Review

The Atlanta Classification of acute pancreatitis revisited

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Background: **In a complex disease such as acute pancreatitis, correct terminology and clear definitions are important. The clinically based Atlanta Classification was formulated in 1992, but in recent years it has been increasingly criticized. No formal evaluation of the use of the Atlanta definitions in the literature has ever been performed.**

Methods: **A Medline literature search sought studies published after 1993. Guidelines, review articles and their cross-references were reviewed to assess whether the Atlanta or alternative definitions were used.**

Results: **A total of 447 articles was assessed, including 12 guidelines and 82 reviews. Alternative definitions of predicted severity of acute pancreatitis, actual severity and organ failure were used in more than half of the studies. There was a large variation in the interpretation of the Atlanta definitions of local complications, especially relating to the content of peripancreatic collections.**

Conclusion: **The Atlanta definitions for acute pancreatitis are often used inappropriately, and alternative definitions are frequently applied. Such lack of consensus illustrates the need for a revision of the Atlanta Classification.**

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Introduction

Over the past five decades, several classification systems on pancreatitis have emerged from interdisciplinary symposia[1–4]. The most recent international meeting on this topic, the 1992 Atlanta symposium, produced a clinically based classification system[4,5]. Definitions of acute pancreatitis, its severity, organ failure and the local complications 'acute fluid collection', 'pancreatic necrosis', 'pseudocyst' and 'pancreatic abscess' were proposed. The Atlanta Classification attempted to introduce uniformity in the assessment of clinical severity and the various complications of the disease. This is the only widely accepted classification system used by clinicians and radiologists.

With increasing knowledge of the pathophysiology of pancreatitis and the development of new means of intervention, several authors have pointed out shortcomings in the Atlanta Classification[6–13]. A recent review demonstrated that terminology abandoned by the Atlanta symposium, for instance 'phlegmon' and 'infected pseudocyst', is still used frequently in the literature, and that various new terms, such as 'organized pancreatic necrosis' and 'necroma', have been introduced since 1993[14]. A critical evaluation of the use of the Atlanta Classification in the literature has never been performed. The present review assesses whether the definitions of the Atlanta Classification are accepted in the literature and evaluates the extent of variation in interpretation of these definitions.

Methods

A Medline search of literature published between 1993 and 2006 was performed using the following terms:

The Editors have satisfied themselves that all authors have contributed significantly to this publication

Table 1 Summary of the 1992 Atlanta Classification

	Definition
Acute pancreatitis	An acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems Associated with raised pancreatic enzyme levels in blood and/or urine
Severity	
Mild acute pancreatitis	Associated with minimal organ dysfunction and an uneventful recovery; lacks the features of severe acute pancreatitis. Usually normal enhancement of pancreatic parenchyma on contrast-enhanced computed tomography
Severe acute pancreatitis	Associated with organ failure and/or local complications such as necrosis, abscess or pseudocyst
Predicted severity	Ranson score ≥ 3 or APACHE II score ≥ 8
Organ failure and systemic complications	
Shock	Systolic blood pressure < 90 mmHg
Pulmonary insufficiency	$Pa_{O_2} \leq 60$ mmHg
Renal failure	Creatinine ≥ 177 μmol/l or ≤ 2 mg/dl after rehydration
Gastrointestinal bleeding	500 ml in 24 h
Disseminated intravascular coagulation	Platelets $\leq 100,000/mm^3$, fibrinogen $< 1{\cdot}0$ g/l and fibrin-split products > 80 μg/l
Severe metabolic disturbances	Calcium $\leq 1{\cdot}87$ mmol/l or $\leq 7{\cdot}5$ mg/dl
Local complications	
Acute fluid collections	Occur early in the course of acute pancreatitis, are located in or near the pancreas and always lack a wall of granulation of fibrous tissue. In about half of patients, spontaneous regression occurs. In the other half, an acute fluid collection develops into a pancreatic abscess or pseudocyst
Pancreatic necrosis	Diffuse or focal area(s) of non-viable pancreatic parenchyma, typically associated with peripancreatic fat necrosis Non-enhanced pancreatic parenchyma > 3 cm or involving more than 30% of the area of the pancreas
Acute pseudocyst	Collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, which arises as a result of acute pancreatitis, pancreatic trauma or chronic pancreatitis, occurring at least 4 weeks after onset of symptoms, is round or ovoid and most often sterile; when pus is present, lesion is termed a 'pancreatic abscess'
Pancreatic abscess	Circumscribed, intra-abdominal collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma Often 4 weeks or more after onset Pancreatic abscess and infected pancreatic necrosis differ in clinical expression and extent of associated necrosis

APACHE, Acute Physiology And Chronic Health Evaluation; Pa_{O_2}, arterial partial pressure of oxygen.

'acute pancreatitis and review' and 'acute pancreatitis and guidelines'. From the identified guidelines and reviews, cross-references were retrieved. The search included all types of publication (reviews, guidelines, original studies, case reports and editorials), but excluded those not in English and animal experimental studies. One author (T.L.B.) performed the selection and reviewed all full-text papers to assess whether the original Atlanta definitions (*Table 1*) or other definitions were used for the following five components of the Atlanta Classification: diagnosis (cut-off levels of pancreatic enzymes lipase and amylase); predicted severity (predictive scoring systems, cut-off levels of scoring systems); actual severity (distinction between mild and severe pancreatitis, distinction between predicted and actual severity); organ failure (determinants of individual failing organ systems, cut-off levels of determinants, distinction between single-organ failure and multiorgan failure); local complications (pancreatic necrosis and peripancreatic necrosis, infection of necrosis, morphological aspects and distinction of different types of collection).

If different definitions for the components were identified, this was double checked by one of two other authors (H.C.v.S., M.G.B.). All disagreements were resolved by discussion among the authors. In addition, study results leading to new insights that might have influenced the interpretation of the Atlanta Classification were recorded and are discussed. As a large number of references were retrieved, for each component of the Atlanta Classification that was assessed only the three most recent articles are cited here; the remaining references are published in *Appendix 1* (available as supplementary material online at www.bjs.co.uk).

Results

A total of 447 articles was reviewed, including 12 guidelines and 82 reviews. These articles reported on studies that were not specifically designed to evaluate the Atlanta Classification; they merely mentioned Atlanta definitions (for example a randomized trial comparing two treatment strategies with the outcome 'pseudocyst'). Therefore, an assessment of methodological quality was deemed inappropriate. *Table 2* gives an overview of the papers according to type of article and impact factor of the journals in which they were published. The most important discrepancies for the five components of the Atlanta Classification and discrepancies in the 12 guidelines are discussed in order.

Diagnosis

The Atlanta Classification provides no cut-off value for pancreatic enzyme levels. In 116 studies, the diagnosis of acute pancreatitis was defined as a characteristic clinical history of abdominal pain and an increased level of pancreatic enzymes to three or more times the upper limit of normal. However, 31 studies used different thresholds, ranging from two or more[15–17] to more than four[18–20] and more than five[21–23] times the upper limit of normal.

Predicted severity

A total of 283 articles provided criteria for predicting severity in acute pancreatitis. Some 86 reports used the severity scoring systems proposed by the Atlanta symposium[16,17,23]. However, 197 studies used a different cut-off level for defining severity, or used different or additional scoring systems, such as computed tomography (CT) severity index, Imrie (Glasgow) score, Simplified Acute Physiology score, Sequential Organ Failure Assessment or severity predictors (such as C-reactive protein)[15,24,25]. Cut-off values for severity stratification differed considerably between reports. For the CT severity index, the most established radiological scoring system developed by Balthazar and colleagues[26] in 1990, the cut-off value to differentiate between mild and severe disease ranged from three or more to eight or more points[27–29]. In 32 studies, threshold values for Acute Physiology And Chronic Health Evaluation (APACHE) II score (other than eight or more) varied from five or more to 11 or more, whereas the time for calculating the score varied from day of admission to 24 and 48 h after admission[30–32]. Eleven studies used different threshold values for the Ranson criteria (other than three or more), ranging from more than three to more than five[32–34].

Since the Atlanta symposium in 1992, many studies have identified new predictors of severity and these have been incorporated in several guidelines. Such predictors include age (over 55[6], over 70[35] or over 80[36] years), obesity (body mass index over 30 kg/m^2)[11,24,37], pleural effusion (left or bilateral) on chest radiograph[38–40], raised haematocrit level[6,41,42] and C-reactive protein level greater than 150 mg/dl after 48 h[43–45].

Actual severity

Of 297 articles providing definitions for severe acute pancreatitis, 195 defined severe disease according to the Atlanta Classification, although 61 merely stated that the Atlanta criteria were used without specification[46–48]. The remaining 102 articles used definitions of severe disease other than those of the Atlanta Classification. These definitions were based on admission to an intensive care unit, length of intensive care unit or hospital stay, complications requiring medical or operative intervention, mortality or various other, additional or non-specified criteria[17,49,50]. The authors of 45 articles used the absence and presence of

Table 2 Characteristics of retrieved articles (1993–2006) specified according to impact factor of journal

		Impact factor		
	Total no. of studies (n = 447)	High (> 5·0) (n = 89)	Intermediate (1·5–4·9) (n = 273)	Low (< 1·5) (n = 85)
Meta-analyses	3	2	1	0
Randomized controlled trials	34	13	18	3
Prospective series	144	28	99	17
Retrospective series	147	23	95	29
Reviews	82	10	44	28
Guidelines	12	5	5	2
Editorials	5	2	3	0
Other	20	6	8	6

pancreatic necrosis broadly synonymously with mild and severe acute pancreatitis respectively[47,51,52]. Some reports, however, pointed out that patients with the morphological diagnosis of interstitial pancreatitis may develop clinically severe disease[44,53,54].

The relationship between the development of organ failure and pancreatic necrosis (the most important determinants of severe acute pancreatitis) is contentious. Several reports noted that only 51–55 per cent of patients with pancreatic necrosis manifested organ failure[55–57]. In the study by Lankisch and colleagues[53], 15 per cent of patients with acute oedematous pancreatitis developed organ failure. In a recent study, organ failure was the main risk factor for mortality, regardless of the presence or absence of pancreatic necrosis[23]. Conversely, other studies showed a good correlation between organ failure and the extent of pancreatic necrosis[16,58,59].

Finally, in 38 articles, the differentiation between 'predicted severe' acute pancreatitis (Ranson, Imrie or APACHE II score) and 'actual severe' disease (systemic or local complications) was not apparent from the published data[17,28,60]. The difference is important, because in recent studies less than 50 per cent of patients with predicted severe disease eventually turned out to have actual severe disease according to the Atlanta criteria[25,46]. This lack of distinction may account for the variation in incidence of severe acute pancreatitis among institutions.

Organ failure

Criteria for organ failure were found in 149 articles. In 35 reports the exact Atlanta definitions for organ failure were specifically stated and used[23,61,62]. Seven articles restricted organ failure to two of the four Atlanta determinants for organ failure: respiratory and renal insufficiency[63–65]. However, 107 articles used additional criteria for organ failure and systemic complications, such as leucocytosis, temperature, coagulopathy, nervous system failure, hepatic failure, systemic inflammatory response syndrome or sepsis, or used altered thresholds or adjustments for the Atlanta definitions of organ failure[52,66,67]. The remaining articles gave no definition of organ failure, or simply noted that the Atlanta criteria were used, without specification.

In recent years, multiorgan failure has been acknowledged as a major determinant of mortality. However, no uniform definition for multiorgan failure exists: 20 reports defined it as failure of two or more organ systems[31,46,49], and eight as failure of three or more organ systems[23,68,69], although most studies did not define multiorgan failure.

The dynamic process of organ dysfunction is increasingly recognized, and several authors differentiated between transient and persistent organ failure[70–72]. In addition, several studies showed that early and progressive organ failure was associated with high mortality, but most patients with transient organ failure had an uncomplicated course[72–74]. The recent UK guidelines on acute pancreatitis state that organ failure in the first week resolving within 48 h should not be considered an indicator of severe disease[43].

Since 1993, several new organ failure grading systems have been developed (Goris score, Marshall or multiple organ dysfunction score, Bernard score, Sequential Organ Failure Assessment and logistic organ dysfunction syndrome score) that take into account the number of organ systems involved and the degree of dysfunction of each individual organ. Some systems also include the need for inotropic or vasopressor agents, mechanical ventilation and dialysis that the Atlanta symposium did not account for. Several studies have shown that dynamic scoring systems (such as the delta APACHE II score) or scoring systems that account for the physiological response to treatment (such as the delta organ failure score or cumulative Marshall score) are better predictors of outcome than static scoring systems[31,32,71].

Local complications

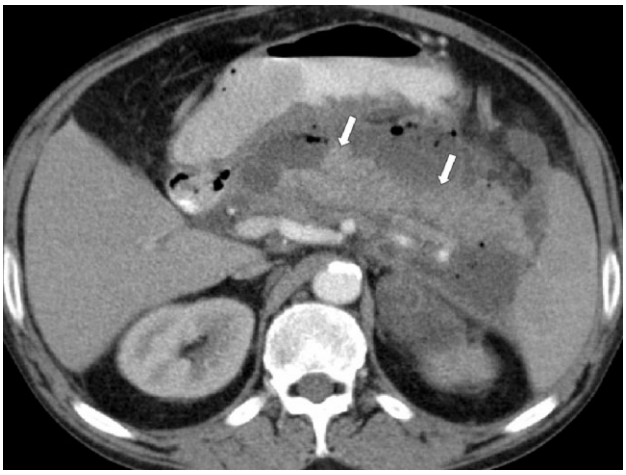
In a recent interobserver agreement study on the Atlanta definitions regarding the various local complications, interobserver agreement was poor: five radiologists agreed on the respective Atlanta definition in only three of 70 collections depicted by contrast-enhanced CT (CECT)[8].

Acute fluid collection

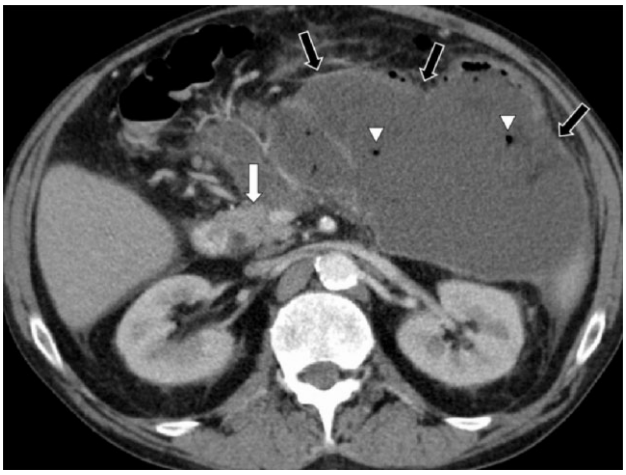
In 64 articles, a definition was given for an 'acute fluid collection'. The following terms were used to describe acute fluid collections: '(peri)pancreatic fluid collections'[75–77], 'peripancreatic effusions'[78], 'extrapancreatic fluid collections'[61,79,80], 'immature pseudocyst'[81,82] and 'exudates'[54]. (Peri)pancreatic fluid collection was also used as an overall descriptive term for all types of collection related to acute pancreatitis[83–85].

In most reports, the differentiation between acute fluid collection and pseudocyst was made after 4 weeks from onset of disease (as proposed by the Atlanta Classification). In eight reports, however, a different time period was used as a criterion for this distinction, varying from 3 weeks[75,86,87] to 6[88,89] and even 8[90] weeks. Moreover, they did not adequately describe whether acute fluid collections consisted of fluid alone or whether they may have contained necrotic debris[85,91,92].

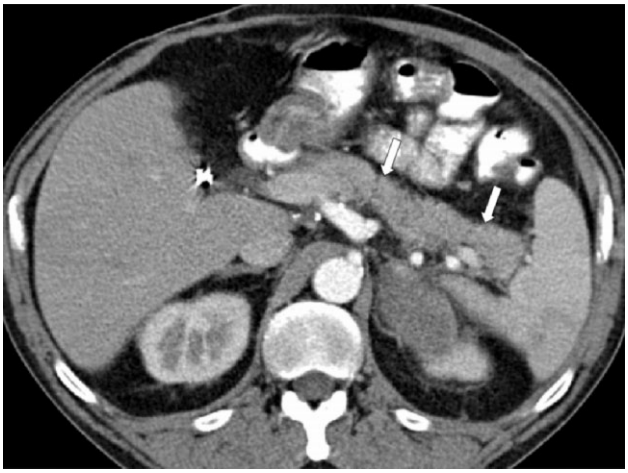
Authors of 17 articles regarded the occurrence of an acute fluid collection to be a local complication and so a



a Normal enhancement of the pancreas



b Large fluid collection with gas bubbles



c Follow-up 6 months after operation

Fig. 1 Contrast-enhanced computed tomography (CT) of a patient with acute pancreatitis 22 days after onset of symptoms, **a** with normal enhancement of the pancreas (white arrows) and **b** surrounded by a large heterogeneous and encapsulated fluid collection (black arrows) with gas bubbles (arrowheads) suggesting secondary infection. Some would call this 'necrotizing pancreatitis', but others would call it 'interstitial pancreatitis' because there is no evidence of pancreatic parenchymal necrosis (only peripancreatic necrosis). A large amount of fat necrosis was debrided during operation. **c** Follow-up CT 6 months after operation reveals a normal enhancing pancreatic parenchyma (white arrows)

sign of 'severe disease'[46,62,93]. However, most others did not include acute fluid collection either in the definition of local complication or in that of severe disease.

Pancreatic necrosis

Of 152 articles that gave a specific definition for 'pancreatic necrosis' or 'necrotizing pancreatitis' (*Fig. 1*), 47 used the Atlanta criterion of more than 30 per cent parenchymal necrosis to define necrotizing pancreatitis[28,61,94]. However, 85 defined necrotizing pancreatitis as any evidence of pancreatic parenchymal necrosis (including less than 30 per cent parenchymal necrosis)[47,95,96]. A third definition of necrotizing pancreatitis, reported in 20 papers, was the appearance of pancreatic necrosis or extrapancreatic necrosis, or both, on CECT (and a serum C-reactive protein value of more than 150 mg/dl)[52,86,97].

In the Atlanta Classification, the definition of pancreatic necrosis requires pancreatic parenchymal non-enhancement on CECT[4]. However, some clinicians questioned whether non-enhancement on CECT meant irreversible damage and necrosis[86,98,99]. For instance, Traverso

and Kozarek[86] defined pancreatic necrosis as devitalized tissue found at operation. This was supported by Takeda and colleagues[100–102], who noted that pancreatic parenchymal perfusion was maintained during intra-arterial angiography, while CECT showed pancreatic non-enhancement. In contrast, several studies demonstrated a good correlation between parenchymal non-enhancement on CECT and the presence of pancreatic necrosis (confirmed at operation)[103–105].

Data on the accuracy of CECT in diagnosing extrapancreatic or peripancreatic fat necrosis are conflicting. Although eight groups claimed that fat necrosis could not be determined reliably by CECT[92,106,107], several studies demonstrated a good correlation between extrapancreatic findings on CECT and the presence of fat necrosis at operation or autopsy[104,108,109].

The Atlanta Classification includes both infected and sterile necrosis within the definition of 'pancreatic necrosis'[4]. Several groups claimed that pancreatic parenchymal necrosis without infection is not a major morbidity risk[110–112]. This was supported by studies showing an uncomplicated course in the presence of necrosis without infection[23,55,56]. Beger and colleagues[81,113] were the first to emphasize that necrosis is a potential nidus for secondary infection occurring in 40–70 per cent of patients. Recent studies confirmed this, demonstrating infected necrosis as the primary cause of late mortality[58,114,115]. However, definitions of 'infected necrosis' were also conflicting. Some authors regarded the presence of parenchymal necrosis as a prerequisite for the diagnosis of infected necrosis[116–118], but others defined infected necrosis as infection that could occur in parenchymal necrosis or peripancreatic fat necrosis (in other words, in the absence of parenchymal necrosis), or both[67,76,119].

Pseudocyst

A specific definition for the term 'pseudocyst' was provided in 87 articles, and all were similar to that of the Atlanta Classification. Some controversies, however, remain. Thirty-eight articles included collections containing both fluid and necrotic debris under the heading of pseudocyst (*Fig. 2*)[120–122]. Yet Baron[123] and others[85,124] have stated that pseudocysts should be devoid of solid necrotic debris. Evidence has shown that therapeutic strategy and outcome differed between collections containing fluid alone and those containing necrosis and fluid[84,125,126]. Bradley[127] considered that mischaracterization of (peri)pancreatic fluid collections as pseudocyst by CECT was an extremely common error in contemporary diagnostic radiology. This mischaracterization has two potentially dangerous consequences: first, by instrumentation of a sterile collection containing both fluid and necrosis, infection may be introduced[6,120,128]; second, a delay in appropriate intervention may occur[33,120,129].

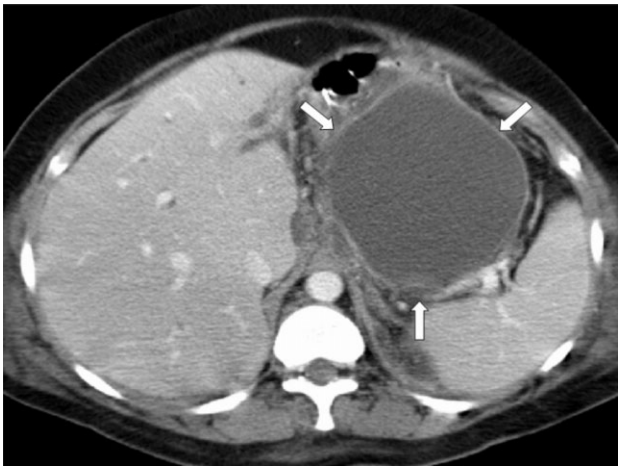


Fig. 2 Contrast-enhanced computed tomography (CT) of a patient with acute pancreatitis 30 days after onset of symptoms. The fluid collection seems to be homogeneous and encapsulated (white arrows) and could be interpreted as a 'pseudocyst' according to the Atlanta Classification. However, at operation the collection was found to contain large amounts of necrotic debris that CT had not shown

The incidence, natural history and options for management differed between acute and chronic pseudocysts. Several authors emphasized that the results of treatment of pancreatic fluid collections in the literature were difficult to interpret, because often no distinction was made between pseudocysts and acute fluid collections, or between pseudocysts that complicated acute and chronic pancreatitis[122,128,130]. Thirty-one original articles on the treatment of pseudocysts were reviewed but only five dealt exclusively with pseudocysts after an episode of acute pancreatitis[89,120,131]. The remaining 26 articles reported results of the treatment of pseudocysts complicating acute and chronic pancreatitis[121,132,133].

Pancreatic abscess

Some 68 articles provided a definition of 'pancreatic abscess', which was generally in line with the original Atlanta definition. Nine original articles after 1993 were identified that reported on the treatment of 'pancreatic abscesses', and the Atlanta definition (collection of pus and virtually no necrotic debris, more than 4 weeks after onset) was strictly applied in three of these[134–136]. The others included collections that contained, in addition to pus, solid necrotic debris[137–139] or that were treated within 4 weeks of onset of disease[140] or after surgery[141,142].

Table 3 Overview of definitions for organ failure and predicted severe acute pancreatitis in guidelines for acute pancreatitis published after 1993

Guideline	Definitions for organ failure	Definitions for predicted severe acute pancreatitis
ACG 1997[157]	Refers to Atlanta Classification 1992	Ranson score ≥ 3 after 48 h APACHE II score > 8 after 48 h
UK 1998[158]	Refers to Atlanta Classification 1992	Ranson/Glasgow ≥ 3 CRP > 210 mg/l (first 4 days) or > 120 mg/l at 1 week APACHE II score ≥ 9 (severe acute pancreatitis) or ≥ 6 (includes all severe cases, but PPV of 50%)
SSAT 1998[159]	Not addressed	Not stated
Santorini 1999[160]	Not addressed	BMI > 30 kg/m^2 Pleural effusion APACHE II score ≥ 6 (at 24 h) APACHE (obesity) score ≥ 6 CRP > 150 mg/l
French 2000[36]	Renal failure: creatinine > 170 μmol/l Shock: systolic BP < 90 mmHg despite fluid replacement Pulmonary insufficiency: $Pa_{O_2} \leq 60$ mmHg on room air Glasgow Coma Score < 13 Platelets < 80 g/l	At admission Age > 80 years BMI > 30 kg/m^2 Chronic renal failure Pre-existing severe illnesses At 24–48 h Presence of organ failure by using simple measures or use of scoring system (e.g. SOFA) Ranson/Imrie score > 3 CECT: CT severity index ≥ 4 (48–72 h) CRP > 150 mg/l *Note: 'The non-specific scores (APACHE II, SAP II, etc) are not recommended by the Jury'*
WCG 2002[35]	SIRS ≥ 1 vital organ dysfunction ARDS Renal failure: increased serum creatinine $> 0{\cdot}5$ mg/dl (44 μmol/l) or 50% above baseline or reduction in calculated creatinine clearance $> 50\%$ or need for dialysis Hypotension: mean arterial pressure < 60 mmHg DIC Acute adrenal insufficiency Acute hepatitis Metabolic encephalopathy Ileus	At admission Age > 70 years Clinical assessment BMI > 30 kg/m^2 Pleural effusion/infiltrates CECT: $> 30\%$ non-enhancement of the pancreas APACHE II score ≥ 8 Presence of organ failure At 24–48 h Clinical assessment Glasgow score (no cut-off value provided) CRP > 150 mg/l Presence of organ failure
IAP 2002[161]	Not addressed	Not stated: surgical guideline
JSAEM 2002[162]	Not addressed	Clinical signs CRP (48 h: no cut-off value provided) BMI (no value provided) CECT: necrosis Scoring system, like JMHW, APACHE II at 24 h or Ranson/Glasgow at 24–48 h: no cut-off values provided Japanese score ≥ 2
Nathens 2004[148]	Refers to the guidelines for intensive care unit admission, published in 1999[163]	Elderly (age not specified) BMI > 30 kg/m^2 Patients requiring ongoing volume resuscitation CECT: $> 30\%$ non-enhancement of the pancreas Clinical assessment *Note: 'Disease-specific scoring systems or severity scores are useful adjuncts to identify patients at high risk of a complication, but should not replace serial clinical assessments. In addition, there is a recommendation against the use of markers such as CRP or procalcitonin to guide clinical decision making or predict clinical course of acute pancreatitis or to triage patients'*

(Continued)

Table 3 (Continued)

Guideline	Definitions for organ failure	Definitions for predicted severe acute pancreatitis
UK 2005[43]	Refers to Atlanta Classification 1992	At admission Clinical assessment BMI > 30 kg/m^2 Pleural effusion APACHE score > 8 At 24–48 h Clinical assessment Glasgow score ≥ 3 APACHE II score > 8 Persistent organ failure for 48 h (especially if multiple and progressive) CRP > 150 mg/l *Note: 'Organ failure present within 1 week, which resolves within 48 h, should not be considered an indicator of a severe attack of acute pancreatitis'*
ACG 2006[6]	Refers to Atlanta classification 1992 *Note: 'Criteria of organ failure will change in the future: gastrointestinal bleeding will undoubtedly be deleted'*	At admission Age > 55 years BMI > 30 kg/m^2 Presence of organ failure Pleural effusion/infiltrates 24–48 h APACHE II score ≥ 8 Serum haematocrit ≥ 44% *Note: 'Ranson signs are no longer advocated, due to a comprehensive evaluation of 110 studies that concluded that Ranson signs provided very poor predictive power of severity of acute pancreatitis'*
JSAEM 2006[164,165]	Pulmonary insufficiency: dyspnoea Shock Central nervous system disorders Bleeding tendency Negative base excess failure: rise of blood urea nitrogen level and creatinine level	Japanese score ≥ 2

ACG, Practice Parameters Committee of the American College of Gastroenterology; APACHE, Acute Physiology And Chronic Health Evaluation; UK, Working Party of the British Society of Gastroenterology, Association of Surgeons of Great Britain and Ireland, Pancreatic Society of Great Britain and Ireland, and Association of Upper GI Surgeons of Great Britain and Ireland; CRP, C-reactive protein; PPV, positive predictive value; SSAT, Society for Surgery of the Alimentary Tract; Santorini, Santorini Consensus Conference; BMI, body mass index; French, French Consensus Conference on Acute Pancreatitis; BP, blood pressure; Pa_{O_2}, arterial partial pressure of oxygen; SOFA, Sequential Organ Failure Assessment; CECT, contrast-enhanced computed tomography; SAP, Simplified Acute Physiology; WCG, World Congress of Gastroenterology; SIRS, systemic inflammatory response syndrome; ARDS, adult respiratory distress syndrome; DIC, disseminated intravascular coagulation; IAP, International Association of Pancreatology; JSAEM, Japanese Society of Emergency Abdominal Medicine; JMHW, Japanese Ministry of Health and Welfare; Nathens, Consensus Statement regarding the management of the critically ill patient with severe acute pancreatitis.

The diagnosis of pancreatic abscess on CECT is also controversial. In ten articles, the 'air bubble' phenomenon was considered 'diagnostic of a pancreatic abscess'[93,143,144]. In 31, however, gas bubbles in a heterogeneous collection on CT were regarded as highly indicative of infected pancreatic necrosis (*Fig. 3*)[61,67,145]. Varying hypotheses exist on the aetiology of pancreatic abscess. Some authors considered 'postacute pseudocysts' and pancreatic abscesses as late consequences of necrotizing pancreatitis[146–148]. In contrast, others maintained that pancreatic abscesses occurred exclusively in interstitial pancreatitis with a normal enhancing pancreas on CECT[117,149,150].

Apart from 'infection of a pseudocyst', several authors hypothesized that pancreatic abscesses evolved from progressive liquefaction of necrotic pancreatic and peripancreatic tissues, in time resulting in complete liquefaction[76,123,151]. According to the Atlanta Classification, most pancreatic abscesses arise at least 4 weeks after onset of symptoms[4], although others diagnose 'pancreatic abscesses' after 1[50,152], 2[153,154] or 3[86,146,147] weeks. Interestingly, when performing operative necrosectomy

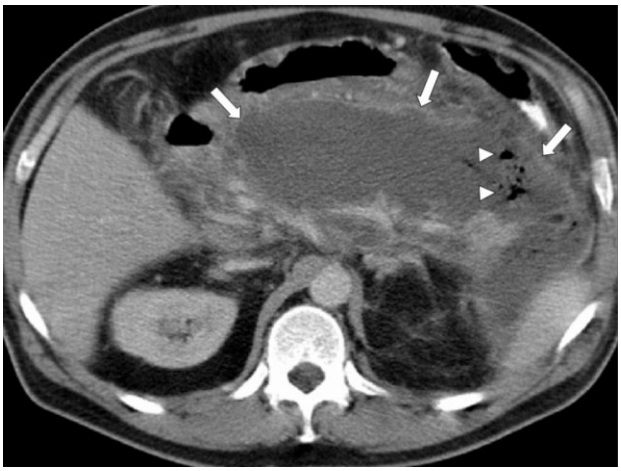


Fig. 3 Contrast-enhanced computed tomography of a patient with acute pancreatitis 36 days after onset of symptoms. The body and tail of the pancreas are largely non-enhancing. Adjacent to the pancreatic bed is a large collection with predominately fluid-like attenuation (white arrows). Because of the gas bubbles (arrowheads), some would call this a 'pancreatic abscess' but others would call it 'infected pancreatic necrosis'

several months after the onset of severe acute pancreatitis, Morgan and colleagues[10], Howard and Wagner[155] and others[156] observed different degrees of liquefaction of necrotic tissue. Several authors acknowledged this evolving process, and they postulated that a collection may represent a transitional entity from (infected) pancreatic necrosis to an (infected) pseudocyst or pancreatic abscess, as they encountered both pus and necrotic debris in these (infected) collections[7,12,139].

Guidelines

The greatest discrepancies in the 12 guidelines[6,35,36,43,148,157–165] on acute pancreatitis related to the definitions of organ failure and those of predicted severe disease. These are summarized in *Table 3*.

Discussion

The present review has demonstrated that the Atlanta definitions of severity and local complications of acute pancreatitis are being used inconsistently, and that several components of the classification have received considerable criticism. By providing definitions, the result of consensus by over 40 experts based on the data available in 1992, the Atlanta symposium improved the management of acute pancreatitis and clinical research relating to the condition. However, the past 20 years have seen not only new insights in pathophysiology and therapeutic strategies but also improved imaging techniques. Clearly, the time has come to revise the classification of acute pancreatitis.

The various predictive scoring systems have not improved substantially since the Atlanta symposium. They are only moderately accurate in predicting severe disease in an individual patient. As McKay and Imrie[166] have noted, predictive systems were developed initially to allocate patients within clinical trials and not to assess severity in an individual. Defining severity based on the presence or absence of organ failure also has its limitations. It is increasingly recognized that persistent organ failure (for more than 48 h) is the most important determinant of morbidity and mortality, which are predominantly related to the number of organ systems failing, the degree of dysfunction of the organs involved and the duration of organ failure.

The definition of necrotizing pancreatitis is controversial because it incorporates both sterile and infected necrosis, and covers both pancreatic parenchymal necrosis and peripancreatic fat necrosis. Interpretations of pseudocyst and pancreatic abscess vary widely because necrotic debris within these collections is often not accounted for. This might be explained by the incapacity of CECT to detect necrotic debris in collections predominantly containing fluid, and its incapacity to discriminate between sterile and infected collections[7,10,12,92,167]. Although magnetic resonance imaging (MRI) and (endoscopic) ultrasonography may be of additional value in classifying these collections[10,168,169], their applicability in severely ill patients has been questioned[92,170].

Although the Atlanta Classification incorporates a pathological and morphological description of different local complications, it does not provide exact radiological criteria for each. The recently demonstrated poor interobserver agreement on the Atlanta Classification of local complications[8] highlights the need for new descriptive morphological terms to describe CECT findings. The existing radiological grading system, the CT severity index, is a numerical scoring system that combines quantification of extrapancreatic changes with the extent of pancreatic necrosis[26]. Although the CT severity index has clear prognostic value with regard to morbidity and mortality[26,171–174], it does not characterize the local complications of acute pancreatitis.

Much of the persisting controversy over the natural course of (peri)pancreatic collections is due to a lack of prospective data from large patient series. The authors of this review, therefore, advocate a collaborative international study to clarify pathophysiology, natural course and optimal management of (peri)pancreatic

collections. The present review has aimed to give an overview of the controversies regarding the Atlanta Classification in the literature. There are virtually no studies addressing the validation of the definitions proposed by the Atlanta Classification. Consequently, hardly any original data on this topic are available to analyse. This review, therefore, has merely categorized applications and interpretations of the Atlanta definitions. Correct terminology and standardized definitions are important for adequate communication in clinical practice and for comparing interinstitutional data for clinical research. The continuing failure to use standardized definitions for predicted and actual severe acute pancreatitis, organ failure and the local complications, and the heterogeneity of inclusion criteria of patients in clinical trials, have hampered the progress of evidence-based recommendations. This review has identified many studies that have improved insight into the natural course of the disease. These new insights should be used to design a new classification.

The authors propose the following recommendations for revision of the classification of acute pancreatitis. First, the diagnosis should incorporate two of the following three items: upper abdominal pain, amylase and/or lipase levels at least three times the upper limit of normal (as this cut-off is used most frequently in the literature), and CT or MRI findings compatible with acute pancreatitis. Second, persistent organ failure (for at least 48 h) should have an important role in defining severity of acute pancreatitis. Third, it should be decided which predictive scoring system(s), including cut-off value, should be used to define predicted severe acute pancreatitis, based on a systematic review of the available data. Fourth, future studies should always make a clear distinction between predicted severe and actual severe disease, with *a posteriori* validation of the disease severity. Fifth, a systematic review should demonstrate which organ failure scoring system should be used, and definitions for organ failure should take into account the number of organ systems failing, the duration (less or more than 48 h) of organ failure, and the need for specific therapy (such as inotropic or vasopressor agents, mechanical ventilation and dialysis). Sixth, peripancreatic fat necrosis without pancreatic parenchymal necrosis should be regarded either as a separate entity or as necrotizing pancreatitis. Seventh, infected necrosis should be regarded as a separate entity. Eighth, a term should be appointed for encapsulated collections containing both fluid and necrotic debris. Ninth, in order to diagnose a collection that contains fluid only (such as pseudocyst), MRI or (endoscopic) ultrasonography should be performed first to exclude necrotic debris in the collection. Tenth, a new set of descriptive morphological terms should be designed to describe local complications on CT.

Such a new classification system should be evaluated in high-quality interobserver and prospective clinical studies. Adjustments should be made every few years, based on new data. Most importantly, clinicians and radiologists worldwide should comply with the new classification in clinical practice and research. Progress in the field of acute pancreatitis is hampered greatly when various author groups use their own idiosyncratic definitions. When journal referees are requested to peer-review manuscripts, they should pay special attention to the correct use of definitions as defined by a new classification.

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