recruited as three volunteers from the first set of experiments were not happy to swallow an intraduodenal tube.

Fluctuations in basal SMABF between different test days do, in our review, reflect acceptable physiological variations. As all determinations were performed by the same investigator (CS), the fluctuations cannot be attributed to an interobserver variability.

(3) We have measured postprandial secretin, cholecystokinin, and gastrin concentrations for many years.⁸⁻¹⁰ We did not measure these in the present study, because we felt that the plasma concentrations did not add any new pertinent information. We acknowledge the references stated with regard to glucagon values found postprandially. Even if the glucagon dose employed induced an increase at the upper limit of physiological glucagon concentrations, we still believe that our conclusions are valid and this is underlined by the lack of change in SMABF observed in our study with this dose of glucagon.

We cannot comment on the unpublished data discussed in the letter, as they are not available yet.

In summary, we have presented data on specific mechanisms regulating postprandial mesenteric artery blood flow using the echo-Doppler flowmetry. Quantitative studies of postprandial hyperaemia are important for the understanding of normal physiological processes. We therefore hope that future research by various groups (as the group of Dr Wells) will be able to shed more light into the physiology and pathophysiology of intestinal blood flow regulation.

CORNEL SIEBER
Hepatic Hemodynamic Laboratory 111/7,
VAMC and Yale Medical School,
West Haven, CT 06516, USA
CHRISTOPH BEGLINGER
Division of Gastroenterology
and
KURT JAEGER
Division of Angiology,
University Hospital,
CH-4031 Basel, Switzerland

Correspondence to: Dr Beglinger.

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Helicobacter pylori infection in healthy people

SIR,—We have recently published the results of an epidemiological study in *Gut* reporting discrepancies between active *Helicobacter pylori* (Hp) infection determined by means of the ¹³C-urea breath test and the prevalence of anti-Hp antibodies in healthy volunteers. Further developments in serological tests make it necessary to report additional information and to reconsider our conclusions based on the serological data presented in the paper.

A systemic humoral immune response to *H pylori* has been searched for in many studies (including our own) by similar serological tests,¹⁻⁵ some of which have become commercially available. They have all in common that whole bacterial cells were primarily used as antigen (acid glycine extracts or sonicated whole cell preparations or formalin treated bacteria). The antigen used in these tests, however, contains flagella proteins that are well known to share antigenicity with flagellae of other bacteria, especially *Campylobacter jejuni*.^{6,7} False positive serological test results can therefore not be excluded. Thus new serological tests using purified high molecular outer membrane proteins of *H pylori* and urease as antigens have recently been developed.^{8,9} These second generation serological tests may be more specific for *H pylori* infection.

We have investigated the sensitivity and specificity of several different serological tests in a population of patients in whom the presence or absence of *H pylori* infection was unequivocally established. These patients all had upper gastrointestinal tract endoscopy with antral mucosal biopsies that were used for microbiological *H pylori* culture, and a quick urease test (CLO test) and they all underwent a ¹³C-urea breath test. Sera were used only from patients in whom either all three tests were positive (*H pylori* infection present) or all three tests were negative (infection absent). These latter patients were also questioned about treatment with antibiotics within the past six months and included in the present analysis only if the response was negative. Sera from this population were tested for anti-Hp antibodies with our own enzyme linked immunosorbent assay ELISA and two commercially available, first generation serological tests (anti-Hp IgG EIA Roche, Hoffman-La Roche, Basel, Switzerland, and GAP test IgG, Bio Rad Laboratories, Glattbrugg, Switzerland) and a new second generation serological test that uses a well characterised, highly immunogenic, purified Hp-specific multicomponent antigen preparation free of cross reacting flagella proteins (anti-Hp IgG EIA Roche second generation, Hoffman-La Roche). Sera from 223 patients were tested; 64 patients had *H pylori* infection and 159 did not. The sensitivity and specificity of the four serological tests are shown in the Table.

	Meyer et al ¹	Roche 1st	GAP	Roche 2nd
Sensitivity	100	93	94	97
Specificity	80	85	85 (96)*	93

*If test results were included that reacted slightly or strongly (specificity within parentheses) positive.

Firstly, it is obvious that the three first generation tests react positively in 15-20% of people who have no active *H pylori* infection. If the same people are, however, tested with the second generation test, only 7% have anti-Hp

without active infection. The cumulative percentage of patients reacting with either one of the three first generation tests amounted to 29%, resulting in a specificity of only 71%. These findings support the hypothesis that with first generation serological tests in an important fraction of people, anti-Hp antibodies may be due to non-specific binding to the antigen in the test kit rather than to a specific response to *H pylori* infection in the past. While it is still possible that healthy people eventually eliminate *H pylori* spontaneously, this conclusion may not be drawn from our results based on the first generation serological test that was used. Similar caution, however, should be used in the interpretation of virtually all studies that reported *H pylori* prevalence data based on first generation serological tests.¹⁻⁵ Epidemiological studies designed to gather information on the prevalence of *H pylori* should preferentially use direct proof of infection rather than serology.

B MEYER-WYSS
C BEGLINGER
L BASELGLIA
Division of Gastroenterology,
University Hospital,
CH-4031 Basel, Switzerland
H MERKI
E RENNERT
Division of Gastroenterology,
Inselspital,
CH-3010 Bern, Switzerland

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Medical treatment of bleeding peptic ulcer: old drugs, new regimens

SIR,—Haemorrhage from peptic ulcer is due to the erosion of artery at the ulcer base by the combined digestive actions of acid and pepsin. Platelet plug and clot formation (both factors being pH sensitive) seal the bleeding artery. Dissolution of the clot is the most important factor for peptic ulcer rebleeding.¹ Intra-gastric acidity prolongs the duration of bleeding² as the gastric juice contains fibrinolytic substances³ and a pH <7 results in inhibition of platelet aggregation and dissolution of the clot.⁴ Understandably then, attempts to counteract the acid^{5,6} or pepsin⁷ or to inhibit fibrinolysis⁸ should result in stabilisation of the clot and prevention of rebleeding. Yet to date the efficacy of none of the above mentioned drugs