The diagnostic criteria for disseminated intravascular coagulation (DIC) have been proposed by Colman (1), the Japanese Ministry Health and Welfare (JMHW) (2), the International Society on Thrombosis and Hemostasis (ISTH) (3) and the Japanese Association for Acute Medicine (JAAM) (4). According to these diagnostic criteria, global coagulation tests such as prothrombin time, platelet count, fibrinogen and fibrin and fibrinogen degradation products or D-dimer are mainly used in scoring for haemostatic abnormalities. The sensitivity of the JAAM DIC diagnostic criteria is highest among the four criteria scores, but the specificity for DIC is not clear. The ISTH overt DIC diagnostic criteria are not very sensitive for DIC and the establishment of non-overt DIC diagnostic criteria is under evaluation (5).

Recent clinical trials for severe sepsis (6–8) indicated that the mortality in severe sepsis is about 35–45%, and it is higher in patients associated with DIC than in those without. The frequency of DIC in severe sepsis was 40.7% in the KyberSept trial (antithrombin) (6) and 22.4% in the PROWESS study (recombinant activated protein C) (7). DIC is frequently associated with sepsis and with poor outcome in sepsis (8). Therefore, an early diagnosis and treatment of DIC is necessary to eventually prevent a fatal outcome in septic DIC.

In the current issue of Thrombosis and Haemostasis, S. Kushimoto and his study group compared the outcome, organ failure and haemostatic abnormalities in DIC patients with sepsis and those with trauma diagnosed by JAAM between sepsis and trauma (9). Patients with trauma are usually admitted to an intensive care unit and are frequently associated with DIC, like patients with sepsis. The 28-day mortality rate is significantly higher in sepsis patients than in trauma patients (9). Coagulation disorders including DIC are well known complications in patients with trauma, especially head trauma. In another prospective study (10) of head trauma, the outcome was poorer in patients with DIC than those without DIC. Sepsis patients had a lower platelet count, higher prothrombin time ratio, higher Sequential Organ Failure Assessment score in comparison to trauma patients. On Day 3, a significantly higher percentage of trauma patients showed improvement of DIC in comparison to sepsis patients (9).

Both DIC due to trauma and sepsis have sometimes been considered to have similar pathophysiology but the mechanism of DIC due to trauma might be different from that due to sepsis. The DIC subcommittee of the Scientific Standardized Committee (SSC) of the ISTH emphasized the vascular endothelial cell injury and inflammatory response in sepsis as the definition of DIC (3, 11). DIC occurs acutely after trauma when the brain, fat, amniotic fluid, or other strong thromboplastins enter the circulation. It occurs subacutely when endothelial inflammation or failure reduces clearing of activated coagulation factors allowing microthrombi to cause secondary injury and to exacerbate DIC (12). More than 50% of the JAAM DIC patients with sepsis who died within 28 days could not be detected by the ISTH DIC criteria during the initial three days, thus suggesting that the ISTH overt-DIC diagnostic criteria might not be sufficiently sensitive for early DIC due to sepsis. The establishment of non-overt DIC diagnostic criteria is also required.

The findings by Kushimoto et al. (9) suggest that coagulation abnormalities, organ dysfunction and the outcome of DIC differ between patients with sepsis and trauma.
References