Partner Veterinary

Surviving Sepsis:

A Deep Dive into Diagnosis and Stabilization of the Septic Abdomen

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Lecture Objectives

- To walk through a septic peritonitis case from presentation to post-op
- In doing so, we will review principles of sepsis, shock, and resuscitation
- DISCLAIMER – To everyone's dismay I'm not a surgeon and this is not a surgery lecture

A fair amount of this lecture is derived from this single AMAZING review – HIGHLY ENCOURAGED READING!

INVITED REVIEW | Open Access

Diagnosis and surgical management of septic peritonitis in small animals: A review

Bonnie G. Campbell DVM, PhD, DACVS, Shana K. O'Marra DVM, DACVECC

First published: 18 October 2025 | https://doi.org/10.1111/vsu.70024 | VIEW METRICS

Before we jump into the case…What is SEPSIS?

SPECIAL ARTICLE | Open Access

Defining sepsis in small animals

Stefano Cortellini DVM, MVetMed, DACVECC, DECVECC, Amy E. DeClue DVM, MSc, DACVIM, Massimo Giunti DVM, DECVECC, Robert Goggs BVSc, PhD, DACVECC, DECVECC ... See all authors

First published: 13 February 2024 | https://doi.org/10.1111/vec.13359 | VIEW METRICS

TLDR: vet medicine doesn't really have a universally agreed upon, formal definition of sepsis

"Traditional" definitions were often something like: SIRS + infection

Human med has **revised definition three times since 1991 to try and more precisely identify SEPSIS** versus sterile/non-infectious SIRS conditions

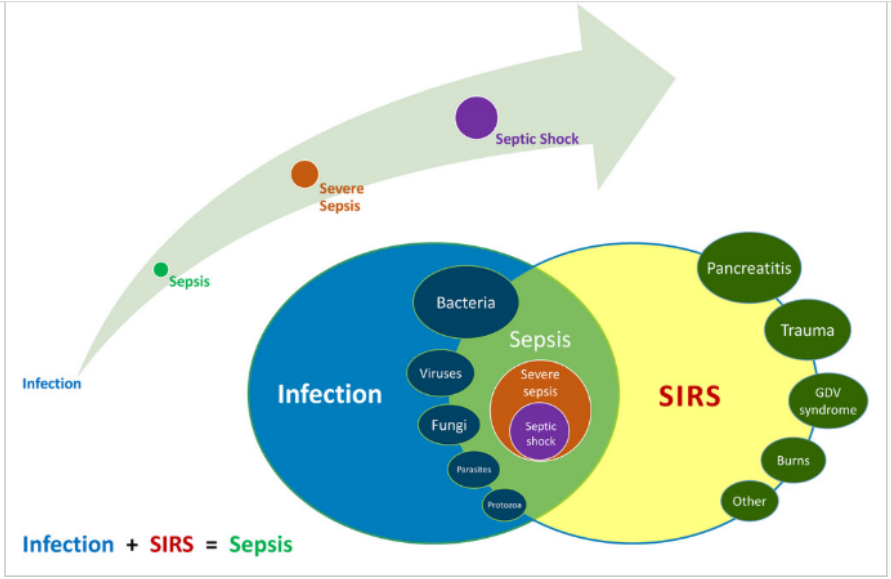


FIGURE 1 Open in figure viewer | PowerPoint

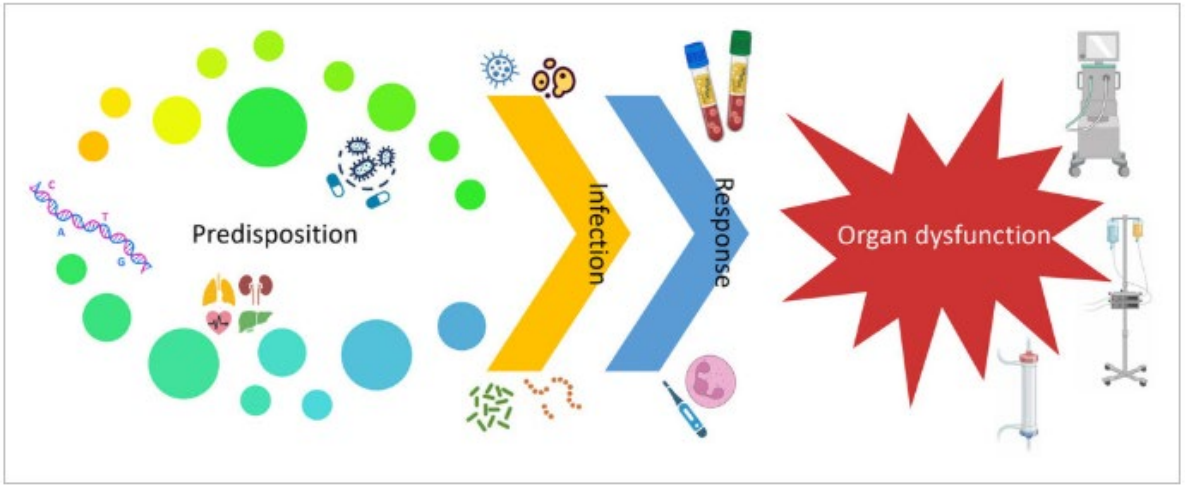
A schematic representation of sepsis as defined by the 1991 Consensus Conference. Per the 1991 (Sepsis-1) consensus definitions, sepsis is the result of the systemic inflammatory response to infection. The systemic

Not all patients become septic from the same infection and not all septic patients present the same way!

In 2001, human med introduced the PIRO concept

Predisposing factors
Infection factors
Host **R**esponse
Organ dysfunction

Some even consider sepsis a genetic disease!



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Brief note on DAMPs and PAMPs because it's fun to say

- PAMPs = pathogen associated molecular patterns
- DAMPs = damage associated molecular patterns (injured or dying host cells)

- BOTH PAMPs and DAMPs bind PRRs (pattern recognition receptors) to initiate and amplify the inflammatory response (cytokine storm)

- DAMPs fuel inflammation and organ dysfunction even AFTER the pathogen is gone → **host-dependent response**

The UPDATED definition of sepsis in human medicine (sepsis 3)…

Sepsis = life-threatening organ dysfunction secondary to host's dysregulated response to infection

Table 1. The Sequential Organ Failure Assessment (SOFA) score.

Respiratory system	Nervous system	Cardiovascular system	Liver	Coagulation	Kidneys	SOFA score
PaO2/FiO2 (mmHg)	Glasgow Coma Scale	Mean arterial pressure (MAP) OR administration of vasopressors required	Bilirubin (mg/dl) [μmol/L]	Platelets × 103/m	Creatinine (mg/dl) [μmol/L]; urine output	
>400	15	MAP > 70 mmHg	<1.2 [<20]	>150	<1.2 [<110]	0
<400	13–14	MAP < 70 mm/Hg	1.2–1.9 [20–32]	<150	1.2–1.9 [110–170]	1
<300	10–12	Dopamine ≤5 μg/kg/min or dobutamine (any dose)	2.0–5.9 [33–101]	<100	2.0–3.4 [171–299]	2
<200 with respiratory support	6–9	Dopamine >5 μg/kg/min OR epinephrine ≤0.1 μg/kg/min OR norepinephrine ≤0.1 μg/kg/min	6.0–11.9 [102–204]	<50	3.5–4.9 [300–440] (or urine output <500 mL/day)	3
<100 with respiratory support	<6	Dopamine >15 μh/kg/min OR epinephrine >0.1 μg/kg/min OR norepinephrine >0.1 μg/kg/min	>12.0 [>204]	<20	>5.0 [>440]; urine output <200 mL/day	4

MAP, mean arterial pressure; SOFA, Sequential Organ Failure Assessment.

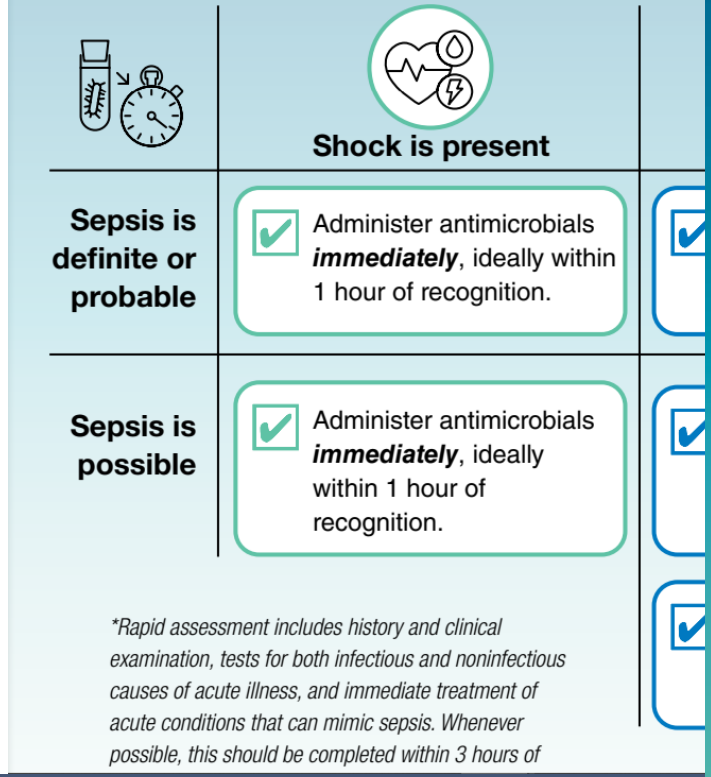
The Sequential Organ Failure Assessment (SOFA) score.

Everyone loves a good furniture acronym – they use the SOFA score (sequential organ failure assessment)

How does sepsis/inflammation cause organ failure? The most pathophys-heavy slide of the day

- **Direct cytotoxic effects of inflammatory mediators**
- **Hemodynamic instability** – (mal)distributive shock and hypotension from inflammatory mediators (like inducible nitric oxide synthase) result in venous pooling and reduced critical organ perfusion (systemic arteriole pressure drives perfusion)
- **Endothelial failure**→ organ ischemia is consequence of microvascular thrombosis and tissue edema
  - **Inflammation causes hypercoagulability!**
  - **Glycocalyx is disrupted or denuded**
  - "Endothelium is the motor of MODS" – *This is maybe my favorite quote of all time*
- **Cytopathic hypoxia/mitochondrial dysfunction** → reactive oxygen species damage mitochondria, rendering cells unable to use oxygen for energy production even if it makes it to the cells

"Surviving Sepsis" Bundles (ie: protocolized care)



Vasoactive Agent Management

	Use norepinephrine as first-line vasopressor.
For patients with septic shock on vasopressors	Target a MAP of 65 mm Hg.
	Consider invasive monitoring of arterial blood pressure.
If central access is not yet available	**Consider** initiating vasopressors peripherally.*
If MAP is inadequate despite low-to-moderate norepinephrine	**Consider** adding vasopressin.
If cardiac dysfunction with persistent hypoperfusion is present despite adequate volume status and blood pressure	**Consider** adding dobutamine or switching to epinephrine.

A nice veterinary “bundle” resource

TVP-2024-1112_Algorithm_Sepsis-1.pdf

Let's Present THE CASE

- **Time/Place**: Monday morning, 10am
- **The Patient:** Murphy, 7 y/o MN golden doodle (30kg), presenting for a 3d history of hyporexia, lethargy, and weakness.
- **The Exam:** Weakly ambulatory and dull on presentation, temp 103.2, MMs muddy and tacky with prolonged CRT (3-4s), tachycardic (HR 200bpm) with synchronous but weak pulses, thoracic auscultation WNL, abdomen tense and palpation elicits mild discomfort
- **Past Relevant History:** Started to get picky about his food on Friday night, vomited a sock on Saturday but no further vomiting, minimal interest in food Saturday or Sunday, today didn't want to get up.
- **Current Medications:** Rimadyl 50mg PO q12h for hip osteoarthritis (has been on an NSAID for about 3 years)

Is Murphy in SHOCK?

- Shock = inadequate cellular energy production; oxygen delivery < oxygen consumption
  - Signs of shock?

- Differentials (audience participation)

Things we can do IMMEDIATELY for Murphy (or almost any shock patient)

- PLACE AN IVC
  - Bonus points if you can grab some blood off that IVC placement
    - Glucose
    - PCV/TS
    - Lytes
    - Lactate
- Initiate a fluid bolus
- Put him on ECG
- Obtain a blood pressure
- POCUS exam

About our FLUID BOLUS

- **WHY** do we give a fluid bolus?
- What **VOLUME** of fluids do you choose?
- What **TYPE** of fluids do you use?
- In which SHOCK patients is an initial fluid bolus **contraindicated?**

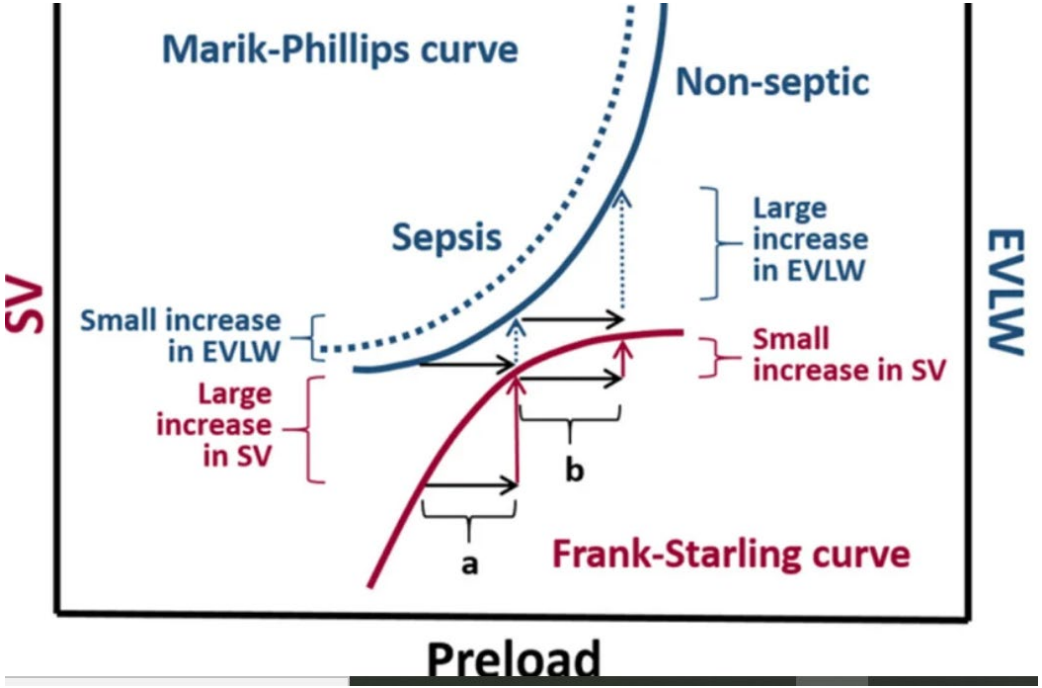
The Art of the IVF Bolus

- Frank Starling curve → stroke volume (and cardiac output) is related to pre-load or filling pressure…to a point

- Patients can be divided into “fluid responders” and “fluid non-responders”

- Fluid responders will hemodynamically improve with fluid boluses; non-responders won’t improve and may start to rapidly develop edema

But let's take that one step further …



TLDR:
If you need fluids, increasing preload (with IV fluids) improves your cardiac output…

In healthy patients, once you're no longer fluid deplete, IVFs will start to go into extravascular spaces (including lung interstitium)

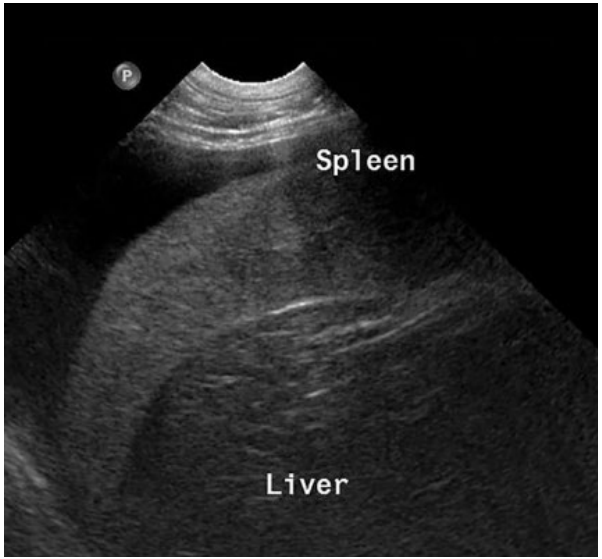
SEPTIC PATIENTS ARE VERY LEAKY

Other things you'd like to know about Murphy (history, exam, etc)?

Pick your own adventure

Additional Exam and Diagnostic Findings

- POCUS:



BP: 70mmHg (doppler)
Glucose: 55mg/dL
Lactate: 7.0
PCV/TS: 62%, 5.3g/dL
CBC: neutrophils 0.4K, PLT 75K
Chem: BUN 62, creat 2.6, albumin 2.7, ALT 360
iCa: 1.0 (low)
PT: 36s
PTT: 145s

https://todaysveterinarypractice.com/radiology-imaging/ultrasonography-peritoneal-retroperitoneal-spaces-abdominal-lymph-nodes/

Worth noting – rads vs ultrasound

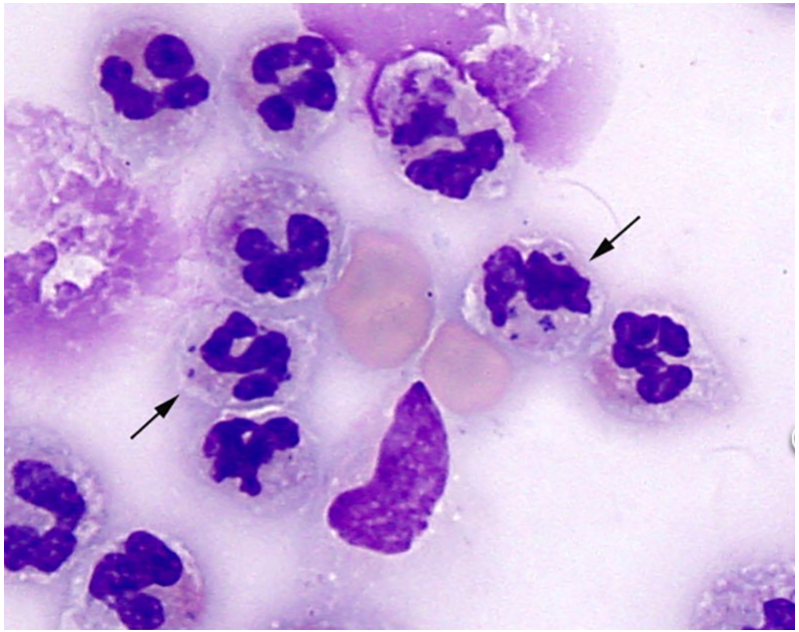
Point of care ultrasound (POCUS) is commonly used to identify free abdominal fluid and often incorporated into initial patient assessment.[89] Small amounts of peritoneal fluid are more reliably detected with ultrasound (≥6.6 mL/kg) than radiographs (≥8.8 mL/kg).[90] Similar to humans, POCUS reliably detected free abdominal fluid in dogs and cats with recent trauma compared to the gold standard of computed tomography (CT).[91] Ultrasound may be even more sensitive at detecting small volumes of free air, reliably identifying 0.4 mL of air in

Pneumoperitoneum was present in about 57% dogs with perforated GIT

Free fluid may be easier to detect (and sample) with AUS than radiographs

Ultrasound may also have utility in identifying dehiscence but that's likely user-dependent (ie I can't do that)

You tap the abdominal effusion and obtain the following:



https://eclinpath.com/atlas/cytology-2/canine-peritoneal-fluid/

Lactate (fluid): 14
Glucose (fluid): 26mg/dL

Interpret the cytology?

A note on upper GI perforations…you won't always see bacteria!

NB!! Comparative Glucoses and Lactates

We often measure comparative glucoses and lactates on abdominal effusion vs peripheral blood

A significantly lower glucose on effusion vs peripheral blood (high WB-EGD) and a significantly HIGHER lactate support SP

POC glucometers are not always accurate (over-estimate the effusion glucose) so a high WB-EGD (>37mg/dL difference) supports SP but a normal or low difference does not rule it out

Comparative lactate levels in cats (effusion and whole blood) are less reliable

And this

lower than blood glucose in septic effusions.[46] A subsequent study in dogs with systemic inflammatory response syndrome (SIRS) and peritoneal effusion found 89.5% sensitivity and 66.7% specificity with ≥37 mg/dL whole blood–effusion glucose difference (WB-EGD),[112] suggesting that while a high WB-EGD should raise a strong suspicion of SP, lower WB-EGD should not be used to rule out SP.

Point of care glucometers overestimate effusion glucose due to low cell mass, leading to low sensitivity to detect SP via WB-EGD.[10], [113] Using plasma-effusion glucose difference instead of WB-EGD improves sensitivity and specificity.[113]

Although most studies described above included patients with neoplasia, clinicians should note that non-septic neoplastic effusions have been reported to have high lactate and low glucose.[117]

SEPTIC VS STERILE SIRS – NOT ALWAYS EASY TO TELL

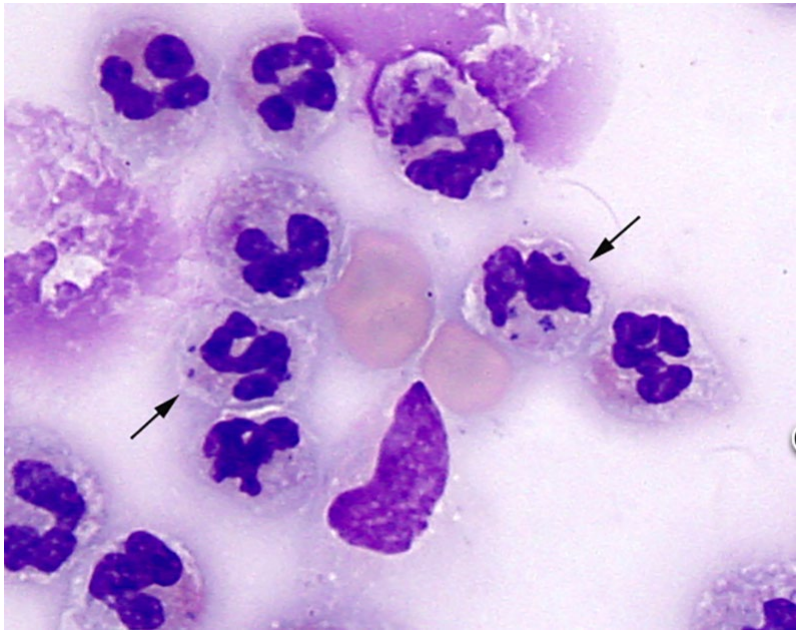
Fortunately, there is always a new biomarker that shows promise in discriminating sepsis vs non-septic SIRS (though with further investigating, most are proven to not differentiate that great)

- C-C motif chemokine ligand 2
- Cell-free DNA
- Probably not CURRENTLY relevant to those of us practicing in the real world?

Is Murphy SEPTIC?

- Yes! No biomarkers needed

- Diagnosis: septic peritonitis



What's the prognosis for a "septic abdomen" in vet med?

that extended at least into the last 15 years, survival rates for dogs and cats with SP of any cause ranged from 36.4% to 100%.[1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26] The gastrointestinal tract (GIT) was the source in >75% of cases, with survival rates of 36.4%–88.5%.[1, 2, 3, 4, 12, 16, 17, 18, 19, 21, 24, 25, 26, 27] Survival rates for dogs and cats developing recurrent SP were 0%–43.9%.[12, 15, 18, 26, 27, 28, 29, 30, 31]

Any prognostic value from intake blood work?

No consistent associations with WBC counts and mortality

No consistent associations with platelet count and mortality

Hypoglycemia not associated clearly with prognostication in dogs; **HYPERGLYCEMIA may be a negative** prognostic indicator in **cats**

Lactate → failure to clear pre/post-operatively is associated with mortality in dogs

Severe **POST-OPERATIVE hypoalbuminemia** = **increased mortality** risk in dogs

Hypocholesterolemia = increased all-cause mortality in septic people and dogs

Ionized hypocalcemia = **increased mortality in DOGS,** common in septic cats but not associated with increased mortality; CAN'T SUBSITUTE WITH TOTAL CALCIUM

Increased creatinine = AKI is associated with mortality; HOWEVER can be hard to differentiate AKI from pre-renal

Any prognostication with coags?

If you remember two things from this lecture, this probably isn't going to be one of those two things HOWEVER it's interesting nonetheless: **DIC occurs on a spectrum → typically STARTS hypercoagulable and ends hypocoagulable**

THERE IS CRITICAL CROSS-TALK BETWEEN INFLAMMATION AND COAGULATION!!

Bad inflammatory states typically start out as PRO or HYPERCOAGULABLE; inflammatory cells express tissue factor → tissue factor activates thrombin generation/ propagation and platelet activation via the cell-based model of coagulation

- Thrombin is very PRO-INFLAMMATORY – tells inflammatory cells to RAMP UP production of inflammatory cytokines, etc.
- anti-coagulant proteins are also ANTI-INFLAMMATORY

VERY BAD inflammatory states may END with a hypocoagulable phenotype as clotting factors are consumed, etc.

The data about coag says:

range: 75%–135%). Similar to human studies, pre- and postoperative ATIII and PC levels were associated with survival in this population.[44], [76] Specifically, ATIII activity >41.5% preoperatively and >22.5% postoperatively, and PC activity >60% preoperatively and >49% postoperatively, were each predictive of survival.[44] In addition to anticoagulant activity, ATIII and PC possess anti-inflammatory properties.[77] Higher ATIII and PC in survivors may reflect less severe DIC, however their activity also impacts the underlying inflammatory state driving sepsis and its complications. Activated partial thromboplastin time (aPTT) was prolonged in non-survivors.[44] Thromboelastography (TEG) revealed shortened R time in all patients with SP, and otherwise hypercoagulable or largely normal TEG tracings.[44] Patients with hypercoagulable tracings were more likely to survive. The authors suggest that survivors had a compensated hypercoagulable phenotype, while non-survivors had a decompensated phenotype characterized by lower ATIII and PC levels and a deficiency of procoagulant factors. This interpretation is supported by a study showing that dogs in DIC with hypercoagulable TEG tracings had improved survival compared to those with hypocoagulable TEGs.[78]

So back to the case, what IMMEDIATE treatments do we want to add to our IVF bolus?

- Antibiotics – get something IV on-board
- Dextrose – will help augment our resuscitation efforts
- Note that antibiotics are not the DEFINITIVE treatment for septic abdomen HOWEVER your resuscitation goals are to lessen the SYSTEMIC manifestation of septic peritonitis – these cases warrant **surgical explore**

J Vet Emerg Crit Care (San Antonio). 2015 Jan-Feb;25(1):152-9. doi: 10.1111/vec.12273. Epub 2014 Dec 26.

Impact of appropriate empirical antimicrobial therapy on outcome of dogs with septic peritonitis

Amy E Dickinson[1] Jennifer E Summers Jamie Wignal Amanda K Boag Iain Keir

Conclusions: In this population, appropriateness of empirical antimicrobial choice was not associated with survival to discharge. Previous antimicrobial administration or abdominal surgery was associated with subsequent inappropriate empirical antimicrobial selection.

Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock*

Kumar, Anand MD; Roberts, Daniel MD; Wood, Kenneth E. DO; Light, Bruce MD; Parrillo, Joseph E. MD; Sharma, Satendra MD; Suppes, Robert BSc; Feinstein, Daniel MD; Zanotti, Sergio MD; Taiberg, Leo MD; Gurka, David MD; Kumar, Aseem PhD; Cheang, Mary MSc

suspected pathogens within the first hour of documented hypotension was associated with a survival rate of 79.9%. Each hour of delay in antimicrobial administration over the ensuing 6 hrs was associated with an average decrease in survival of 7.6%. By the second hour after onset of persistent/recurrent hypotension, in-hospital mortality rate was significantly increased relative to receiving therapy within the first hour (odds ratio 1.67; 95% confidence interval, 1.12–2.48). In multivariate

SEPTIC PERITONITIS IS A SURGICAL DISEASE – SOURCE CONTROL IS CRITICAL

PREPARE PREPARE PREPARE

Step 1: Optimize Murphy's metabolic and hemodynamic status PRE-ANESTHETICALLY

Step 2: Instrument him adequately for anesthetic interventions (minimum of two IVCs)

Step 3: Create a safe anesthetic plan – I often plan for TIVA (many of these patients get a "wiff" of anesthesia and decompensate) and consider blood products to augment my fluids (plasma, etc)

Step 4: Have ALL of your monitoring equipment ready, calculate your emergency drugs (including reversals) AND have a norepinephrine CRI ready to go – THIS CAN GO THROUGH A PERIPHERAL IV CATHETER FOR A SHORT PERIOD OF TIME

Step 5: Have a post-operative plan in-place with the owner; my unsolicited/unfounded opinion: **most of these cases die post-operatively, fewer die "on the table"**

Stabilize sounds easy enough…

But how do we stabilize the persistently hypotensive patient? THIS IS ITS OWN LECTURE! But here's my cheat sheet

Blood pressure = cardiac output (stroke volume x HR) X systemic vascular resistance (vessel tone)

The trick is to determine if patient hypotensive from LOW VOLUME, FAILING PUMP (poor systolic function), or VASODILATION

Here's what I ask myself (or intern) every time:

1. Is the patient **hot or cold**?
2. Are the gums **pale or injected?**
3. Is the **CRT fast or prolonged**?

Needs Volume	Needs a Pressor
Pale gums, prolonged CRT, cold	Injected gums, fast CRT, warm

When do we reach for a colloid? REFRACTORY HYPOVOLEMIA!

- Answer: if BP, HR, CRT, pulse quality, etc. aren't improving with crystalloids AND/OR if total solids are low
- Colloids contain LARGE MOLECULES and are meant to stay within the intravascular space (unlike crystalloids, which redistribute)
  - Patients with low protein have less oncotic pressure holding fluid inside vessels
  - Even if some colloids don't have a ton of protein, they may still be "hyperoncotic" to a sick patient
- Colloids **DO increase intravascular volume**
  - Improvement in circulatory volume should improve blood pressure and cardiac output
- Colloids **DO NOT pull fluid back into vessels** from the interstitium (will NOT "fix" edema)
- Colloids **DO NOT rehydrate**
- Examples of colloids = plasma, canine albumin, vetstarch, hetastarch

Obligatory Slide on the synthetic colloid CONTROVERSY…

- In human medicine, use of synthetic colloids appears to be independently associated with increased risk of AKI and mortality in septic patients
- Associated with osmotic nephrosis (swelling of renal tubular epithelial cells due to accumulation of the starch)
- In veterinary medicine, the jury is still out!

ORIGINAL ARTICLE

Hydroxyethyl Starch 130/0.42 versus Ringer's Acetate in Severe Sepsis

Anders Perner, M.D., Ph.D., Nicolai Haase, M.D., Anne B. Guttormsen, M.D., Ph.D., Jyrki Tenhunen, M.D., Ph.D., Gudmundur Klemenzson, M.D., Anders Åneman, M.D., Ph.D., Kristian R. Madsen, M.D., Morten H. Møller, M.D., Ph.D., Jeanie M. Elkjær, M.D., Lone M. Poulsen, M.D., Asger Bendtsen, M.D., M.P.H., Robert Winding, M.D., et al., for the 6S Trial Group and the Scandinavian Critical Care Trials Group*

CONCLUSIONS

Patients with severe sepsis assigned to fluid resuscitation with HES 130/0.42 had an increased risk of death at day 90 and were more likely to require renal-replacement therapy, as compared with those receiving Ringer's acetate. (Funded by the Danish Research Council and others; 6S ClinicalTrials.gov number, NCT00962156.)

Changes in Serum Creatinine Concentration and Acute Kidney Injury (AKI) Grade in Dogs Treated with Hydroxyethyl Starch 130/0.4 From 2013 to 2015

N.E. Sigrist,[✉] [1] N. Kälin, [1] and A. Dreyfus [2]

Conclusions and Clinical Importance

HES-130/0.4-treated dogs were not more prone to develop AKI than HES-untreated, but the number of HES days was significantly associated with an increase in AKI grade within 10 days post-HES administration. The time frame of HES treatment should be kept short. Prospective, randomized clinical trials are required to assess the effect of HES on renal function in dogs.

Synthetic Colloids Continued…

-Species-specific differences in how synthetic colloids are metabolized

-Dogs and cats with sepsis typically get MUCH LOWER DOSES (hospitalized for much shorter times) than people

-No survival advantage in sepsis, so still not recommended in those patients

criteria in septic vs. non-septic patients (111). It
veterinary patients, as there are species-specific differences in HES metabolism. In addition, dogs and cats with sepsis are typically hospitalized for shorter durations compared to human patients, and receive far less total colloid. Several retrospective studies have evaluated HES use in dogs with conflicting results, although none have specifically evaluated a population of dogs with sepsis (112–114). Two retrospective studies in non-azotemic cats failed to show an association between HES administration and acute kidney injury or mortality (115, 116). In one of these studies, cats with sepsis were specifically evaluated and there was no HES-associated acute kidney injury noted; only 14 cats were in this group and the authors cautioned against making recommendations until a larger group of septic cats could be evaluated (115). Given that there is no clear survival advantage for synthetic colloids in patients with sepsis and there is potential risk of renal injury, use of these fluids in this patient population is not recommended. For further discussion of the controversies

PRESSOR TIME (DON'T BE AFRAID)!

- If **persistently hypotensive or hemodynamically unstable despite volume resuscitation** (crystalloids AND/OR colloids), consider pressor (norepinephrine)

- "Pressors" treat pathologic vasodilation → **limbs are warm, MMs injected, CRT may be rapid** → by **constricting arterioles, they increase blood pressure**

- By constricting veins, pressors **RECRUIT blood volume that's pooling** in places it shouldn't (ie splanchnic circulation) and dump it into the central venous pool to improve cardiac output and perfusion of vital organs (brain, heart, kidney)

- Human studies may show some benefit to starting norepinephrine CRIs sooner in resuscitation of septic patients as NE appears to augment volume resuscitation (and minimize fluid-overload)

How to give a pressor and for how long?

- To keep it simple, **<u>at time of presentation, norepinephrine is IN and dopamine is OUT</u>**
  - Norepi is a fairly clean vasoconstrictor (arterioles and veins) –MOST of the desired activity is via alpha-1 receptors, only has a little bit of beta activity (this means you really don't get as many arrhythmias with NE as with dirty dopa)

- Norepi is given as a CRI (requires dilution): I usually start low @ **0.05-0.1mcg/kg/min** and **increase by 0.1mcg/kg/min every 5-15 minutes until I get an adequate blood pressure** (systolic >90mmHg, MAP >65mmHg) **without exceeding 1-1.5mcg/kg/min**; PERSONALLY if I'm approaching 0.5mcg/kg/min I'm typically re-evaluating my plan
  - Do I need something else?
  - Are my electrolytes okay?
  - Acid-base okay?
  - iCa okay?

- Some patients only need pressor therapy for peri-anesthetic support (vasoplegia that comes with anesthesia needs to be counteracted with it)

- Other patients may need a day or more of vasopressor therapy – in those patients, a central line is the SAFEST way to administer it (extravasation is a big risk)

PUMP FAILURE – BRIEF NOTE

Not spending much time on it, but there is a lesser category of myocardial depression/systolic dysfunction; in a small subset of septic patients, we may need inotropic support (**DOBUTAMINE CRI**)

Back to the Case…Murphy has received:

(3) 10mL/kg LRS boluses; BP would transiently improve then drop again

(1) 4mL/kg HTS bolus; BP improved to 80mmHg but not further

You elect to start him on a **plasma CRI @ 2mL/kg/hr** for some additional intravascular and coag support, anticipating a hemostatic challenge and low protein (maybe will even help to patch up that glycocalyx)

AND he's been started on **norepinephrine CRI titrated up to @ 0.2mcg/kg/min**

HIS BLOOD PRESSURE IS **100mmHg (systolic) and his HR is 90bpm**

GREAT JOB!! THIS IS OUR SURGICAL WINDOW!!

Refer or not refer?

- Speaking as a surgical idiot, I rely ENTIRELY on smarter/better people than me for the surgery part
- FOR YOU may depend on: your anesthesia comfort and your surgical comfort – R&A common…hopefully not but possibly Billroth Type 1

We want to get to surgery…what's your anesthetic plan?

- **Drugs I love:**
  - ***Fentanyl CRI*** for analgesia is my favorite (alternatives: methadone, hydromorphone); buprenorphine and torb don't give you enough pain control
  - ***Midazolam*** to lessen the amount of propofol/alfax/
  - ***Alfaxalone*** (maybe less cardiodepressant but needs a benzo with it) OR ***propofol*** titrated to effect
  - ***Lidocaine*** (bolus and CRI) – I use this for the sedating/analgesic effects for TIVA and for its reactive oxygen species scavenging (we know we're re-perfusing tissue when patients present in shock)

- **Drugs I like (may be helpful):**
  - LOW-DOSE ***dexmedetomidine CRI*** – 1mcg/kg/hr
  - Low-ish dose ***ketamine*** bolus and CRI – may have immunomodulating effects, cautious with dosing to avoid myocardial stress

- **Drugs I would avoid:**
  - Acepromazine
  - High dose inhalant anesthesia

Animals (Basel). 2024 Mar 14;14(6):892. doi: 10.3390/ani14060892

Impact of a Dexmedetomidine Intravenous Infusion in Septic Dogs: Preliminary Study

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Abstract

The purpose of this study was to determine if a continuous rate infusion (CRI) of dexmedetomidine decreases vasopressor requirements in septic dogs undergoing surgery. Vital parameters, sequential organ failure assessment (SOFA) score, vasopressor requirement, and 28-day mortality were recorded. Dogs were randomly divided into two groups: a dexmedetomidine (DEX) (1 mcg/kg/h) group and a control group (NaCl), which received an equivalent CRI of NaCl. Dogs were premedicated with fentanyl 5 mcg/kg IV, induced with propofol, and maintained with sevoflurane and a variable rate fentanyl infusion. DEX or NaCl infusions were started 10 min prior to induction. Fluid-responsive hypotensive patients received repeated Ringer's lactate boluses (2 mL/kg) until stable or they were no longer fluid-responsive. Patients that remained hypotensive following fluid boluses received norepinephrine at a starting dose of 0.05 mcg/kg/min, with increases of 0.05 mcg/kg/min. Rescue adrenaline boluses were administered (0.001 mg/kg) if normotension was not achieved within 30 min of starting norepinephrine. The NaCl group received a significantly higher dose of norepinephrine (0.8, 0.4–2 mcg/kg/min) than the DEX group (0.12, 0–0.86 mcg/kg/min). Mortality was statistically lower in the DEX group (1/10) vs. the NaCl group (5/6). Results of this study suggest that a 1 mcg/kg/h CRI of dexmedetomidine decreases the demand for intraoperative vasopressors and may improve

TLDR: LOW doses of dexmed (1mcg/kg/hr) as CRI may help to maintain normotension and reduce norepinephrine requirements

What are the surgical goals?

- Source control – resect non-viable or perforated tissue
- EXPLORE for other areas of devitalization
- Evacuate infected fluid

*Playing the game - is that tissue viable?

Visual and palpatory assessment of intestinal viability have not proven reliable in human or animal studies.[138] Prediction by human surgeons of whether an anastomosis would later leak had fairly low sensitivity (38%–62%), specificity (46%–52%), and predictive value (64%).[139] While palpation and visualization were adequate guides to remove all non-viable bowel (89% accuracy), this resulted in unnecessary resection of viable tissue in 46% of people.[140]

*Fully admit I haven't done surgery since my residency; I don't play this game, I would not win this game

Fluorescent angiography??

Some promise shown in injecting various dyes IV and assessing for them in the tissues… area of opportunity for vet med?

R&A – Fast Facts (all pulled from the review paper)

- More likely to dehisce if they have a septic abdomen going in (proteases, unhealthy tissue, microbial dysbiosis, malnutrition, GI edema, poor tissue perfusion, etc)
- **Some studies have found a significantly lower dehiscence rate for stapled compared to handsewn anastomoses**
- Intersuture probing with hemostats is more sensitive than saline leak test, but SLT is still standard in vet med
- Omentalization – multi-mechanistically aids in healing, theoretical risk of stricture with 360 wrapping, but not a significant risk otherwise
- Serosal patching - blood supply and fibrin seal by connecting healthy bowel to less healthy bowel- trend towards better outcomes, should be considered
- Collagen-based biomaterial patching – significant decrease in leaking in humans

Lavage – Do or Don't?

- Pros
  - Reduce microbial burden
  - Reduce inflammatory mediators
  - Remove foreign material
  - Reduce adhesions?
- Cons
  - May introduce microorganisms into places they weren't already
  - Saline is irritating/inflammatory to the mesothelium
- Advice
  - Still performed commonly
  - **Remove all residual fluid before closing and be MINDFUL of contact time throughout surgery of peritoneum with that saline lavage**
  - May change recommendations to balanced crystalloid
  - **Take a culture (pre or post is likely okay though post is fewer organisms)**

Open Abdomens or Closed-Suction Drains?

In people with **severe septic peritonitis and/or severe shock**, not uncommon to do DAMAGE CONTROL SURGERY – stop the leak, clean up the mess but RESECT TISSUE WITHOUT FULL ANASTAMOSIS at initial surgery → temporary cutaneous enterostomy and/or manage as "open abdomen"

Allows for shorter anesthesia time, less risk of abdominal compartment syndrome, and provides time for viable vs non-viable to declare (used to be standard of care to re-explore all septic abdomens 24-48h post-op, but they've since determined unnecessary in some patients)

Some open abdomens maintained with VAC

In vet med, open abdomens are rarely performed/maintained (very difficult to do so), more common to put JP drain in

of cultures were positive at drain removal.[237] In a clinical study, the survival rate of SP dogs with closed suction drains (53%) was significantly lower than without drains (77%); however, dogs with drains had higher illness severity scores, making a direct comparison difficult.[8]

Post-Op – A game I FREQUENTLY PLAY - What else can go wrong? Hope for the best, monitor for the worst

- Organ Dysfunction
  - Important to monitor and address organ injury (ie GI ileus (can be profound), lung injury, acute kidney injury, arrhythmias, hypotension, etc)
- Fluid balance and metabolic derangements
  - Try not to let these guys get profoundly fluid overloaded → easy to do, edema makes healing much harder and exacerbates organ dysfunction
  - Monitor electrolytes and acid/base
- Coag derangements (can be SUBTLE)
  - Recall inflammation and coag go hand in hand → some of these patients may show signs of excessive clotting (or bleeding) and require intervention
- Dehiscense
  - 3-5 day window is peak but can happen 1-17 days post-op
  - Increased volume of abdominal effusion
  - Recurrent clinical decompensation

Take-home points

- Pay attention to your critically ill patients – treat early and- when in doubt- treat aggressively
- Sepsis is a dysregulated response to infection
  - Hemodynamic optimization is often achieved with fluid and pressor therapy
  - Assume the endothelium/endothelial glycocalyx is unhealthy (leaky and procoagulable)
  - That combination of hypercoagulability and poor oxygen delivery/utilization results in propagating organ edema/cellular dysfunction/failure
  - As with any suspected sepsis, early antibiotic administration is critical
  - Septic peritonitis is a SURGICAL DISEASE
- The post-operative period is as critical and challenging as the intra-operative (may be more so)
- Recurrent SP is associated with worse prognosis than initial SP

Questions??

This presentation is dedicated to my dog Emerson, who died from post-op complications of a septic abdomen.

As a pit-hound from Arkansas, he was a truly terrible pet, but he loved his life, the outdoors, his brother Conrad, and his family very much.

Thank you!