reporting times should be resisted if it entails dissecting poorly prepared specimens. The key to good microscopic diagnosis is in the macroscopic examination of properly fixed specimens. Most resected bowls are the patient's definitive treatment and "delay" of a report to allow correct fixation rarely affects acute postoperative management.

**Method for improving lymph node retrieval from gastrectomy specimens**

The ACP broschure 133 by Scott et al outlined the general procedures used in their department for gross examination of the stomach. We agree with the methods they describe but wish to suggest an alternative approach for dealing with gastrectomy specimens which gives high lymph node yields.

This method is a modification of the Japanese Research Society for Gastric Cancer guidelines, as outlined in a paper in the Japanese Journal of Surgery. We recently started using it at the request of one of our surgeons who had a major interest in surgery for gastric cancer.

A summary of the method we use is as follows. The specimens are received fresh, unopened. The anatomical location of the various lymph node groups related to major arteries is identified on the gross fresh specimen (figure) and the relevant piece(s) of fat/omentum containing each lymph node group is then dissected off the specimen. This is easy when the specimen is fresh and each piece(s) representing a specific lymph node group is then placed in a separate labelled pot with 10% formalin for fixation. When all the fat/omentum has been removed the stomach is opened along the greater curve, pinned to a cork board, and floated upside down in a formalin bath until the next day. As all the fat has been removed these specimens fix very well with overnight fixation.

After fixation the specimen is photographed. The relevant measurements and blocks from the stomach are taken as described by Scott et al. Each piece of fat/omentum is then finely sliced using a sharp knife, and also palpated. All lymph node groups found are sampled and identified as belonging to a specific anatomical lymph node group. A form is issued with each report giving details of the number of lymph nodes found in each group and the number in each group with metastatic disease together with a summary of the number of lymph nodes involved in the N1, N2, and N3 groups.

In our department this method is currently used for all radical gastrectomies because we believe this increases the yield of lymph nodes. The table shows the number of lymph nodes retrieved and the number with metastasis from each of the cases received to date.

The advantages to this system of handling are as follows:

1. There is a high yield of lymph nodes as each piece of fat/omentum is small, no longer attached to the stomach, is easy to handle, and can therefore be quickly and thoroughly sliced.
2. The number and anatomical location of all nodes recovered can be accurately detailed to regional areas. The anatomical identification is done at the initial examination when the fat/omentum containing the lymph node groups is separated. This is important with lymph node groups along vascular pedicles—for example, left gastric artery—as the anatomy of these nodes may alter when fixed attached to the main specimen, especially after it has been opened.
3. Once all the fat and omentum has been removed from the stomach, the stomach fixes quicker and is easier to handle and take blocks from as there is no fat.
4. This method can be modified to deal with any partial or total gastrectomy for malignancy in order to maximise lymph node yield. Please note that if there is suspected serosal disease the removal of fat and omentum will not affect assessment of this unless it is exceptionally vigorous.

In summary, we feel this is a valuable and worthwhile procedure which improves lymph node recovery and gives detailed anatomical location of all lymph nodes removed. With a little practice the initial dissection in the fresh state is relatively easy to perform and the total time involved for complete dissection is comparable to conventional methods.

**Diagrammatic representation of the lymph node groups along major arteries.**

**Table 1:** Number of lymph nodes recovered and number with metastasis from each case

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<th>Type of total gastrectomy</th>
<th>No of lymph nodes retrieved</th>
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**Xanthogranulomatous gastritis: an entity or a secondary phenomenon?**

In their paper, Guarino et al seem to imply that the "xanthogranulomatous gastritis" associated with their case of xanthogranulomatous cholecystitis (XGC) is an independent entity. Albeit with the same cause, they postulate that this is related to "a peculiar composition of bile," damaging the mucosa of both the stomach and the gall bladder. They state that they cannot unambiguously attribute similar causes of an association between XGC and xanthogranulomatous inflammation in the bowel wall, but believe that "it is conceivable that deepening of the xanthogranulomatous process in the gall bladder and adhesions in the stomach may result in a transmural involvement of the gastric wall".

It is now accepted that xanthogranulomatous pylonephritis (XPN), a condition analogous to XGC, can produce deep sinuses or fistulae both within and beyond the abdominal cavity to sites such as the small or large bowel, the diaphragm, the lung or skin. Similarly, in a study of only 13 cases of XGC, Roberts and Parsons found three cases of fistulae originating in the gall bladder, two of them extended to the duodenum (close to the stomach, which was not affected) and one to the skin.

Sinus or fistula formation is therefore quite uncommon, although a frequently unrecognised, complication of xanthogranulomatous inflammation in the gall bladder, kidney and, even the appendix. In my experience nodules of xanthogranulomatous tissue, separated by bands of fibrous tissue, can often be found along the track of the extension of this inflammation as it "points" to other organs. Guarino et al describe the gall bladder in their case to be "adherent to the gastric antrum by means of fibrous tissue," I am convinced, therefore, that the gastric xanthogranulomatous inflammation recorded by Guarino et al is the result of extension of primary XGC, and as such should not be described as the proposed separate entity of "xanthogranulomatous gastritis". Despite this, to my knowledge, extension of XGC into the stomach has not been recorded before.
NEQAS parasitology scheme

The recent report of the NEQAS parasitology scheme by Hawthorne et al raised several points of concern. As a participant in both the main parasitology scheme and the parasitology included in the general haematology NEQAS, it seems to me that double standards are operating. The parasitology NEQAS for blood parasites has developed a complex scoring system that seems to be removed from clinical reality in relation to malaria. The haematology NEQAS asks us only for species identification or estimation of the parasitaemia in Plasmodium falciparum infections (the standard practice of the African laboratories that I have worked in).

In table 2 of Hawthorne’s paper the “correct” parasitaemia is given by the reference laboratory without 95% confidence limits to compare individual laboratories’ performance. This is obviously an important area for two reasons: as one assessment of the severity of infection; and as whether to further therapeutic intervention is necessary, such as exchange transfusion. Confusing Plasmodium vivax for Plasmodium ovale is a common problem, but clinically less important because the therapeutic approach is usually the same. In routine practice we always look at both thin and thick films in this laboratory (the latter are not distributed as part of the NEQAS exercises). We usually also have the benefit of more clinical information and often multiple samples over a short period of time which can be of diagnostic use. Getting a sample to the laboratory is the most critical step for laboratory diagnosis: once malarial parasitaemia has been documented, there are relatively few problems. My plea is for one blood parasite scheme with a clinically reasonable scoring system.

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Dr Guarino et al comment:
We find no contradiction between our interpretations and Dr Parsons’ statements. Some points, however, deserve specification. The conclusion drawn by Dr Parsons that the gastric lesion which we described is the result of extension of xanthogranulomatous cholecystitis is exclusively based on the finding of sinususes and fistulae formation in the cases which has studied, none of which affected the stomach. Although we agree that adhesions to surrounding tissues are not uncommon in xanthogranulomatous cholecystitis, in our case sinususes or fistulae were absent and, therefore, we find the comparison made by Dr Parsons inappropriate.
As we pointed out in our paper, we suppose that the gastric lesion may result by adhesion and deepening of the primary gall bladder lesion by Dr Paul’s theory, but there is no definite proof of such an event. Although the cellular mechanisms underlying the collection of xanthomatous histocytes and other cellular elements is basically the same, the causes of xanthogranulomatous inflammation may be very different in various anatomic sites, suggesting caution in drawing general conclusions on aetiology. Therefore, the conviction of Dr Parsons that xanthogranulomatous lesion we studied certainly results from extension of xanthogranulomatous cholecystitis could be an oversimplification.
Finally, the objection made by Dr Parsons on the term of clinical context doesn’t carry any meaning, in our opinion. Indeed, even supposing that the gastric lesion originates from extension of primary xanthogranulomatous cholecystitis, the term xanthogranulomatous gastritis seems to be adequate and is as applicable as the extension to the colon of Crohn’s disease is named granulomatous colitis.

Dr Chiodini et al comment:
We are delighted to note that Dr Paul is enrolled in the parasitology subscheme as well as the general haematology NEQAS. The schemes, however, have slightly different objectives. The haematology scheme distributes blood films four times a year and our communications with that scheme have indicated that the haematologists wish to maintain and sharpen diagnostic skills within the whole breadth of haematology. The inclusion of more blood parasite films to the scheme would weigh against a bias out of proportion to the prevalence of such abnormal films in a district general hospital. Thus the level of input and sophistication of the scoring will inevitably differ between the haematology scheme and the parasitology subscheme where the whole undertaking is devoted to blood parasites. Nevertheless, the parasitology subscheme supplies both the material and the teaching sheets for the haematologists in respect of blood parasites. As I understand it, the haematologists do not have an actual score for their blood film reports, but the mailing is analysed by participant comments. Expert malaria diagnosis, however, is not a consensus of peer opinion but should be assessed against the actual material sent by the designated expert centre against which performers must match their attainment.

The parasitology subscheme asks for species identification or estimation of the parasitaemia in Plasmodium falciparum infections (as do the haematologists). In addition, however, the parasitology subscheme does expect an indication of the species, for example, gametocytes only of Plasmodium falciparum would be managed differently from trophozoites alone.
As for Dr Paul’s comment on parasitology we have the 95% confidence limits for estimates of parasitaemia and participants are scored according to the difference in their report in terms of standard errors from the actual parasitaemia. We entirely agree with Dr Paul that parasitology is important and we are therefore disappointed with the poor performance of participants in this area.
We agree that thick and thin films would be an ideal specimen to send, but at the present time we are sending only obvious thin films and have to be realistic in terms of our expectations, particularly as performance, even with these films, is far from satisfactory. Standard thick films, even when stained are unfixed, and therefore additional concerns over biohazard would be raised.
In our routine practice we too have the benefit of more clinical information and multiple samples over a short period of time but this is clearly impractical when one is considering a NEQAS scheme and we have all to accept that it is impossible to reproduce the exact clinical situation in any external scheme, though we feel that we do come close.
We feel that our scoring system is clinically reasonable and would like to point out that the NEQASAP for parasitology to which the parasitology subscheme reports gave the organiser permission to act on severity of clinical errors if it was felt that individual laboratories were scoring in such a way as to constitute a clinical danger. A scheme which takes account of significant errors in estimation of parasitaemia or missing potentially fatal infections such as Plasmodium falciparum is, we feel, clinically relevant. It is clear that Plasmodium ovale and Plasmodium falciparum have the same treatment and thus the severity of error here is less significant, but none the less we are aiming at precision and it is important for participants to know how well or otherwise they are doing.


Dr Guarino et al reply:
We wish to emphasize that the extension of xanthogranulomatous cholecystitis to the stomach is not an uncommon phenomenon. Of the 67 cases which have been studied, 17 have presented with symptoms and signs of gastric involvement.