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Response

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We thank Dr Lacout and colleagues for their interest in our study (1). As they note, benign nodules can decrease in size, especially nodules with a cystic portion. Shrunken benign nodules can show suspicious features such as shadowing and regular peripheral black and white halo at US (2) as well as solidity, ill-defined margins, and taller-than-wide shape. These features are considered suspicious malignant features. Thus, thyroid nodules with these features are considered as suspicious. Regular peripheral black and white halo and shadowing without macrocalcifications are features that are considered to indicate shrunken benign nodules. However, a definite differentiation between shrunken benign nodules and suspicious nodules can be done only if the suspicious nodules are located at the corresponding area of the cystic nodules observed at a previous US examination and if there are no other nodules. In patients with these findings, we do not perform fine-needle aspiration (FNA). However, if there is no previous US examination, FNA is performed because there is no evidence of shrunken benign nodules. In terms of vasculature, malignant nodules more frequently show no vascularity than benign nodules (3). Thus, vascularity is not a useful trait to decide whether FNA should be performed.

TIRADS is based on initial US features (1). However, if nondiagnostic thyroid nodules show decreased size at follow-up US, repeat FNA is not performed regardless of initial TIRADS category.

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Severe Acute Pancreatitis: Is Organ Failure Enough?

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Editor:

In the July 2015 issue of *Radiology*, Dr Meyrignac and colleagues (1) showed that the volume of extrapancreatic necrosis is an early predictor of several outcomes in patients with acute pancreatitis, including organ failure and infected necrosis. However, the authors defined organ failure with use of an antiquated scoring system that is not used in clinical practice or research due to the inclusion of low Glasgow Coma Scale scores and platelet counts because neither are specific for nor commonly seen in acute pancreatitis (2). More important, they fail to recognize that the mere presence of organ failure does not define severe acute pancreatitis. Rather, persistent organ failure (ie, organ failure lasting >48 hours) defines severe acute pancreatitis, and this was the basis for the recent revised Atlanta and determinant-based classification systems for acute pancreatitis (3). Transient organ failure (<48 hours) is not associated with increased mortality (4). The authors' definition of infection has similar flaws because it encompasses both extrapancreatic and pancreatic infections, which each have a different impact on prognosis. Nearly 25% of patients with acute pancreatitis have extrapancreatic infections that are associated with low mortality (5). However, pancreatic infection or infected necrosis increases mortality in the late phase of hospitalization and is considered by many to be equivalent to persistent organ failure as a determinant of severe acute pancreatitis (6). In addition, the reduced use of early surgical débridement in favor of percutaneous drainage catheters and minimally invasive "step-up" approaches in patients with infected necrosis has substantially decreased mortality (7). Because the authors did not use widely accepted definitions of organ failure and infection in severe acute pancreatitis or provide numeric data to show how many patients in their cohort actually had persistent organ failure and/or infected necrosis, it is difficult to generalize their findings to clinical practice. We suspect that extrapancreatic necrosis volume would not be superior to any of the other radiologic scoring systems if the outcomes were defined as above. In addition, the authors provide no information about parenchymal pancreatic necrosis in their co-

hort. This information is important because patients with extrapancreatic necrosis have been shown to have better outcomes than patients with combined parenchymal and extrapancreatic pancreatic necrosis (8).

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Response

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We sincerely thank Dr Kamal and colleagues for their comments on our study (1). They raised the central point of how severe acute pancreatitis is defined, questioning our choice of considering transient organ failure, low platelet count, and extrapancreatic infection in our definition of severe acute pancreatitis.

Although we agree that a persistent organ failure criterion should be used in future studies (2), we would like to address the other two definitional concerns. Authors have shown that aggravated coagulation parameters such as low platelet count enable the prediction of fatal outcome in patients with acute pancreatitis (3,4) and that extrapancreatic infections are also associated with fatal outcomes. Specifically, in the study by Besselink et al (5) cited by Dr Kamal and colleagues, the mortality rate was higher in patients with bacteremia and pneumonia (36% and 34.6%) than in those with infected necrosis and pancreatic necrosis (30% and 23.4%). Moreover, in our study, every scoring system was evaluated with the same criteria. Modifying those criteria would certainly change the descriptive values of absolute scoring systems (eg, sensitivity and specificity). However, the ranking between areas under receiver operating characteristic curves should remain the same.

With regard to minimally invasive "step-up" approaches in patients with infected necrosis, we agree that it has decreased acute pancreatitis mortality, especially when one includes endoscopic approaches (6). Nevertheless, patients who undergo these kinds of procedures still suffer from severe acute pancreatitis and this should not change the initial severity evaluation.

We agree that the presence of parenchymal pancreatic necrosis is important information. However, in our study, 10 patients had more than 50% parenchymal necrosis. Only one of these patients had less than 100 mL of extrapancreatic necrosis. That patient did not develop organ failure, not even transient organ failure or infection (including extrapancreatic infection). Extrapancreatic necrosis was greater than 200 mL in the other nine patients. Nonetheless, we believe that no scoring system is self-sufficient and that pancreatic necrosis evaluation and clinical scoring systems should still help assess acute pancreatitis severity along with extrapancreatic necrosis volume. Finally, looking beyond any definitional concerns, our study clearly demonstrated a correlation between length of hospitalization and extrapancreatic necrosis volume that we believe is useful for clinical practice.

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No Association between Intravenous Contrast Material Exposure and Adverse Clinical Outcome: Are Prevention Protocols Effective?

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Editor:

We read with great interest the article by Dr McDonald and colleagues in the December 2014 issue of *Radiology* (1), in which they suggested that intravenous contrast material exposure is not associated with dialysis and mortality. The large sample size and the definition of a control group (contrast material–enhanced computed tomographic [CT] scans were compared with unenhanced CT scans) are noteworthy. Their results are consistent with those from a previous article by the authors, based on the same dataset, showing no relationship between contrast material exposure and acute kidney injury (AKI) (2).

Conversely, contrast material exposure has been associated with adverse outcomes in numerous other studies (3). Incidence rates of contrast-induced AKI up to 30% have been reported. The risk of contrast-induced AKI is probably lower after intravenous as compared to intraarterial contrast material administration, although the evidence is scarce (4). Several strategies have been described to prevent contrast-induced AKI, including intravenous volume expansion and temporarily stopping nephrotoxic medication (4). According to guidelines, most hospitals have now implemented contrast-enhanced AKI prevention protocols. However, the effectiveness of these prevention protocols has not been investigated.

In the study by Dr McDonald and colleagues, it is unclear whether patients were treated according to a prevention protocol. Assuming that a prevention protocol was followed in a substantial proportion of the patients, one would expect a relatively low incidence of AKI after contrast material exposure. The fact that no difference in AKI was found after contrast-enhanced versus unenhanced CT in this study could be interpreted as showing that prevention protocols are effective. Ideally, it would be interesting to study the occurrence of contrast-induced AKI before and after implementation of a prevention protocol.

Furthermore, it is possible that AKI was overestimated in the noncontrast group. Patients were only included if creatinine levels were measured before and after CT. This suggests a selection of patients with co-morbid conditions (eg, hospitalization) that may be related to adverse outcomes and may explain the very high 30-day mortality rate (8%) in their study. Moreover, despite propensity score analysis, bias may be introduced in the noncontrast group, as physicians will be inclined to avoid contrast material in patients who are, in their opinion, at high risk to develop AKI (3).

The study shows that dialysis dependency after intravenous contrast material administration is rare. Patient selection and treatment according to prevention protocols may contribute to this reassuring outcome. Nevertheless, it cannot be precluded from the data that intravenous contrast material exposure can be an infrequent cause of contrast-induced AKI.

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Response

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We appreciate Drs Gramberg and Penne's interest in our recent article on clinical outcomes associated with contrast material–associated AKI. In our propensity score–matched study, we were unable to find evidence that intravenous contrast material administration was a significant independent risk factor for AKI, dialysis, or mortality (1). The evidence in support of a causal association between iodinated contrast material exposure and adverse clinical outcomes may appear robust but is largely comprised of uncontrolled studies of intra-arterial iodinated contrast material administration (2). Such uncontrolled studies are of limited value as they cannot establish causality, yet such data confound discussions of both the true incidence and severity of contrast-associated AKI (3).

At our institution, there is no protocol for outpatient CT contrast-asso-