OF MINUTES (3 Minutes)**
   January 7, 2019 Minutes— Zeke Foster, MD (Attachment A)

5. **STANDING REPORTS**
   5.1. Trauma System—Shanna Kissel (Attachment B)
   5.2. STEMI System— Dan Sitar (Attachment C)
   5.3. Stroke System— Dan Sitar (Attachment D)

6. **Other Reports**
   6.1. EMCC Report – Trevor Douville

7. **DISCUSSION ITEMS, UNFINISHED & NEW BUSINESS (60 Minutes)**
   7.1. CQI Update – Lisa Madrid (Attachment E)
   7.2. Education / Policy Update – Misty Plumley (Attachment F)
   7.3. Provider Recognitions – REMSA Clinical Team / Trevor Douville
   7.4. PMAC Membership Structure – Dr. V. (Attachment G)
   7.5 Airway Management in OHCA – Misty Plumley / Dr. V.
   7.6 Policy Updates proposed for Fall 2019 Implementation – Misty Plumley (Attachment H)
   7.6.1 Drowning/Submersion Policy – Misty Plumley, Emily Craig (Attachment I)

8. **REQUEST FOR DISCUSSIONS**
   Members can request that items be placed on the agenda for discussion at the following PMAC meeting. References to studies, presentations and supporting literature must be submitted to REMSA three weeks prior to the next PMAC meeting to allow ample time for preparation, distribution and review among committee members and other interested parties.

Loma Linda University Med. Center Murrieta
1-Kevin Flaig, MD
4-Kristin Butler

Menifee Valley Medical Center
1-Todd Hanna, MD
4-Janny Nelsen

Kaiser Permanente Moreno Valley
1-George Salameh, MD
4-Katherine Heichel-Casas

Palo Verde Hospital
1-David Sincavage, MD
4-Carmelita Aquines

Parkview Community Hospital
1-Chad Clark, MD
4-Guillean Estrada

Rancho Springs Medical Center
1-Zeke Foster, MD (Chair)
4-Sarah Young

Riverside Community Hospital
1-Stephen Patterson, MD
4-Sabrina Yamashiro

Riverside County Fire Department
5-Scott Visyak
8-Tim Buckley

Riverside County Police Association
7-Sean Hadden

Riverside University Health System Med. Center
1-Michael Mesisca, D
4-Kay Schulz

San Gorgonio Memorial Medical Center
1-Richard Preci, MD
4-Trish Ritarita

Temecula Valley Hospital
1-Pranav Kachhi, MD
4-Jacquelyn Ramirez

Trauma Audit Comm. & Trauma Program Managers
2-Frank Ercoli, MD
3-Charlie Hendra

Ex-officio Members:
1-Cameron Kaiser, MD, Public Health Officer
2-Reza Vaezazizi, MD, REMSA Medical Director
3-Bruce Barton, REMSA Director
4-Jeff Grange, MD, LLUMC
5-Phong Nguyen, MD, Redlands Community Hospital
6-Rodney Borger, MD, Arrowhead Regional Medical Center

9. ANNOUNCEMENTS (15 Minutes)

This is the time/place in which committee members and non-committee members can speak on items not on the agenda but within the purview of PMAC. Each announcement should be limited to two minutes unless extended by the PMAC Chairperson.

10. NEXT MEETING / ADJOURNMENT (1 Minute)

July 22, 2019—4210 Riverwalk Parkway First Floor Conference Rooms

11. CASE REVIEW SESSION (60 Minutes)

This is the time/place in which committee members and invited parties will participate in case review of sentinel events, or cases that are part of trends in patient care in the EMS System. Closed case review session for PMAC members and invited personnel.

Members are requested to please sit at the table with name plates in order to identify members for an accurate count of votes

Please come prepared to discuss the agenda items. If you have any questions or comments, call or email Misty Plumley at (951) 201-4705 / mplumley@rivco.org. PMAC Agendas with attachments are available at: www.rivcoems.org. Meeting minutes are audio recorded to facilitate dictation for minutes.

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TOPIC	DISCUSSION	ACTION
1. CALL TO ORDER	Misty Plumley called the meeting to order at 9:04 a.m. and reviewed housekeeping items before turning the meeting over to PMAC Chair Dr. Zeke Foster.	
2. PLEDGE OF ALLEGIANCE	Dr. Zeke Foster led the Pledge of Allegiance.	
3. ROUNDTABLE INTRODUCTIONS	Dr. Zeke Foster facilitated self-introductions.	
4. APPROVAL OF MINUTES		The October 22, 2018 PMAC meeting minutes were approved with no changes.
5. STANDING REPORTS		
5.1 Trauma System Updates	ImageTrend trauma registry will be going to the Board of Supervisors for approval this month. Upon approval, there will be education and training for the trauma centers prior to implementation. IVMC and RCH completed their American College of Surgeons (ACS) survey in November and both passed successfully. Now three of four trauma centers in Riverside County are ACS verified. Penetrating trauma protocol will be reviewed at TAC in March and brought back to PMAC for discussion in April.	Information only.
5.2 STEMI System Updates	State STEMI regulations (Title 22) were approved by the EMS Commission in September 2018 with an anticipated implementation in April 2019. The Riverside STEMI EMS Critical Care System is currently compliant with these pending regulations. Regulations also includes requirements for non-STEMI centers to participate and submit data to REMSA. 2018 year in review included quarter STEMI coordinators meeting and case reviews, the elimination of STEMI diversion, except during internal disasters, and treatment policies modified to expedite transport of STEMI and OHCA patients. Goals for 2019 includes: The implementation of the ImageTrend STEMI registry and the on boarding of required data registrars for STEMI centers. Anticipated start date begins first quarter 2019. Continued focus on recent policy changes regarding STEMI center destinations, outcome data regarding false/positives, and designing and implementing an education model to promote STEMI education to field providers.	Information only.

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	The next STEMI meeting is on January 17th, 2019.	
5.3 Stroke System Updates	State Stroke regulations were also approved by the EMS Commission in September 2018 with an anticipated implementation in April 2019. The Riverside Stroke EMS Critical Care System is near compliance with these pending regulations. 2018 year in review included AHA stroke guidelines published, then partially retracted, quarterly stroke committee meetings and case reviews, the change of stroke scale from Cincinnati to mLAPSS, LAMS was also added late 2018, and data reporting for hospitals aligned with GWTG and Coverdell elements. Goals for 2019 includes: Data in the future to determine stroke triage with updated stroke center designation criteria to include designation of facilities with neurointerventional capabilities. Creation of a Stroke Continuation of Care policy. Designing and implementing an educational model to promote stroke education to field providers. Begin submitting data to the California Stroke Registry and on boarding required stroke data registrars in first quarter of 2019. Dr. Vaezazizi emphasized the importance of adhering to strict hospital requirements to be compliant with Title 22 regulations. The next stroke meeting is on February 21st, 2019.	Information only.
6. OTHER REPORTS		
6.1 EMCC Report	No updates on EMCC now, next meeting postponed to January 23rd, 2019. Kristen Clements announced she is resigning from her position from EMCC effective immediately.	Information only.
7. DISCUSSION ITEMS, UNFINISHED & NEW BUSINESS		
7.1 CQI Update	From the last CQILT meeting on December 20th, 2018, discussions included the future of data to aid in system enhancements, which includes CORE measures changes at the EMSA level. Anticipated standardized process and manual end of January 2019. Integrating CPR report cards in case review discussions and data analysis.	Information only.

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	CQI of trauma data from ImageTrend and existing registries revealed some data issues in collection of elements related to trauma triage criteria patient met for transport to a Trauma Center and trauma risk factors that may have guided EMS providers to choose transport to a Trauma Center if trauma criteria not met above. Discussed updating the validation rule in hopes to capture all information in the future. The next CQILT meeting is on Thursday, March 21st, 2019.	
7.2 Education/Policy Update	Fall 2018 Policies and Procedures Update Courses concluded by mid-December 2018. We are now preparing for Spring 2019 PUC courses with train to trainer dates to come. PMAC had previously approved a series of policy additions; edits and comment phases have opened and closed. 2019 policy updates were included in attachment F, however the calculation chart edits for push dose epinephrine was missed on the list in attachment F, but will be included in 2019 updates. Going into 2019, we anticipate policy changes for Fall, so that we can work in the appropriate flow to push education and training before policies go into effect. Cal Fire had a question regarding standard drug and equipment list that missed the public comment phase before changes were made. They asked if fentanyl, ketamine or morphine had to all be carried or just at least one of them. Misty answered, Fentanyl should be listed as priority and Dr. suggested for carriers to all carry ketamine as well. All protocols say fentanyl or ketamine, however, ketamine is not always readily available due to a shortage. The provider asked if they could write a letter of leniency if there is a shortage of the drug listed. Dr. V answered yes, of course they can. We also would slowly like to move away from morphine for prehospital use and look towards alternative that may include acetaminophen.	
7.3 Provider Recognitions	Recognizing outstanding performance from our providers, Misty Plumley congratulated and thanked first responders and their team for exceptional service in patient care from three separate incidents: Norco, Menifee and Murrieta calls. Awards of Excellence were given to the recipients below:	Information only.

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	• Norco Call
	  o Michael Moore, Paramedic
	  o Aaron Nimmo, EMT
	  o Jonathan Blood, EMT
	  o Kevin Molloy, Paramedic
	  o John Shirokawa, EMT
	  o Rebecca Marodi, EMT
	  o Salvador De La Cruz, Paramedic
	• Menifee Call
	  o Michael Yardley, Paramedic
	  o Ryan Slagle, EMT
	  o Stacey Ballard, EMT
	  o Alexander Garcia, Paramedic
	  o Jeremy Brambila, EMR
	  o Michael Merrick, Paramedic
	• Murrieta Call
	  o Caleb Barone
	  o Richard Martinez, Paramedic
	  o Ryan Roufs, Paramedic
	  o Mathew Bentley, Paramedic
	  o Tammy Lord, Paramedic
	  o Richie Leyba, EMT	

7.4 PMAC Membership Structure	Discussion was had amongst PMAC members to revisit and change the PMAC membership structure to allow for a more balanced representation of voting members. A current draft proposal was sent out to Misty, to request for more representation from fire agencies. PMAC debated however, adding more fire agencies would only skew the members towards one side as opposed to redesigning to fill in the gaps. Discussion continued with ideas on building a constituency that encompasses all agencies that includes Hospital (Specialty Care/Non Specialty Care), Fire, Ambulance (ALS/BLS), Physicians, Medical Directors, Schools, Law Enforcements, Behavioral Health and any agency that would benefit from the system. In addition, each voting member would have two representatives, a primary and alternate for attending meetings and voting. Discussion concluded with the agreement for REMSA to bring a draft proposal to the next PMAC meeting in April. Misty agreed that if the public would like to see the current proposal received from Fire, she could send it out for review. Any agency or individual who would like to submit their ideas/suggestions, please email Trevor Douville.	Discussion only.
7.5 Use of King Airway in OHCA Management	Dr. Vaezazizi discussed the use of King Airway in OHCA Management. There is a current perception that King Airway is being used more and more often as a primary	

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	airway for cardiac arrest patients in the field. However, due to the potential of King Airway stopping the blood supply to the brain, Dr. would like to retrain paramedics to use high quality CPR and BVM instead. King Airway should only be used as a rescue airway if bagging or intubation does not work.	
7.6 Advanced Resuscitation Training	In discussion about Advanced Resuscitation Training, whether agencies are using AHA or ART, we would still like to focus on the basic principles of high quality CPR. We use AHA as our standard requirement, which is in our protocol; however, we are permissive to other training platforms as long as they go beyond the basics we require. A suggestion was made to focus on a new training piece regarding using end tidal CO2 as a target to defibrillation timing. We would be open to exploring this new concept; however, there is anxiety of a fragmented system if field providers are not using the same treatment protocols for patients cohesively regarding resuscitation. We can update our protocol to embrace a new training in addition to AHA, since this is a part of ART, but it would have to be done system wide as a whole and monitored as a trial system. Members raised a concern in an operational standpoint, on how to monitor and CQI this. It would be possible, but would take time. In the essence of time and proper training needed for adding this piece, the members agreed to move forward with everything else in ART and leave this piece for CQILT to review first and will discuss further about implementation at a future date.	
8. REQUEST FOR DISCUSSIONS	There were no requests at this time.	
9. ANNOUNCEMENTS		Information only.
10. NEXT MEETING/ADJOURNMENT	April 22nd, 2019 from 9:00 – 11:00 a.m. 4210 Riverwalk Parkway First Floor Conference Rooms.	Information only.

FOR CONSIDERATION BY PMAC

DATE: March 15, 2019

TO: PMAC

FROM: Shanna Kissel, RN, Assistant Nurse Manager

SUBJECT: Trauma System

1. *Tentative* training on ImageTrend trauma registry June 2019.
2. Trauma continuation of care policy updated per TAC request.
3. TAC made a recommendation for REMSA to review penetrating traumas within the appropriate REMSA protocols. Suggesting to remove transport to closest PRC with time frame and transport penetrating traumas to closest Trauma center. No changes made at this time.

ACTION: PMAC should be prepared to receive the information and provide feedback to REMSA.

FOR CONSIDERATION BY PMAC

Date: April 22nd, 2019

TO: PMAC

FROM: Dan Sitar, Specialty Care Consultant, RN

SUBJECT: STEMI System

1. State STEMI regulations (Title 22) were reopened for a short public comment period that ended February 7th. Implementation projected for July 2019. The Riverside STEMI EMS Critical Care System is currently compliant with these pending regulations. Please note that the regulations continue to include requirements for non-STEMI centers to participate and submit data to REMSA.
2. An EMS plan update for the STEMI Critical Care System is due six months following the implementation of the final regulations.
3. Image Trend STEMI Registry in the final stages of the purchasing process. Planning will occur through April with planned on-boarding of facilities in May. The development of a full registry greatly improves the data-driven CQI requirement of the Title 22 regulation language.
4. Policies:
    a. Continuation of STEMI Care policy updated to reflect changes across the continuation of care processes.
    b. ACS/STEMI treatment policy update.

Next STEMI Committee meeting is on July 18th, 2019 in the Vineyard room

Action: PMAC should be prepared to receive the information and provide feedback to the EMS Agency

FOR CONSIDERATION BY PMAC

Date: April 22nd, 2019

TO: PMAC

FROM: Dan Sitar, Specialty Care Consultant, RN

SUBJECT: Stroke System

1. State Stroke regulations were reopened for a brief public comment period that ended February 7th. Implementation anticipated July 2019. Riverside's Stroke EMS Critical Care System is now in compliance with these pending regulations.
2. An EMS plan update for the Stroke Critical Care System is due six months following the implementation of the final regulations.
3. Implementation of the California Stroke Registry continues. REMSA has also acquired a local Image Trend Stroke registry that allows for a backup data repository should the CSR project experience funding difficulties. Both registries will aid in correlating EMS care with hospital outcomes.
4. Policies:
    a. Stroke Center Designation policy finalized. Stroke centers interested in elevating to a Comprehensive or Thrombectomy-capable level may now apply for such designation. The application is available on the remsa.us website.
    b. Stroke Continuation of Care policy finalized. This policy does not limit interfacility transfers of any patient, but rather facilitates and expedites stroke transfers to designated stroke centers.

Next Stroke Committee meeting is on May 16th, 2019 in the Vineyard room

Action: PMAC should be prepared to receive the information and provide feedback to the EMS Agency

FOR CONSIDERATION BY PMAC

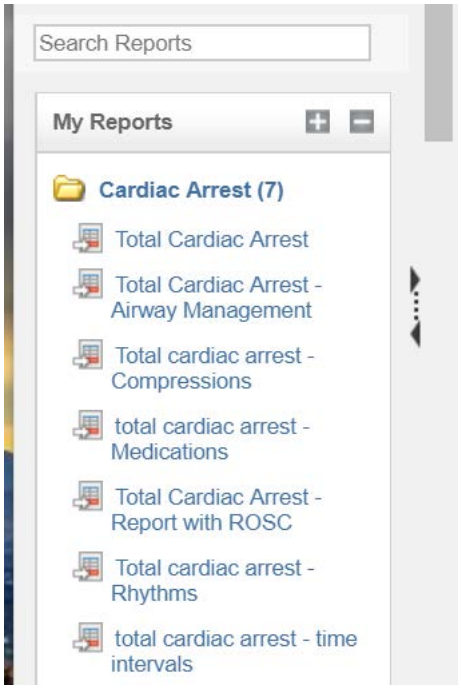
DATE: December 21, 2018

TO: PMAC

FROM: Lisa Madrid, EMS Specialist

SUBJECT: CQI

- The CORE measures manual for 2018 has not been released from the state as of 4-1-19. As soon as the manual is released the REMSA Data team will work on the changes to the report and then running and putting together the reports for EMSA.
- REMSA has built seven cardiac arrest reports. A peer review for cardiac arrest in the CQI Module has been created, if you would like to use this please let me know and I will add you as a reviewer. We will be closely auditing/monitoring this topic. The reports for cardiac arrest have been shared and are available for everyone to use/view.



- REMSA will also be monitoring the recent changes to the 2019-202 policy manual such as the uses of Push – Dose Epi.
- Over the next several months you can be on the lookout for CQI reports to be added to the SCOPE page.
- Our next CQILT meeting is on June 20th at 10:00 a.m.

FOR CONSIDERATION BY PMAC

DATE: April 22, 2019

TO: PMAC

FROM: Misty Plumley, Senior EMS Specialist

SUBJECT: Training and Education Update

First Quarter PUC 2019 updates were released, and training completed by providers prior to April 1, 2019.

Murrieta Fire & Rescue hosted a Resuscitation Academy training with speakers including their Medical Director – Dr. Foster, speakers from AMR, Riverside County FD/Cal Fire, and REMSA.

The Riverside County Emergency Management Department, in collaboration with Region VI partners, Emergency Managers and OC EMS Agency will be hosting our Southern California Preparedness Summit on May 8, 2019 at the Riverside Convention Center. Registration is free, breakfast and lunch are provided. We have an EMS Track this year as well – with 7 hours of free EMS CE credit available. Registration: http://www.cvent.com/events/southern-california-preparedness-summit/event-summary-30989ac23652472b9510da5bf02e2a27.aspx?i=fdecd906-c618-4082-a321-b787a8fe9ee1 and summit info: https://rivcoemd.org/summit

The CFED Conference will be hosted in May, http://www.cvent.com/events/cfed-conference-expo-2019/custom-21-91618233db0549209a6ea0ed211ddb8c.aspx

New Training/Education/Policy Updates:

In an effort to update several policies to current standards of care and address system CQI issues REMSA will be conducting a 4th Quarter training initiative focused on additional policy and procedure updates continuing our momentum from Spring 2018.

Proposed Policy Update: Pain Management Protocol

- Discussion of addition for Fall 2019 or implementation in Spring 2019

These next policy updates plan to address the policies, with adjunctive education for the below:

- REMSA 4102 Universal Patient Protocol (glucometer use with the EMT)
  - Remove the requirement of a paramedic being present to check BGL.
- REMSA 7501 Use of the Glucometer
  - Update for consistency for EMT use
- REMSA 4302 Traumatic Injuries and 4303 Burns
  - Removal of BHO for situations where morphine and fentanyl are combined
  - Removal of BHO for situations where ketamine and morphine/fentanyl are combined

FOR CONSIDERATION BY PMAC

- REMSA 4408 Respiratory Distress
    - Addition of magnesium sulfate for treatment of status asthmaticus
    - Addition of glucagon for foreign body airway obstruction unable to be relieved with direct laryngoscopy and Magill forcep usage
- REMSA 7310 Defibrillation
    - Clarification of verbiage: allowing stacked shocks in cases of witnessed VF/pulseless VT

Training and Education would also address the policies below:

- REMSA 4406 Cardiac Arrest

ACTION: PMAC recommendation needed as above. Information should be reviewed and a PMAC recommendation issued.

FOR CONSIDERATION BY PMAC

DATE: April 22, 2019

TO: PMAC

FROM: REMSA Clinical Team / Stakeholder Comment Compilation

SUBJECT: Proposed Updates for Fall Implementation 2019

REMSA
PREHOSPITAL MEDICAL ADVISORY COMMITTEE

APPOINTMENTS

MEMBER NAME	MAC POSITION	# OF REPS	APPOINTING AUTHORITY
	Trauma Hospital Physician	2	Trauma Center/TAC
	Pediatric Critical Care Physician	1	Pediatric Critical Care Hospital
	Non-Trauma Base Physician	2	Non-Trauma Base Hospitals
	Non-Base Hospital Physician	1	Non-Base Hospitals
	Public Transport Medical Director	1	Public Transport Providers
	Private Transport Medical Director	1	Private Transport Providers
	Fire Department Medical Director	1	Fire Chiefs
	PLN Committee Representative	1	PLN Committee
	EMS Officers	1	EMS Officers Committee
	Public Transport Medical Rep (Paramedic/RN)	1	Fire Chiefs
	Private Transport Medical Rep (Paramedic/RN)	1	Ambulance Association
	Specialty Center Medical Director	1	Specialty Center Committees
	Specialty Center (Trauma/STEMI/Stroke) Coordinator	1	Specialty Centers Committees
	Air Transport Medical Director	1	Air Transport Providers
	Medical Examiner's Office Pathologist	1	Riverside County Coroner's Office
	PSAP Medical Director	1	PSAPs
	Training Program representative	1	Training program
	Medical Director Appointee		REMSA Medical Director

FOR CONSIDERATION BY PMAC

DATE: April 22, 2019

TO: PMAC

FROM: Misty Plumley, Senior EMS Specialist

SUBJECT: Proposed Updated for Fall Implementation 2019

New Training/Education/Policy Updates:

In an effort to update several policies to current standards of care and address system CQI issues REMSA will be conducting a 4th Quarter training initiative focused on additional policy and procedure updates continuing our momentum from Spring 2019.

These next policy updates plan to address the policies, with adjunctive education for the below:

- REMSA 4102 Universal Patient Protocol (glucometer use with the EMT)
  - Remove the requirement of a paramedic being present to check BGL.
- REMSA 7501 Use of the Glucometer
  - Update for consistency for EMT use
- REMSA 7310 Defibrillation
  - Clarification of verbiage: allowing stacked shocks in cases of witnessed VF/pulseless VT

Training and Education would also address the policies below:

- REMSA 4406 Cardiac Arrest

Proposed New Policy/Policy verbiage addition related to Drowning/Submersion patient care.

ACTION: PMAC recommendation needed as above. Information should be reviewed and a PMAC recommendation issued to move above proposed policy changes to stakeholder comment phase.

FOR CONSIDERATION BY PMAC

DATE: April 22, 2019

TO: PMAC

FROM: Misty Plumley, Senior EMS Specialist
Emily Craig, Senior EMS Specialist RVCFD/Cal Fire

SUBJECT: Proposed Updates for Fall Implementation 2019

Proposed New Policy Updates:

Proposed New Policy/Policy verbiage addition related to Drowning/Submersion patient care.

Points of discussion for PMAC:

- Giving 5 initial breaths in victims of submersion who are unresponsive or altered can help reverse laryngospasm and may help prevent full respiratory or cardiac arrest
- Ventilating through foam rather than waiting for suction
- "Stopping the drowning process"
- Gastric decompression
- Encouraging transport even if asymptomatic or with minimal symptoms

I've attached some of the research/references that Emily compiled to support the discussion.

ACTION: PMAC recommendation needed as above. Information should be reviewed and a PMAC recommendation issued to move above proposed policy changes to stakeholder comment phase.

Resuscitation 82 (2011) 799–800

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Editorial

Rescue and resuscitation or body retrieval—The dilemmas of search and rescue efforts in drowning incidents

Drowning is the 3rd leading cause of unintentional injury death worldwide, accounting for nearly 400,000 deaths annually.[1,2] Global estimates may significantly underestimate the actual public health problem related to drowning. The care of the submersion victim is complex. It often involves a multi-agency approach with several different organisations being independently responsible for different phases of the victims care from the initial aquatic rescue, on scene resuscitation, transfer to hospital and hospital care.

A key question in the prehospital phase of care is the duration of submersion beyond which the chance of survival becomes negligible. Beyond this point the focus of care should shift from rescue and resuscitation to body recovery. Variation in practices between rescue and healthcare agencies has led to anecdotal reports of the opening of body bags and recommencement of resuscitative efforts on transfer of a victim from one agency to another. It is therefore timely that in this issue of *Resuscitation*, Michael Tipton and Frank Golden present the outcome of a multi-agency workshop set up to develop a guideline for the search, rescue and resuscitation of submersion victims.[3]

The authors conducted a pseudo systematic review of the literature. The term "pseudo" is used to reflect the fact that the approach differed from the approach advocated by organisations such as the Centre for Evidence Based Medicine[4] and the International Liaison Committee on Resuscitation.[5,6] Systematic reviews aim to collate all evidence that fits pre-specified eligibility criteria in order to address a specific research question. Systematic reviews aim to minimise bias by using explicit, systematic methods to identify and appraise evidence. The first step in a systematic review is to define the research question. The PICO approach (Population, Intervention, Comparator [if necessary], Outcome) provides a standardised format for asking focused questions and facilitates the literature search. The next step involves defining a search strategy and identifying the sources/databases from which evidence will be sought. In the ILCOR process, the Cochrane Database of Systematic Reviews and Register of Controlled Trials; Medline; EMBASE and AHA Master EndNote library were searched. Studies are selected on the basis of review of the title and abstracts and if relevant full text of the articles according to pre-defined inclusion and exclusion criteria. The level of evidence (in this setting using the levels of evidence for prognostic studies) and quality of studies are then summarised. The purpose behind this systematic approach is to produce a reproducible summary of evidence and to minimise bias in article selection and subsequent summary of evidence.

In the current review, the precise question for the review is not clearly defined. Search terms are defined although precisely how these were applied is unclear. The databases searched comprised a mix of peer reviewed (Cochrane, Medline, PubMed) and non-peer reviewed articles (Internet-based press cuttings and other media or news websites). The articles included in the review appear to be from English language journals only. The precise inclusion and exclusion criteria for including articles in the review is unclear. This may explain why some additional uncited articles were identified during the peer review process and leaves the reader uncertain as to the comprehensiveness of the literature review.

The clinical decision rule that was derived through the evidence appraisal and multi-agency consensus meeting prompts rescuers to undertake an initial assessment of water temperatures and duration of submersion. Rescue responses are then dichotomised on water temperature above or below 6 °C. For those in water temperatures below 6 °C guidance is provided to continue attempts at search and rescue for up to 90 minutes. For those in water temperatures above 6 °C (which includes coastal waters in many part of the US and Europe) survival is considered unlikely after 30 minutes implying that cessation of search and rescue efforts can be considered. Victims trapped in submerged vehicles are excluded from the decision rule as the possibility of a water pocket in a vehicle makes it difficult to estimate the duration of submersion/asphyxia.

Whilst recognising the need for common algorithms between rescue agencies and balancing the risks of on-going search and rescue efforts, the case for selecting solely water temperature and submersion duration is not described clearly. Whilst submersion duration is linked to survival in some case series[7,8] this association is not universal.[9] The interaction between environmental factors (e.g. water temperature, salinity) patient (e.g. age, co-morbidities, precipitating factors) and treatment factors (e.g. intubation, CPAP, bypass) further limit the prognostic value of single or pairs of factors. In the setting of such uncertainty, many care providers err on the side of caution in the development of clinical decision rules and protocols. It is therefore a surprise that the consensus group selected reducing the duration of rescue attempts from 90 to 30 minutes in victims found in water above 6 °C. This recommendation differs from those advocated by the US Lifesaving Association and Brazilian Resuscitation group which advise search and rescue should persist for 60 minutes from submersion. The Joint Royal College Ambulance Liaison Committee in the UK recommended efforts should continue for 90 minutes.

The attempt to reach consensus on this important topic is commendable, but the process through which the evidence presented in the review informed the clinical decision rule is unclear. The biological plausibility that a child could survive submerged for 30 minutes

0300-9572/$ – see front matter
doi:10.1016/j.resuscitation.2011.05.001

in 6 °C water as in the case series reported by Eich *et al* [10] yet not in water a degree or two higher is questionable. The fact that the review only identified three cases in water temperatures above 6 °C implies the evidence upon which this cut off is based is small. The gaps in the evidence highlight the need for further research in this area. However pending more definitive evidence it may be premature to abandon search and rescue efforts 30 minutes after submersion in water above 6 °C.

Conflict of interest

No conflict of interest to report.

References

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Letter to the Editor

Comments on editorial "Rescue and resuscitation or body retrieval—The dilemmas of search and rescue efforts in drowning incidents"

Sir,

Having referred to our "*pseudo systematic review*", Professor Perkins outlines the methodology for conducting a systematic review of the literature. We are aware of this methodology, however when examining the literature we were simply looking for the details of the scenario associated with prolonged underwater survival. Given the small number of relevant reports, and that the data relating to circumstance are often omitted, or are impossible to assess in terms of accuracy, we took the decision to summarise all of the papers we reviewed in Table 1 (i.e. present the raw data); this goes someway further than that required of a systematic review.

In Section 2.3 of our paper we discuss the difficulties of reviewing a body of the literature for specific pieces of information that may not be the central theme of that literature. Our considerations are not acknowledged in the editorial. Indeed, a recurring theme in the editorial is the use as criticisms, of our own considerations of the limitations, assumptions and caveats associated with the production of guidance in this area. For example, (i) The recommendations of various international lifesaving organisations are presented in the editorial as differing from our recommendations. It is not acknowledged that we present the policies of the UK, US and Brazil in our paper and explain that they have no evidence-base. We actually state that we corresponded with relevant international experts about these policies (Section 3.1), and we acknowledge their help. (ii) We are told in the editorial that several factors influence outcome and, by implication, we are criticised for using only duration and water temperature. However, our considerations of the usefulness of age, water salinity etc. in predicting outcome in cold water are ignored (see Section 4; Discussion). (iii) Our use of 6 °C in the guide is criticised in the editorial as being based on a small number of cases – this is a circular argument. We explain why we think few cases of prolonged survival exist in water above 6 °C (Section 4; Discussion). Thus, the "cut off" at 6 °C was created by the literature, and not imposed upon it.

The editorial concludes that "pending more definitive evidence it may be premature to abandon search and rescue efforts 30 min after submersion in water above 6 °C." Given all that is said in the editorial, it comes as something of a surprise to read a statement that is made without any attempt to assess, research or provide evidence to support the continuation of search and rescue beyond 30 min. That is, the existing recommendations appear to be accepted without scrutiny?

We undertook this work to assist the emergency services, desperate for some more evidence-based and consistent guidance in this area. We expected our paper to provoke a response and possibly identify new information that may require the guidance to be amended and refined. Until this occurs we are content with the recommendations we have made, with the associated qualifications and caveats that can be found in our original paper.

Conflict of interest statement

The authors have no conflict of interests to declare.

Yours faithfully,

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Letter to the Editor

Drowning: guidelines extant, evidence-based risk for rescuers?

Sir,

In recent editions of Resuscitation there has been an interesting and constructive debate on the topic of the rescue and resuscitation of submerged victims. This was prompted by a paper we wrote[1] which itself was prompted by a request for clear guidance on the issue from the emergency services.[2] Our guidance was clear, qualified and specific to those that are **submerged**; as evidenced by the title "*A proposed decision-making guide for the search, rescue and resuscitation of submersion (head under) victims based on expert opinion*".

On the basis of the available literature we concluded that, if water temperature is warmer than 6 °C survival/resuscitation is extremely unlikely if submerged longer than 30 min. If water temperature is 6 °C or below, survival/resuscitation is extremely unlikely if submerged longer than 90 min.[1]

In a recent edition of Resuscitation, in an Editorial under the heading "*Drowning: more hope for patients, less hope for guidelines*"[3] Professor Deakin reviews our guidelines in the light of two papers published in the same edition.[4,5] In contrast to Professor Deakin's conclusion, we can find nothing in the papers that changes our view, indeed just the opposite. It is important to understand how this position can come about, lest the issue gets clouded by semantics and confuses those still in search of clear, evidence-based guidance.

In his editorial, Professor Deakin uses "immersion" and "submersion" interchangeably; the editorial also includes a somewhat ambiguous statement "*.. it may be premature to abandon search and rescue efforts as soon as **30 min after submersion** in water above 6 °C*". We must be clear about this; it is submersion (head under) that we are interested in rather than immersion (head out), and our paper refers to those who ***remain submerged*** in water warmer than 6 °C and have not been found despite 30 min of searching. Professor Deakin's statement could be misunderstood as meaning that if an individual has had a brief period of submersion at the start or end of an immersion, resuscitative efforts should not be undertaken if 30 min have elapsed – this is not our position. We are interested in when the risk to rescuers – which is likely to increase with time - may outweigh the likelihood of finding a *submerged* individual who can be resuscitated. It follows that this guidance is particularly pertinent in conditions that place rescuers at high risk.

What do the papers reviewed by Professor Deakin actually add to the debate on the rescue and resuscitation of **submerged** victims? The answer is that neither paper provides data associated with prolonged submersion and subsequent survival that changes our original conclusion. The data of Claesson et al[4] are insufficient to make conclusions concerning water temperature, however when reporting accidents where divers were required (submersions), they state (page 4, under "3.3.3. Survival") "*The median submersion time was 15 min, no survivors were found after this time in the group with water temperatures >15 °C*". This would seem to provide some support for our original conclusions which were based on cases of prolonged underwater survival (Fig. 1.[1]). Further support is provided in a recent review on drowning[6] (Table 2) in which the risk of death or severe neurological impairment after hospital discharge is given as "nearly 100%" when the duration of submersion exceeds 25 min. Finally, the findings of Wanscher et al[5] are probably irrelevant to the current debate as they conclude that in the cases they review, which occurred in 2 °C salt water without submersion, *"circulatory arrest was most likely due to hypothermia rather than asphyxia (due to salt water aspiration)"*.

In his editorial Professor Deakin adds the median time taken for the rescue services to arrive at the scene in Sweden (8 min) to an *immersion* time of 20 min for survivors to conclude that this is a bit close ("*pessimistic*") to 30 min, and warrants a longer rescue phase. However, our advice in this area, which arose as a recommendation (R Hackwell, Maritime and Coastguard Agency) from the consensus conference we ran on this subject,[1] is that the clock used to determine elapsed time is not started until the emergency services arrive on the scene. This is, necessarily, standard practice when a submersion is not witnessed (often the case[4]), and even if it is witnessed, this approach avoids relying on the memory of those that may be stressed and distracted at the time. In connection with this, Professor Deakin reports[3] that Claesson et al[4] found that "*all survivors at one month* (after cardiac arrest due to drowning) *had been found within 20 min of arrival of the emergency services and 75% of these within 10 min*". For those in this category that were submerged outside of a vehicle ($n = 5$, Table 4[4]), four were found "*immediately*" and one within 10 min. One person was found in a vehicle within 10 min, but we advise caution when dealing with these cases as they may be submerged in an air pocket and therefore have an extended underwater survival time[1].

So what are the facts at this time?

a. Despite the large number of submersions that occur, we are still seeking evidence that individuals submerged for longer than 30 min in water warmer than 6 °C survive. Until we obtain this evidence we remain of the view that this is "extremely unlikely" and believe our guidance remains extant.
b. That something does not happen (survival) when it has frequent opportunities to happen (accidental submersion >30 min in water >6 °C), constitutes strong evidence that it (survival) is improbable.
c. No evidence has been presented for the requirement to search for 60 min or 90 min in water warmer than 6 °C. This means that when conditions are extreme, rescuers may be put at risk without foundation.

We re-iterate our previous conclusions [1,7]: what we have produced should only be regarded as a guide, and local circumstances and/or clinical signs may dictate an alternative course of action to

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the senior medical responder at the scene. It is likely to be of most use when rescuers are placed at high risk by continuing a search and subsequent rescue attempt. We expected our paper to provoke discussion and possibly identify new information that may require the guidance to be amended and refined. The papers referenced by Professor Deakin do not appear to provide such evidence, we therefore remain content with the recommendations we have made, with the associated qualifications and caveats that can be found in our original paper.

Conflict of interest statement

Professor Mike Tipton: Patron of the SARbot UK charity, Member of the RNLI's Medical & Survival Sub-Committee.

Dr Frank Golden: Nil return.

Dr Patrick Morgan: Registrar Anaesthesia and Critical Care. Gloucestershire Royal Hospitals NHS Trust, Gloucester. Great Western Air Ambulance. COI: RNLI Medical and Survival Sub-committee, Medical advisor SLSGB.

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WILDERNESS MEDICAL SOCIETY PRACTICE GUIDELINES

Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Drowning

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The Wilderness Medical Society convened a panel to review available evidence supporting practices for the prevention and acute management of drowning in out-of-hospital and emergency medical care settings. Literature about definition and terminology, epidemiology, rescue, resuscitation, acute clinical management, disposition, and drowning prevention was reviewed. The panel graded evidence supporting practices according to the American College of Chest Physicians criteria, then made recommendations based on that evidence. Recommendations were based on the panel's collective clinical experience and judgment when published evidence was lacking.

Key words: drowning, submersion, immersion, cold water submersion, hypothermia

Introduction

With an estimated annual worldwide human mortality of approximately 372,000, the burden of drowning as a global disease is self-evident.[1] Drowning often affects the young and can have dire personal, emotional, and financial consequences for patients, their families, and society. The goal of these practice guidelines is to reduce the burden of drowning through improvements in prevention, rescue, and treatment. We present preferred drowning terminology and a review and evaluation of the literature regarding acute care for the drowning patient in out-of-hospital and emergency medical care settings, with particular focus on the wilderness context. The experience and knowledge of a panel of wilderness and emergency medicine practitioners was used to make recommendations when little or unreliable evidence was available.[2]

Methods

A panel of reviewers was convened twice in 2013. Members were selected based on clinical and research experience. The panel included 5 emergency physicians and 1 pediatric physician, all of whom have practical wilderness medical experience, and several of whom have extensive experience in drowning prevention, education, and training.

Relevant articles were identified through PubMed, MEDLINE, and Google Scholar using a keyword search appropriate to each topic. Randomized controlled trials, observational studies, case series, and review articles were reviewed, and evidence was assessed. Abstracts for which the full article could not be obtained were excluded. If no relevant studies were identified, recommendations are based on the panel's clinical experience and judgment about potential risks of the recommended intervention vs its potential benefits. Recommendations are graded using the American College of Chest Physicians classification scheme, in accordance with prior versions of the Wilderness Medical Society Practice Guidelines (Table 1).[3]

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EPIDEMIOLOGY

The highest risk age group for drowning is children 1 to 4 years old in residential pools; the next highest risk group is adolescents and young adults in natural bodies of water. There were 46,419 recorded drowning deaths in the United States from 1999 to 2010, including

Table 1. American College of Chest Physicians classification scheme for grading evidence in clinical guidelines

Grade	*Description*	*Benefits vs risks and burdens*	*Methodological quality of supporting evidence*
1A	Strong recommendation, high-quality evidence	Benefits clearly outweigh risks and burdens or vice versa	RCTs without important limitations or overwhelming evidence from observational studies
1B	Strong recommendation, moderate-quality evidence	Benefits clearly outweigh risks and burdens or vice versa	RCTs with important limitations or exceptionally strong evidence from observational studies
1C	Strong recommendation, low-quality or very low-quality evidence	Benefits clearly outweigh risks and burdens or vice versa	Observational studies or case series
2A	Weak recommendation, high-quality evidence	Benefits closely balanced with risks and burdens	RCTs without important limitations or overwhelming evidence from observational studies
2B	Weak recommendation, moderate-quality evidence	Benefits closely balanced with risks and burdens	RCTs with important limitations or exceptionally strong evidence from observational studies
2C	Weak recommendation, low-quality or very low-quality evidence	Uncertainty in the estimates of benefits, risks and burden; benefits, risk and burden may be closely balanced	Observational studies or case series

RCT, randomized, controlled trial.

boating accidents; an average of 3868 deaths per year, or about 10 per day.[4] Based on World Health Organization and Centers for Disease Control and Prevention (CDC) systems for classifying drowning statistics, these numbers exclude deaths occurring during floods and other natural disasters. In 2010, there were 12,900 emergency department (ED) visits for drowning, with 20% of patients admitted to the hospital. Drowning deaths were 48% more likely on weekends. Fifty-three percent of all male and 26% of all female drowning deaths occurred in natural bodies of water.[4,5]

TERMINOLOGY

The standard definition for drowning, as defined by the World Congress on Drowning in 2002, is "the process of experiencing respiratory impairment due to submersion or immersion in liquid." Inspired by the Utstein Style for reporting cardiac arrest data, the standard definition allows for only 3 outcomes after drowning: 1) morbidity, 2) no morbidity, and 3) mortality. The following modifier terms should *not* be used to categorize "drowning" patients and events: near, wet, dry, active, passive, salt-water, freshwater, or secondary. Although previously thought to be of physiologic relevance (salt vs fresh, wet vs dry), years of data related to human drowning pathophysiology show that these are not valid distinctions because the final common pathway is hypoxemia and eventual cardiopulmonary arrest.[2,6,7] By understanding and using the standard definition for drowning and abstaining from using outdated terminology, communication between medical practitioners, data collection agencies, researchers, and policy makers may become more consistent, thereby more accurately reflecting the true incidence, prevalence, and sequelae of drowning.

Rescue of the Drowning Patient

RESCUER SAFETY

Rescuer safety is paramount during all rescue operations; in the aquatic environment, a specific set of skills, training, and physical capabilities is required. Technical rescue in the aquatic environment can range from swift-water to ocean, lake, scuba, and ice rescue, each requiring different sets of equipment and training. Few studies objectively measure effectiveness of in-water rescue techniques, and much of the literature on this topic is based on the experiences and policies of the writer or organization authoring the text. There is evidence for a high prevalence of fatal and nonfatal drowning of untrained persons attempting to perform in-water rescues.[8–10] Hazardous water conditions that led to the initial person drowning often still exist and place a well-intentioned rescuer at risk for becoming an additional drowning patient.[11] Rescue by untrained persons should be attempted without entering hazardous conditions by reaching to the drowning patient with a paddle or branch, throwing a rope, buoy, cooler, or any floating object, or safely rowing a boat, canoe, or paddleboard to the patient. Trained rescue personnel should

operate according to their level of training, expertise, equipment, and comfort level. Based on the inherent risk of performing any technical rescue without appropriate training and equipment, entering the water to effect a contact rescue should be attempted only by persons with specific training to operate in that environment.

Recommendation: Given the risks associated with in-water technical rescue, persons without formal water rescue training should only attempt rescues from a safe location by reaching, throwing, or rowing to the drowning patient. Persons with formal water rescue training should perform in-water rescues according to their level of training and with appropriate personal protective and safety equipment. (Recommendation grade: 1C)

REACHING THE PATIENT

Persons not formally trained in technical rescue in specific aquatic environments (eg, swift water, ice, open water) should avoid entering the water or making direct contact with a drowning patient and seek alternative means for accessing them. The mantra of "Reach, Throw, Row, Don't Go" should be used; these principles may include use of buoyant objects from the surrounding area or piloting a vessel to the patient. Few studies have been conducted on the effectiveness of different water safety devices (eg, rescue tubes, rescue cans, throw bags, life rings), but what has been demonstrated is that proper and effective use of these devices requires basic knowledge of their function and regular practice.[12] Any trip in which water rescue devices may be used should be preceded by competency training for all participants.

Recommendation: Persons without formal technical rescue training in aquatic environments attempting a water rescue should do so by avoiding water entry and direct patient contact, according to the mantra "Reach, Throw, Row, Don't Go." There is insufficient evidence to recommend specific rescue devices; if any specialized rescue equipment is to be used during an excursion, participants should be made familiar with the location and purpose of this equipment, and designated rescue personnel with proper training in its use should be tasked with its deployment in the case of a water rescue. (Recommendation grade: 1C)

PATIENTS IN SUBMERGED VEHICLES

Death caused by entrapment and drowning in submerged vehicles is often not classified as a drowning death, confounding attempts to accurately track the epidemiology of this type of drowning.[13] Studies suggest that 10% of drowning deaths may be caused by entrapment in submerged vehicles and that in the case of inland flooding, as many as 10% of motor vehicle crashes result in a drowning death.[14–18] There is a small body of medical and rescue literature on the topic of vehicle submersions.[16,19–23] A formal review of educational and public service information identified "three probable significant contributors to [the] high fatality rate [of drowning in submerged vehicles]: 1) 'authorities' provide an inadequate description of vehicle sinking characteristics; 2) contradictory and inadequate advice is often provided; and 3) [there is] a poor public perception of how to escape."[23] Specifically, several sources recommend questionable practices without any supporting evidence for efficacy. These include allowing the passenger compartment to fill with water so that it will be easier to open doors, waiting until the vehicle sinks to the bottom of a body of water to maintain orientation, relying on kicking out the windshield or opening doors after the vehicle has fully sunk, and relying on breathing trapped air in the passenger compartment. In a formal survey, more than half of the general public identify an option that involves staying in a vehicle while it sinks to the bottom as being the safest option when trapped in a submerging vehicle; this advice appears in the popular media.[23] However, research experiments and data derived from 35 vehicle submersions conducted in diverse locations and seasons suggest that this advice is erroneous and that the best time to escape from a submerging vehicle is immediately during the initial floating phase, ideally during the initial 30 seconds to 2 minutes after water entry when most vehicles remain partially above the surface.[23] One US-based proprietary prehospital dispatch system has created an additional card addendum to its standardized protocols instructing emergency medical dispatchers not to persist in getting a location for a caller in a submerging vehicle as would be the case for all other callers. Instead, it recommends that a caller exit the vehicle immediately if it is submerging, before using precious time to determine location.[24]

Recommendation: The safest time to escape from a submerging vehicle is immediately after it enters the water, during the initial floating phase. If it remains floating, persons should climb out and remain on top of the vehicle. If it is sinking, they should move away from the vehicle and toward safety after exiting. (Recommendation grade: 2C)

IN-WATER RESUSCITATION

The primary physiologic insult in a drowning patient is that of cerebral hypoxia; its rapid reversal is the primary objective of drowning resuscitation. There are situations in which a rescuer reaches the drowning patient in the water and is faced with the decision to extricate the patient or to initiate resuscitation while in the water. For

the purpose of these guidelines, in-water resuscitation (IWR) is defined as an attempt to provide ventilations to a drowning patient who is still in the water; this does not apply to chest compressions. It is not possible to perform adequate chest compressions while in the water and they should not be attempted.[25] Successful use of IWR was first described in 1976, then verified through feasibility studies on a manikin in 1980; however, the first clinical study to show a positive patient outcome was not published until 2004.[26–28] Available outcome data for IWR with ventilations are based on a single retrospective analysis of lifeguard rescues in Brazil, and show significant improvement in survival and neurological outcome in persons receiving IWR. These rescues were performed by trained, professional lifeguards in the ocean environment. Lifeguards had helicopter backup and would frequently tow the patient beyond breaking waves and perform mouth to mouth ventilations while awaiting helicopter pickup.[28] Subsequent studies, primarily using manikins, evaluated ease of performing this task in controlled aquatic environments and found that IWR increases overall rescue time, subjective rescue difficulty, number of submersions, and water aspiration.[29,30] A single study comparing lifeguards with lay rescuers when using IWR found that lifeguards showed improved rescue times and decreased estimated pulmonary aspiration.[31] Consensus statements from the International Lifesaving Federation, United States Lifesaving Association, American Red Cross, and the Young Men's Christian Association recommend IWR by trained rescuers when a patient is rescued in shallow water or in deep water when a flotation device is present.[32,33]

Rescuer safety and prevention of communicable diseases are of utmost importance, so consideration should be given to the use of barrier devices during IWR. US Food and Drug Administration–approved, IWR-specific devices are available that use a self-purging mechanical one-way valve instead of the paper valve on standard cardiopulmonary resuscitation (CPR) masks.[34]

Recommendation: The decision to perform IWR should only be considered by a rescuer with the adequate training and ability to check for a pulse in the water and to safely perform the skill. To benefit from rescue breathing alone, the drowning patient must have a pulse and be unconscious with inadequate or absent respirations. The aquatic conditions must be sufficiently safe for the rescuer to perform IWR, and the point of extrication (boat, shore, etc) must be sufficiently distant to warrant an attempt of this technically difficult task. If conditions are too hazardous to safely perform the task, or if the patient is pulseless, rapid removal from the water is indicated without a delay for IWR. Chest compressions should not be attempted in the water; all drowning patients without a pulse should be extricated as quickly and safely as possible so that early, effective chest compressions and ventilations can be initiated. (Recommendation grade: 1C)

Initial Resuscitation

HYPOTHERMIA

Water is thermally neutral at approximately 32.8°C (91°F), and most patients will drown in water at a temperature lower than this, so concomitant hypothermia is common in drowning.[14] Reversal of hypothermia is paramount in initial resuscitation of a drowning patient. Beyond initiation of basic warming measures, the details of hypothermia treatment, including augmented advanced life support measures, are beyond the scope of these guidelines. Readers are encouraged to review the Wilderness Medical Society Practice Guidelines for the Out-of-Hospital Evaluation and Treatment of Accidental Hypothermia: 2014 Update.[35]

Recommendation: Treat hypothermia aggressively with active and passive measures dependent on patient conditions and available resources. (Recommendation grade: 1C)

CARDIOPULMONARY RESUSCITATION AND PRIORITIZATION OF AIRWAY

Because of the central role of hypoxemia in drowning, initial resuscitation should focus on establishing and maintaining a patent airway and providing oxygen. Recent updates to CPR algorithms, specifically for the lay rescuer, include recommendations for compression-only CPR and prioritization of compressions before airway maneuvers.[36,37] Owing to the underlying pathophysiology of drowning, these changes do not apply to the drowning patient. If the airway is overlooked in initial resuscitation, ongoing hypoxemia leads to decreased survival and worse neurological outcomes.

Recommendation: Interruption of the drowning process as quickly as possible by supplying oxygen to the brain is critical to successful resuscitation of the drowning patient. Establishing an airway and providing oxygen are priorities in the initial resuscitation of a drowning patient. For the patient in cardiac arrest, provide positive-pressure ventilations in addition to chest compressions using the traditional Airway-Breathing-Circulation model of resuscitation. If an advanced airway is available and properly placed, provide breaths at specified time intervals (every 6 to 8 seconds) while continuous compressions are administered. (Recommendation grade: 1C)

OXYGENATION

Few large-scale studies have evaluated different airway adjuncts applied to drowning patients. Although ideal delivery of rescue breaths includes supplemental oxygen and a positive-pressure delivery device, any amount of oxygen delivery (eg, mouth-to-mouth, bag-valve-mask [BVM] with ambient air) is better than none if supplemental oxygen is not available. As a result of direct pulmonary injury and airway edema from drowning, certain supraglottic airway devices may be difficult to use for oxygenation based on leak pressures; instead, a BVM should be used if it achieves adequate chest rise.[38]

Recent resuscitation data have brought into question the benefit of providing high oxygen concentrations in the acute setting of out-of-hospital cardiac arrest and stroke, primarily based on data correlating hyperoxia after return of spontaneous circulation (ROSC) with increased mortality. Most of these data focus on the period after ROSC in the intensive care unit setting, and no studies focus specifically on cardiac arrest associated with drowning or other primary respiratory events. A single retrospective case-control study involving arterial blood analysis during CPR provides support for using high levels of supplemental oxygen. This study showed a significant increase in survival to hospital discharge with increasing levels of arterial oxygenation, in all cardiac arrest patients, even at levels that would be considered hyperoxic.[39]

Recommendation: For resuscitation of a drowning patient, oxygen should be delivered at the highest concentration available based on the patient's tolerance and available resources or provider training. For the patient in respiratory distress or arrest, providing positive-pressure ventilations is preferred over passive ventilation. If multiple modalities are available, the method that most effectively delivers the highest concentration of oxygen should be used. If a modality or device fails, BVM or mouth-to-mouth ventilation should be attempted. (Recommendation grade: 1C)

AUTOMATED EXTERNAL DEFIBRILLATOR

Although cerebral anoxia is the primary cause of morbidity in the drowning patient, hypoxic myocardial injury may also occur. Drowning patients typically experience sinus tachycardia as a result of the initial struggle, followed by bradycardia, pulseless electrical activity, and then asystole, owing to the hypoxic nature of the event.[40] In drowning patients, ventricular fibrillation (VF) is rare, occurring in less than 10% of patients in the published literature; thus, reversal of hypoxia with ventilations and compressions should not be delayed in an attempt to apply an automated external defibrillator (AED).[40–46] Early application of an AED may be beneficial, given the possibility of a VF as the cause or result of drowning, and should be considered if available. In any drowning patient, if global myocardial hypoxia persists, attempts at defibrillation may be unsuccessful without concomitant oxygenation and ventilation.

Experimental animal models have shown that as long as AED pads are placed firmly on a patient's chest and a rescuer is not in direct contact with that patient, use of an AED in a wet environment does not pose increased risk to the patient or rescuers.[47–50] AEDs have been tested and noted to correctly detect simulated arrhythmias and deliver shocks on moving boats.[51]

Recommendation: Ventricular fibrillation is rare in drowning, so incorporation of an AED in the initial minutes of drowning resuscitation should not interfere with oxygenation and ventilation. If available, an AED should be used during resuscitation of a drowning patient, and its use is not contraindicated in a wet environment. (Recommendation grade: 1A)

HEIMLICH MANEUVER

Drowning involves water obstructing the airway, causing cerebral hypoxia; in some cases, small amounts of water are aspirated into the lungs. This can cause atelectasis, direct cellular injury, and pulmonary edema. Even after unconsciousness, reflex swallowing of water from the hypopharynx into the stomach may occur. Heimlich advocated the use of abdominal thrusts in initial treatment of the drowning patient, claiming that aspirated water must first be cleared from the airway to allow proper ventilations.[52–54] In the 30 years since his original report, a great amount of concern has been raised about this recommendation, resulting in an Institute of Medicine report and 3 systematic literature reviews by the American Red Cross.[55,56] All of these investigations failed to identify quality data to support use of the Heimlich maneuver before providing ventilations. Its use during initial resuscitation delays delivery of ventilations and prolongs hypoxemia.[55]

Recommendation: Owing to the possibility of delaying ventilations, the Heimlich maneuver is not recommended for resuscitation of the drowning patient. (Recommendation grade: 1B)

CERVICAL SPINAL IMMOBILIZATION

Retrospective studies of drowning patients found the incidence of cervical spine injuries was low (0.5%–5%) and that most injuries were related to diving from a height.[57,58] Without obvious signs of trauma or a known fall or diving event, routine cervical spine

immobilization is unnecessary and may distract rescuers from the critical role of oxygenation and ventilation. In accordance with the Wilderness Medical Society Practice Guidelines for Spine Immobilization in the Austere Environment,[59] spinal immobilization should be considered in the setting of blunt trauma in association with any of the following:

1. Significant mechanism for cervical spine injury
2. Altered mental status (Glasgow Coma Scale [GCS] < 15; evidence of intoxication)
3. Focal neurological deficit
4. Significant distracting injury

Recommendation: Spinal immobilization should be considered in patients with evidence of spinal injury, such as focal neurological deficit or history of high-risk activity, and in patients who exhibit altered mental status. Spinal immobilization should not take priority over initial resuscitation of a patient with severe respiratory distress who requires aggressive airway management. (Recommendation grade: 1C)

Postresuscitation Management

VENTILATION

Mechanical Ventilation

No literature is available comparing out-of-hospital or in-hospital mechanical ventilation strategies for the drowning patient. Current practice recommends a lung-protective ventilation strategy similar to that used for patients with acute respiratory distress syndrome (ARDS), on the premise that the lung injury pattern after drowning is similar.[6,60,61] This includes mechanical ventilation starting with a tidal volume (Vt) of 6 to 8 mL/kg, augmentation of Vt and respiratory rate to maintain plateau pressure less than 30 mm Hg, and augmentation of positive end-expiratory pressure (PEEP) and fraction of inspired oxygen (F_{IO_2}) to maintain arterial partial oxygen pressure (Pa_{O_2}) at 55 to 80 mm Hg.[62]

Recommendation: Mechanical ventilation for the drowning patient should follow ARDS protocols. (Recommendation grade: 1C)

Noninvasive Positive-Pressure Ventilation

Noninvasive positive-pressure ventilation (NIPPV) has been used successfully in the prehospital setting. There are case reports describing its successful use in drowning.[63–65] However, caution should be used with NIPPV in the drowning patient with altered mental status because there may be an increased risk of vomiting and aspiration. Drowning patients who have mild to moderate hypoxemia and are being treated in prehospital and emergency medical systems familiar with NIPPV may benefit from this therapy.

Recommendation: NIPPV may be used in the alert patient with mild to moderate respiratory symptoms. Caution should be taken with any patient displaying altered mental status or active emesis because of the potential for aspiration. (Recommendation grade: 2C)

DIAGNOSTICS

Radiologic Testing

Several retrospective ED studies of drowning patients found that the initial chest radiograph did not correlate with arterial blood gas levels, outcome, or disposition.[66,67] A study of admitted drowning patients showed that those who went on to exhibit acute lung injury or ARDS had abnormal chest radiograph findings within the first few hours, but not necessarily on arrival to the ED.[60] Head computed tomography (CT) imaging has been studied in an attempt to quantify anoxic brain injury in drowning patients. Retrospective studies have found that patients with abnormal initial CT scans all went on to experience severe brain injury or die, whereas initially normal head CT scans had no prognostic value.[68]

Recommendation: Initial chest radiograph findings do not correlate with arterial blood gas measurements or outcome; chest radiographs may be useful in tracking changes in patient condition, but not for determining prognosis if obtained at the time of presentation. A normal initial head CT scan does not have prognostic value in the drowning patient. Routine use of neuroimaging in the awake and alert drowning patient is not recommended unless dictated by a change in clinical status. (Recommendation grade: 1C)

Laboratory Testing

Canine studies performed in the 1960s showed clinically significant hemodilution and red blood cell lysis associated with salt, chlorine, and freshwater drowning.[69–71] These studies were based on instilling up to 44 mL/kg of fluid into the trachea of anesthetized dogs, far greater than the 1 to 3 mL/kg typically aspirated by human drowning patients. Electrolyte abnormalities and hemodilution only occurred in dogs that had 11 mL/kg or greater instilled. No studies have identified clinically significant electrolyte or hematologic abnormalities in drowning patients that help guide initial therapy or provide prognostic information. In patients with altered mental status or decreased level of consciousness, laboratory evaluation for alternative causes that may have led to the drowning event, such as hypoglycemia or

intoxication, may be helpful. Arterial blood gas analysis in symptomatic patients may be used to help guide initial respiratory resuscitation.

Recommendation: Routine use of complete blood count or electrolyte testing in the drowning patient is not recommended. Arterial blood gas testing in patients with evidence of hypoxemia or respiratory distress (eg, cyanosis, low oxygen saturation, tachypnea, persistent tachycardia) may be indicated to guide respiratory interventions. For patients whose mental status fails to respond to resuscitation or in whom the initial cause of submersion is unknown, laboratory testing for causes of altered mental status should be considered. (Recommendation grade: 1C)

OTHER TREATMENTS

Antibiotics

Although microorganisms present in aspirated water may eventually cause pneumonia, no studies to date have shown benefit from empiric administration of antibiotics in drowning patients. This is related in part to the fact that microorganisms found in drowning-associated pneumonia are atypical bacteria or fungi and are often resistant to standard empiric treatments.[72–74] Aspiration of even small volumes of water can produce abnormalities on chest radiograph that may mimic pneumonia. The trauma of the drowning event and hypoxemia can cause leukocytosis from stress demargination as well as fever from inflammation and irritation caused by water in the airways, making it difficult to differentiate inflammatory from infectious pneumonitis.[75] The decision to administer antibiotics should be made after initial resuscitation and ideally be based on expectorated sputum or endotracheal aspirate bacterial culture, blood cultures, or urinary antigen tests.[72–74] As these tests are not available in the wilderness setting, treatment should be initiated for symptoms consistent with pulmonary infection (eg, fever, increased sputum, abnormal lung auscultation) that continue after initial resuscitation and treatment phases.

Recommendation: There is no evidence to support empiric antibiotic therapy in the treatment of drowning patients. After initial resuscitation, if pneumonia is present, treatment should be guided by expectorated sputum or endotracheal aspirate bacterial culture, blood cultures, or urinary antigen tests. In the absence of these tests, decision to treat should be based on clinical examination focusing on physical evidence of pulmonary or systemic infection (eg, fever, increased sputum, abnormal lung auscultation). (Recommendation grade: 1A)

Corticosteroids

Corticosteroids were historically used in drowning patients to facilitate pulmonary recovery and surfactant production. A systematic review of 35 years of literature found no randomized controlled trials regarding their use; of the pertinent studies reviewed, all but one were retrospective or case studies. Low study patient numbers and varying corticosteroid regimens further hindered comparisons. There is not sufficient evidence to support empiric corticosteroid administration for drowning patients.[76]

Recommendation: Given limited data, corticosteroids should not be routinely administered specifically for treatment of drowning patients. (Recommendation grade: 1C)

Therapeutic Hypothermia

Mild therapeutic hypothermia (TH) has been shown to decrease cerebral oxygen utilization and improve neurologically intact survival in patients with witnessed VF cardiac arrest.[77] Current American Heart Association/International Liaison Committee on Resuscitation guidelines recommend that survivors of out-of hospital cardiac arrest with an initial rhythm of VF be cooled to 32°C to 34°C (90°F to 93°F) for 12 to 24 hours.[78] Many institutions have extrapolated these data to include non-VF causes of cardiac arrest.

The 2002 World Congress on Drowning provided a consensus statement recommending TH of 32°C to 34°C (90°F to 93°F) for patients achieving ROSC after cardiac arrest caused by drowning.[79] Our literature search yielded multiple case reports and retrospective reviews supporting neurologically intact survival in hypothermic patients, but several older studies showed no benefit.[80–92] There are no prospective studies comparing TH with normothermia after ROSC in drowning patients. There may be benefit to discontinuing rewarming interventions after a hypothermic drowning patient has reached TH temperature range, but this has been insufficiently studied to support an evidence-based recommendation.

Recommendation: Although current literature shows there may be benefit to TH in witnessed VF arrest, there is insufficient evidence to either support or discourage induction or maintenance of TH in drowning patients. (Recommendation grade: 2C)

Disposition in the Wilderness

DECISION TO EVACUATE

If a patient survives a drowning event in the wilderness, objective physical examination findings may assist in the decision to evacuate the patient to advanced medical care. A single large retrospective study of nearly 42,000 ocean lifeguard rescues serves as the primary evidence for

on-scene decision-making.[93] This study found that patients who experienced a drowning event but had no symptoms other than mild cough and did not have abnormal lung sounds had 0% mortality. As symptoms worsened and abnormal lung sounds appeared, mortality increased. A subsequent drop in blood pressure (to systolic blood pressure $<$ 90 mm Hg or mean arterial pressure $<$ 60 mm Hg) accounted for the next largest increase in mortality (Table 2). In a retrospective study of children who experienced nonfatal drowning, any clinical deterioration occurred within the first 4 hours in patients presenting with mild symptoms and GCS $\geq$ 13.[66] These findings are similar to those from another retrospective study of pediatric patients in which new symptom development after arrival to the hospital occurred within 4.5 hours in all but 1 patient; the final patient exhibited symptoms in 7 hours and had a good outcome.[94]

Recommendation: Any patient with abnormal lung sounds, severe cough, frothy sputum, or foamy material in the airway; depressed mentation; or hypotension warrants evacuation to advanced medical care if risks of evacuation do not outweigh potential benefit. Any patient who is asymptomatic (other than a mild cough) and displays normal lung auscultation may be released on scene. If evacuation is difficult or may compromise the overall expedition, patients with mild symptoms and normal mentation should be observed for 4 to 6 hours. Any evidence of decompensation warrants prompt evacuation if the risks of evacuation do not outweigh the potential benefit. If evacuation of a mildly symptomatic patient has begun and the patient becomes asymptomatic for 4 to 6 hours, canceling further evacuation and continuing previous activity may be considered. (Recommendation grade: 1C)

CEASING WATER-BASED RESCUE AND RESUSCITATION EFFORTS

A search and rescue team may range from a small group of untrained participants with no equipment to a highly trained team with extensive resources. In the wilderness setting, available resources, risk to rescuers, and team safety must be considered when deciding how much time to search for a submerged patient. Although each drowning episode has unique patient and environmental factors, the most important predictor of outcome is duration of submersion.[46,95,96] Available evidence shows that prognosis is poor with submersion times greater than 30 minutes, regardless of water temperature.[97] There are also case reports of survival with good neurologic outcome despite prolonged submersion, predominantly in children 6 years and younger in water less than 6°C (43°F), and with use of advanced treatment modalities, such as extracorporeal membrane oxygenation.[98–103] For the purpose of these guidelines, recommendations are based on available evidence relevant to a typical drowning patient, and on the probability of neurologically intact survival in specific conditions. A literature review of 43 cases serves as the evidence for water-based rescue.[104] The report concludes that there is minimal chance of neurologically intact survival with submersion time greater than 30 minutes in water greater than 6°C (43°F), or greater than 90 minutes in water less than 6°C (43°F). It is important to note that "submersion time" was defined as beginning on arrival of emergency services personnel as total submersion time is often unknown.

If a drowning patient is removed from the water and resuscitation takes place, it may be necessary to decide when to cease resuscitation efforts if no signs of life return. Based on available evidence, primarily retrospective studies, submersion times of greater than 10 minutes appear to correlate with increased mortality or survival with severe neurological dysfunction.[46,96,105] In addition, more than 25 minutes of resuscitation or prolonged time to advanced medical care also correlate with negative outcomes, but without the statistical significance of submersion time. In a Dutch retrospective review of 160 hypothermic drowning patients younger than 16 years, 98 children received

Table 2. Prehospital management and classification of drowning patients

Grade	*Pulmonary examination*	*Cardiac examination*	*Mortality (%)*
0	Normal auscultation, - cough	Radial pulses	0
1	Normal auscultation, + cough	Radial pulses	0
2	Rales, small foam in airway	Radial pulses	0.6
3	Acute pulmonary edema	Radial pulses	5.2
4	Acute pulmonary edema	Hypotension	19
5	Respiratory arrest	Hypotension	44
6	Cardiopulmonary arrest		93

Adapted from Cushing et al.[13]

CPR for more than 30 minutes, with only 11 surviving to discharge, all of whom were neurologically devastated.[97,105–107]

Recommendation: Based on resources, it may be reasonable to cease rescue and resuscitation efforts when there is a known submersion time of greater than 30 minutes in water greater than 6°C (43°F), or greater than 90 minutes in water less than 6°C (43°F), or after 25 minutes of continuous CPR. If at any point during search and rescue efforts the safety of the rescue team becomes threatened, rescue efforts should be ceased. If resources are available and recovery team safety is maintained, body recovery efforts may continue beyond the search and rescue period with the understanding that resuscitation attempts will likely be futile. (Recommendation grade: 1C)

Disposition in the Emergency Department

Although many studies have addressed prognostic factors for neurological survival at hospital discharge, only a few have addressed the question, "Which patients can be safely discharged from the ED?" The first, a prospective study of primarily pediatric patients, included follow-up phone interviews with 33 patients who were either released on scene or discharged from the ED within 1 to 6 hours of arrival, and found that none of these patients experienced delayed effects.[108] A retrospective review of 48 pediatric drowning patients who presented to a single ED with a GCS of at least 13 studied whether factors predicting safe ED discharge could be identified.[66] Initial chest radiograph did not correlate with severity of disease, and all patients who deteriorated did so within 4 hours of ED arrival. The authors concluded that patients could be safely discharged home if normalized and there was no deterioration in respiratory function after 4 to 6 hours of observation in the ED. A retrospective review of hospitalized pediatric patients found that in all patients who were initially asymptomatic, but who went on to exhibit symptoms during their stay, these symptoms developed within 4.5 hours in all but 1 patient, and within 7 hours in the final patient.[94]

Recommendation: After an observation period of 4 to 6 hours, it is reasonable to discharge from the ED a drowning patient with normal mental status in whom respiratory function is normalized and no further deterioration in respiratory function has been observed. (Recommendation grade: 2C)

Prevention

Analogous to smoking cessation and wearing seatbelts, prevention holds the potential to save far more lives than rescue or treatment of a drowning person. A comprehensive prevention program includes participant screening for medical diseases that increase risk of drowning, swimming ability, use of safety devices, and use of safe practices when in and around water.

PARTICIPANT SCREENING

Retrospective studies have linked coronary artery disease, prolonged QT syndrome, and seizure disorders with higher than normal rates of drowning and drowning deaths.[41,109–116] Preparticipation screening should focus on uncovering any medical or physical condition that may potentially impair decision making, physical abilities, and thus, swimming ability.

Recommendation: All patients with coronary artery disease, prolonged QT syndrome, seizure disorders, or other medical and physical impairments should be counseled about their increased risk of drowning and about steps to mitigate the risk should they choose to participate in water activities. (Recommendation grade: 2C)

SWIMMING ABILITY

Common sense dictates that an adolescent or adult who is a competent swimmer and has the neurocognitive ability to make appropriate decisions about water safety has a decreased likelihood of drowning. However, the best ages to learn technique and specific swimming skills that reduce a person's chance of drowning are not well understood. Most available literature evaluates infant and pediatric populations for the effects of swimming and "infant survival" lessons on drowning and mortality.[117,118] There is concern that by providing swim lessons to young children, parents may develop a false sense of security in their child's swimming ability, which may lead to an increase in drowning incidents.[119–121]

The American Academy of Pediatrics (AAP) has always maintained that children should learn to swim at some point in their life. Previous recommendations were against formal swim lessons for all children 4 years and younger. The most recent review by the AAP acknowledges lack of evidence surrounding pediatric swimming lessons and currently does not formally recommend for or against lessons for children younger than 4 years.[117]

There is considerable debate regarding the definition of "swimming" or "survival-swimming" and what constitutes the most protective approach to swim instruction. Although the ability to swim farther distances can be perceived as increased swim ability, for the purpose of swimming as a tool for drowning prevention, the distance of 25 m (82 feet) has been adopted by

international lifesaving agencies and a large population-based study in Bangladesh.[122–124]

Despite the lack of definitive evidence showing a clear benefit to formal swim lessons, panel members agree that familiarity with and, more importantly, confidence in an aquatic environment would be beneficial in the event of accidental immersion or submersion. In addition, unique aquatic environments such as whitewater should be approached only after focused instruction on swimming techniques specific to that environment.

Recommendation: All persons who participate in activities conducted in or around open water should have, at a minimum, enough experience and physical capability to maintain a floating position, tread water, and make forward progress for a distance of 25 m (82 feet). (Recommendation grade: 2C [pediatrics], panel consensus [adults])

PERSONAL FLOTATION DEVICES

Under the category of personal flotation devices, devices such as lifejackets, manually or automated inflation systems, and neoprene wetsuits are available. Currently, lifejackets are the only devices with injury prevention data available and will, therefore, be used as the prototypical model for this category. In 2013, according to US Coast Guard data, drowning was the cause of death in more than 75% of fatal boating accidents.[125] In addition, 85% of these fatalities were not wearing lifejackets. Three other retrospective studies have found an association between lifejacket use and decreased mortality in boating accidents.[126–128] One of these studies specifically compared drowning deaths before and after increased lifejacket regulations, revealing improved survival rates after regulations went into effect. These data suggest that activities in and around water, especially while boating, should include lifejacket use.[126]

Recommendation: Properly fitted lifejackets that meet local regulatory specifications should be available for participants when boating or engaging in any water sports for which lifejackets are recommended, and should always be worn while engaged in the activity. (Recommendation grade: 1C)

DROWNING PREVENTION STRATEGIES

Alcohol is a known contributing factor to drowning deaths. Data have been obtained primarily from telephone studies, and likely underrepresent the true burden of alcohol in causing drowning. In 2013, alcohol was a contributing factor to 16% of boating-related deaths.[125] A 2004 review found that 30% to 70% of drowning fatalities have a measurable blood alcohol level, with 10% to 30% of deaths being directly attributed to alcohol use.[129]

There are no specific peer-reviewed studies on the utility of lifeguards on expeditions or wilderness trips.[130] A 2001 CDC working group report recommends lifeguards for drowning prevention in open water settings. In 2013, the US Lifesaving Association reported 6,725,264 preventive actions and 68,320 rescues covering a population of 339,049,941 beachgoers. There were 23 reported drowning deaths at guarded beaches compared with 92 deaths at beaches without lifeguards.[131] Among nationally recognized lifeguard certifying agencies (Ellis & Associates, American Red Cross, Starfish Aquatics Institute, and National Aquatic Safety Company), there are no specific guidelines or recommendations for the number of lifeguards per number of participants in an event or at an aquatic facility.

Recommendation: Alcohol and other intoxicating substances should be avoided before and during water activities. Despite lack of definitive evidence, all groups operating in or near aquatic environments, regardless of size, should consider water safety during planning and execution of excursions. This includes contingencies for prevention, rescue, and treatment of drowning persons. In high-risk environments or large groups, consider including personnel with technical rescue training and appropriate rescue equipment. (Recommendation grade: 1C)

Special Situations

COLD-WATER SURVIVAL

No single recommendation can address all possible scenarios that a person may encounter in a water setting. An unintentional fall into a swiftly moving river, deep offshore ocean, inland waterways, or backyard swimming pool, or falling through the ice into static or moving water, are all treated according to the skill level, preparation, and equipment available to both patient and rescuer. Immediate attention must always be given to self-rescue and extricating oneself immediately from a hazardous environment. After immersion in cold water, a person has a limited amount of time before fatigue and incapacitation render self-rescue impossible. Likelihood of survival is increased by having appropriate gear and training and by dressing for water temperature, not just air temperature, in the event of immersion.

Extensive controlled trials of cold-water survival are lacking, and the available literature is not generalizable to all scenarios. For example, the presence of a lifejacket, sea state, weather, physical fitness, clothing, and mental preparedness all contribute to survivability in cold water.

Survival in whitewater is different from still water or in the ocean in polar regions. A single large literature review serves as the source for recommendations about cold-water survival under "ideal" conditions and must be interpreted according to the level of training, preparation, and situation presented to the patient.[132]

After immersion, the most important decisions a person must make are 1) presence of any potential immediate life threats and 2) whether to swim to safety or await rescue. Should a person choose to await rescue, preventing loss of body heat becomes paramount. By positioning the body to protect major areas of heat loss, a patient may lengthen immersion survival time. A position that has been proven in a laboratory setting to decrease heat loss is the Heat Escape Lessening Position (HELP). This position is maintained by flexing the hips and knees and hugging the knees to the chest; it is important to note that to maintain this position, a lifejacket or similar flotation device is necessary. In the event of a group immersion, a huddle formation has been recommended to lessen heat loss, assist injured or weak persons, and improve group morale. Although this position has been shown to decrease cooling in participating individuals in a controlled environment, the effort needed to assist debilitated individuals in a actual emergency may result in increased heat loss (Figures 1, 2).[133]

Swimming or treading water should be limited to minimize heat loss. Lifejackets should be worn to aid insulation and flotation. If possible, the ideal location to await rescue is out of the water, even if only partially, to reduce heat loss and delay onset of hypothermia. Prolonged cold-water exposure eventually results in cognitive and motor disabilities, which can appear within 10 minutes of immersion, making advanced maneuvers or decision making difficult. For this reason, it may be beneficial to affix one's body or clothing to a floating object using rope, or freezing clothing to icy surfaces.

Figure 1. HELP position. Reprinted from *Wilderness Medicine*, 6e, Auerbach PS (ed.), Submersion Injuries and Drowning, page 1502, Copyright 2012, with permission from Elsevier.

Figure 2. Huddle formation. Reprinted from *Wilderness Medicine*, 6e, Auerbach PS (ed.), Submersion Injuries and Drowning, page 1502, Copyright 2012, with permission from Elsevier.

Should a person decide to swim to safety, some important physiologic changes may occur. The initial cold shock, which lasts seconds to a few minutes, may prompt gasping and hyperventilation, and can have a disorienting effect, making self-rescue attempts difficult. On immersion in cold water, if no immediate life threats are present, a person should focus on remaining calm and controlling breathing. Once a person is able to obtain his or her bearings, he or she may have far less than 10 minutes of effective swimming, and up to 1 hour of consciousness, before succumbing to hypothermia. All of these statements assume the person is wearing an appropriate lifejacket. Further detailed discussion of the science behind cold-water immersion is available in Chapter 6 of *Wilderness Medicine* (6th ed) by Auerbach.[14]

Recommendation: On falling into cold water, distancing oneself from any immediate life threats (eg, fire, sinking vehicle, whitewater, hazardous waves, rocks) is paramount. An attempt should then be made to remain calm and focused. The person should then consider physical capabilities, location, resources, and chances of rescue to determine whether or not to swim to safety. If a decision is made to swim to safety, this should be done as soon as possible before physical and mental capabilities deteriorate from the effects of cold stress. If a decision is made to await rescue, an attempt should be made to remove as much of the body from the water as possible. All clothing should remain on, unless it hampers buoyancy. If the person remains immersed, the HELP position should be maintained if possible. In a group, the huddle position may be used. If prolonged rescue is expected, it would be beneficial to attach oneself to buoyant objects or to a surface out of the

water to improve the chance for survival once incapacitated. (Recommendation grade: 2C)

SWIMMING-INDUCED PULMONARY EDEMA

During the past 30 years, numerous case reports and studies have described a syndrome of acute shortness of breath and bloody or pink, frothy sputum after strenuous exercise, such as military training, triathlons, and long-distance swims.[134–138] For these guidelines, we focus on this spectrum of symptoms as related to surface swimming, not scuba diving. Acute pulmonary edema secondary to surface swimming is thought to be a combination of increased cardiac output, redistribution of circulating blood volume to the central circulation and pulmonary arteries as a result of immersion and cold-water vasoconstriction, and possibly overhydration. Although the incidence of this syndrome varies greatly as cited in studies, there were few reports of severe residual disease or poor outcomes. Studies have found temporary changes in pulmonary function testing lasting up to 1 week without changes in cardiac function.[135]

Recommendation: Patients experiencing symptoms consistent with pulmonary edema after swimming should cease further strenuous activity until fully recovered and be advised of the possibility for temporary changes in lung function. Patients experiencing this syndrome usually recover well without treatment, and in the absence of severe respiratory symptoms, evacuation is not warranted. (Recommendation grade: 2C)

Conclusions

Drowning is a process with outcomes ranging from no morbidity to severe morbidity, and eventually death. As with other injuries encountered in the wilderness environment, the best treatment for drowning is prevention. This includes a multitiered approach including swim lessons, appropriate supervision, use of suitable lifejacket or personal flotation device, knowledge of water conditions and weather patterns, and avoidance of drugs and alcohol. When prevention fails, or circumstance leads to the drowning process, then the most important aspect of treatment is to reverse cerebral hypoxia by providing oxygen to the brain by whatever means available.

Conflicts of Interest

The authors wish to report the following disclosures: A.S. and J.S. are directors of Lifeguards Without Borders; S.H. is the medical director of Landmark Learning, the medical director of Starfish Aquatics Institute, the medical director of NC State Parks, owner of Hawk Ventures, the Medical Director of Burke County EMS; and the executive editor of *Wilderness Medicine*; and T.C. is a board member of the Wilderness Medical Society, and chair of the WMS Practice Guidelines Committee. A.A. and P.A. have no conflicts of interest to declare.

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Physiology Of Drowning: A Review

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Drowning physiology relates to two different events: immersion (upper airway above water) and submersion (upper airway under water). Immersion involves integrated cardiorespiratory responses to skin and deep body temperature, including cold shock, physical incapacitation, and hypovolemia, as precursors of collapse and submersion. The physiology of submersion includes fear of drowning, diving response, autonomic conflict, upper airway reflexes, water aspiration and swallowing, emesis, and electrolyte disorders. Submersion outcome is determined by cardiac, pulmonary, and neurological injury. Knowledge of drowning physiology is scarce. Better understanding may identify methods to improve survival, particularly related to hot-water immersion, cold shock, cold-induced physical incapacitation, and fear of drowning.

A recent report of the World Health Organization (WHO) labels drowning as one of the world's leading causes of death (164). Although standard WHO statistics report almost 375,000 persons drowning each year, the actual figure for worldwide drowning is probably four or five times as high. Low- and middle-income countries, where most drownings occur, have incomplete recording of drowning events. Furthermore, drowning as a result of flooding, ferryboat accidents, and accidents with boat-carrying refugees and migrants are not reported in the standard drowning statistics (140, 164).

Drowning is formally defined as the process of experiencing respiratory impairment from submersion/immersion in liquid (273). The exact physiological mechanisms of this process are complex and largely unknown, and have only been speculatively described in drowning reviews and studies over recent decades (29, 108, 132, 195, 257). Although there are clinical differences between submersion and immersion (30, 275), the details of the physiological processes have not been reviewed. The present work provides an overview of the current understanding of the physiological mechanisms that may occur during a submersion or immersion incident. Since human drowning mechanisms per se are difficult to study, observations from animal experiments, case series, and forensics have been included where helpful.

The objective of the review is to summarize the physiological mechanisms associated with drowning and to explore whether future physiological studies may contribute to the prevention, treatment, and forensic investigations of drowning.

Immersion

Hot-Water Immersion

"Thermoneutral" is the term for the water temperature at which heat loss equals heat production (53, 238). Most drowning events occur at water temperatures below the point of thermoneutrality, which is 35°C ± 0.5. Some drownings, however, occur in hot-water tubs, while pouring hot water over the head, or during diving or competitive swimming in warm water.

Ofuro bathing is a component of Japan's national culture and identity. It is believed that healthy persons may benefit from the physiological effects of hot-water immersion (HWI) on the body's homeostatic systems (16, 25, 48, 53, 81, 118). People may sit and soak up to the shoulders or neck in deep hot (38-43°C) tubs for 5–15 min (96, 118). The high incidence of Japanese hot-water tub fatalities suggests that HWI may lead to drowning (2, 109, 175, 191, 211, 291).

Thermoregulation during HWI differs from thermoregulation in hot ambient air. In ambient air, elimination of body heat occurs mainly by sweat evaporation. The phase-change from a liquid to a gaseous state removes heat from the skin and cools the body. In HWI, the high humidity of the ambient air around a hot tub, with only the head and neck skin above the water, allows limited evaporation of sweat above the water. Sweating, however, also occurs under water. The secretory pressure of sweat glands allows sweat to flow outward to dissolve in the water. This sweat fails to evaporate and thus does not contribute to body cooling. When skin temperature increases, cutaneous warm thermoreceptors located in sensory nerve (unmyeli-

nated C-type fiber) endings interact with keratinocytes through transient receptor potential vanilloid cation channels and convey signals, via the spinal dorsal horn and trigeminal nerve, to hypothalamic thermoregulatory centers mainly situated in the pre-optic area (220, 235, 237). From the pre-optic area, autonomic efferent information is forwarded to the skin and causes, among other effects, cutaneous vasodilatation (118, 169, 182). Under normal circumstances, external hydrostatic pressure results in bradycardia. In HWI, however, the temperature effects overcome this (16, 34, 236, 251), because decreased peripheral vascular resistance raises heart rate (34, 48, 175, 259, 284).

Increased HR may trigger ventricular arrhythmias, potentially hazardous in combination with peripheral vasodilatation and increased blood viscosity (229). The associated dehydration increases likelihood of thrombosis, particularly in the elderly (158, 239). HWI drowning is most likely to occur during protracted immersion at high temperature and when leaving the tub (1, 117). The loss of the hydrostatic squeeze on leaving a bath and assuming an upright posture can cause a gradual or sudden decrease in blood pressure (44, 109, 118, 185, 211, 229, 291). The HWI-related cardiovascular changes may be important in the elderly and in those with coronary artery disease, hypertension, or congestive heart failure (7, 25, 48, 81, 117, 175, 211, 291).

Drownings have also been attributed to pouring hot water (>39°C) over the head. Tactile and temperature stimuli can trigger reflex epilepsy in individuals with aberrant thermoregulation or genetic defects (24, 165, 228).

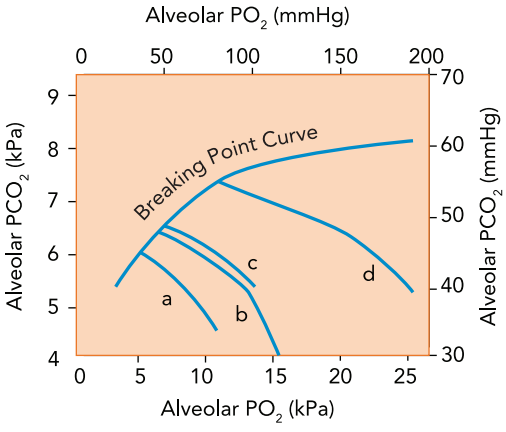

FIGURE 1. **The breaking points of breath-holding in different settings**
The "breaking point curve" defines the values of alveolar P_{O_2} and P_{CO_2} at the breath-hold break point when starting from different states. The normal alveolar starting point is shown. This point is displaced by different maneuvers, and the length of the arrows gives an indication of the changes of the breath-hold duration. See Refs. 73, 196. a, After breathing 15% oxygen; b, after hyperventilation; c, normal alveolar point; d, after breathing 30% oxygen.

Competitive swimming in warm water can cause a marked increase in deep body temperature and insidious hyperthermia. The pathophysiology of endurance swimming in warm water has been considered after a death during warm-water competition (260). Hyperthermia during diving in tropical waters also can pose a drowning risk (208, 260).

Cold-Water Immersion

Most drownings occur in water colder than thermoneutral temperature, thus initiating physiological responses associated with cooling. In cold water, the responses that act as precursors to drowning are evoked by skin cooling (cold shock), then cooling of superficial nerves and muscles in the limbs, and finally cooling of deep body tissues (hypothermia).

Cold Shock

After a fall into cold water, any intention to breath-hold can be overcome by cold shock (261, 263). The response starts in water ~25°C and peaks somewhere between 15 and 10°C; it peaks in the first 30 s of immersion and attenuates during the next 2–3 min (268). It is evoked by cold receptors located in the superficial sub-epidermal layer of the skin; below ~19°C, cold nociceptors contribute to the response with a sensation of intense cold pain being experienced in water below ~5°C (40, 163). The cold-shock response may be decreased but is still present in those with a high body temperature (159).

Cold receptors respond to the sudden decrease in skin temperature resulting from immersion in cold water with a dynamic response that evokes gasping, hyperventilation, increased cardiac output, peripheral vasoconstriction, and hypertension. These responses, along with a generalized increase in muscle tension, can increase metabolic rate on initial immersion by a factor of four (98). This would, on its own, decrease breath-hold time during initial immersion because the hypoxic and hypercapnic thresholds for the breakpoint of breath-holding would be reached earlier (FIGURE 1). More important, thermo-afferents from the peripheral cold receptors dramatically increase respiratory drive via direct stimulation of the respiratory center (121), with a reflex stimulation at the spinal level of α-motoneurons innervating the intercostal muscles and diaphragm (166, 263). As a consequence, the gasp response and hyperventilation cause an inability to breath-hold. Maximum breath-hold time generally is 60–90 s at a comfortable air temperature and is reduced to just a few seconds in water colder than ~15°C. The inability to breath-hold represents the most hazardous response to cold-water immer-

sion, increasing the chance of aspiration and drowning.

At the same time, upon initial immersion in cold water, the incidence of arrhythmias increases from 2% during cold water immersion with head-out-of-water free-breathing to 82% if the cold immersion is associated with face immersion and maximum breath-holding (see section *Diving Response* and *Autonomic Conflict* below).

Superficial Tissue Cooling

After the skin has been exposed to cold water, the next tissues to cool are superficial nerves and muscles. Those in the arms are particularly susceptible due to the surface area-to-mass ratio of the arms and the relatively superficial anatomical location of nerves and muscles. Low muscle temperature can affect chemical and physical processes at the cellular level. This includes metabolic rate, enzymatic activity, calcium and acetylcholine release and diffusion rate, and series elastic components of connective tissues (278). At muscle temperatures below 25°C, fatigue occurs because cooling impairs superficial muscle fibers leaving a smaller number of fibers to produce the same force (50). Maximum dynamic strength, power output, jumping, and sprinting performance have been related to muscle temperature with reductions ranging from 4 to 6% per degree fall in muscle temperature down to 30°C (27, 213).

At nerve temperatures below ~20°C, nerve conduction is slowed and action potential amplitude is decreased (62). Ulnar nerve conduction velocity falls by 15 m/s per 10°C decrease of local temperature. Nerve block may occur at a local temperature of between 5 and 15°C for 1–15 min and lead to a dysfunction that is equivalent to peripheral paralysis (22, 50).

The detrimental influence of peripheral and deep-tissue cooling on physical performance has recently been reviewed (43). It is noteworthy that drowning caused by physical incapacitation can occur before deep body tissue temperature falls below 35°C.

Deep-Tissue Cooling: Hypothermia

With regard to drowning, the most significant consequence of hypothermia is the loss of consciousness (LOC) with deep body cooling. This prevents individuals from undertaking physical activity to maintain a clear airway.

The progressive signs and symptoms are shivering (36°C), confusion, disorientation, introversion (35°C), amnesia (34°C), cardiac arrhythmias (33°C), clouding of consciousness (33-30°C), LOC (30°C), ventricular fibrillation (VF) (28°C), and death (25°C). Below a cardiac temperature of 28°C, the heart may suddenly and spontaneously arrest. VF may result from rough handling of the casualty at deep body temperature of ~28°C (88, 89). Hypothermia affects cellular metabolism, blood flow, and neural function. In severe hypothermia, the patient will be deeply unconscious. The decreased oxygen requirement of cold cells and organs causes decreased respiratory and heart rates. This makes it difficult to detect vital signs in the field. Tendon reflexes are absent and the pupils dilated: this may give the appearance of death (88).

A distinction should be made between induced hypothermia for clinical purposes and accidental hypothermia. Successful resuscitation has occurred following induced hypothermia down to deep body temperatures as low as 5°C (32). In contrast, in accidental hypothermia, it is not uncommon for death to occur at a body temperature of 24–28°C. Therefore, the circumstances of cooling and rewarming resuscitation, and associated changes in physiology in themselves, can be important determinants of survival. In addition, different physiological functions have different susceptibilities to cooling. The Q_{10} temperature coefficient is a measure of the rate of change of a biological or chemical system as a consequence of increasing/decreasing temperature by 10°C. For example, metabolic and rhythmic processes are particularly depressed by hypothermia (Q_{10} of ~3); contractile processes have a Q_{10} of ~2. As hypothermia progresses, metabolic and rhythmic processes are depressed two to three times more than the rates of diffusion of different metabolites (152). Some of the mechanisms underpinning the functional changes associated with hypothermia are briefly outlined in (see Table 1). Although presented separately, it should be apparent that these changes are interrelated.

The signs and symptoms of progressive hypothermia are not strongly correlated with temperature. The temperatures above in parentheses are only rough approximations. Great variation exists between individuals in both the rate of cooling and the lowest deep body temperature compatible with life or consciousness (5). The rate of cooling depends on a wide range of internal thermal factors including subcutaneous fat thickness (119, 213) or nonthermal factors like motion illness (167) and external factors such as water temperature and sea state (88). Table 2 provides an overview of the risk factors for development of immersion hypothermia.

As noted, during accidental hypothermia, the deep body temperature associated with death is ~25°C (5), but the lowest deep body temperature recorded to date following accidental exposure to cold air and with a beating heart and full recovery was 12.7°C in a 28-mo-old child (19,

Table 1. *Overview of the physiological changes with moderate-severe hypothermia compared to normothermia*

Decreased spontaneous depolarization of heart pacemaker cells	Progressive bradycardia that accompanies increasing hypothermia is characterized by a relatively greater prolongation of systole than diastole. It affects both atrial and ventricular rates and is approximately linearly related to the fall in body temperature. There is a direct effect of cold on the pacemaker tissue (18, 75). Myocardial conductivity is uniformly and progressively depressed (103).
Altered activity of membrane ion channels	Alterations in the myocardial membrane action potential are thought to be due to changes in ion fluxes across the myocardium sarcolemma (153). Such alterations are thought to produce the characteristic J-deflection recorded in both induced and accidental hypothermia (186, 241).
Renal function and glomerular filtration depression, augmented by osmotic diuresis	Hypothermia has a depressant effect on all aspects of renal function. There is a progressive fall in renal blood flow and increase in renal vascular resistance. At a deep body temperate of 28°C, renal blood flow may have been reduced by as much as 50% (221). Decreased renal tubular function appears to be a result of the direct effects of cold (12). Hypothermia inhibits many of the enzymatic processes in the renal tubular cells, negating the kidneys' role in acid-base control (221). Increased sodium excretion during hypothermia is evidence of depressed tubular transport of sodium; this also impacts the ability of the kidneys to contribute to acid-base regulation (sodium-hydrogen exchange) (221). There is a cold-induced inhibition of the tubular reabsorption of water, which contributes to the diuresis seen on cooling (222).
Fluid shift into the extravascular compartment	Hypothermia depresses many of the mechanisms involved in the regulation of body fluid balance and causes abnormal fluid shifts between body compartments (107).
Impaired hepatic metabolism	The hypothermic liver is less able to utilize glucose or remove excess lactate or other products of muscle metabolism; this contributes to the metabolic acidosis seen in hypothermia (221).
Impaired respiratory function	At deep body temperature below ~32°C, spontaneous respiratory activity is decreased. This, plus the increased solubility of carbon dioxide in the body fluids, may combine to produce respiratory acidosis (249).
Diminished endocrine function	The release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary is depressed in proportion to the severity of hypothermia; the action of ACTH on the adrenal cortex also falls in proportion to deep body temperature below 32.2°C (79). The depression of human adrenocortical function intensifies at a deep body temperature of 28°C (112). The adrenal medulla production of adrenaline and noradrenaline become significantly depressed at a deep body temperature of 28°C (113). Endogenous production of insulin falls (23, 52). Below a deep body temperature of 30°C, the glucose carrier mechanisms of the cell membrane appear to be inhibited, and glucose utilization is severely reduced, resulting in hyperglycemia even in the presence of insulin (35). Conversely, hypoglycemia can increase the likelihood of hypothermia by inhibiting metabolism (202).
Impaired cerebral function	Cerebral blood flow decreases due to falling cardiac output and blood pressure, and a rise in blood viscosity and cerebrovascular resistance. The metabolic rate of the brain and spinal cord are depressed. There is a progressive slowing of electrical activity, with cerebral electrical activity ceasing in some individuals at 17°C (197).
Impaired peripheral neural function	When expressed as a semi-logarithmic function, all peripheral motor and sensory nerve fibers have a Q_{10} of 1.51 (59); voluntary grip strength has a Q_{10} of 1.2 (64).
Decreased gastrointestinal motility and function	Multiple acute submucosal hemorrhages are common in the region of the pylorus (155, 183). The absorption of drugs from the bowel is impaired during hypothermia (193, 219).
Blood alterations	Hypothermia results in increased blood viscosity (120), which can interfere with the microcirculation. There is a severe fall in leukocyte count below a deep body temperature of 28°C (269). There is a linear fall in platelet count with deep body cooling. Thrombocytopenia becomes pronounced at a deep body temperate below 28°C. The fall in the number of platelets is associated with a tendency for abnormal bleeding (282).
Collapse of the microvasculature	Hypothermia can result in a loss of microcirculatory control, vasomotor paralysis, and hypoperfusion (similar to "shock"). Capillary sludging and microcirculatory stasis can result and threaten survival (100). Circulatory failure prevents acids formed in the tissues, due to hypothermia-induced hypoxic metabolism, from being buffered. When the microcirculation improves with rewarming, metabolic acidosis may rapidly increase as the acid products of anaerobic metabolism are returned to the circulation (187).

217). The coldest adult survivor of cold-water immersion followed by submersion had a body temperature of 13.7°C (86).

The variation in the rates at which people cool in water below thermoneutral temperatures, and the poor association between the signs and symptoms of hypothermia and actual deep body temperature, make the determination of time of useful consciousness and survival time "more of an art than a science" (88). It also follows that the

signs of hypothermia may be unreliable indicators of deep body temperature. For example, the presence and absence of shivering have variously served as indicators that body temperature is normal, under threat, or profoundly hypothermic. However, shivering is dependent on functioning neuromuscular pathways, blood glucose levels, and a local supply of substrate (82); these factors may change, independent of deep body temperature. Hypoglycemia following consumption of alcohol, a common factor in immersion victims, can suppress shivering independent of body temperature (94). Substrate depletion is more likely in chronic hypothermia than in the acute hypothermia observed in immersion victims.

A final problem with hypothermia is that, in the field and sometimes in the emergency department, the absence of a reliable measure of deep body temperature makes the direct and accurate assessment of the degree of hypothermia in an immersion victim difficult (150, 258).

Submersion

Sympathetic Activation, Fear of Drowning

Fear of drowning as a mechanism that results in drowning is most often reported in the gray literature and social media. Several triathletes mention excessive panic, notably during the mass start of swimming. The panic is accompanied by complete inability to swim. The fear of drowning urges them to go back to shore or get attached to a buoy or lifeboat. Approximately 80% of triathlon deaths occur during the swim, and it is speculated that several drownings during triathlon swimming may be due to the results of these panic attacks (39, 260, 264). Also, competitive swimmers may panic when swimming in open water where they are confronted with a different setting than the Olympic pool and the need to use different swimming strokes than the strokes they are trained for (260, 264). Recreational swimmers in open water encounter similar panic experiences when suddenly confronted with cold water, rip currents, or unexpected underwater objects. Some swimming instructors have experienced students who refused to enter the water or almost drowned when in the water, paralyzed by this fear of drowning. Special training programs have been developed for these students and are also recommended for experienced swimmers (114, 181). Divers with self-contained underwater breathing apparatus (SCUBA) also may panic when experiencing the sensation of cold and streaming water, losing visual contact with the bottom (blue orb syndrome), observation of large or dangerous fish, entanglement, entrapment, or equipment malfunction. This is sometimes combined with a reduction of muscle force (see below). An unknown, but probably significant, contributor to SCUBA drowning may be panic that completely incapacitates the diver both mentally and physically (181, 210).

Table 2. ***Risk factors for immersion hypothermia (88, 119, 167, 213)***

- Water temperature: effects being most significant during cold water immersion
- Water movement: faster-moving liquids increase convective heat loss
- Surface area-to-mass area: the higher this ratio, the more cooling is facilitated
- Age: children cool faster than adults due to their lower levels of subcutaneous fat and higher surface area-to-mass ratio
- Body stature: tall, thin individuals cool faster than do those short and obese
- Body morphology: body fat and nonperfused muscle are good insulators
- Gender: females tend to have more subcutaneous fat than men but a weaker shivering response
- Fitness: high fitness level enables greater heat production
- Fatigue: exhaustion results in decreased heat production
- Nutritional state: hypoglycemia attenuates shivering and accentuates cooling
- Intoxication: alcohol and other drug depressants affect metabolism
- Lack of appropriate/specialized clothing

The psychological aspects in these situations also includes concern by the person in the water about a sudden onset of previously diagnosed and treated minor physical problems (such as cardiac problems, hypertension, diabetes) and other frightening thoughts, leading to sensory deprivation, illusions, flashbacks, and thoughts of catastrophic outcome. It is well known that panic leads almost instantaneously to irrational logic and cognition. Problem-solving capacities are decreased.

There is limited physiological literature on this phenomenon, although many reports also mention a physical component, most of all paralysis or loss of muscle strength. This may be due to the hyperarousal of the sympathetic activation during panic in the water. This will lead to a combination of physical and psychological stressors that could potentiate cold shock, disable swimming ability, or at least create the feeling that swimming ability has seriously decreased. Notable in people with an overreactive anxiety state, the stressful or unexpected event may result in a panic-induced hyperarousal, resulting in submersion.

Breath-Holding

Breath-holding has served as an intervention to discover more about control of breathing and determinants of breath-hold time. A long history of excellent work has examined breath-holding physiology (78, 104, 200). Under normal circumstances in air, an initial period occurs with little respiratory afferent activity and therefore also with little effort required to maintain a breath-hold ("easy going phase"). This ends due to afferent neural input to the respiratory centers arising from the respiratory musculature, creating an increasing drive for respiratory movement: the struggle phase (FIGURE 2). Respiratory movement, in the form of rebreathing into a bag at the point of maximum breath-hold, can double the time spent without fresh air (78). The respiratory movement associated with rebreathing decreases the afferent neural input arising from the respiratory musculature and extends the breath-hold time to the point where blood oxygen and carbon dioxide tensions drive respiration. Swallowing can extend breath-hold time by causing some movement of the respiratory musculature, perhaps explaining why some drowning victims have water in their stomachs when rescued.

Under normal circumstances, typical alveolar P_{CO_2} at the breakpoint ranges between 43 and 53 Torr and occurs 60–90 s after breath-holding with ambient air (FIGURE 2). The breath-holding time can be influenced by several factors, including those listed in Table 3 (196).

In water, important additional physiological factors decrease breath-holding duration, including alcohol intoxication, water temperature below ~15°C, and the cold shock response that intensifies respiratory drive. Other factors that can influence breath-hold time include voluntary liquid aspiration such as occurs in suicides.

As with many physiological responses that involve a combination of autonomic and conscious input, the variation between individuals in maximum breath-hold time is large. Even in the same cold water temperature, the breath-hold time can range from <10 to >100 s (262). In warm water, the average maximum breath-hold time is ~45 s, but some trained breath-hold divers can achieve over 20 min (149, 252a).

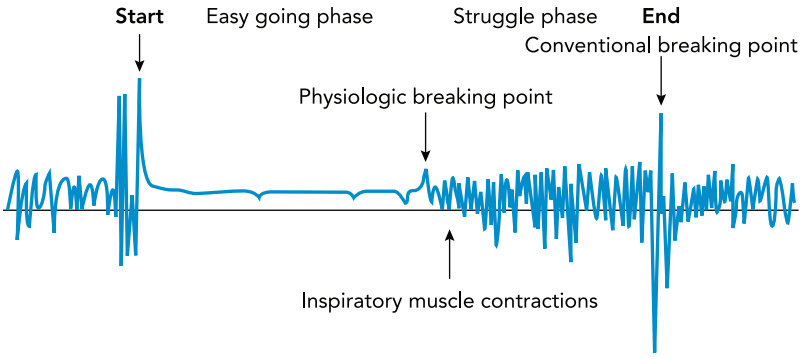

FIGURE 2. **Human thoracic movements measured by electromyography (EMG) during maximal breath-holding in an untrained, non-immersed subject**
"Easy going phase" and "struggle phase" are distinguished by the absence or presence of respiratory muscle activity that must be suppressed during breath-holding. Figure is from Ref. 72 and used with permission from Saunders.

Diving Response

Whereas there is evidence that the diving response conserves oxygen during apneic diving or cold exposure of the face, only limited and indirect evidence defines the role of the diving response during drowning. The diving response is one of the commonly proposed mechanisms to explain why some drowning victims survive for prolonged periods underwater (91). However, the response is probably much less important as a protective mechanism than is rapid selective brain cooling caused by the supply of cold carotid blood to the brain and cooling of the heart caused by the aspiration and ingestion of ice-cold water (51, 90, 266, 270).

Research into diving birds, reptiles, mammals living in or underwater, and other hypoxia-tolerant animals shows that the diving response is an autonomic response that serves as an endogenous hypoxia defense mechanism to preserve life. The diving response is better developed and has a faster onset in diving mammals and children than in adult humans (47, 77, 91, 106, 137). Rats have been trained to dive to allow study of the diving response (106, 161, 162, 198).

Studies with infants reveal that, up to 6 mo of age, all children have the ability to achieve the diving response. This is decreased to 90% of all children at 12 mo of age (87, 207). The diving response could be triggered in 66% of adult volunteers, but with large interindividual differences in its effects (99).

Human studies are mostly related to breath-hold diving (9, 95, 97, 102, 133, 134, 137) and facial immersion in cold water (38, 126). The diving response is considered one of the most powerful autonomic responses, particularly in children (42, 198). For this reason, clinically relevant studies on the diving response have involved treatment of paroxysmal atrial tachycardia, diabetic cardiovascular autonomic neuropathy, and rheumatoid arthritis (42). Because the diving response is an oxygen-conserving response, it is also used as a potential model to study endogenic neuronal protection effects at the molecular level (105, 227). It is also used to teach integrative physiology to students (45).

The diving response can be activated by apnea alone or by facial immersion alone, but their combination enhances the response (4, 11, 42,

45, 97). Most important is the presence of cold water and a large ambient air-to-water temperature gradient (77, 233). The diving response involves simultaneous activation of sympathetic and parasympathetic responses leading to peripheral vasoconstriction, hypertension, and bradycardia (FIGURE 3) (42, 203). Individual papers debate whether apnea, laryngospasm, or contractions of the spleen are part of the response (87, 232). The effects have been consistently observed in studies, with large individual differences. The diving response decreases metabolism selectively, mainly in the vasoconstricted tissues and heart, resulting in an overall decrease in oxygen consumption and a slower desaturation during apnea. This is in contrast to protective hypothermia that decreases the metabolism and oxygen consumption of all organs. The factors that influence the effects of the diving response are summarized in Table 4.

Small involuntary breathing movements during the struggle phase of prolonged apnea do not influence the response in the presence of hypoxia and hypercapnia (9). Animal and human experiments have shown that the sympathetic and parasympathetic components of the diving response can be separately influenced by atropine and vasodilators. No chemoreceptor influence comes from acidic, asphyxic blood (203). The sympathetic effects, occurring within 10–40 s after cold water touches the face in humans, are affected by input from facial cold-receptors and chemoreceptors. Next comes selective vasoconstriction, with less perfusion measured in the peripheral vascular beds of the skin, muscles, and viscera (9, 77, 102). Metabolism in these organs is decreased, and they shift to anaerobic metabolism, leading to increased lactate. Vasoconstriction causes hypertension (77). Increased carotid artery blood flow and vasodilatation in the brain results in better cerebral perfusion. Brain hypoxia also enhances cerebral perfusion. The result is that the oxygenation of this most oxygen-sensitive organ remains preferentially sustained underwater (38, 126).

Table 3. ***Factors influencing breath-holding duration in air***

Metabolic rate during breath-holding
Prebreathing with hyperoxic or hypoxic gas mixtures
Carbon dioxide and oxygen storage capacity
Prior hyperventilation
Experience and psychological tolerance of unpleasant sensations arising during breath-holding

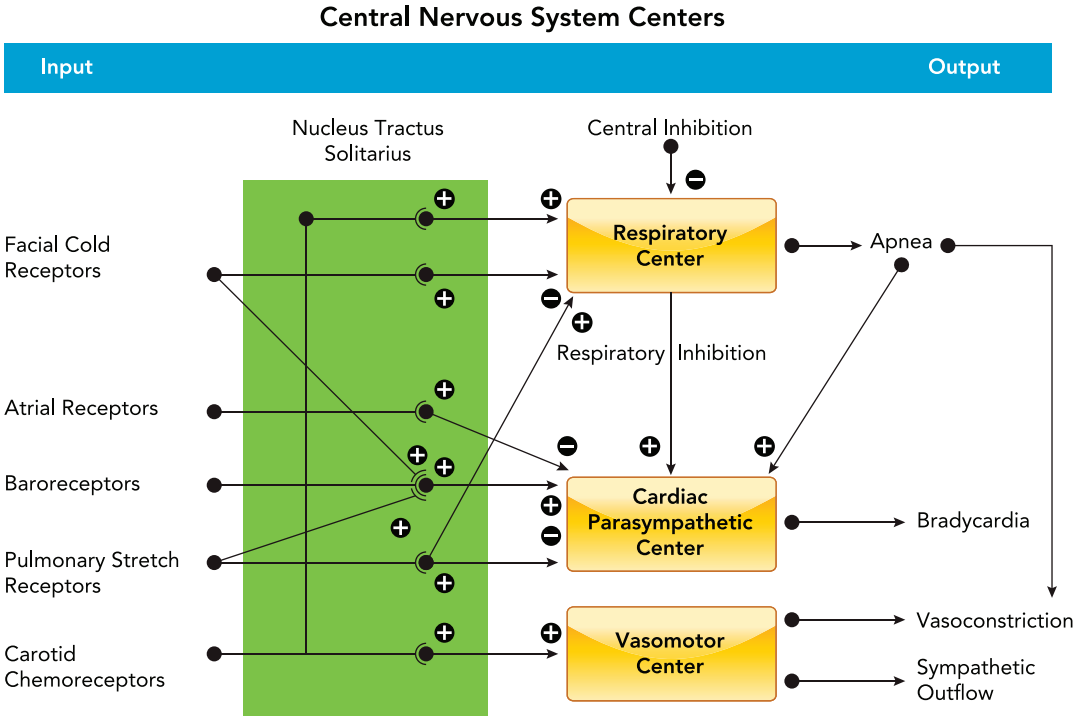

FIGURE 3. **Schematic figure of the diving response**
Input appears on the *left* and output at the *right*. In between are the neural connections located in the nucleus tractus solitarius and central nervous system control centers. The response is activated through the chemoreceptor sensitivity of the cold receptors of the skin and the unmyelinated C-fibers of the ophthalmic branch of the nervus trigeminus. For more details on the neurological pathways of the diving response, see also Refs. 162, 199, 226, 234. +, Excitatory neural connections, −, inhibitory neural connections. Figure is from Ref. 77 and used with permission from *Scandinavian Journal of Medicine and Science in Sports*.

Table 4. *Factors that increase effects of the diving response (10, 11, 17, 42, 77, 134, 231, 233)*

Precooling of the face
Contact of cold material with the face (water, air, cold packs)
Large air/water temperature gradient
Increased hypoxia
Prolonged or deeper submersion
Posture in the water
Smaller vital capacity lung volume
Previous breath-hold diving experiences
A series of repeated apnea dives or apnea exercises
Physical fitness

In addition, the diving response induces, after ~30 s, vagal-mediated bradycardia. Initially, bradycardia is a baroreceptor response due to vasoconstriction and later a chemoreceptor response due to hypoxia. Bradycardia also results from the decreased cardiac output related to decreased oxygen demand in vasoconstricted tissue. Heart rate can decrease to 30–40% of resting levels. During underwater diving competitions, heart rates as low as 20 beats/min have been attributed to the diving response. Bradycardia adds to the oxygen-saving effects through decreased myocardial oxygen consumption (17). The rate-dependent fall in contractility can be counterbalanced by the increase in sympathetic tone. Sometimes ectopic beats occur, either as escape arrhythmias or due to the simultaneous co-activation of the sympathetic and parasympathetic nervous systems. This may result in a vagal cardiac arrest (95, 102, 133).

Human studies with experimental head immersion in cold water and during apneic diving have shown that the diving response decreases oxygen consumption, slows arterial desaturation, and prolongs the duration of breath-holding or diving and the duration before asphyxia becomes life-threatening (9, 77, 134, 137). In the context of drowning, some consider the reflex fantastic physiology, others physiological fantasy.

Apnea and face immersion in cold water, alone or in combination, may occur in some drowning scenarios. Under these circumstances, the diving response may occur. But many drowning scenarios will involve neither voluntary apnea nor cold water. Other mechanisms may also cause interference. For example, when a drowning victim is able to take a full breath before disappearing under water, the increased intrathoracic pressure may result in decreased cardiac output and hypotension (9). Also, the physical effort associated with the predrowning struggle to maintain the airway clear of the water may significantly decrease breath-holding and negate the diving response.

Bradycardia and hypertension may be indicators that the diving response has been active, information rarely available at the rescue site. Therapeutic interventions at the scene, in the emergency department, and in the intensive care department will limit exploration of the diving response in clinical settings.

Despite these reservations, the possibility remains that the diving response has a role in the prevention of fatal drowning, notably in very young children. It is important to realize that not all persons who engage in the struggle of drowning will experience the physiological processes of a diving mammal or human.

Autonomic Conflict

Interplay between sympathetic and parasympathetic components of the autonomic nervous system has led to the theory of "autonomic conflict" to account for the genesis of cardiac arrhythmias and dysrhythmias (244, 265). Arrhythmias arise due to simultaneous and conflicting positive and negative chronotropic signals to the heart. Human data, and data from isolated hearts, indicate that arrhythmias are most likely to occur in a situation of cyclical vagal stimuli to the heart, for example, with the reinstatement of breathing post-breath-holding, against a background of sympathetic stimulation due to cold, exercise, anger, or anxiety (264) (FIGURE 4).

Arrhythmias are usually observed within 10 s of the cessation of breath-holding. The fact that they occur on cold immersion without face immersion (57) indicates that the release of breath-holding is, in itself, an arrhythmogenic trigger, due to neural responses associated with the release of stimulation of the cardiac vagal neurons. The incidence of an arrhythmia increases further if breath-holding is coincident with submersion in cold water (57, 267). This is probably due to the greater vagal drive seen with face immersion and trigeminal nerve stimulation. This is a powerful pro-arrhythmic stimulus that, on initial immersion, occurs at a time when the QT interval does not match the underlying heart rate, further increasing the likelihood of cardiac arrhythmias (286). Cardiac arrhythmias are predominantly supraventricular and junctional but can include short bursts of ventricular tachycardia interposed between periods of bradycardia, supraventricular ectopic beats, or even atrio-ventricular blocks. Arrhythmias tend to occur when heart rhythm changes from tachycardia (sympathetic predominance) to bradycardia (vagal predominance) (57, 267). The appearance of arrhythmias immediately post-breath-holding on immersion in cold water may also depend on the timing or magnitude of this rhythm in relation to the cessation of breath-holding. While autonomic conflict commonly results in arrhythmias, these descend into fatal arrhythmias much less commonly and probably only in the presence of a

range of predisposing factors that may include ischemic heart disease, long QT, channelopathies, and atherosclerosis (FIGURE 4) (244).

It is possible that arrhythmias caused by cold water submersion could result in death but go undiagnosed, mainly because electrical disturbances to the heart that result in fatal arrhythmias are undetectable postmortem. Also, even if the primary problem on immersion is cardiac, spontaneous terminal gasping may result in water entering the lungs, giving the appearance of drowning.

Upper Airway Reflexes

The upper airway is composed of the nose, pharynx, larynx, and extrathoracic portion of the trachea, and has many reflexes relevant for life, including maintenance of an open airway and airway defense. Mild irritation of the laryngeal mucosa may lead to a laryngeal closure reflex as a protective reflex against materials entering the tracheobronchial tree. Laryngospasm is the closure of the aryepiglottic folds, false vocal cords, and true vocal cords. This reflex responds to direct laryngeal stimulation from secretions, blood, or a foreign body (41, 188–190, 285). Other upper-airway reflexes include the pharyngoglottal closure reflex, esophagoglottal closure reflex, and aerodigestive reflex (65, 243).

During the drowning process, laryngospasm may prevent the entrance of water into the lungs, but this remains controversial. The existence of laryngospasm, also known as glottis spasm, was mentioned in the earliest drowning studies to explain why 10–20% of all dead drowning victims had macroscopically dry lungs (147, 178). Laryngospasm as an explanation for dry lungs seems logical (131). However, dry-drowning and dry-lung have been variably explained not only by laryngospasm but also by vago-vagal cardiac inhibition, pulmonary reflexes, absorption of aspirated fresh water into the circulation, and various reflexes triggered by contact of the body with water (36, 61, 178, 230, 250). Some of these deaths were also labeled "hydrocution" or "atypical drowning" in the early medical literature (288). Critical appraisal of the original literature from the 1930s, as well as clinical observations, has concluded that dry-drowning as result of a laryngospasm is nonexistent. If a laryngospasm may initially have occurred, it will cease to operate as a result of progressive hypoxia of the laryngeal muscles while under water breathing efforts are sustained (178, 194).

Some morphological forensic studies, also using microscopic tracers of the drowning liquid, indicate that penetration of liquid into the lungs occurs in almost all drowning deaths (148), even in those with macroscopically apparent dry-lung. Actually, dry-lungs with no evidence of liquid penetration can be found only in bodies dumped into water after death on land (148, 178). Mechanisms other than aspiration may, however, also lead to lung changes mimicking liquid aspiration. Case studies describe mild pulmonary edema after swimming, snorkeling, and diving, especially in cold water (144). Subclinical pulmonary edema has resulted from marginal hyperbaric stress under hypoxic conditions (83), from intense exertion by athletic and military swimmers (144) and from an increase in catecholamines due to hypoxemia and stress, leading to pulmonary vascular overload. Another potential mechanism is negative-pressure pulmonary edema caused by extreme negative intrathoracic pressure from inspiration efforts against a

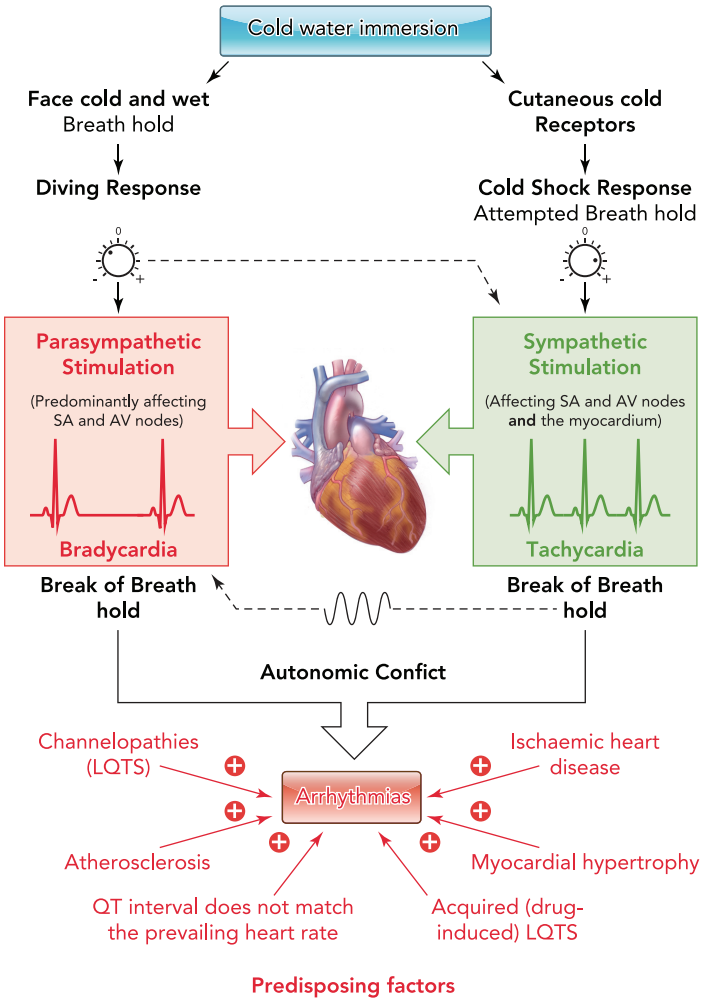

FIGURE 4. **Autonomic conflict**
Cold-water immersion activates two powerful responses: the diving response (on facial immersion) and the cold shock response (on the activation of cutaneous cold receptors). The magnitudes of these responses can vary with a range of factors including water temperature, clothing, and habituation. The diving response triggers a parasympathetically driven bradycardia, whereas cold shock activates a sympathetically driven tachycardia. It is hypothesized that together these conflicting inputs to the heart can lead to arrhythmias, particularly at the break of breath-hold, which increases parasympathetic tone that varies with respiration. The substrate for arrhythmias is enhanced by various predisposing factors. Figure is from Ref. 244 and used with permission from *The Journal of Physiology*.

closed glottis (60, 184, 279). On the other side, clinical studies report that the majority of drowning resuscitation survivors, whom can be assumed to have been under water for a sustained interval, have no clinically relevant pulmonary complications (76, 274).

One problem in gathering evidence for this debate on upper airway reflexes is that knowledge of laryngeal muscle function is limited. Some information can be extracted from studies on speaking, singing, and swallowing (65, 243, 292). The muscles of the larynx have largely involuntary medulla-mediated tasks during swallowing, breathing, and coughing, and volitional cortex-mediated tasks during speaking and crying for help. For each of its functions, the different vocal-fold movements need rapid and precise control by intrinsic and extrinsic laryngeal muscles. All these muscles can be actively controlled.

Older studies assume that the afferent end-organs in the larynx can respond to pressure, flow, respiratory drive, osmolarity, temperature, and chemical irritants (285). More recent studies on the pharyngoglottal closure reflex, relevant for prevention of food aspiration, show that the laryngeal vocal folds close when water is injected rapidly toward the posterior pharyngeal wall (65, 243). Anatomic coordination exists between the larynx muscles, respiratory reflexes, and cough control through stimulation of the same internal branches of the superior laryngeal nerve (8, 242). Because all four functions of the laryngeal muscles (swallowing water, breathing, coughing, crying for help) are relevant to the drowning process, these muscles must play an active but still poorly studied role in drowning (142).

Although there is on-going discussion about the existence of laryngospasm during drowning, it will only be protective in those few patients where the spasm has been activated and is still active at the moment of rescue from the water.

Aspiration of Water

The lung is an immediately vulnerable target during the drowning process. During laryngospasm, forceful ventilatory movements against a closed glottis may cause mechanical damage. Furthermore, during the drowning process, both hypertonic and hypotonic aspirated liquids cause changes to the pulmonary surfactant and to the alveolocapillary barrier that result in systemic hypoxemia.

Confusion exists about the volume of water aspirated in drowning and how these volumes contribute to respiratory impairment. Speculative extrapolations of data from experimental and postmortem studies to clinical settings have contributed to this confusion. Aspirated volumes are also reported in milliliters per pound in some studies and in milliliters per kilogram in others (90, 177, 179, 180).

Hypotonic liquid, when reaching the alveoli, damages and dilutes pulmonary surfactant. The increase in the alveolar surface tension, along with diminution of pulmonary compliance, causes alveolar instability and atelectasis that alters the ventilation-to-perfusion ratio. Because a large part of the lung is not adequately ventilated, more venous blood bypasses the lungs, and the shunt fraction increases. Aspiration of 2.5 ml/kg of sea water causes the pulmonary shunt fraction to increase by 75% (206). Hypotonic fresh water tends to be absorbed into the pulmonary circulation and distributed throughout the body. Aspiration of hypertonic seawater draws liquid from the plasma into the alveoli and also causes damage to surfactant (215). In both situations, the supranormal hydrostatic forces over the alveolar-capillary membrane will disrupt its integrity. Plasma enters the alveoli, incapacitating normal gas exchange. Plasma in the alveoli may also generate foam that further decreases pulmonary efficiency (147). This results in a local adult respiratory distress syndrome-like clinical picture (85, 92, 176, 274).

Spontaneous ventilation persists after submersion and causes liquid penetration into the lungs. Moreover, terminal shock induced by a variety of natural causes can produce pulmonary stasis and edema mimicking a wet lung indistinguishable from that observed in actual drowning. It is therefore impossible macroscopically or microscopically to assert whether a lung contains or does not contain aspirated liquid. One way to assess, and eventually quantify, the penetration of drowning liquid into the lungs is to study the presence of microscopic tracers of the drowning liquid (147).

Swallowing of Water

Swallowing water during the drowning process may increase the risk for vomiting, spontaneously or during resuscitation, eventually leading to aspiration of gastric content. Swallowing water may also contribute to life-threatening electrolyte disorders.

Under normal conditions, the process of swallowing liquid includes an oral phase, a pharyngeal phase, and an esophageal phase (69, 160, 174, 252). Swallowing is triggered by cortical inputs integrated into the swallowing central pattern generator (SCPG) of the brain stem (69, 70, 115, 129, 174). The SCPG sends efferent innervations to over 30 muscles involved in swallowing. Afferent pathways originate from chemical or mechanical receptors in the upper-airway mucosa and from lung and intercostal muscles. Information is conveyed via

cranial nerves V, IX, and X to the brain stem and the nucleus tractus solitarium. The main efferent pathways are via the ambiguous and hypoglossal nuclei (XII) (189).

Coordination between breathing and swallowing prevents liquid aspiration (189). During swallowing, elevation of the soft palate, tilting of the epiglottis, and SCPG-mediated inhibition of airway reflexes interrupt respiration for 0.5–1.5 s during the inspiration-expiration transition or the expiratory phase (37, 63, 127, 190, 205, 240).

During drowning, swallowing of liquid usually occurs during partial head-out immersion or during breath-holding (see *Sympathetic Activation, Fear of Drowning* above). Under these circumstances, active and passive swallowing differ from the normal physiological processes (56). Uncontrolled premature entry of liquid into the pharynx can cause aspiration and swallowing, accentuated by a cough reflex. Stress, increased P_{CO_2}, decreased P_{O_2}, respiratory- and lung-volume changes, and unconsciousness hamper coordination between swallowing and respiration may cause swallowing during inspiratory and expiratory phases, with a consequent risk for aspiration (123, 124, 173, 188–190, 223).

Water swallowing during drowning has long been a subject of investigation (71, 218), but with little high-quality data. Experiments in the 1970s in rats, when electrolyte disorders were considered important for drowning outcome, suggested that the ratio of aspirated to swallowed liquid differed between fresh (1:1) and salt water (1:3) (58). The relevance of this to conscious humans is unknown.

Some authors maintain that drowning victims swallow much more water than they inhale (195). At this moment, autopsy data are still inconclusive (141, 230). Based on one of the author's (P. Lunetta) investigation of over 2,000 fatal drownings, the stomach of a drowning victim is either empty, or contains watery fluid, liquid mixed with food, or exclusively food. Postmortem liquid penetration into the stomach or its leakage into the small intestine impedes reliable prospective studies. Presence of water-borne particles in the stomach, such as plankton, is also not conclusive, because they can penetrate postmortem or can be present in food and beverages consumed before the incident (101, 146, 290). One postmortem computerized tomography (CT) study on 28 retrieved drowning bodies revealed gastric distension in 89%, but high-attenuation sediment in only 21% (135). Another series of 10 fatal fresh-water drowning cases examined by postmortem CT, reported a gastric volume ranging from 50 to 1,200 ml, with an average density of gastric contents less than the control group (49). Although some drowning victims clearly have swallowed water, data are limited as to the incidence and clinical relevance, and whether differences exist between fatal and non-fatal drowning.

Emesis

Detailed data on the occurrence of emesis in drowning are also lacking. One study, reported 25–60% of drowning victims vomited (151). Another study revealed that emesis occurred in 86% of drowning victims who required cardiopulmonary resuscitation and in 50% of those who required no intervention (154). Autopsy series have disclosed aspiration of gastric contents in 24% of drowning victims (80). In a large series on out-of-hospital cardiac arrest (CA) with a cardiac and non-cardiac etiology, emesis occurred in 30–35% of all patients (247). The trigger can be the condition underlying the arrest, CA itself, gastric distension caused by artificial ventilation, or improper chest compression that increases intra-abdominal pressure.

The main vagal sensory afferents responsible for emesis originate from mechano-, osmo-, and chemoreceptors activated by gastric distension or mucosal irritation (14, 21). Mucosal chemoreceptors in the stomach can be stimulated by hydrochlorides or hypertonic saline (13). These afferents relay information to the nucleus tractus solitarium and then to the medulla oblongata, where a neural network (central pattern generator) coordinates the efferent response (21, 110). This integration area receives afferents also from the cerebral cortex, the vestibular region, and a chemoreceptor trigger zone located between the medulla and the floor of the fourth ventricle. The chemoreceptor trigger zone detects, within the blood, emetic stimulants, including hypoxia and ketoacidosis. The efferent motor pathways innervate the upper gastrointestinal tract via cranial nerves V, VII, IX, X, and XII, the diaphragm and abdominal muscles via spinal nerves (21, 26, 110).

Emesis includes retroperistaltic activity from the small intestine, relaxation of the pyloric sphincter, downward contraction of the diaphragm with decreased intrathoracic pressure, increase in intra-abdominal pressure, contraction of the abdominal wall muscles, squeezing and contraction of the stomach with elevation of intragastric pressure and closure of the pylorus, relaxation of the esophageal sphincter, and expulsion of gastric contents (130, 172). The extent to which these classical reflex mechanisms explain emesis in drowning is undefined.

During drowning, gastric contents can be aspirated into the airways, resulting in pulmonary infection and chemical irritation (68, 274). Emesis can also interfere with pulmonary resuscitation. In drowning, both vomiting and cardiopulmonary resuscitation may cause gastric mucosal tears, the

frequency of which varies widely among studies but has been detected in as many as 21% of patients (15, 33, 55, 145).

Electrolyte Disorders

Experiments during the 1940s and 1950s have long influenced the concepts of drowning pathophysiology (254–256). These experiments stressed the role of liquid osmolarity in drowning, especially the penetration of hypotonic liquid into the circulation, causing hypervolemia, erythrocyte hemolysis, intravascular potassium release, and subsequent VF. Studies beginning during the 1960s (179, 180) suggested that the volume of aspirated water rather than its osmolarity is the critical factor. Pathophysiological differences between freshwater or saltwater drowning are observable in experimental models. However, in most drowning victims, serum electrolyte changes are of limited importance because liquid redistribution within the body rapidly restores electrolyte balance. Hypo- and hypertonic liquid cause a ventilation/perfusion shift and hypoxemia and metabolic acidosis. These, in turn, cause myocardial depression, pulmonary vasoconstriction, and changes in capillary permeability that worsen pulmonary edema (148). The final common pathway is hypoxemia.

In most environments, drowning is not associated with clinically important electrolyte changes. When such changes do occur, it can be impossible to disentangle the roles of ingestion and aspiration (136). The small intestine absorbs ~80% of ingested liquid via concentration gradients and complex molecular mechanisms (84). When seawater reaches the small intestine, sodium moves mainly through the jejunal mucosal cells of the villi into capillaries. Water diffusion follows the osmotic gradient (245).

In sporadic cases, such as in protracted immersion while wearing a malfunctioning or poorly designed lifejacket, sea-water ingestion following the breath-holding breaking-point directly causes hypernatremia (67). Serum sodium concentrations higher than 145 mM have occurred in pediatric drownings (111). Chlorine ions may also pass the intestinal barrier by concentration gradient, causing metabolic acidosis (67, 111). In specific environments such as the Dead Sea, with its high magnesium and calcium concentration, ingestion of as little as 200 ml may have a significant clinical impact (136). Conversely, swallowing hypotonic liquid thus far has not been reported to result in water intoxication, although swallowing fresh water can cause hyponatremia, especially in children (287).

Electrolyte disorders have been considered a major factor in drowning mortality previously. Current studies suggest that this only occurs in exceptional circumstances.

Neurophysiology

The cerebral physiological response to drowning is poorly understood but is most likely an interaction between hypoxemia, submersion liquid temperature, aspiration, and cold shock. Most information pertaining to cerebral physiological responses to drowning is derived from experimental models simulating CA, which may not be directly relevant. Cardiogenic VF causes abrupt cessation of oxygenated cerebral blood flow (CBF). Asphyxia, in contrast, causes progressive cerebral hypoxia that precedes CA. Antecedent hypoxemia aggravates injury associated with asphyxial CA. In canines, a shorter interval of normothermic asphyxia-induced CA causes more severe injury than an even longer interval of VF CA (271).

A critical event in drowning is loss of consciousness (LOC). This is often attributed to asphyxia following submersion, loss of pulmonary oxygen uptake, brain energy failure, and deterioration of brain function. Hypoxemia in normothermic healthy humans causes an initial cerebral vasodilatory response to preserve oxygen delivery (3). Progressive hypoxemia leads to a depletion of high-energy phosphates and loss of electrocortical activity consistent with LOC (171). The duration of this state defines the severity of injury and reversibility of neurological dysfunction.

The CBF response to sudden cold-water immersion may be adverse. Healthy humans suddenly immersed in 0°C fresh water were monitored with transcranial Doppler (156). In untrained subjects, CBF rapidly decreased by ~50% from normal, followed by loss of sensorium necessary for self-rescue. Immersion also causes hyperventilation at a magnitude consistent with the known depressant effects of hypocapnia on CBF and mental status (122). Respiratory cold-shock responses, alone, may be sufficient to precipitate LOC, later complicated by submersion and asphyxia. Trained suppression of hyperventilation or habituation to cold prevents the CBF decrease and need for rescue (157). Hence, conscious suppression of hyperventilation in cold water may be beneficial in prolonging duration to LOC so as to aid in brain cooling before submersion.

The brain's tolerance to energy deprivation is closely associated with brain temperature (170). Brain cooling before asphyxial CA is important to submersion outcome (46, 86, 246). In the rat, deep hypothermia induced by submersion during an otherwise lethal asphyxial insult is profoundly protective, but only if core temperature decreases rapidly (283). Brain cooling in humans depends on several factors. Surface cooling of the head alone

has little effect (168). At the same time, in oxygenated humans, submersion of the head with the rest of the body accelerates the rate of core cooling by 56% (212). Circulatory function is necessary to rapidly decrease brain temperature. After LOC from asphyxia, hypothermia, or cold shock, ventricular contractions may or may not persist to provide additional brain cooling (143).

Deep hypothermic CA, employed for some surgical procedures, may offer insight into neuroprotective physiology in drowning. Brain cooling is dependent on blood flow, perfusate temperature, and cooling duration (150). Use of pH-stat control of carbon dioxide tension accelerates cooling by allowing cerebral vasodilation (128), but no opportunity for this exists in drowning. Hence, progressive brain cooling would be expected to decrease CBF proportionate to increased blood carbon dioxide solubility and a coupled decrease in cerebral metabolic rate (54, 204). The balance between these factors and water temperature likely defines the speed and depth of brain cooling during immersion and tolerance of hypoxemia.

Pulmonary aspiration may aid brain survival due to cooling of blood flowing past aspirated cold water (289). Lightly anesthetized, spontaneously breathing dogs were submerged in 4°C water with an open airway allowing aspiration (51). In controls with endotracheal tube protected airways, aspiration was prevented. Carotid blood temperature in those dogs with an open airway decreased to 29°C within 2 min, followed by a decrease in respiratory rate. Carotid blood temperature in controls decreased to only 36°C. Delivery of 29°C blood to the brain is sufficient to suppress consciousness (74) but also elevates cellular tolerance to anoxia. Trauma or use of drugs before submersion may affect any of these mechanisms.

Regardless of cause, a sustained decrease in oxygen delivery causes brain energy metabolism failure, inhibited protein synthesis (201), ATP depletion (171), loss of synaptic neurotransmission (204), oxidative stress (281), ionic gradient deterioration (20), and initiation of intracellular cascades leading to apoptosis, autophaghy, or necrosis of neurons and glia (214). Little information is available to distinguish neuropathological responses induced by asphyxial vs. cardiogenic CA. Most data are derived from simulated VF arrest models where vulnerability of brain cell types to deprivation is selective. Hippocampal CA1 pyramidal neurons and cerebellar Purkinje cells are particularly sensitive (125, 209). More severe insults produce wider morphological and functional damage (248).

Many drowning victims survive, sometimes for decades, with permanent brain damage (31, 192, 224, 272, 276). Among the physiological mechanisms activated during drowning, the neurophysiological responses are vital, and their improved understanding is essential to advance intervention success (270).

Future Research

Awareness has been growing that drowning constitutes a neglected epidemic (28, 164). Because 90% of drownings occur in low- and middle-income countries (138), preventative community measures are likely to have the greatest impact (139, 216).

In this review, 14 different physiological mechanisms of drowning have been described. Each of them may play a role in drowning. A decrease in fatal and nonfatal drownings may also be achieved by a better understanding of drowning physiology.

Studies that will result in a better understanding of the mechanisms of hot water immersion, cold shock, cold-induced physical incapacitation, and fear of drowning may contribute to prevention of drowning where these mechanisms play a role. A better understanding of the physiological mechanisms of deep tissue cooling, water aspiration, hypoxic cardiac arrest, and neurophysiology may contribute to better treatment options and outcome. Better knowledge of the physiology of drowning is also crucial in forensic pathology for: interpretation of events leading to death by drowning; assessment of medical liabilities in fatal incidents; and analysis of postmortem findings. Research on breath-holding, diving response, upper airway reflexes, and autonomic conflict in drowning settings can be used as models for physiology studies. Based on these assumptions, research directives are suggested.

Research That May Help to Prevent Drowning

Hot water immersion. A large case-control study in different age groups, especially the elderly with preexisting cardiac disease, would determine whether there is an actual excess mortality during hot water bathing and its accompanying risk factors. Focused postmortem investigations can better define cause of death. Studies in human volunteers are required to define the cardiovascular effects of hot water immersion and ambient temperature.

Cold shock. A more detailed understanding of the neurophysiological pathways associated with cold shock is needed, including the relationship between surface area exposed to cold water and the magnitude of the cold-shock response. Some methods of mitigating the dangers associated with cold shock are known, but others, such as pharma-

cological interventions, remain to be investigated. The cold-shock response has undergone little study in children or the elderly. Current knowledge is largely derived from 18- to 39-yr-old subjects studied in the laboratory.

Superficial tissue cooling. The cellular and bimolecular physiology of impaired neuromuscular function during cooling requires investigation. Physical incapacitation is a major threat associated with immersion in cold water and a common precursor to drowning.

Fear of drowning. The lay community indicates this is a phenomenon that has not been appreciated by the medical and scientific community. The physiological and psychological substrates need further study, which may decrease drowning in experienced and inexperienced swimmers.

Research That May Help to Improve Treatment and Outcome of Drowning

Deep tissue cooling. Rewarming methodologies and devices need systematic appraisal of physiological and clinical efficacy in profoundly hypothermic victims.

Aspiration of water. Data from forensic and clinical studies on the frequency, severity, and clinical consequences of aspiration during and after drowning are conflicting and require more investigation. Understanding of the role of aspiration of water may be important in decision making such as: *1*) When should a drowning victim be allowed to return home from the beach or the emergency department? *2*) Which drowning victims may or may not benefit from extracorporeal membrane oxygenation? Studies should define the contribution of respiratory failure to drowning mortality.

Hypoxic cardiac arrest. Cardiac arrest after drowning is different from cardiogenic cardiac arrest. Immediate post-rescue data are needed to understand heart function between the moments of submersion and the onset of cardiac arrest. This information will inform whether resuscitation after drowning requires different skills or different application of skills. More knowledge is required pertaining to the effects of cardiac compressions on a hypoxic, hypothermic, and acidotic, but still working, heart.

Neurophysiology. Case reports have indicated excellent neurological outcomes from drowning (66, 93, 116, 143, 225, 253, 277). It is now evident that long-term, often disabling, cognitive deficits remain (253, 276). Comprehensive investigation into the nature of persistent cognitive deficits in drowning survivors may serve to inform study of mechanisms of injury and interventions specifically relevant to this population.

Most molecular and cellular biology associated with CNS injury in drowning has been extrapolated from cardiogenic CA or experimental acute disruption of global CBF. CA preceded by anoxia differs markedly from abrupt flow cessation in both severity and recovery (271). Thus CNS research specific to drowning is necessary.

The largest impediment to this is availability of a reliable, highly characterized preclinical CNS-specific drowning recovery model. Emphasis on developing a model is paramount, as is defining confounding influences of requisite anesthetics. Drowning-specific models will allow increased understanding of CNS effects of and interactions among cold shock, autonomic conflict, aspiration and swallowing, temperature, electrolytes, and anoxia. Such understanding will allow investigation into improved CNS resuscitation techniques for drowning. Transfer of knowledge gained from the large body of research already focused on treatment of cardiac arrest, traumatic brain injury, and stroke may then be evaluated specifically in the context of drowning to define relevance and potential for clinical advance. Efficacious therapeutic and preventive concepts will emerge only when the basic injury mechanisms are better understood and preclinical therapeutic efficacy is rigorously characterized (280).

Studies That May Help the Forensic Investigations of Drowning

Aspiration and swallowing of the drowning media. Quantitative postmortem studies on the penetration of water-born exogenous substances (such as planktonic elements, pollutants, electrolytes) in the lung, circulation, and internal organs of victims of drowning can assist the postmortem diagnosis of drowning. Moreover they may provide more accurate estimates of the volume of drowning liquid being aspirated and/or swallowed during the drowning process. First, diagnostic values of the substance concentrations found in victims with verified fatal drowning vs. non-drowning deaths should be defined to discriminate antemortem from agonal penetration or, even, ante- or postmortem contamination. Then, such values can be assessed in a range of drowning situations characterized by factors that may affect the duration of the drowning process; for instance, an acute cardiac event, alcohol or other drug intoxication, or preexisting disease. Assessment of the original concentration of such substances in the drowning media, at the site of drowning, is a prerequisite for such studies.

Postmortem changes of serum electrolyte concentrations in the organism resulting from penetration of the drowning liquid should be further assessed as a function of the duration of the drowning process and the tonicity of the drowning media. A major challenge for future studies re-

mains the discrimination between actual antemortem changes and those occurring postmortem as a result of the decomposition process and the prolonged contact of the body with the water. Research on cellular and molecular changes and markers associated with drowning should focus on those occurring during the short time frame of the drowning process. Control groups of non-drowned individuals who died on dry land and were subsequently disposed in water are vital for such studies.

Comprehensive postmortem investigations, including molecular testing and toxicology tested against controls, should clarify the actual role and pathophysiological mechanisms of preexisting diseases or acute conditions (e.g., arrhythmogenic gene mutations, cardiac disease, alcohol and drug intoxication) for drowning in different settings.

Physiology Studies

Breath-holding. The physiology of breath-holding is well understood. From a drowning perspective, what is less understood is what happens at the break of breath-hold in a submerged individual. How much water enters the stomach, and how much enters the lung? What volumes and conditions are required to produce incapacitation or laryngeal spasm? The answer to these questions is of importance but necessitates (ethically problematic) experimental studies.

Diving response. When detectable substrates of the vagal response can be identified, such markers of parasympathetic activity may be clinically relevant.

Upper airway reflexes. Further studies in this field may help to better understand when and why laryngospasm may or may not occur during drowning and whether the upper airway reflexes affect drowning mortality and morbidity.

Autonomic conflict. It has been relatively straightforward to produce arrhythmias and dysrhythmias in healthy, young humans following breath-holding submersions in cold water. It has proved more difficult to model this in other settings. Thus, although autonomic conflict may produce arrhythmias, as yet, it has not yet been possible to determine what turns these benign arrhythmias into more dangerous waveforms. A model of autonomic conflict will allow precursors that cause the descent from benign to fatal arrhythmias to be identified.

Conclusion

Immersion and submersion, the two entities of drowning, interact with basic physiological factors: temperature and oxygen. Little is known about these mechanisms when they occur under the extreme and lethal circumstances that result in drowning. Few studies reveal how these mechanisms interact, whether directly or indirectly, and how they are influenced by autonomous protective (diving response, breath-holding, acute hypothermia) and life-threatening (cold shock, autonomic conflict, aspiration) responses.

The theories to explain how drowning happens via these mechanisms have been taken for granted for several decades. A critical appraisal, based on current understanding and knowledge, suggests that little is definitively known about the pathophysiological events associated with drowning. Such knowledge is not just of academic interest; it can guide in preventative measures, assist in the clinical treatment of drowning fatalities, and aid in forensic studies. Increased appreciation of the prevalence of drowning-related death should foster major research efforts specific to this population. ■

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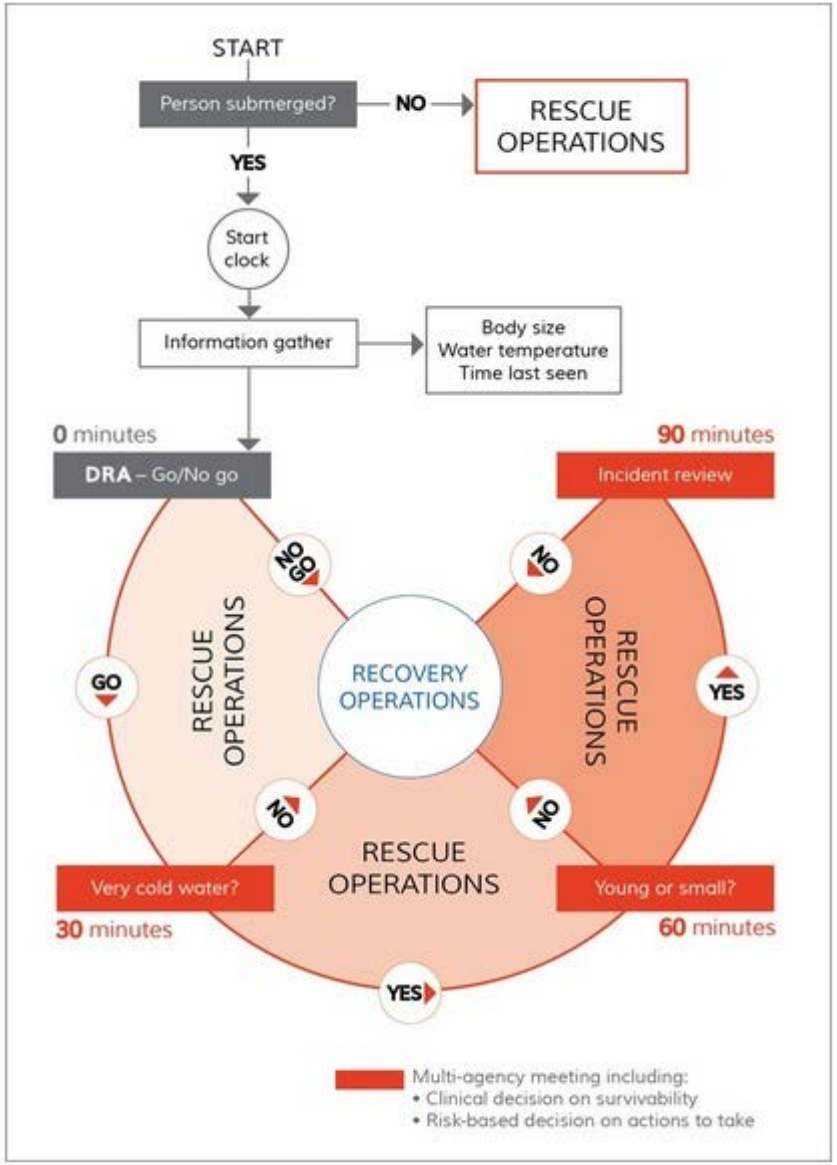
National Operational Guidance Programme

Local Government Association | CFOA Chief Fire Officers Association | in partnership with LFB London Fire Brigade



The model is designed to give casualties every reasonable chance of rescue and resuscitation and is balanced against the risk of harm to responders when carrying out rescues. DRA = dynamic risk assessment.

Available medical evidence suggests that water temperatures in the region of 6-7°C or less are required for prolonged survival times in submerged casualties.

http://www.ukfrs.com/Blog/Post/65/Water-rescue-and-flooding