Matters arising

hominis could be pathogenic. Because of
space limitations, this is only implicit in the
biotyping scheme presented in Warsaw
(Marples 1981) where a taxonomic col-
lection, mostly for non-clinical sources, was
studied. The isolates were ornithine decar-
boxylase positive and two were from the
blood. At that time, we had not recognised
any anomalies in clumping factor tests,
though we have become aware of these in
this and other species of coagulate negative
staphylococci.

Recognition that tests for clumping may
not be specific for S aureus when rapid bench
kits are used is important, as it may affect
treatment and management. In our case,
the initial misidentification as an anomalous S
aureus was not of clinical importance
because appropriate treatment was given.
There continues to be a need to identify
organisms causing endocarditis.

Comparison of latex and haemolysin tests for
determination of anti-streptolysin (ASO)
antibodies

Curtis et al compared latex and standard
haemolysis inhibition tests for determining
ASO antibodies.1 May we offer one obvious
explanation for the discrepancies in results
referred to in their table 3? They make no
distinction between antistreptolysin
antibody (ASO) and the entity that we have
designated as antistreptolysin factor (ASF).2
The latter is produced by the activity of
cholesterol esterase on low density
lipoproteins and is contained in peptide
fragments where cholesterol is spatially
orientated in such a manner that it is capable
of binding to streptolysin O.3 For years we
have routinely distinguished between ASO
and ASF by measuring activity before and
after precipitation of lipoproteins with dex-
tran sulphate. Only the latter fraction con-
tains true antibody activity and titres are
almost always lower than they are before
precipitation. Raised ASF titres may be
found in some patients with chronic sta-
phylococcal infection, in rheumatoid arthritis,
and in glomerulonephritis. Even in patients
with Lancefield group A infections,
the titre measured by haemolysis inhibition is
usually due to ASF with true ASO only
about two thirds that of ASF. Occasional
contaminated sera give high ASF concen-
trations due to production of cholesterol
esterase by Staphylococcus aureus or
Pseudomonas aeruginosa. Both produce high
ASF concentrations when grown in serum broth mixtures.

We tried to use absolute ethanol to replace
acetone in the mixture. Spreading of the
marker was noted, and we concluded that
acetone was not a good substitute for acetone
for this purpose.

Reference

1 Paterson DA, Davies JD. Marking planes of
surgical excision on breast biopsy specimens:
use of artists' pigments suspended in acetone.

Marking planes of surgical excision on
specimens with mixture of India ink and
acetone

In a recent paper by Paterson and Davies,1
the use of artists' pigments suspended in
acetone to mark planes of surgical excision
on breast specimens was advocated. The
pigment preparations were reported to be
better than India ink in that spreading did
not occur and drying was much quicker.
These authors did not mention the use of
India ink in acetone or other volatile liquids
in their methods nor in their discussions. In
our laboratory the pathologists routinely use
India ink (Pelikan AG, West Germany) to
mark planes of surgical excision. The possi-
bility of improving the performance of
India ink by mixing it with a suitable volatile
reagent immediately came across our minds.
We were unaware of anyone who had
explored this possibility.

We mixed equal parts of acetone with
India ink (Pelikan AG, West Germany) and
compared the application of this mixture
with undiluted India ink on two breast
specimens. When the India ink and acetone
mixture was applied with a brush, no spreading
of the marker beyond the area painted
was noted. Drying was quick and completed
in less than five minutes. The mixture was
easy to prepare and was free from potential
health hazards associated with the artists'
pigments.1 If the mixture was allowed to dry,
however, a hardened gel would result that
could be very difficult to break up. The
undiluted India ink was slow to dry and
tended to spread beyond the area painted.
The intensity of labelling using the two
preparations was indistinguishable when
viewed under the microscope.

Dr Davies et al comment:

We were intrigued to see the above letter
from Drs Chan et al about the use of India
ink mixed with acetone. A brief trial in this
laboratory confirms the apparent benefit
of their novel method, although the very
fluidity of the mixture—admittedly an asset
in rapidly coating a biopsy specimen—would
make differential marking difficult. Indeed,
we must be pointed out that by itself India ink,
even admixed with acetone, precludes
microscopic identification of more than one
plane of resection. Coloured pigments are
necessary for this purpose. Use of the many
older, if more muted artists' pigments would
avoid the potential toxic hazards of cad-
mium, cobalt, and other bright modern
colours. Furthermore, the use of India ink
diluted in acetone does not prevent the
unfortunate tendency for the marker to
penetrate between lobules of fat. The main
advantage of acetone as an ancillary moun-
tant for India ink is that it shortens the
drying time. Like Chan et al, we, too, found
in pilot studies that ethanol was a poor
substitute for acetone.

Reference

1 Harley RD. Artists' pigments c. 1600–1835.
London: Butterworth Scientific, 1982:43-
180.
Comparison of latex and haemolysin tests for determination of anti-streptolysin (ASO) antibodies.

K C Watson and E J Kerr

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