protein (CRP) concentrations in serum (1) has sensitivity, specificity, and accuracy comparable with those of latex agglutination (2). We compared the results obtained by the fat-agglutination test for 488 sera with those by nephelometry (Hyland laser nephelometer PDQ). Lipemic or hemolyzed sera unsuitable for nephelometry were quantified by radial immunodiffusion (RID; Behring LC Partigen).

We used goat antibodies to human CRP (Atlantic Antibodies, Scarborough, ME), diluted 40-fold in isotonic saline, standardizing the nephelometry with Atlantic Calibrator 7, the test results being interpolated automatically. For the fat-agglutination test we mixed one part of Intralipid (20%) with four parts of 0.1 mol/L CaCl₂ solution, then mixed 30 μL of this with an equal volume of test sera, on a slide, and examined the results visually 10 min later. Tests were scored as positive or negative for agglutination. Unequivocal agglutination indicated CRP > 10 mg/L. Higher concentrations are considered pathological (3).

The fat-agglutination test gave unequivocal results for 448 samples; a further 42 showed weak agglutination, which we also scored as positive. False-positive results were recorded in 13 cases (2.9%) and false negatives in four (1%), with all the false negatives having CRP < 20 mg/L by nephelometry (confirmed by RID). There was no variation in scoring among four laboratory personnel. These results reflect a sensitivity of 97.6%, a specificity of 95.6%, and an accuracy of 96.4%.

Although not directly quantifiable, the extent of fat agglutination reflects the CRP concentration in serum. No prozone effect appeared up to 400 mg/L, and lipemic or hemolyzed samples and samples containing rheumatoid factors (titer 1:320) caused no interference. Reagent costs are less than a thirtieth as great as for nephelometric determination (in-house costs). About half of our requests for CRP quantification give results reported as insignificant (< 10 mg/L). The desirability of rapid CRP determination is well documented (4, 5). We find an inexpensive, rapid, and reliable screening procedure useful in a busy department.

References

No Catalase Isoenzymes in Serum, László Góth (Dept. of Lab., Municipal Hosp., H-8330 Sümeg, Hungary)

We used our polarographic method (1) for catalase (EC 1.11.1.6) to look for diseases with increased catalase activity in serum, finding it in acute pancreatitis (2) and hemolytic (3) and liver diseases (1). This enzyme is localized in a soluble form in erythrocytes, and mitochondria and peroxisomes of liver are rich in it (4).

To increase the diagnostic specificity, we attempted to detect catalase isoenzymes in serum by electrophoresis on cellulose acetate membrane (Electrophoresis Strip; Sartorius, F.R.G.). We applied 2-μL samples onto a 4 × 7 cm strip and electrophoresed for 5 h (4 mA per strip in phosphate buffer, 60 mmol/L, pH 7.4). We then placed the strips on the surface of starch gel (50 g/L in the phosphate buffer) for 2 h. After blotting the samples, we made catalase in the starch gel visible by a simple negative-staining method (5). With this method we could detect catalase activities between 30 and 9000 kU/L. The catalase bands were characterized by their mobility (Rf) relative to serum albumin.

We assayed sera with above-normal catalase activity from 29 patients with acute pancreatitis (mean and range) 436.4 (197–959) kU/L; 54 with hemolytic diseases—hemolytic anemia, pernicious anemia, Zieve’s syndrome, or polyglobulinemia—1374.6 (126–1485) kU/L; 54 with liver diseases—toxic hepatitis, cholestasis syndrome, liver cirrhosis, or liver congestion due to cirulatory failure—[575.9 (111–6690)] kU/L; and 57 with other diseases, including diabetes mellitus, myocardial infarction, bronchopneumonia, arteriosclerosis, and others [296.0 (153–988)] kU/L. For comparison, we also assayed 68 sera from a control group with normal catalase activity [61.8 (34.8–98.4)] kU/L; 50 homogenates of human liver [2670 (1380–5980)] kU/L; 30 homogenates of human pancreas [286 (182–394)] kU/L; and 50 hemolysates of human erythrocytes [6580 (4540–8670)] kU/L.

Serum catalase activity as determined by our polarographic method (1) gave the following normal value [mean (and range)]: 56.7 (14.1–99.3) kU/L.

Sera from the control group, with normal catalase activity, showed, electrophoretically, one catalase band with an Rf value of (mean ± SD) 0.516 ± 0.034. This catalase band was localized in the second half of the β-globulin region, in front of the γ-globulin band. Sera with pathological activities also had only one catalase band, characterized by the same (P > 0.1) Rf value: 0.525 ± 0.029 in hemolytic diseases, 0.529 ± 0.02 in acute pancreatitis, 0.524 ± 0.024 in liver diseases, and 0.530 ± 0.029 in other diseases.

We also detected only one catalase band in human tissues, but with the following mean (SD) Rf values: 0.664 ± 0.021 in erythrocyte hemolysates, 0.662 ± 0.027 in liver homoge-

![Fig. 1. Electrophoretic pattern of catalase enzyme activity](image-url)
nates, and 0.656 ± 0.021 in pancreas homogenates. These catalase bands were localized in the β1-globulin region with the same electrophoretic mobility (P >0.1). The bands for catalase in serum and tissue homogenates can be easily distinguished, either by visualization or statistically (P <0.001).

Given that some enzymes (e.g., γ-glutamyltransferase, creatine kinase) undergo modification in serum, we supposed a similar effect of serum for catalase. To prove this, we incubated mixtures of sera and erythrocyte hemolysates (5:1 by vol) for 30 min before electrophoresis. The high catalase activity (2000 kU/L) yielded only one catalase band, its mobility (0.536 ± 0.051) being the same (P >0.1) as that of serum catalase. Similar results were obtained for mixtures of serum and purified erythrocyte catalase.

Thus serum catalase can be regarded as a post-translational modification of tissue catalase. This modification takes place in the serum and the new catalase variant, with changed electrophoretic mobility, is supposedly tissue catalase plus some serum component(s).

We conclude that electrophoresis does not enhance the diagnostic specificity of serum catalase determinations.

References

Correlation between Serum Urea and Salivary Urea,
Kyaw Tun Sein and Geetha Arumainayagam (Dept. of
Chem. Pathol., School of Med. Sciences, Universiti Sains
Malaysia, 11900 Penang, Malaysia)

Previous studies have shown that urea in parotid saliva is
directly proportional to that in blood (1–3), and measure-
ment of the former has been suggested as a simple means of
following the progress of hemodialysis (4). We report here
our findings on the correlation between serum urea and
mixed salivary urea in healthy volunteers (ages 21–71 r),
in patients with either hypertension, diabetes, or chronic renal
failure (ages 24–75 r), and in patients undergoing hemodi-
yalys (ages 16–54 r). Subjects were asked to rinse their
mouths twice with distilled water, and the (unstimulated)
saliva produced within the next 15 min was collected. We
then determined serum and salivary urea by the urease/glut-
amate dehydrogenase method (4), using Roche reagent kits
in a Cobas-Bio centrifugal analyzer. Salivary urea was
determined within 30 min after specimen collection. Within-
day precision (CV) for four different samples of either serum
or mixed saliva samples was ±1.5% (n = 20). For healthy
volunteers (serum urea 2.98–7.00 mmol/L, n = 56) the
correlation coefficient between serum urea and mixed sali-

vary urea was 0.735 and the regression equation was y
(salivary urea) = 0.615x + 0.609. The Syp was 0.778. Figure
1 shows the correlation between serum urea and salivary
urea in the patients. The high correlation (r about 0.99)
between serum urea and mixed salivary urea that we have
obtained in both groups of patients agrees well with the
recent observations of Akai et al. (5), who could also show
a strong correlation between these two parameters with use of
a reagent-strip method.

References
1. Forland M, Shanon IL, Katz FH. Parotid fluid urea in nitrogen
2. Dahlberg WH, Sreebny LM, King B. Studies of parotid saliva

Fig. 1. A. Correlation between serum urea and mixed salivary urea in
patients with either hypertension, diabetes, or chronic renal failure
r = 0.999, y (salivary urea) = 0.900x – 1.424, and Syp = 0.778. (n = 56, serum
urea, 5.40–36.10 mmol/L)

B. Correlation between serum urea and mixed salivary urea in hemodi-
alys patients before and after dialysis
r = 0.990, y (salivary urea) = 0.865x – 0.847, and Syp = 0.669. (n = 50, serum
urea 4.02–22.94 mmol/L)