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• SAGES Position Statement on endoscopy and PPE requirements during COVID-19 Pandemic

INNOVATION
• Rapid emergent changes in the Upper Gastrointestinal Endoscopy Service routines at Groote Schuur Hospital with COVID-19

REVIEW ARTICLES
• Non-Steroidal Anti-Inflammatory Drug-Related Gastrointestinal Tract Adverse Effects
• Toxic megacolon
• Is Azithromycin a reasonable option for Helicobacter pylori eradication therapy?

THE GASTROENTEROLOGY FOUNDATION OF SOUTH AFRICA
WORLD DIGESTIVE HEALTH DAY
• Omeprazole 40 mg increases acid control and symptom relief *3

• In some duodenal ulcer patients refractory to other treatment regimens, 40 mg once daily may be effective 4

Editorial

This issue of South African Gastroenterology Review comes at a time, unprecedented in most lives. Overnight, a coronavirus, called SARS CoV-2, has completely changed our normal functioning. The virus, first observed in the last quarter of 2019 in Wuhan, China, is responsible for coronavirus disease 2019 or Covid-19. It has spread, to achieve pandemic status, through rapidly engulfing the entire planet. The only land mass yet to report a case is Antarctica. At the frontline of fighting and pushing back against this pandemic, are healthcare workers. Consequently, it is us who are at enormous risk of exposure and contracting the virus. Current data suggests several facts that seem to be duplicated around the world. Firstly, morbidity and mortality are a function of age and concomitant diseases in the host. Secondly virulence of SARS CoV-2 is high and the R₀ (reproduction number, expected number of cases directly generated by one case in a population) is thought to be between 2 to 5. Thirdly, a significant percentage of people infected are asymptomatic yet infectious. Hence, healthcare workers warrant protection given their proximity to infection as evidenced by the large numbers of doctors and nurses who have succumbed globally to COVID-19. If not done, the available healthcare worker pool would be significantly affected rendering them potentially unavailable to deliver care to growing numbers of patients. South Africa’s first positive case was reported on March 5th 2020. At the time of writing, we have > 6000 confirmed infections and > 130 deaths. It would seem we likely face a slow yet incremental rise in our caseload over a longer period of time with a flattened curve.

Gastroenterology, with its many invasive endoscopic diagnostic and therapeutic procedures, poses a significant Covid-19 infection risk to the operator. In the Journal, Chinnery and Scriba describe their experience in addressing the issue in their Hospital. Their approach and solutions represent out of the box thinking and solution seeking tactics. They highlight that much of what is happening both here and in other countries, represents practical implementation of solutions by experienced clinicians, so as to avoid paralyzing service to patients who need care. Going forward they underpin the need for shared experiences and solutions as we all learn from one another.

Two superb reference articles by Professors’ Setshedi and Watermeyer, respectively review NSAID toxicity and the Gut and Toxic Megacolon. These are both reviews that insightfully cover these topics and serve as outstanding summaries for trainees and reviews for practicing colleagues. Almost 3 decades after its described association with ulcer and malignancy, the most effective eradication therapy for Helicobacter pylori, remains under debate. Cost issues are important and Dr Dion Levin eloquently argues that the evidentiary basis for the use of Azithromycin, rather than Clarithromycin, is as policy in the Western Cape, does not necessarily hold up to scrutiny.

From various blogs and reports provided, 2019 was clearly a busy year. The landscape for 2020 looks decidedly different and the discovery of applications like Zoom or Microsoft Teams by many, is reshaping how we function. This may well be the new normal for some time yet. Sadly, given these events, inevitably the annual SAGES Congress for August this year has been cancelled. This is the first time there will be no meeting in 58 years. Supportive educational activities will occur in due course but in the meantime, with all due credit to Queen Elizabeth II in her recent address, we would concur that “We should take comfort that while we may have more still to endure, better days will return. We will be with our friends again, we will be with our families again, we will meet again”. In the meantime, Stay Safe!

Mark Sonderup
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**SAGES Seretariat**
- Karin Fenton
  PO Box 13241
  Mowbray
  7705
- Tel: 021-404-3062 (9am - 2pm)
- Fax: 021-447-0582
- Email: Karin.fenton@uct.ac.za
- Website: www.sages.org.za

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SAGES Position Statement on endoscopy and PPE requirements during COVID-19 Pandemic

COVID-19 pandemic is rapidly spreading and affecting all spheres of previous standard medical practices, including endoscopy. There is also a rapid rate of new information and ever-changing/evolving guidelines.

Most of major Societies’ have released position statements or recommendations including:
- BSG/JAC/ACPGBI/AUGIS/PSGBI/UKI-EUS/BSGAR
- Joint Gastroenterology Society Message (AASLD/ACG/AGA/ASGE)
- ESGE/ESGENA

All recommendations will be based on international norms, standards and also to protect all HCW and patients.

A significant proportion of Health care providers (HCP) have become infected with COVID-19. The spread of COVID-19 can occur from asymptomatic carriers, HCP or patients. The main route of virus transmission is via aerosolized droplets. All endoscopic procedures should be considered aerosol-generating procedures (although risk stratification does vary with upper endoscopy greater than lower endoscopy).

Personal Protective Equipment (PPE) is only part of the strategy to prevent injection. General Infection Control Prevention (IPC) measures, including hand hygiene and social distancing, must not be neglected.

**Pre-Procedure**
- All staff involved in endoscopy must be appropriately trained and informed on IPC strategy
  - Practice Donning and Doffing process
  - Doffing sequence essential
  - This needs to be done on an continual basis and needs to be critique to improve any overt deficiencies
- Clearly identify area for endoscopy
  - Clear flow in and out
  - Identify holding area before endoscopy
  - Recovery area after endoscopy
- Appropriate environmental cleaning before and after the procedure
- In endoscopy area minimize staff and equipment
- Risk stratification of endoscopy
  - Only emergency or urgent endoscopy should be performed
  - All routine, non-urgent and elective endoscopy should be deferred (accurate recording of all cases for catch up purposes post restriction of services)

**Intra-Procedure**
All members of endoscopy team to wear FULL PPE for all endoscopy procedures
- Hair Net
- Eye protection- Goggles/ Face shield or equivalent
- N95 mask
- Waterproof gown or equivalent
- Double disposable gloves
- Shoe covering or equivalent

**Post-Procedure**
- Safely place endoscope for reprocessing
- Safely remove PPE- Clear doffing procedure
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Rapid emergent changes in the Upper Gastrointestinal Endoscopy Service routines at Groote Schuur Hospital with COVID-19

GE Chinnery, MF Scriba, EG Jonas
Surgical Gastroenterology Unit, Division of General Surgery, University of Cape Town Health Sciences Faculty and Groote Schuur Hospital, Cape Town, South Africa

Community spread of COVID-19 is now established in South Africa and recent data from China suggests up to 80% of infected individuals possibly being asymptomatic and thus an important possible source of contagion. Upper gastrointestinal (GI) endoscopy is considered a high-risk procedure, as it carries the potential of aerosolizing the SARS-CoV-2 virus. This has prompted our local endoscopic practice to change dramatically in a short space of time.

All patients arriving at the endoscopy unit are formally screened for exposure and symptoms of COVID-19. Indirect measures to decrease risk of exposure to healthcare workers, decrease community spread and allow for staff redeployment have led us to cancel all elective and non-urgent upper and lower GI endoscopies with immediate effect. Every endoscopic procedure is now assessed on a case-by-case basis, with the vast majority of diagnostic endoscopies deferred. At present our only considered indications for gastroscopy are upper GI bleeding needing endoscopic intervention, oesophageal stenting or dilatation for high-grade dysphagia and gastric outlet obstruction amenable to endoscopic intervention. Biliary decompression in patients with obstructive jaundice is limited to patients with cholangitis or symptomatic jaundice refractory to medical management. Where possible percutaneous transhepatic cholangiography (PTC) with biliary drainage, a lower risk procedure, is preferred. Endoscopic retrograde cholangiopancreatography (ERCP) is reserved for patients with contraindications for PTC, or after failed PTC. Further indirect measures include restricting staff exposure to endoscopy with only a limited number of designated, experienced endoscopists who rotate on a daily basis preforming these urgent services. Presently we no longer perform any endoscopic training and allow only the endoscopist and two nursing staff into the endoscopy suite.

We believe a safe working environment for our endoscopic team is of paramount importance. In view of the potentially asymptomatic, yet infective, patient population, the suboptimal sensitivity of the available screening tests, and the significant contagious nature of this novel coronavirus, and in line with international guidelines, every Upper GI endoscopic procedure performed in our unit is done with full personal protective equipment (PPE). The ESGE guidelines suggest the following PPE requirements for endoscopy in all high-risk patients: disposable hairnets, face shields/goggles, N95 (or similar) respirator masks, waterproof disposable gowns and two pairs of disposable gloves. PPE is now a globally scarce resource with needs likely to escalate. As in many countries, in particular those with resource-constrained health systems sourcing of some PPE materials has become the responsibility of the endoscopists and innovative on-site fashioning of own PPE kits has become widespread. In our setting we were from the onset faced with limited availability of some PPE items, such as appropriate protective eyewear, N95 masks and disposable gowns. We thus sourced our goggles from agricultural and hardware stores (these are actually designed for pesticide crop spraying and seal well around the eyes). We have decided...
to reuse our N95 masks in a 1-in-4 sequential daily rotation system (Figure 1). As per a SAGES advisory statement, this involves wearing one mask for the entire endoscopy, and then allowing it to hang for 72 hours before being used again. In confirmed COVID-19 cases, N95 masks are not reused.

Our main concern remains the lack of available disposable waterproof gowns. We have thus taken to fashioning our own gowns using plastic bin liners, as has previously been described from areas of high COVID-19 incidence. Figures 2 and 3 demonstrate our production and donning thereof. A plastic apron is then worn over the "gown", with gloves and apron being changed between patients. If a suspected or confirmed case presents for endoscopy, the entire "gown" is changed. The donning technique is of utmost importance and requires an assistant with scissors slitting the "gown" from behind such that it can be removed in one piece without contaminating the wearer or environment (Figure 3). Standard cleaning techniques for the reprocessing of endoscopes and accessories with commonly used virucidal disinfectants are adequate in inactivating the COVID-19 virus, and as such should be performed as per previously published guidelines. Single-use endoscopic devices should not be reused.

In addition, in aiming to reduce aerosolization of secretions, all endoscopies are performed at a minimum with conscious sedation. This is presently achievable due to the significant decrease in the number of endoscopies performed in our unit. Confirmed or suspected COVID-19 positive...
patients should ideally be done in a negative pressure room (if available) under a general anaesthetic with endotracheal intubation. Due to resource constraints we reserve general anaesthetic with endotracheal intubation for longer procedures, such as complicated ERCPs. In addition, we routinely add a further secretion barrier, by placing a clear plastic sheet over the patient’s face during the endoscopy, with only a small slit cut over the mouth to allow for passage of the endoscope (Figure 4). In order to reduce patient anxiety, adequate sedation and nasal prong oxygen are mandatory before laying the plastic sheet over the patient’s head.

Although to our knowledge there is no documented case of transmission to the endoscopist during colonoscopy, we support the recommendations that during prolonged exposure with potentially virally contaminated faeces, that full PPE as above is also advised.

In these unprecedented times, working in a high-risk and anxiety-provoking environment, team morale is influenced with possible consequent burnout. We are of the view that the simple initiative of making and sourcing protective gear ourselves and proactively attempting to ensure our own safety, has empowered our entire endoscopy team (Figure 5). As an endoscopy community, we need to share ideas or suggestions with each other in order to improve patient and staff welfare. Furthermore, we need to be adaptable with directives and guidelines in a fast-changing environment where evidence is still sparse, most importantly adapting to our own environments with best possible practice.

References

1. Day M. Covid-19: four fifths of cases are asymptomatic, China figures indicate. BMJ 2020;369:m1375. Doi: https://doi.org/10.1136/bmj.m1375 (Published 02 April 2020)


5. SAGES Webmaster: N95 Re-use Strategies. Updated and re-released 03 April 2020. Accessed online at: https://www.sages.org/n-95-re-use-instructions/


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<tr>
<th>Question</th>
<th>Yes/No</th>
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<tr>
<td>Mesalazine is released throughout the GI tract?</td>
<td>Yes(^1,2)</td>
</tr>
<tr>
<td>Release of mesalazine at all pH values in the GI tract?</td>
<td>Yes(^1)</td>
</tr>
<tr>
<td>Does diarrhoea affect the release of mesalazine?</td>
<td>Yes(^3)</td>
</tr>
<tr>
<td>Any peak plasma concentrations of mesalazine?</td>
<td>No(^4)</td>
</tr>
<tr>
<td>May be taken with or without food?</td>
<td>Yes(^1,2)</td>
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**References:**
Non-Steroidal Anti-Inflammatory Drug-Related Gastrointestinal Tract Adverse Effects

M Setshedi
Department of Medicine, Division of Gastroenterology, University of Cape Town, Cape Town, South Africa

Introduction
Non-steroidal anti-inflammatory drugs (NSAIDs) are well known and commonly prescribed agents for their analgesic, antipyretic and anti-inflammatory effects (in high doses) while having the benefit of being non-narcotic and non-addictive.1-4 As a group they are one of the most prescribed drugs worldwide, with global prescriptions at 30 million daily5 excluding over the counter NSAIDs. NSAIDS have multi-organ adverse effects;6 the gastrointestinal (GI) and cardiovascular (CV) effects are more concerning in terms of their frequency and seriousness. The occurrence of adverse effects affecting both organ systems, together with the risk profile of patients requiring these agents, the high frequency of prescriptions, and relative ease with which these agents are acquired, makes safe prescribing for these patients an ongoing challenge. This focused review aims to highlight key issues around NSAID use and gastrointestinal system, with specific reference to the clinical presentation, pathophysiology, and approach to initial prescribing and gastroprotective therapies for a patient who requires long-term NSAIDS.

Gastrointestinal adverse effects
Acute, but more commonly long-term use of NSAIDS (defined as more than 4 weeks) causes adverse GI symptoms and pathology involving the entire GI tract.7-9 NSAID users with various risk profiles compared to matched controls have a 1.5 to 7.2-fold increase in serious GI side effects.10,11 The majority of patients (10-60%) present with upper GI symptoms i.e. dyspepsia (in 25-50% of patients), heartburn and others.12,13 In 15-30% there is evidence of endoscopic disease with ulcers, which may be symptomatic or asymptomatic.14 Approximately 50% of patients with symptoms have no mucosal lesions, whereas more than 50% who present with serious peptic ulcer disease (PUD) complications had no previous warning symptoms.14,15 Thus, clinical symptoms do not correlate with endoscopic findings or complications, as such risk assessment of individual patients and the presence of alarm symptoms should guide the performance of endoscopy. Four to five percent of patients have clinically significant ulcers with a complication rate of 1-1.5% in the first year; these patients present with bleeding, perforation, obstruction and even death.10,16,17 Mortality related to NSAIDS is high (16,500 patients); this was third to leukaemia (20,197 patients) and HIV (16,685 patients)16, although the data is rather old. Newer studies have not evaluated mortality but given the high rates of prescription of these drugs and the prevalent use of NSAIDS in older populations with high-risk comorbid factors, it is likely that mortality from NSAID use remains high. Mortality ranges from 5-12% and is usually due to cardiopulmonary complications and attendant multi-organ failure than directly as a result of NSAIDS.10,16

The Spectrum of NSAID-GI Mucosal damage
The whole of the gut can be adversely affected by NSAIDS. The upper GI tract is affected in 84.5% of cases, and patients present with oesophagitis, petechial submucosal haemorrhages, erosions and gastroduodenal ulcers. The small and large intestines are affected in the remaining proportion of cases and in the case of enteropathy patients present with anaemia, ulcers, strictures and protein-losing enteropathy. In colopathy, patients present with colitis, ulcers, strictures, and collagenous colitis.8 Importantly, NSAIDS may result in flares of inflammatory bowel disease in patients with quiescent disease18, thus should be avoided if possible in this case. There are in addition, reports of NSAID-induced diverticulitis.19

Pathophysiology
NSAIDS can be broadly classified into salicylates, traditional NSAIDS (NSAIDS) and the COX-2 selective inhibitors (coxibs). Aspirin (the oldest acetylated salicylate) inhibits both COX-1 and COX-2 irreversibly by acetylation.20 NSAIDS work by inhibiting prostaglandin synthesis via...
the arachidonic acid and cyclooxygenase pathway. Both COX-1 and COX-2 are inhibited by the NSAIDS with equal affinity. COX-1 is constitutive (present at low doses) and is responsible for normal physiologic gastroprotection, maintenance of blood flow in the gastric mucosa and production of bicarbonate. COX-2 on the other hand, is induced by cell damage and various anti-inflammatory cytokines. NSAID-induced gut inflammation is mainly caused by COX-1 inhibition. Non-selective NSAIDS include aspirin, and have the most toxic GI toxicity; however they are still in development and being tested.

Consequently, selective COX-2 inhibitors were developed, to reduce GI toxicity. A third group of NSAIDS includes the nitric oxide and hydrogen sulfide releasing NSAIDS, which donate nitrates and sulfide, thereby they potentiate their effects of potent vasodilatation and increased mucosal protection. These agents, therefore, have less GI toxicity.

**Risk factors for NSAIDS-related GIT complications**

The highest risk for peptic ulcer complications includes a history of a complicated ulcer (OR=15.4), followed by an uncomplicated ulcer (OR=5.9) compared to no prior history. The use of multiple (OR=9), or high dose NSAIDS (OR=7), anticoagulants (OR=6.4) compared to controls, and age >70 (OR=5.6) (compared to those less than 40), are additional risk factors. Notably those taking combined NSAIDS and aspirin have a two-fold increase of upper GI bleeding, which is dose-independent compared to either drug alone and greater than 9-fold increased risk than in controls. Although the coxibs reduce the risk of GI bleeding, the risk of bleeding is nonetheless 4-fold increased when aspirin is co-prescribed, compared to coxibs alone. Concomitant use of NSAIDS and anticoagulants worsens the risk of peptic ulcer bleeding by a factor of 13 or 3, compared to controls or NSAIDS users alone respectively. Antiplatelet drugs e.g. clopidogrel, used together with NSAIDS increase GI events, especially in patients with a prior history of peptic ulcer disease. Steroids alone appear safe however concomitant use with NSAIDS results in a two-fold increase in serious GI complications, and greater than 10-fold risk of death than with NSAIDS alone. Other risk factors include chronic renal failure, cardiovascular disease and hepatic impairments.

Helicobacter Pylori has been shown to play a significant independent role in the risk of GI bleeding (OR=1.79) and an additional risk (OR=3.5) above the risk associated with NSAIDS alone (OR=4.85). H. Pylori infection and NSAIDS use synergistically increase the risk of PUD and ulcer bleeding. In the same study the proportion of patients who are NSAIDS users and H. Pylori positive was more than double that in H. Pylori negative NSAIDS users. In a meta-analysis done of H. Pylori eradication in the prevention of peptic ulcers in NSAIDS users, the odds ratio (0.3) favoured H. Pylori eradication.

### Evidence for NSAID GI and CV Toxicity

The GI toxicities of aspirin, NSAIDS and coxibs are summarized and compared in Table 1. With respect to aspirin, the effects are dose-dependent, nonetheless even a 10mg dose of aspirin is sufficient to inhibit prostaglandin synthesis. A dose of aspirin of ≤325mg, carried ORs of 2.6, 2.7 and 3.1 for plain, enteric-coated, and buffered aspirin respectively, whereas the risks were doubled in each group for users of aspirin at doses ≥325mg.

The odds of GI bleeding risk with combined low-dose aspirin and NSAIDS was 5.6 (4.4-7.0) compared to with low-dose aspirin alone. 2.6 (2.2-2.9). The higher the COX-1 selectivity, the higher the GI adverse events; therefore ibuprofen has the lowest risk, followed by diclofenac and naproxen (intermediate risk). Overall, the coxibs reduce the risk of upper GI ulceration and bleeding by 50-60% compared to NSAIDS. Notably, although the risk of GI complications is significantly reduced with coxibs it is not completely aborted, and dosage and duration of therapy still has to be considered. Furthermore, coxibs offer no additional benefit than NSAIDS for dyspepsia without ulceration. Combining aspirin with either NSAIDS or coxibs is not advised, as this increases the risk of complications. The risk of GI bleeding enhances when patients already on antiplatelet therapy using thienopyridines, like clopidogrel, are co-prescribed with NSAIDS to reduce adverse cardiovascular events. In terms of cardiovascular risk of NSAIDS, Naproxen 500mg twice daily had a more favourable profile compared to placebo, while Diclofenac and Ibuprofen increased the risk of myocardial infarction. In the same vein, Naproxen had lower CV risk compared to the coxibs. The mechanism for increased risk of myocardial infarction, heart failure, stroke and hypertension is likely due to the fact that coxibs do not block the production of platelet thromboxane, because platelets do not contain COX-2, but instead suppress endothelial prostacyclin (which is an intrinsic vasodilator and platelet inhibitor).

### Table 1. Comparison of clinical features of NSAIDs by type

<table>
<thead>
<tr>
<th>Clinical phenotype</th>
<th>Aspirin</th>
<th>NSAIDS</th>
<th>Coxibs</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical phenotype</strong></td>
<td>PUD 10-25% (at endoscopy)</td>
<td>Dyspepsia 50%</td>
<td>Decreases symptomatic ulcers by 51%</td>
</tr>
<tr>
<td><strong>Bleeding</strong></td>
<td>Small bowel ulcers Haemorrhage (including diverticular)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>GIT location</strong></td>
<td>Mainly foregut</td>
<td>Entire gut, 25-50% beyond duodenum</td>
<td>Less small bowel injury than NSAIDS</td>
</tr>
<tr>
<td><strong>Rate of serious complications</strong></td>
<td>0.5-2% per year (rebleeding) 15% (death) 5-10% (death)</td>
<td>1-5% 1 in 1200 (death)</td>
<td>Decreased by 45%</td>
</tr>
<tr>
<td><strong>Pooled risk ratio for GI complications</strong></td>
<td>3.2 (any dose)</td>
<td>3.8</td>
<td>1.42 (celecoxib)</td>
</tr>
</tbody>
</table>
Caused by COX-1 inhibition. Non-selective NSAIDs, however, are still in development and being tested. These agents, therefore, have less GI toxicity; their effects of potent vasodilation and increased mucosal protection. These agents donate nitrates and sulfide, thereby they potentiate cytokines. NSAID-induced gut inflammation is mainly induced by cell damage and various anti-inflammatory maintenance of blood flow in the gastric mucosa and affinity. COX-1 is constitutive (present at low doses) and in patients with a prior history of peptic ulcer disease. Steroids alone appear safe however concomitant use is significantly reduced with coxibs it is not completely aborted, and dosage and duration of therapy still has to be considered. Furthermore, coxibs offer no additional effectiveness. The importance of H. Pylori eradication cannot be overstated; it reduces the risk of upper GI complications when starting NSAIDS and in those already taking aspirin and improves the response of coxibs. For patients with a history of an ulcer complication who require subsequent therapy with an NSAID or aspirin, H. Pylori eradication alone may not be a sufficient risk reduction strategy. Co-therapy with a PPI in such patients at high risk for recurrence of an ulcer complication is recommended, in both aspirin and non-aspirin users. Recommendations also suggest eradication before starting NSAIDs or coxib.

**Assessing GI and cardiovascular risk**

The approach to prescribing NSAIDs for any patient (where deemed necessary) is based on an assessment of their cardiovascular (defined as the need for cardioprotective aspirin) and gastro-intestinal bleeding risk. For initiating NSAID therapy consideration for cardiovascular risk takes precedence over GI risk. The cardiovascular and GI risk factors are shown in Figure 1. If the patient has high CV and GI risk NSAIDs including coxibs are best avoided, but if required, the preferred choice for an NSAID is Naproxen, which has a safer cardiovascular profile than other NSAIDs; this, however, must be used in conjunction with a PPI. If the patient has low CV risk and high GI risk, coxib alone, or coxib + PPI (preferred) are recommended. If low GI risk and high CV risk, Naproxen ± PPI, and if low GI and CV risk NSAIDs can be “safely” prescribed.

**Summary and conclusions**

Upper GI clinical events occur in approximately 2.5% to 4.5% of NSAIDS users per annum. Major complications (serious bleeding, perforation, obstruction) occur in about 1% to 1.5%. Upper GI events are increased in NSAIDS and coxib users, although the risk is reduced with coxibs. PPIs are effective for the prevention of index and subsequent GI events particularly in H. Pylori positive patients. PPIs should be prescribed for chronic NSAIDs and aspirin users. High risk GI-patients should ideally be prescribed a PPI plus a coxib, or if unavailable/unaffordable a coxib alone. If these options are not available, an NSAIDS with a PPI is a reasonable alternative. Notably, the benefit of coxibs though present is mitigated by aspirin use compared to no aspirin. A risk benefit assessment should always be made each time a patient with cardiovascular and gastrointestinal risk factors requires NSAID therapy. Appropriate preventative measures should be taken to reduce adverse outcomes from chronic NSAID use.

**Initial NSAID prescribing recommendations**

1. Avoid NSAIDS if possible, particularly in patients with GI risk (figure 1).
2. If deemed necessary, choose a “safer” NSAID.
3. Use the lowest effective dose for the shortest period.
4. Avoid concomitant use of steroids, anti-coagulants, and low dose aspirin.
5. Eradicate H. Pylori in patients with GIT bleeding risk prior to NSAIDS commencement.
6. Manage lifestyle factors e.g. weight loss, excessive alcohol intake, and smoking.
7. Choose a gastroprotective strategy (outlined below)

**Gastroprotection – evidence-based therapies**

Any patient with at least one risk factor for GI adverse events or complications should be offered gastroprotection. These therapies are effective in reducing serious adverse events and are proven cost-effective. PPIs have been shown to accelerate endoscopic gastroduodenal ulcer healing, prevent de novo and repeat ulceration (secondary prevention) in NSAID users; this latter group are at highest risk of further complications such as bleeding and perforation with long-term NSAID use. PPIs are superior to H2 receptor antagonists, misoprostol, antacids or placebo in ulcer healing and GI bleeding complications. In addition they have a favourable side effect profile, are relatively easy to access and therefore are the preferred gastroprotective agent. In chronic NSAID and aspirin users concomitant PPI therapy is associated with a significantly reduced risk of NSAID-related complications. NSAID and PPI co-therapy or a coxib, are equivalent in effectiveness although in patients with a previous ulcer bleed these regiments may still have significant risk of recurrent bleeding. Overall, the coxibs reduce the risk of upper GI ulceration and bleeding by 50-60% compared to NSAIDS. One RCT found a significantly lower rate of recurrent upper GI ulcer bleeding with a coxib plus a PPI (0%) compared to the coxib alone (8.9%) over 1 year (P < 0.001). Therefore in these patients a coxib and PPI is recommended and is cost-effective. The importance of H. Pylori eradication cannot be overstated; it reduces the risk of upper GI complications when starting NSAIDS and in those already taking aspirin and improves the response of coxibs. For patients with a history of an ulcer complication who require subsequent therapy with an NSAID or aspirin, H. Pylori eradication alone may not be a sufficient risk reduction strategy. Co-therapy with a PPI in such patients at high risk for recurrence of an ulcer complication is recommended, in both aspirin and non-aspirin users. Recommendations also suggest eradication before starting NSAIDs or coxib.

**References**

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Toxic megacolon (TMC) is a rare, but potentially fatal complication of severe colonic inflammation. It is a medical emergency as it carries a high risk of perforation. It is characterised by non-obstructive colonic dilatation of more than 6cm with signs of systemic toxicity. The dilatation can be either total or segmental. TMC is most commonly associated with inflammatory bowel disease (IBD), especially ulcerative colitis (UC), however any condition that leads to inflammation of the colon can cause TMC. In the immunocompromised patient, in particular those who are HIV positive, cytomegalovirus is the leading cause. TMC in the setting of Crohn’s disease tends to occur early in the course of the disease before fibrosis develops which prevents the colon from dilating.

Pathophysiology
The pathogenesis of TMC is unclear and likely multifactorial. It is likely that transmural mucosal inflammation triggers the process through the release of inflammatory cytokines. They increase production of inducible nitric oxide synthase, which in turn increases nitric oxide. Nitric oxide relaxes smooth muscle leading to dilation of the colon. A study showed that patients with TMC have significantly high levels of inducible nitric oxide synthase in the muscularis propria.

Presentation
A detailed history is essential as this may suggest the underlying cause. It is important to enquire about a known diagnosis of IBD or HIV, recent travel, and recent antibiotic use. Patients with TMC typically present with abdominal pain and distention, nausea, vomiting and diarrhea. They will have features of significant systemic toxicity such as fever, tachycardia, anaemia, leukocytosis with left shift, hypoalbuminaemia, and raised C-reactive protein. On physical exam, abdominal tenderness and decreased bowel sounds are often present. The presence of peritonism raises the possibility of perforation. TMC is often complicated by renal dysfunction, electrolyte abnormalities, and dehydration.

The following criteria are recommended to make the diagnosis:

- Radiographic evidence of the dilation of the colon greater than 6 cm
- At least three of the following:
  - Fever over 38°C
  - Heart rate greater than 120 beats/min
  - A neutrophil count exceeding 10000/micro/L
  - Anaemia
  - At least one of the following:
    - Dehydration
    - Altered sensorium
    - Electrolyte disturbances
    - Hypotension

Special investigations on admission:

- Full blood count (FBC)
- Blood sugar
- Creatinine
- Urea
- Liver function tests
- Electrolytes
- ECG
- Chest X-ray
- Abdominal X-ray
- CT scan of abdomen
- Urgent endoscopy

Correspondence
Gill Watermeyer
e-mail: gillian.watermeyer@uct.ac.za
Toxic megacolon

G Watermeyer
Division of Gastroenterology, Department of Medicine, University of Cape Town and Groote Schuur Hospital, Cape Town, South Africa

Introduction
Toxic megacolon (TMC) is a rare, but potentially fatal complication of severe colonic inflammation. It is a medical emergency as it carries a high risk of perforation. It is characterised by non-obstructive colonic dilatation of more than 6cm with signs of systemic toxicity. The dilatation can be either total or segmental. TMC is most commonly associated with inflammatory bowel disease (IBD), especially ulcerative colitis (UC), however any condition that leads to inflammation of the colon can cause TMC.1-9 TMC can complicate acute bacterial dysentery, C difficile infection, or ischaemic colitis. In the immunocompromised patient, in particular those who are the HIV positive, cytomegalovirus is the leading cause.7 C difficile is the commonest bacterial cause. TMC in the setting of Crohn’s disease tends to occur early in the course of the disease before fibrosis develops which prevents the colon from dilating.8

Pathophysiology
The pathogenesis of TMC is unclear and likely multifactorial. It is likely that transmural mucosal inflammation triggers the process through the release of inflammatory cytokines. They increase production of inducible nitric oxide synthase, which in turn increases nitric oxide. Nitric oxide relaxes smooth muscle leading to dilation of the colon. A study showed that patients with TMC have significantly high levels of inducible nitric oxide synthase in the muscularis propria.10

Presentation
A detailed history is essential as this may suggest the underlying cause. It is important to enquire about a known diagnosis of IBD or HIV, recent travel, and recent antibiotic use. Patients with TMC typically present with abdominal pain and distention, nausea, vomiting and diarrhea. They will have features of significant systemic toxicity such as fever, tachycardia, anaemia, leukocytosis with left shift, hypoalbuminaemia, and raised C-reactive protein.1 On physical exam, abdominal tenderness and decreased bowel sounds are often present. The presence of peritonism raises the possibility of perforation. TMC is often complicated by renal dysfunction, electrolyte abnormalities, and dehydration.11

The following criteria are recommended to make the diagnosis: 1,12

- Radiographic evidence of the dilation of the colon greater than 6 cm AND
- At least three of the following:
  - Fever over 38°C
  - Heart rate greater than 120 beats/min
  - A neutrophil count exceeding 10500/micro/L
  - Anaemia
- At least one of the following:
  - Dehydration
  - Altered sensorium
  - Electrolyte disturbances
  - Hypotension

Table 1. Causes of TMC1-9

| Inflammatory causes: | Ulcerative colitis |
| Infectious causes: | Crohn’s disease |
| Clostridium difficile | Salmonella |
| Shigella | Campylobacter colitis |
| Enterohaemorrhagic Escherichia coli O157 | Cytomegalovirus |
| Entamoeba |

Colonic ischaemia

Other factors that can precipitate toxic megacolon include [1]:
- Electrolyte abnormalities such as hypokalaemia
- Medications (anti-motility agents, opiates, anticholinergics, antidepressants)
- Barium enema
- Colonoscopy & bowel preparations

Correspondence
Gill Watermeyer
email: gillian.watermeyer@uct.ac.za

Special investigations on admission:
- Full blood count (FBC)
Management of TMC
If complications such as perforation are already present at diagnosis surgical intervention is unavoidable. In the absence of complications medical management is successful in approximately 50% of cases. All patients need to be managed upfront by a multidisciplinary team including a physician, colorectal surgeon and stomatherapist. Ideally they should be admitted to a high care unit as they require close monitoring. Aggressive fluid and electrolyte replacement is of paramount importance. Medications which can aggravate TMC, such as opioids and anticholinergics, should be stopped. Initially Abdominal x-ray and blood tests (electrolytes and FBC) should be done 12 hourly. Once the condition stabilizes these can be performed daily. Patients with a known diagnosis of ulcerative colitis should be given intravenous corticosteroids as soon as possible (hydrocortisone 100 mg 6 hourly or equivalent). Due to lack of evidence in the setting of TMC cyclosporine or infliximab are generally not recommended as part of the treatment.1

For most cases the underlying cause is not apparent at presentation and until the aetiology is established patients should be treated for all the common conditions associated with TMC. Initially broad-spectrum antibiotics such as a 3rd generation cephalosporin is recommended due to the high risk of perforation and to cover for bacterial dysentery. Intravenous metronidazole and oral vancomycin should be added to cover for the possibility of C difficile colitis, and intravenous corticosteroid in case this is the 1st presentation of UC. If cytomegalovirus is the suspected, then ganciclovir should be administered as well. Once the underlying aetiology is established treatment can be directed at the specific cause and unnecessary medications discontinued. Lastly, patients should be nil per mouth in case they require emergency surgery. Once the condition stabilizes the patient can gradually start eating to promote gut healing. Patients who fail to improve or in whom there is clinical, biochemical or radiographic deterioration should have an emergency colectomy.14 Timing of surgery in TMC is still controversial.15 Recommendations suggest not persisting with medical therapy beyond 72 hours although some centers are reluctant to continue medical therapy beyond 24 hours because colon perforation carries a worse prognosis, increasing mortality up to 5 fold.

Conclusion
TMC is a potentially lethal complication of any form of severe colonic inflammation. It is most commonly associated with IBD but increasingly infections, in particular C difficile, are responsible. The mechanisms leading to toxic colonic dilatation are incompletely understood. TMC is characterized by signs of systemic toxicity and severe colonic distension. Diagnosis is made by clinical and laboratory evaluation for systemic toxicity and imaging studies depicting colonic dilatation. Management of TMC requires a multidisciplinary team of gastroenterologists and surgeons from the onset. Aggressive medical therapy addressing hydration and electrolyte abnormalities is required, as well as treatment directed at the underlying cause. The optimal timing of surgery for TMC not responding to medical therapy is challenging.

References
Is Azithromycin a reasonable option for Helicobacter pylori eradication therapy?

DA Levin
Division of Gastroenterology, Department of Medicine, Groote Schuur Hospital, University of Cape Town, Cape Town, South Africa

Helicobacter pylori (H. pylori), considered a class 1 human carcinogen by the World Health Organization (WHO), has been implicated in the high prevalence of distal gastric intestinal type adenocarcinoma. Various studies have concluded that gastric cancer rates are directly related to H Pylori infection, with a clear relationship between H Pylori prevalence and a greater incidence of the gastric cancer. H. pylori infection is an independent risk factor for peptic ulcer disease and MALT lymphoma. In addition, this organism is associated with extra-gastric pathologies including iron deficiency anaemia, vitamin B12 deficiency and Idiopathic Thrombocytopenic Purpura.

As an infectious bacterium and given effective antimicrobials, a complete cure or eradication rate of 100% is desirable and achievable. The current standard of care is triple eradication therapy: Clarithromycin 500 mg and Amoxicillin 1g, both twice daily for at least 10, but preferably 14 days, and a proton pump inhibitor twice daily for 14 days. (Maastricht guidelines Statement 11: The treatment duration of PPI-clarithromycin based triple therapy should be extended to 14 days, unless shorter therapies are proven effective locally). An important caveat is that a Clarithromycin based regimen does depends on the local drug resistance pattern.

In the public sector of the Western Cape, an azithromycine-based eradication regimen (500mg daily for 3 days) has been implemented, in lieu of clarithromycin-based therapy. This practice was based on a national essential medicine list medication review process viz. NDOH EDP Azithromycin H Pylori eradication. Adults Medicine review: February 2016 (http://www.health.gov.za/index.php/standard-treatment-guidelines-and-essential-medicines-list/category/410-hospital-level-adults-medicine-reviews?download=1356:azithromycin-h-pylori-eradication-adults-medicine-review-february2016).

The potential use of Azithromycin as an alternative to Clarithromycin is not new and has been debated in the literature over the past 2 decades. The clinical appeal of Azithromycin lies in the proposed mechanism of prolonged drug accumulation in gastric mucosa, redistribution into the mucus layer and then into gastric juice. The concentration of the antibiotic is maintained in the gastric cells for up to 5 days at a level that inhibits H. pylori. Hence, a daily dose of Azithromycin for 3 days has been suggested to have the same eradication efficacy as a twice daily multi week course of Clarithromycin, with a concomitant reduction in cost.

It is important to consider however, two recent expert publications, in 2017 and 2019, respectively:

1. Management of Helicobacter pylori infection—the Maastricht VI/Florence Consensus Report, GUT 2017
2. Reconciliation of Recent Helicobacter pylori Treatment Guidelines in a Time of Increasing Resistance to Antibiotics

Neither of these publications that guide international H. Pylori therapeutic strategies, describe the use of Azithromycin amongst the numerous eradication regimens presented. Although there is always the argument that international experience and therapeutic guidelines may not reflect local experience, the absence of local guidelines requires us to look elsewhere for guidance.

On review of the evidence used in the NDOH document, the following 2 publications were used to support and effect the change in eradication strategy.14 In the first by Dong et al, the meta-analysis, cited as the strongest evidence for policy change, is flawed. The methodology of the included trials was very different and given the very high i2 of 81% should not have been combined. To illustrate this heterogeneity, consider a closer review of five trials included in this meta-analysis, each with 100 or more trial subjects.

1. Laurent et al 2001. 247 patients
Azithromycin 500 mg on day 1 and 250mg day 2 to 5 vs. Clarithromycin based triple therapy for 1 week. The H.
Pylori eradication rate for Azithromycin was 38% vs 72%, clearly favouring a Clarithromycin based regimen. If one argues that Azithromycin was used at a sub-standard dosage, so too was the duration of the Clarithromycin regimen i.e. 7 days and not 14 days.\(^5\)

2. **Ivashkin et al 2002.** 100 patients

This article was used as proof of the efficacy of Azithromycin 1g for 3 days vs. Metronidazole and Amoxil based regimen. Metronidazole and Amoxil based regimen is not standard of care. In addition, the article clearly states: “In Moscow, H. pylori strains with primary metronidazole resistance were found in more than 50% of isolates” (Discussion page 881). This study cannot possibly be used as proof to support eradication superiority with Azithromycin. In any event, the eradication rate was only 72% with the higher dose Azithromycin, which means 28% of patients are at risk of H. pylori pathology.\(^6\)

3. **Trevisani et al 1998.** 160 patients

This study was used to demonstrate an equivalent eradication rate at a dose of Azithromycin 500 mg for 3 days. Azithromycin 500 mg 2-4 days, Tinidazole on day 3 vs. Clarithromycin 250 mg bd and Tinidazole 500 mg bd for 7 days. Both regimens are very peculiar with a clearly suboptimal dose of Clarithromycin that still had a higher eradication rate of 81% vs 73%.\(^7\)

4. **Leri et al 1997.** 123 patients

Azithromycin 500mg daily for 6 days vs. Clarithromycin and Amoxil for 14 days. The Clarithromycin regimen demonstrated a 97% vs 68% eradication success rate.\(^8\)

5. **Iacopini et al 2005.** 184 patients

Azithromycin 500 mg for 7 days vs. Clarithromycin and Amoxil for 7 days demonstrated a 70% vs 76% eradication rate favouring Clarithromycin, again at a sub-standard duration of 7 and not 14 days.\(^9\)

The second paper by Sarkeshikian et al., was used as evidence that Azithromycin is equivalent to a Clarithromycin based regimen.\(^4\) However, the duration of Azithromycin was 10 days and the eradication rate was 75%, resulting in 25% of patients still being at risk of H. pylori related pathology. The Clarithromycin based regimen achieved an 83% eradication rate and by increasing the regimen duration to 14 days would likely have achieved a higher eradication rate.

It is understandable given the high prevalence of Helicobacter pylori infection in the Western Cape, that therapeutic cost containment is important. One cannot dispute the appealing pharmacodynamic and physiological theory put forward for the use of Azithromycin. In addition, one cannot dispute the fact that trials demonstrate that Azithromycin does eradicate the Helicobacter pylori organism. However, numerous trials utilizing Azithromycin based regimens with different methods, drug combinations and treatment durations have demonstrated conflicting, if not suboptimal therapeutic results. As an infectious disease, the aim of H. pylori eradication should be 100%, given the association with such adverse gastrointestinal and non-gastrointestinal pathology. It is therefore difficult to justify replacing an accepted international standard of care regimen, based on heterogenous data and lack of local drug efficacy studies.

**References**


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**Mental Health Matters**

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REVIEW

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This article was used as proof of the efficacy of Abbasciano V. A four-day low dose triple therapy regimen


5. Laurent J, Megraud F, Flejou JF, Caekaert A, Barthelemy P. A


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Western Cape Department of Health and UCT School of Public Health

"Update on COVID-19 surveillance in the Western Cape and South Africa"

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Professor David Boulware is a Professor of Medicine in the Division of Infectious Diseases and International Medicine at the University of Minnesota, US.

“COVID-19 infections in health care workers in the US, the evidence regarding chloroquine and hydroxychloroquine in COVID-19, and the clinical trials he is leading.”

For background reading, his group has recently published a review of chloroquine and hydroxychloroquine in COVID-19 that can be accessed at:


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**Juan Ambrosioni, an Infectious Diseases Specialist**
Hospital Clinic de Barcelona, Spain

"Sgared his experiences of managing COVID-19 patients in Barcelona, and himself recovered from infection."

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**MARVIN HSIAO**
Principle Pathologist of Division of Clinical and Diagnostic Virology Research, National Health Laboratory Services and University of Cape Town


**FRIEDRICH THIENEMANN**
Specialist Consultant at University Hospital Zurich and Honorary Associate Professor, University of Cape Town

Friedrich’s department in Zurich has managed over 150 COVID-19 patients in the last few weeks. He presented two case studies and an overview of his clinical experience with an emphasis on monitoring patients’ respiratory status in the general ward

These past presentations are available as recordings on the Department of Medicine website

http://www.medicine.uct.ac.za/covid19-echo-clinic

You are invited to join every week on Wednesday at 16:00
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Viral Hepatitis in sub-Saharan Africa i-ECHO clinic
Monthly on a Thursday 14h30-15h30

One hour ECHO clinics include a 15 minute didactic lecture followed by a presentation of 2-3 Hepatitis B and C cases for discussion and a management plan. At present we have spokes from centres in Nigeria, Ghana, Ethiopia and Mozambique as well spokes within South Africa presenting cases. We encourage and invite any centres who would like to be part of this Viral Hepatitis program to contact us at wendy.spearman@uct.ac.za or msonderup@samedical.co.za

Professor Lewis Roberts was a recent visitor to the Viral Hepatitis in sub-Saharan Africa i-ECHO Clinic in January 2020. Other visitors to the i-Echo clinic have included Professor Geoff Dusheiko (February 2020) and Professor Harry Dalton (December 2020).

Professor Harry Dalton visited and spoke on Hepatitis E – equally speaking at the annual Liver Update meeting

The ECHO platform uses interactive video technology, to connect groups of community providers with specialists at centers of excellence in regular real-time collaborative sessions. The sessions, designed around case-based learning and mentorship, help local workers gain the expertise required to provide needed services. Providers gain skills and confidence; specialists learn new approaches for applying their knowledge across diverse cultural and geographical contexts.

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The 11th Annual Gastro Foundation Fellows Weekend

In the stunning surroundings of Stellenbosch, on the 17th of January 2020, Spier would once again host the 11th annual Gastro Foundation Fellows weekend. The much anticipated weekend far exceeded the expectations of every Fellow who attended. One thing about the Cape and Stellenbosch region is that when it wants to show off its beauty, it is almost impossible to top. The Gastroenterology Foundation of South Africa could not have got it better in any way, and it was an incredible privilege to attend the weekend.

The weekend started with fellows in gastroenterology making their way down to Cape Town International Airport, not only from all over South Africa but also from our neighbouring northern countries. The logistics around fight cancellations and last minute rescheduling (largely due to the crisis at our national carrier airline) were all met with smiles and a level of organisation that can only come from a very dedicated team. On landing, we were promptly met by our transport shuttle and taken off to the beautiful Spier Hotel and Conference Centre.

After a quick cup of coffee and breakfast snacks we gathered in the conference centre and were welcomed by a passionate Prof Chris Kassianides, the chairman and founding member of the Gastroenterology Foundation of Sub-Saharan Africa. It was clear from the outset how passionate his team is and how privileged we were to be there. The introduction of the academic speakers, guest speakers and the program as a whole vividly reaffirmed this notion.

The morning talks covered a number of broad topics relevant to all disciplines of gastroenterology. Dr Adam Boutall (who is arguably one of the most entertaining speakers) started off by covering large bowel obstruction and, as usual, left most of
the fellows feeling like they somehow had finally grasped a topic that they had spent previous hours poring over in a little under 15 minutes. The rest of the morning sessions addressed chronic pancreatitis, hilar cholangiocarcinoma, liver associated enzyme abnormalities, liver transplant, IBD and the very entertaining “difficult colonoscopy”.

As in previous years, the fellows were then split into two groups for the afternoon sessions. HPB and Upper GIT surgeons formed one group and the physicians and colorectal surgeons the other. Every topic seemed to have been carefully selected and opened itself up to academic discussions that went on well beyond the lectures and into the evening. As per tradition, the evening academia ended off with the very entertaining Team Quiz. Quizmasters, Prof Jake Krige and Prof Ed Jonas, tested the general knowledge of the fellows on a variety of topics. Despite most of the surgeons being disadvantaged by the ratio of history related questions to sport related questions, we were fortunate enough to have an outlier, and I am proud to report that it was a surgical team who won honours in the end. This may have been due to the fact that bribes were accepted and that a surgical team was responsible for marking the winning teams answers.

At the end of the first day, dinner was met with great conversation, numerous newly made friendships and outstanding vino from the region. The late evening was followed by a morning run through the Spier vineyards. It was a great opportunity to spend some time with leaders in the field of gastroenterology on a more personal level away from academia.

On Saturday, Fellows were given the opportunity to work in small groups where relevant topics using case reports as references were discussed. The fellows split up into four groups, namely HPB and UGIT, Colorectal, Medical Gastroenterology and Paediatric Gastroenterology. This was incredibly valuable just prior to exams and afforded one the opportunity to address areas of controversy and new developments. That evening, the fellows and delegates were addressed by guest speakers Prof Jonathan Jansen and Dr Anthony Beeton. These esteemed guest speakers’ much anticipated addresses lived up to every expectation. Prof Jonathan Jansen’s thought provoking address The dilemmas of race in medical science research, was followed by Dr Anthony Beeton’s address Error, Disclosure and Approach to the Dissatisfied Patient.

Sunday morning was a chance to consolidate. Highlights included lectures from Prof Vernon Louw on iron physiology, Dr Anthony Beeton on Patient Blood Management and an incredible account from Prof Jean Botha on “The aging surgeon and our responsibilities”. The variety yet direct relevance of all the guest speakers’ topics to those in the medical field was something very unique to this weekend’s program. The weekend concluded at midday on Sunday the 19th.

Only established in 2006, it is difficult to understand how The Gastroenterology Foundation of South Africa has managed to succeed and surpass its aim in such a short time. The Fellows weekend was outstanding in every way. New friendships were forged and the contributions from leading experts and mentors in gastroenterology will shape the new generation’s practices for years to come. South Africa can be proud of a foundation that is truly contributing to the academic excellence in the field of Gastroenterology. We thank Prof Chris Kassianides, all the experts who generously gave of their time and knowledge, the entire team behind the Gastroenterology Foundation of South Africa, the guest speakers, Karin Fenton, Bini Seale and all the sponsors (Sandoz, Surgical Innovations & Takeda) for their incredibly generous contributions.

Colin Noel  
HPB Surgery, UFS

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A Year in Oxford

I have been back in South Africa for a few months and had the opportunity to reflect on what was a crazy, difficult, enlightening year in Oxford.

Although Oxford is “only” a direct flight to London and short train ride away, the process to get there and get settled was immense.

Getting credentials certified via a US bureau, arranging GMC registration, getting visa sponsorship via the Royal College of Physicians, not to mention the actual visa applications, TB clearance’s, NHS surcharges etc. etc. etc.

Settling in Oxford wasn’t hard. The city is like a movie set! In fact it often is, as the Harry Potter fans can attest. It’s also very small, to the point where family and friends who were visiting got the grand tour in 2-3 hours tops. Oxford is the ‘city of soaring spires’, and the university colleges are amongst the oldest in the English speaking world. As are the pubs! We settled in leafy Summertown, and to cap off the experience, JRR Tolkien’s granddaughter was our landlord!

Working at the John Radcliffe Hospital was something of a culture shock. Although English bureaucracy can be a double-edged sword, it was striking to experience the organization of the National Health Service – there must be 3 or 4 people behind the scenes for every doctor in the ward. As Brexit was the key topic of my time there, it was very noticeable that the workforce represented literally dozens of countries.

Endoscopy services are run with military efficiency and regular input through the Joint Action Group (JAG) who audit and credential endoscopy in the UK. The endoscopy unit performs 70-80 procedures on a daily basis, 6 days a week. The infamous ‘points’ system of endoscopy allocation definitely kept me on my toes. I had the privilege of working with Prof James East and Dr Adam Bailey, interventional endoscopists who feature prominently in the UK guidelines.

As a national service, any patient could end up on anybody’s list, and I had the anxiety of coming face to face with a large right sided polyp in an Oxford Head of Dept who had refused sedation for his colonoscopy - in my very first week! Thankfully the adrenaline cardioverted the anal fibrillation, and it all worked out fine.

The actual practice of gastroenterology was fantastic to experience. The senior staff were handpicked from throughout the world, and the names of the IBD leads, Prof Simon Travis, Prof Jack Satsangi, Dr Oliver Brain and Dr Alissa Walsh should be familiar to anyone regularly reading the literature. Sadly, Prof Satish Keshav, a South African clinician / scientist of distinction, passed away early in my time there.

The IBD service was sensational. As a world-renowned centre, patients are seen from throughout the UK and the world. Weekly IBD MDT’s, histopathology meetings, academic rounds, talks, journal clubs, dinners etc. added to the experience. First order of business upon arriving was acquiring the GCP certificate and ‘cracking on’ with the trials I’d been allocated to – ASTIClite (Crohn’s stem cell transplant), risankizumab, REGENERATE (obeticholic acid NASH trial)

The profile of disease in the general GI clinic was also interesting. At least a few patients a week were seen with eosinophilic oesophagitis or gastroenteritis, bile acid malabsorption (up to 20% of patients with IBS-D on SeHCAT per the BSG), and coeliac disease.

As an intestinal transplant unit, under Dr Phil Allan, on-call admissions could be ‘interesting’.

In general, I was struck by the level of academia, and desire to ‘get involved’ in research throughout the hospital. The Translational Gastroenterology Unit, with labs adjacent to the wards and scientists directly involved in the clinics added to this.

The average registrar / fellow (SPR, specialist registrar) had either completed or was planning to take time off training to complete a DPhil (Oxford’s PhD). Nearly every consultant in the unit (and there were more than 20), had a PhD equivalent. Just about the simplest research project I could be involved in was looking at IBD-PSC metabolomics.

Ultimately the year in Oxford was inspiring. It was at times difficult, especially with small children, but ultimately a brilliant experience. I would encourage any future young gastroenterologists to consider broadening their horizons by working in another country or system – the payoff on a professional and importantly, a personal level is immense.

As always, my gratitude to the Gastroenterology Foundation, and especially to Chris Kassianides for proposing and facilitating this great experience.

Jonathan Bolon
In the pursuit of perfection…

pantoprazole is packed with potential

Efficacy is independent of the patient’s age.

Reduced risk of drug-drug interactions due to a lower affinity for cytochrome P450 system.

Symptomatic improvement (e.g. heartburn, acid regurgitation, pain on swallowing).

No dosage adjustments required in elderly or with renal impairment.

Long-term management and prevention of relapse in gastro-oesophageal reflux disease (GORD).

Does not increase platelet aggregation in patients receiving dual antiplatelet therapy.

The Annual Liver Interest group meeting was held in Cape Town in its usual slot over the last weekend of November 2019. The focus of the Gastroenterology Hepatology Association of Sub Saharan Africa (GHASSA) has been on Viral Hepatitis elimination and by consequence Hepatocellular carcinoma (HCC) awareness.

The theme of this year’s meeting continued on that note with an update on political matters aimed at eradicating Viral Hepatitis from Mark Sonderup. Policy is gradually shifting with more African leaders recognizing the importance of Viral Hepatitis as a contributor to morbidity and mortality in their countries. This has been notable in Egypt with one of the world’s largest hepatitis C treatment programme, used to further and accrue other health benefits in that country.

Fortuitously we were in luck to have Professor Harry Dalton visiting from the UK. Prof Dalton is an international expert on Hepatitis E and was a welcome inclusion into the programme shining a spotlight on a virus that is often forgotten about in the South African context despite our neighboring countries having occasional outbreaks.

One of our most eminent scientists, Professor Anna Kramvis expanded upon why sub-Saharan Africa is so afflicted by the burden of HCC with Prof Eduard Jonas contextualizing why it is so difficult to manage once it has been diagnosed; often late and at an advanced stage, afflicting young people in their prime.

One of the issues affecting widespread screening is the lack of available resources. This then provided an appropriate segue into the after brunch session where we had a focus on Point of Care Ultrasound (POCUS) and its utility for the GI/Hepatologist/Surgeon.

POCUS is a rapidly evolving medical field currently being led by our emergency medicine colleagues. Just as it took many years for Laenec to have the stethoscope catch on as an invaluable piece of medical equipment, POCUS has had a slow birth. Luckily in the modern age, with technology rapidly improving and devices becoming smaller and smaller, it is becoming more a physician choice to pick up the skill rather than a technology burden that is the main barrier.

Our International guest, Dr Matteo Rosselli, completed his medical training in Florence Medical University before moving to London and has established Worldwide Radiology, an international NGO aiming to increase diagnostic imaging capacity in resource limited settings, provided further proof of concept that Contrast Enhanced sonography was a viable modality for us to consider moving forward to assist in the early recognition of HCC in sub Saharan Africa. Newer devices also allow for the implementation for an ECHO model, something that is already established in sub-Saharan Africa.

The day was closed with a panel discussion including Prof Landon Myer from the school of Public Health who was able to give us insights into the HIV/AIDS fight and how we can win similar battles in the viral hepatitis sphere. We certainly have a challenge ahead of us.

As always the team of Karin Fenton and Bini Seale were outstanding in putting together a meeting that went off without a hitch despite additional technology and teleconferencing requirements and credit must go to them and the Vineyard Hotel team for always making these events such successes.

Thanks to the sponsors Ferring, Gilead and Medtronic for making the meeting possible.

Bilal Bobat
Dear SAGES members

Due to the ongoing COVID-19 pandemic and the projected peak to be sometime in July, August into September, it has become necessary to consider the annual SAGES congress. We expect there will still be travel bans and congress restrictions over this time and possibly for some time to come. We have also taken into account the economic challenges that this pandemic has brought to many private practices, therefore, with a heavy heart, we have made the decision to cancel the SAGES congress in August 2020.

Next year will be the planned ASSA SAGES congress, but what we will do to limit cancellation penalties on the venue, we will move the 2020 congress to 2022 at the same venue, the Lord Charles Hotel in Somerset West.

We hope you will all understand and give us your support in this decision. We hope and plan to be able to offer some online virtual SAGES educational activities in 2020 to at least offer some CPD opportunities to our members.

Please keep safe and well

Adam Mahomed
SAGES President
The human gut microbiome contains tens of trillions of microorganisms and over 1,000 known species of bacteria, which have many important functions within the human body. Not surprisingly, there has been considerable interest and even more speculation on the role that gut microorganisms might play in health and disease. Gastroenterologists are frequently called upon to interpret, on behalf of their patients, the latest findings from basic and clinical research. To help sift through and assess the vast, complex, at times confusing, and ever-increasing body of literature and provide some guidance to the practicing clinician and their patients, the World Gastroenterology Organisation selected the Gut Microbiome as the focus of the 15th Annual 2020 World Digestive Health Day (WDHD), celebrated all year long, but highlighted on May 29th.

World Digestive Health Day focuses yearly upon a particular digestive disease or disorder to increase public awareness of the prevention, prevalence, diagnosis, management, and treatment of the disease or disorder worldwide. By increasing awareness worldwide of the role that the gut microbiome may have in diagnosis, and how it can be modulated to treat disease and allay symptoms, we can affect overall human health and, in particular, among low- and middle-income countries.

The WGO global network of member societies, partners, and sponsors is ideally positioned to raise awareness of the role the gut microbiome in human health. WGO invites those who are interested in joining the World Digestive Health Day 2020 initiative to visit www.worldgastroenterology.org/wgo-foundation/wdhd/wdhd-2020, Facebook, Twitter, and Instagram @WorldGastroOrg.

About the World Gastroenterology Organisation
Formed in 1938 and incorporated in 1958, The World Gastroenterology Organisation (WGO) is a federation of 115 member societies and four regional associations of gastroenterology representing more than 50,000 individual members worldwide, focusing on the improvement of standards in gastroenterology training and education on a global scale. WGO’s mission is to promote, to the public and healthcare professional alike, an awareness of the worldwide prevalence and optimal care of gastrointestinal and liver disorders, and to improve care of these disorders, through the provision of high quality, accessible and independent education and training.

About World Digestive Health Day
The first World Digestive Health Day (WDHD) was held on 29 May 2005. Ever since, the World Gastroenterology Organisation (WGO) annually celebrates World Digestive Health Day by initiating a yearlong, worldwide, public health campaign through its 115 WGO Member Societies which reach over 50,000 individuals worldwide, WGO Training Centers, Regional Affiliate Associations, and other WGO global partners. Each year focuses upon a particular digestive disease or disorder in order to increase general public awareness of prevention, prevalence, diagnosis, management, and treatment of the disease or disorder.

To learn more about World Digestive Health Day activities or to join the celebration, please visit: www.worldgastroenterology.org/wgo-foundation/wdhd/ wdhd-2020 or Facebook, Twitter, and Instagram @WorldGastroOrg.
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RALEIGH, NC (April 22, 2020) – The Rome Foundation is proud to release the results of its global study on the worldwide prevalence and burden of twenty-two Functional Gastrointestinal Disorders, otherwise known as Disorders of Gut-Brain Interactions (DGBI), from 33 countries on 6 continents, in a paper soon to appear in the prestigious journal Gastroenterology.

The study was initiated by Dr. Ami Sperber, member of the Rome Foundation Board of Directors, and conducted with the collaboration of key principal investigators in the 33 participating countries. Data collection methods included internet surveys in 24 countries, personal interviews in 7 countries, where Internet surveys were not feasible, and both internet and personal interview methods in China and Turkey, using the Rome IV Adult Diagnostic Questionnaire, the Rome III IBS questions and over 80 other questionnaire items to identify variables associated with these disorders.

The results of the study show that more than 40% of persons worldwide have DGBIs, affecting both quality of life and healthcare utilization rates. The Rome Foundation Global Epidemiology Study is the first large-scale, multi-national study on the prevalence and burden of these conditions. It will have a significant impact on our understanding of these conditions at the global and regional levels including associations with gender, age, culture, diet, and psychosocial factors, as well as their significant burden on quality of life, health care utilization and other health system and economic factors.

“The complexity of this study is reflected in the fact that it took over ten years from the initial idea to the initial publication. Over that period of time, the network of researchers was established, the study questions and design were formulated, the methodology was determined (in particular data collection methods), the project was implemented, the data were analyzed, and the first paper accepted for publication in Gastroenterology. Having said that, this is only the beginning. The depth of the database of over 73,000 respondents from 33 countries in 6 continents and the breadth of the study questionnaire will provide substance for new analyses and multiple papers for time to come.” said Global Study Director, Dr. Ami Sperber.

Profs Sandie Thomson and Mashiko Setshedi from the University of Cape Town are principal investigators for the study in South Africa, stated: “The Rome Foundation Global Epidemiology Study provides us with state-of-the-art reliable data on the prevalence and impact of DGBIs in our country, and how these compare to other countries. The findings in South Africa confirm the need to increase the awareness of these conditions and their relevance with clinicians and health policy decision makers. They will also help to establish needs for further research and for resource allocation.”

“This study reflects Rome Foundation’s continued commitment to advancing the science and moving us forward in understanding Disorders of Gut-Brain Interaction. It also confirms the Rome Foundation as an organization with a truly global reach. The epidemiological basis and additional data analyses that stem from this important study will impact the field for years to come and the Rome Foundation will continue to lead the way in this effort.” said Rome Foundation President, Jan Tack.

About the Rome Foundation

For 30 years, the Rome Foundation has sought to legitimate and update our knowledge of DGBIs. We have accomplished this by bringing together scientists and clinicians from around the world to classify and critically appraise the science of gastrointestinal function and dysfunction. This work has enabled the experts convened by the Foundation to make recommendations for diagnosis and treatment that can be applied in research and clinical practice. These recommendations are provided in the Foundation’s publications and reflected in the Rome diagnostic criteria.

For more information, or to set up an interview with Dr. Ami Sperber, Global Study Director, contact Johannah Ruddy, jruddy@theromefoundation.org