Avoidable, Delayed and Under or Overtransfusion (ADU)

FIGURES FROM THE ANNUAL SHOT REPORTS 2019-2025

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Figure 12.1: Delayed transfusions by year 2011-2025

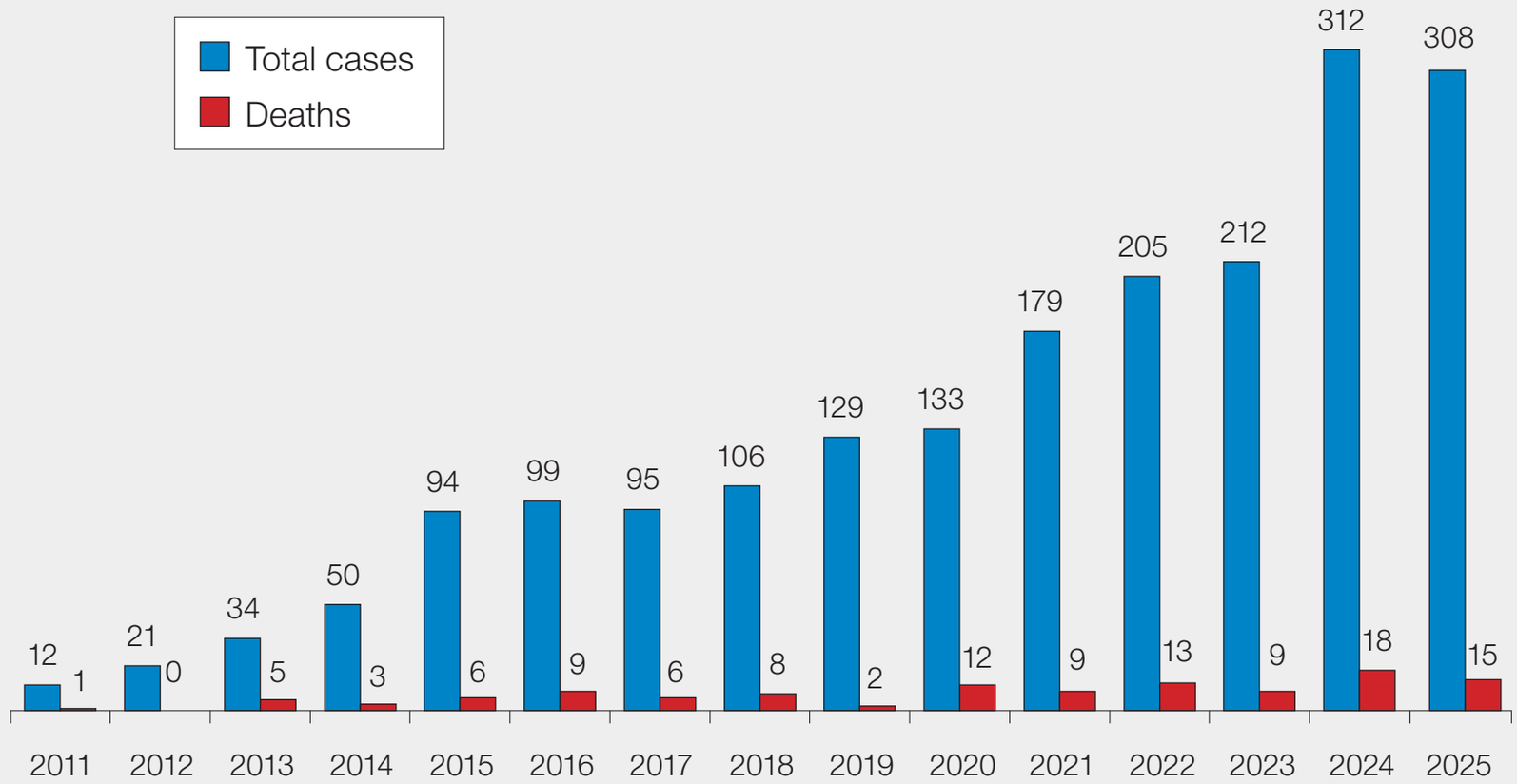


Figure 12.2: Reason for delays in blood transfusion and location of errors associated with Blood Services in 2025 (n=18)

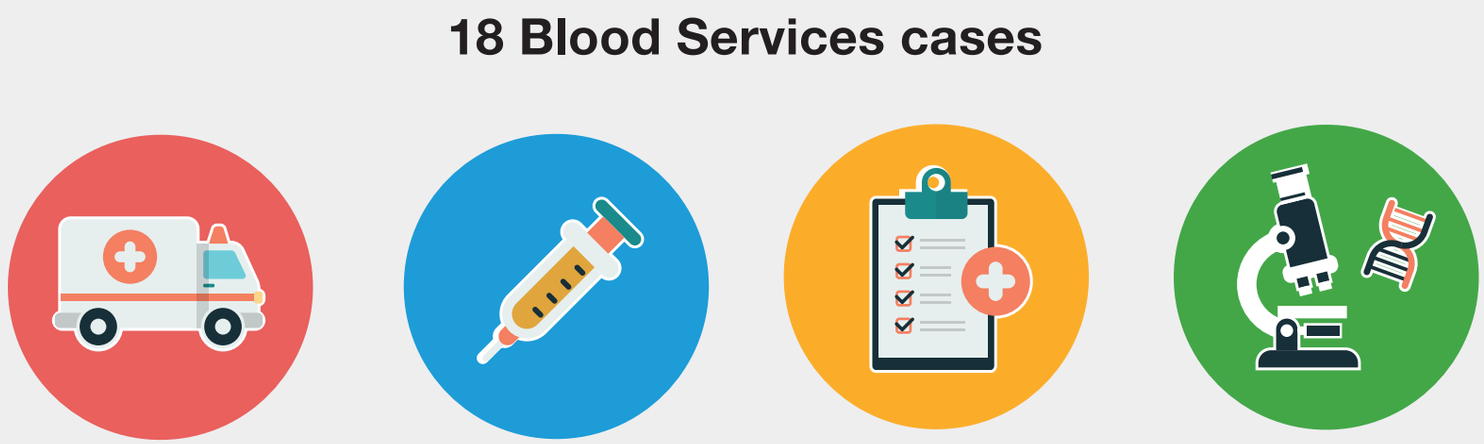


Figure 12.3: Delays associated with MHP 2016-2025, including the proportion of reports resulting in mortality or major morbidity

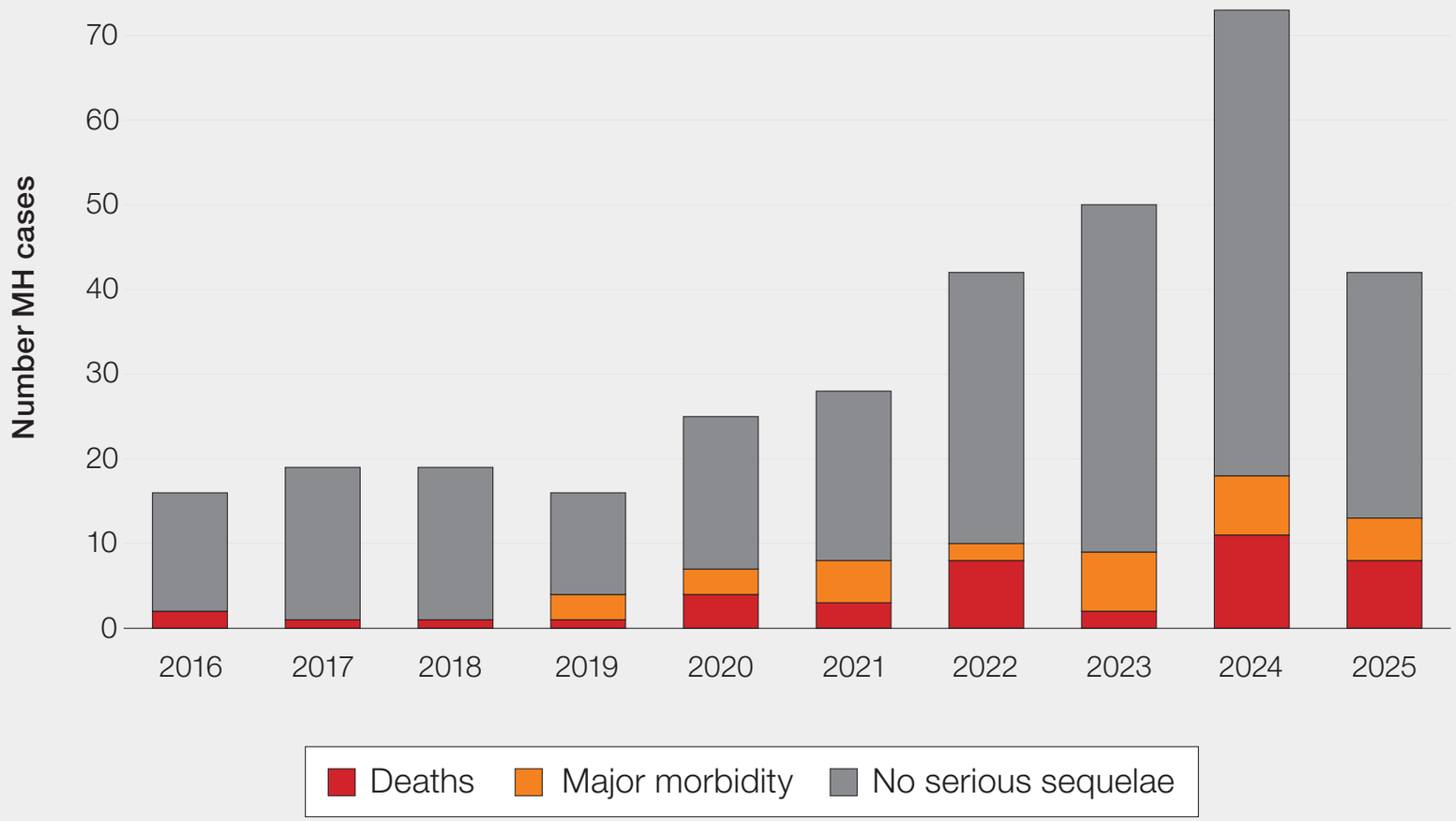


Figure 12.4: Common themes identified that contributed to delays during MH in 2025

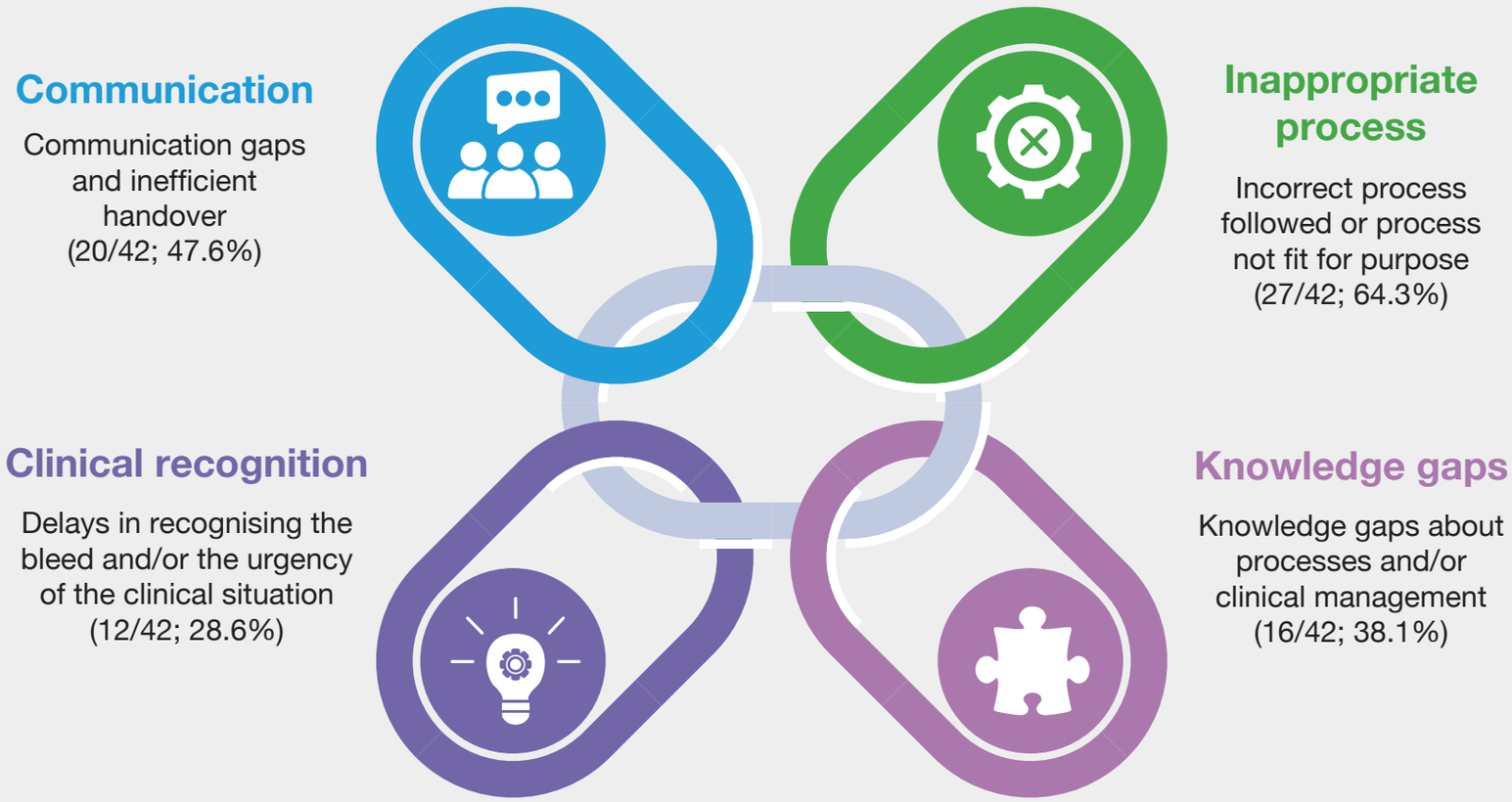


Figure 13.1: Errors during the transfusion pathway resulting in avoidable use of emergency group O (n=61)

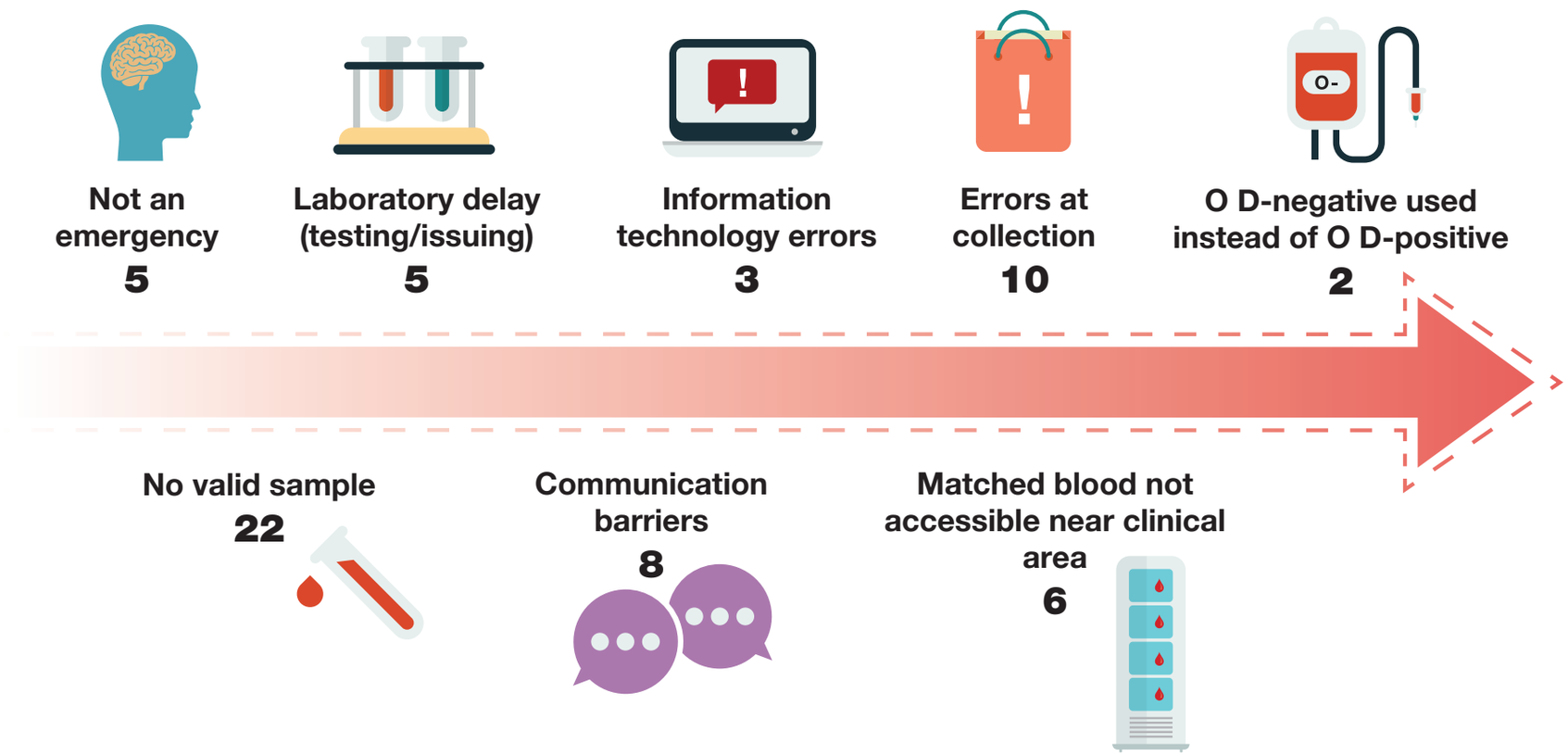


Figure 15.1: Points of delay in the PCC care pathway, 2025

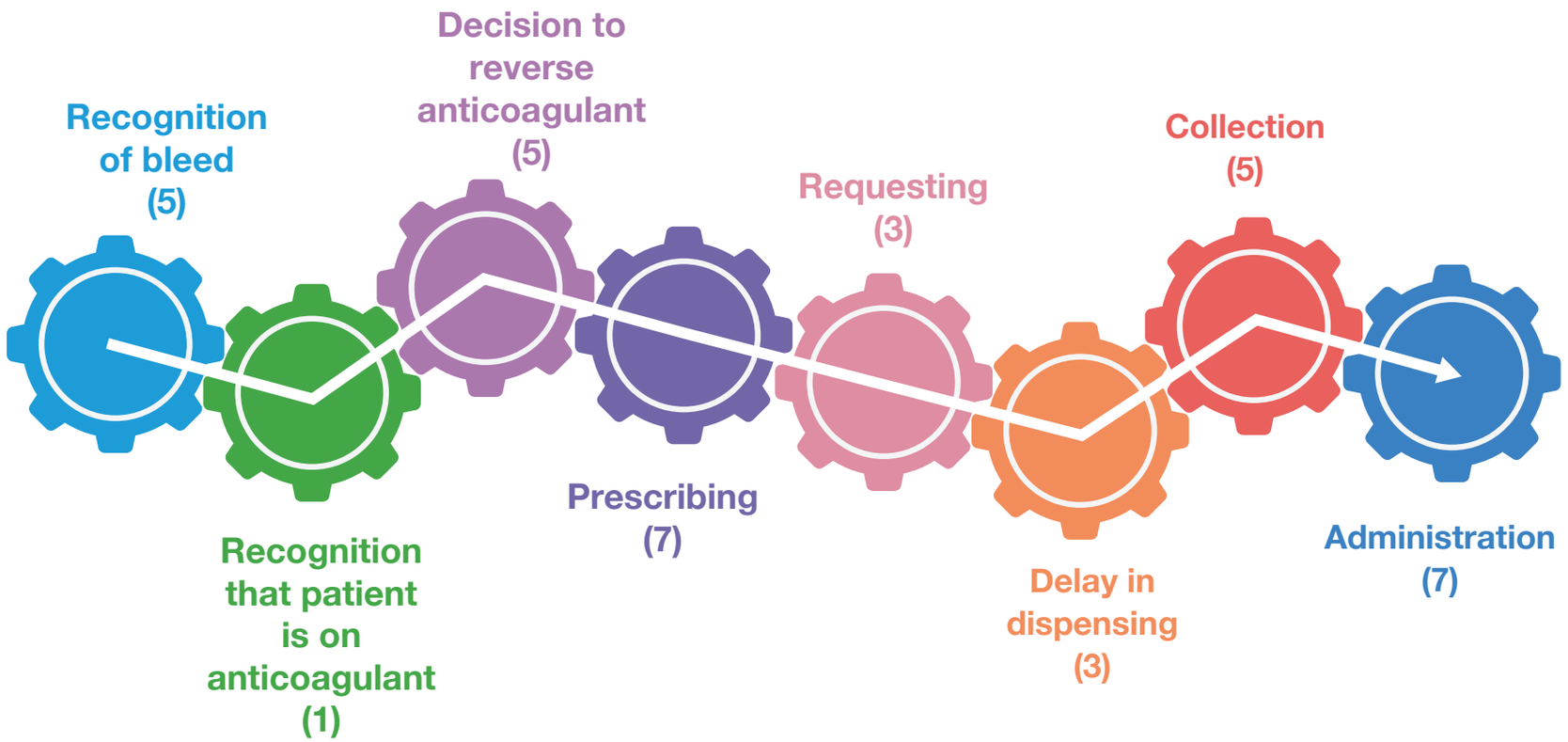


Figure 11.1: Delayed transfusions by year 2011-2024

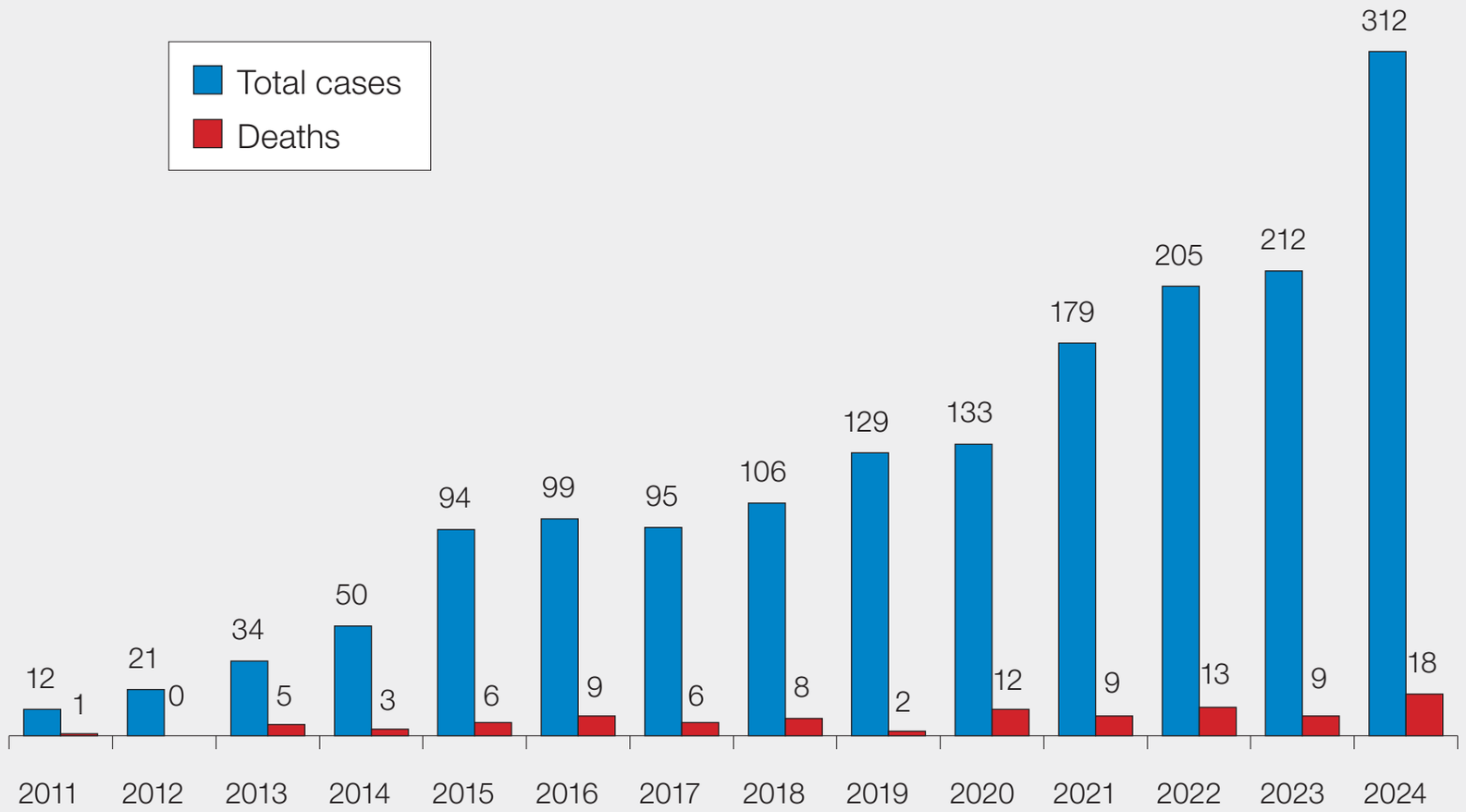


Figure 11.2: Transfusion process step where laboratory errors occurred resulting in transfusion delays in 2024 (n=120)

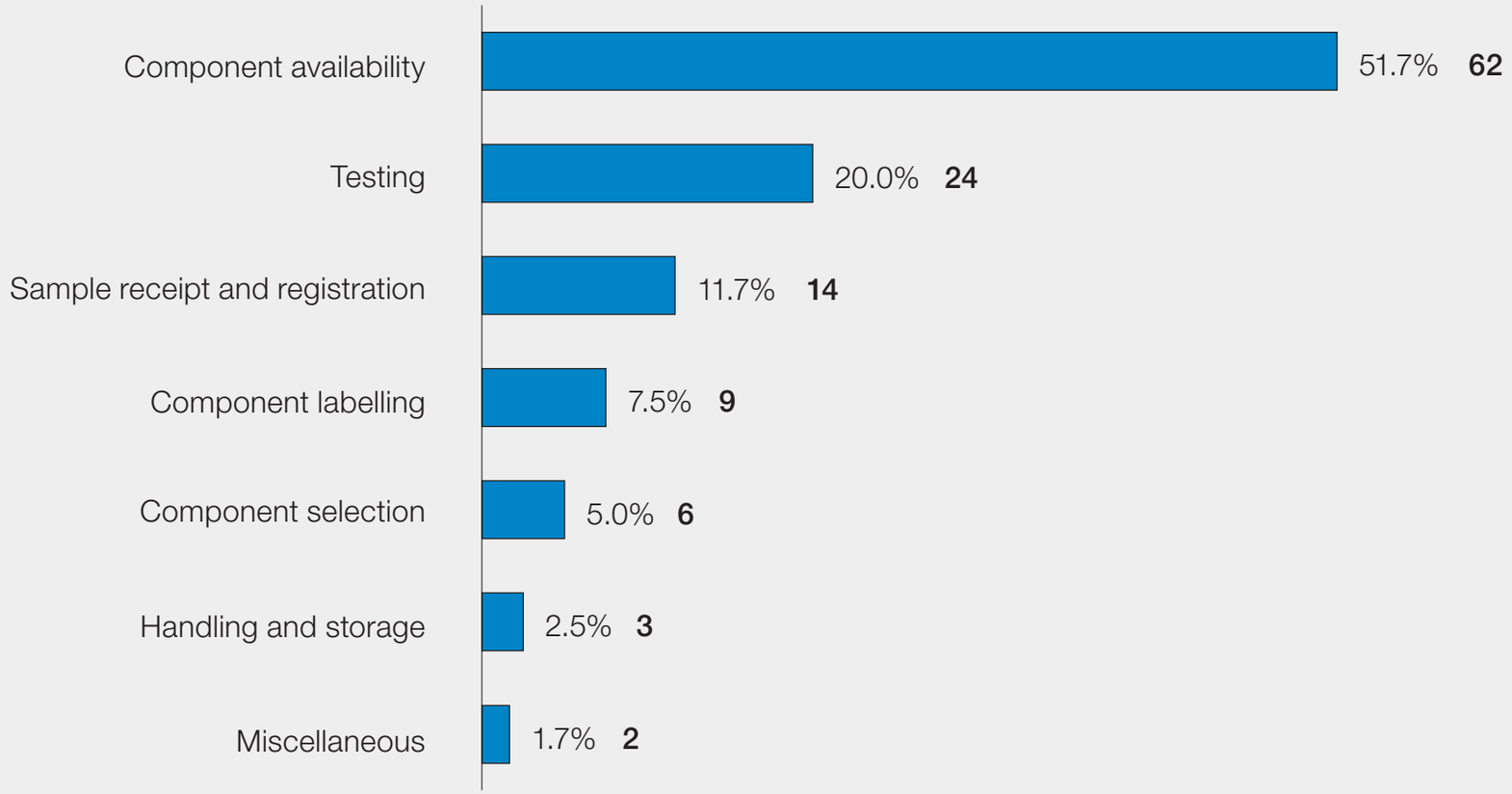


Figure 11.3: Trend in Blood Service-related errors 2019-2024

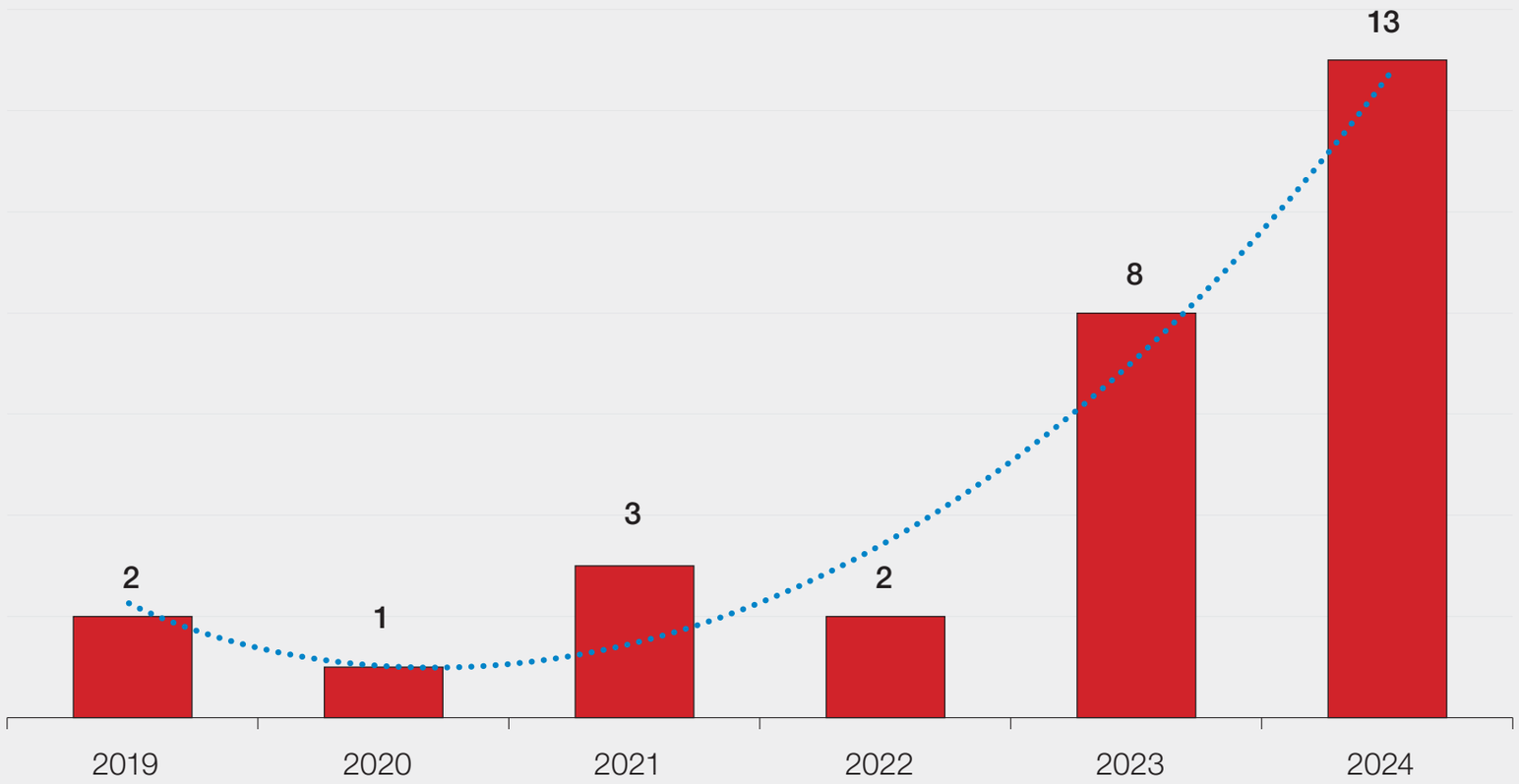


Figure 11.4: Delays associated with MHP 2016-2024

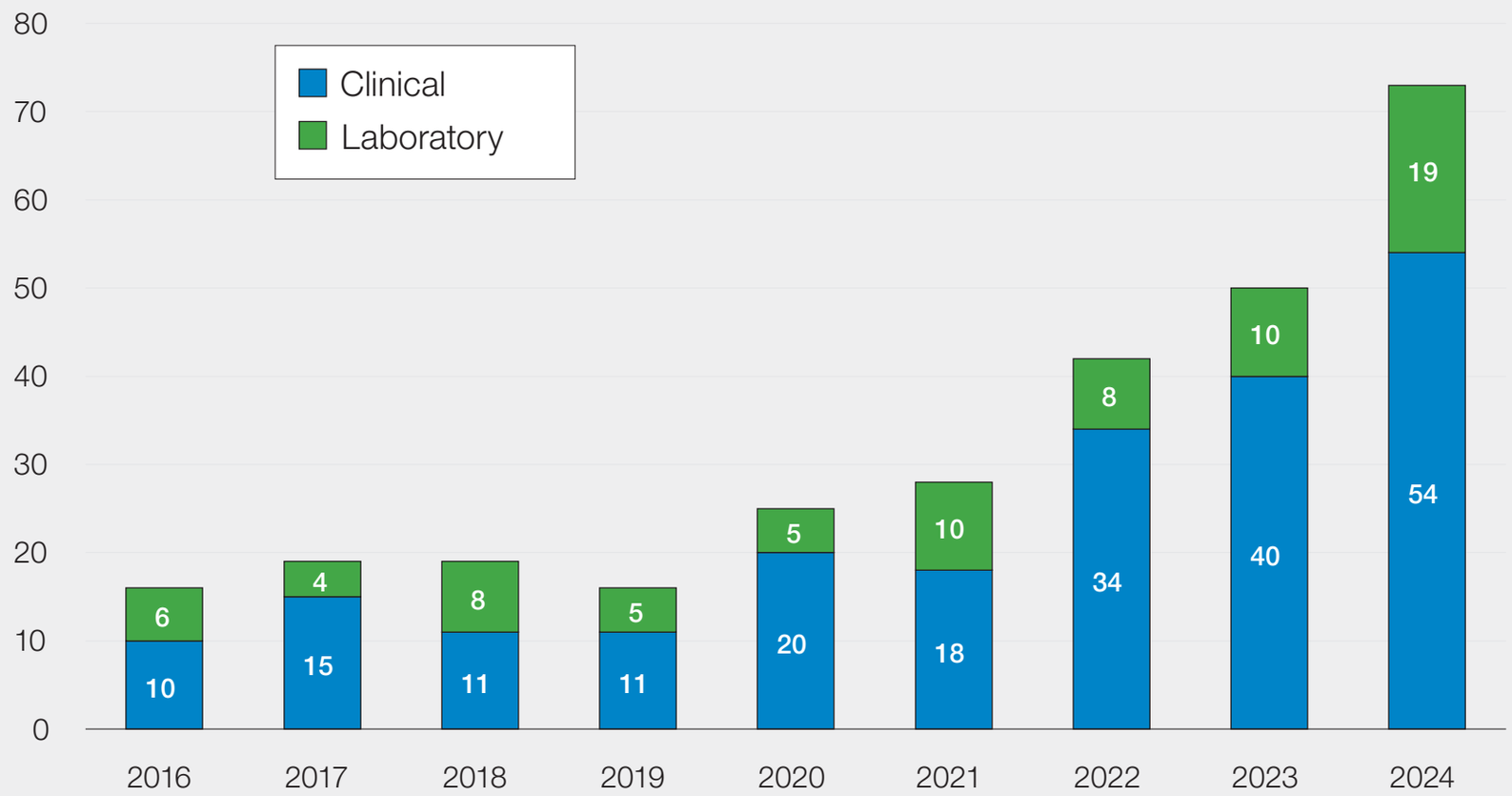
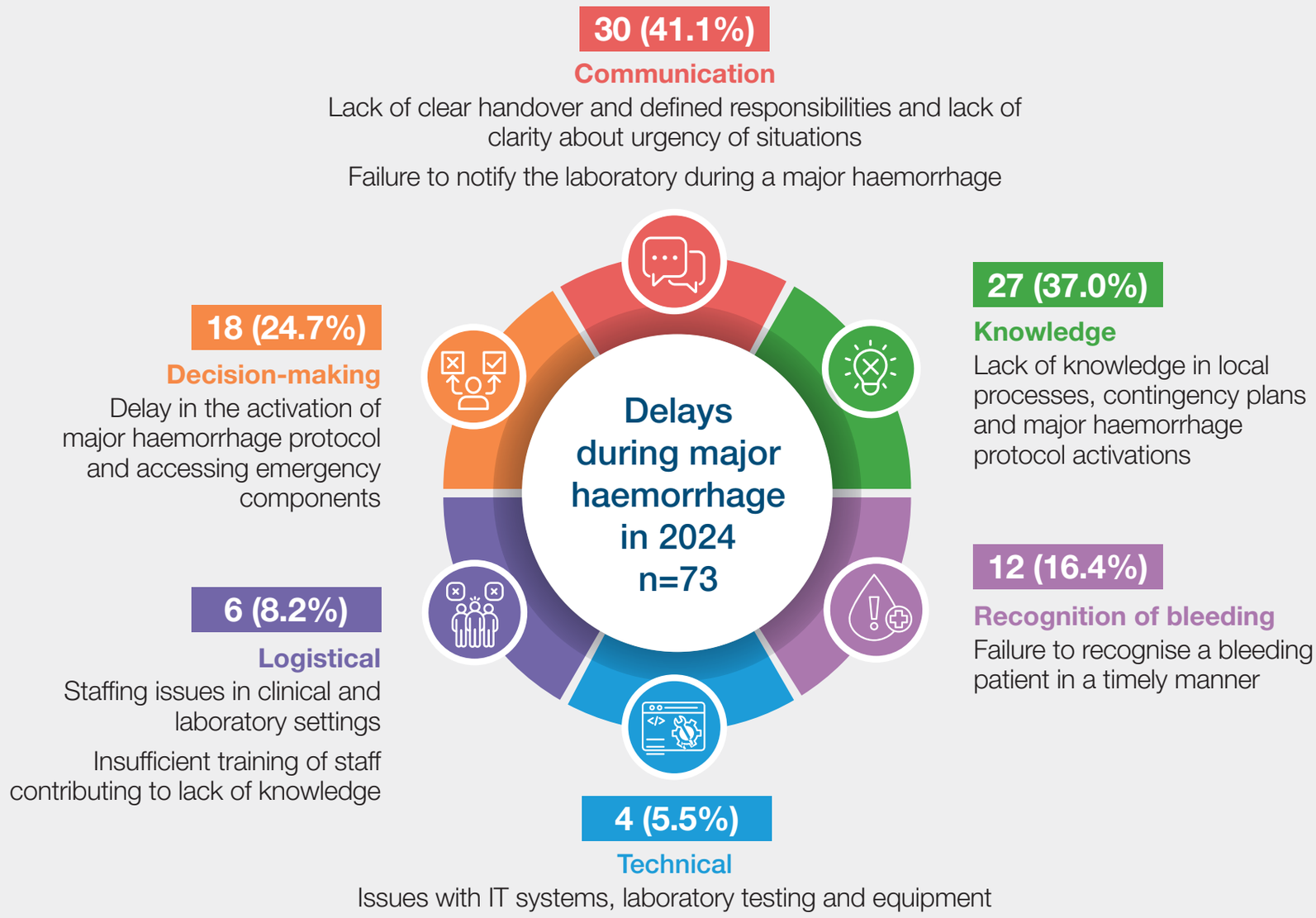
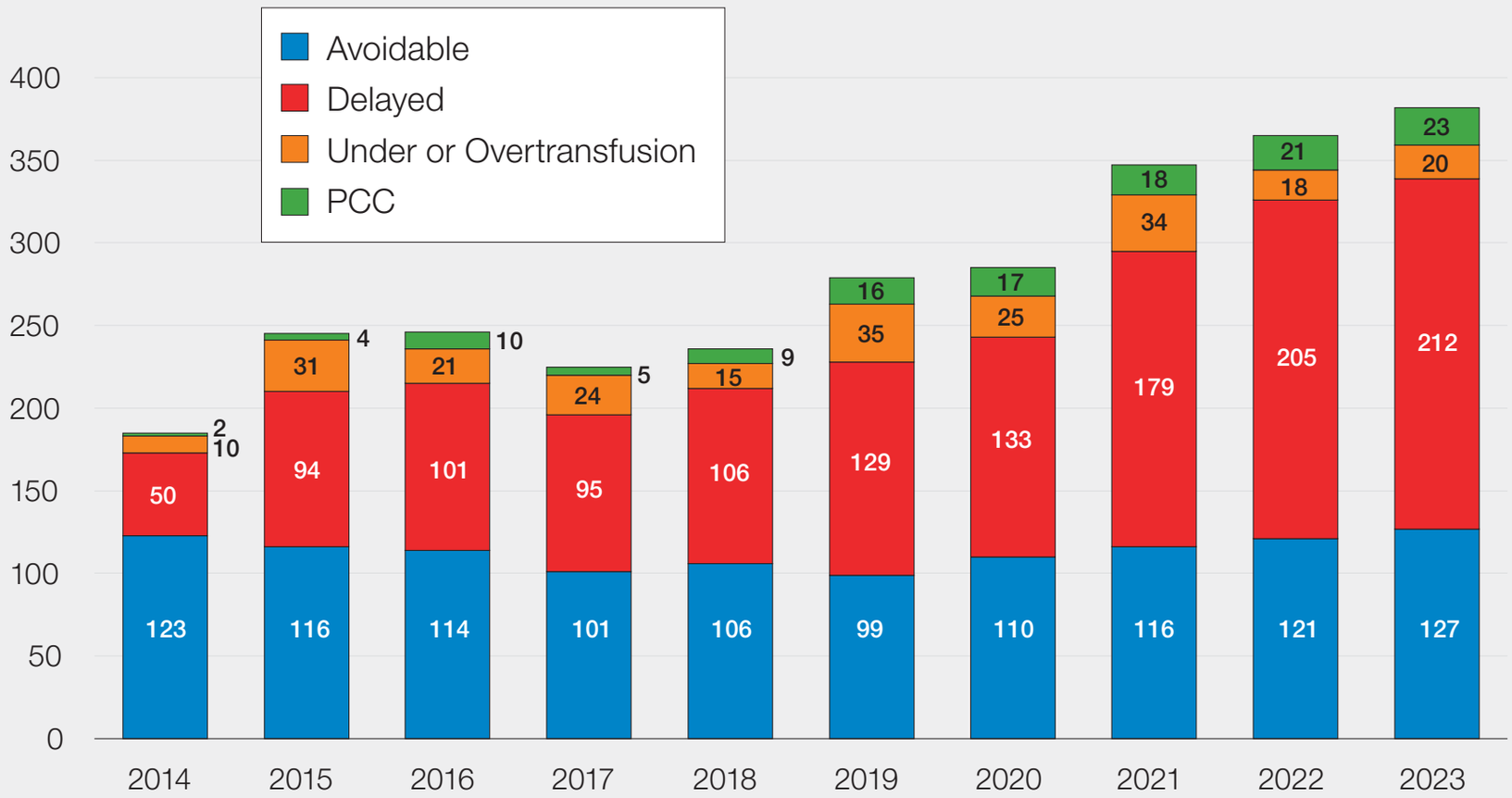


Figure 11.5: An image depicting the multiple contributing factors that resulted in delays during major haemorrhage in 2024 (n=73)



IT=information technology; MH=major haemorrhage; MHP=major haemorrhage protocol

Figure 12.1: ADU reports by category 2014-2023



PCC=prothrombin complex concentrates

Figure 12a.1: Delayed transfusions by year 2011-2023

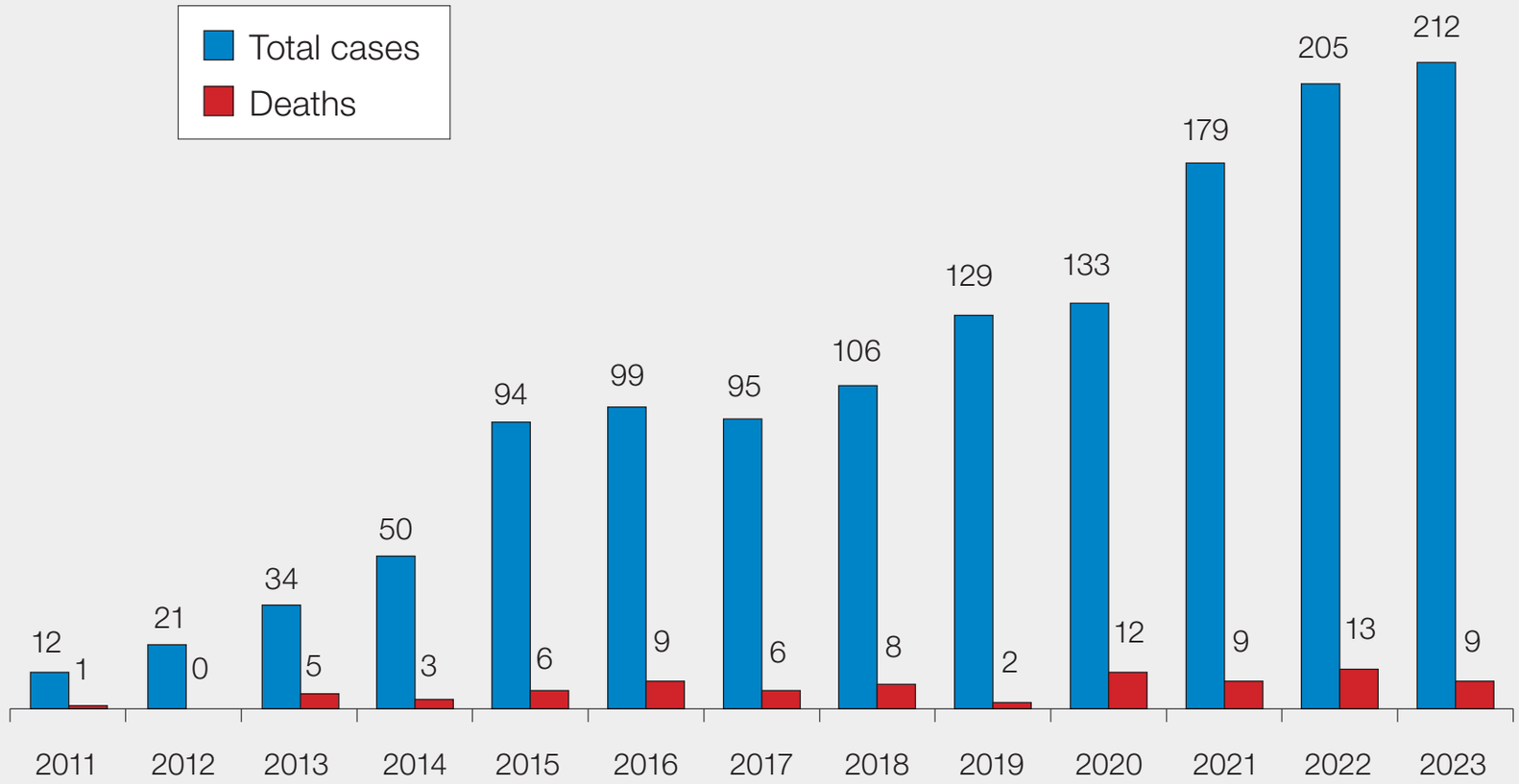


Figure 12a.2: Number of delayed transfusions associated with MHP 2016-2023

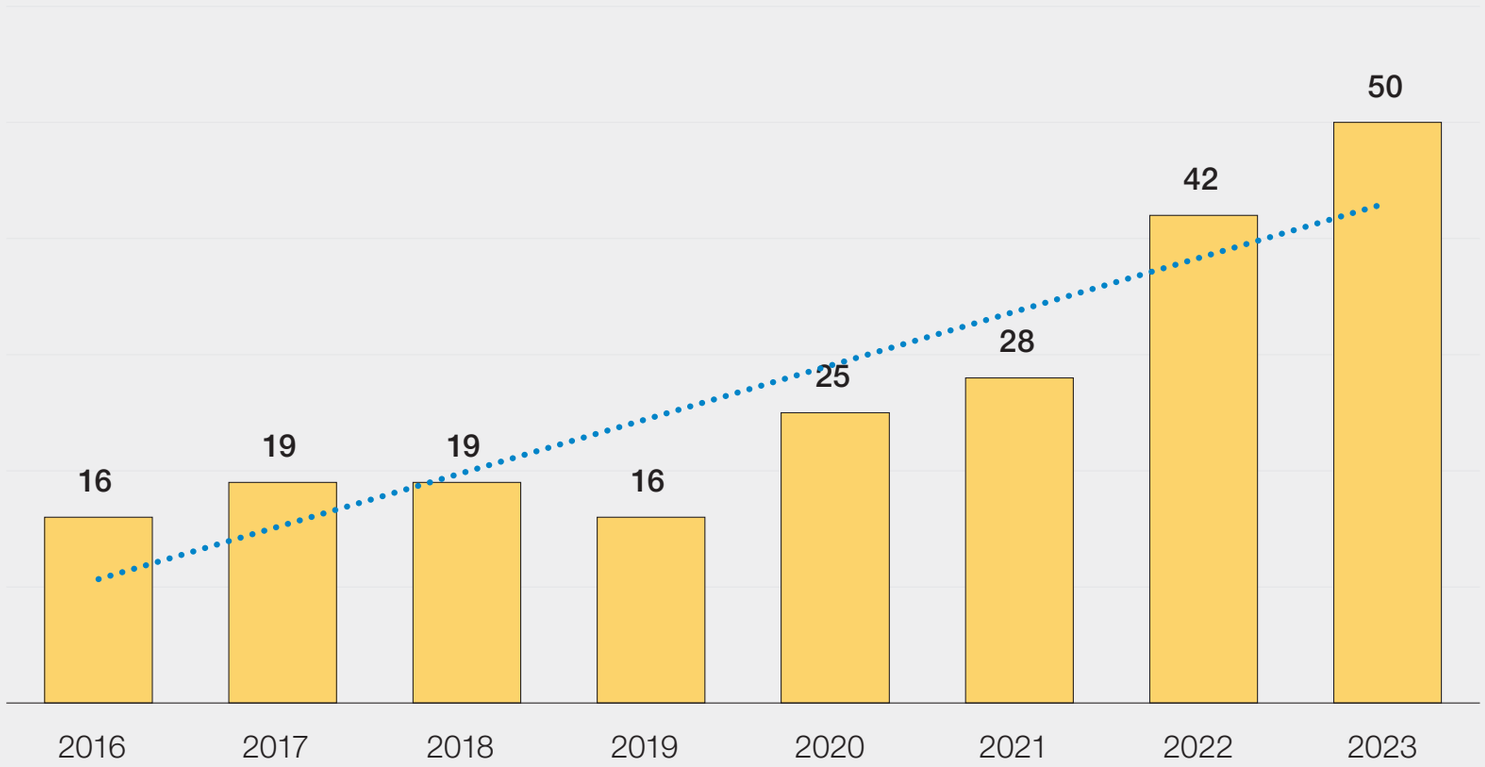


Figure 12a.3: Key factors contributing to delayed transfusions in major haemorrhage in 2023 (n=50)

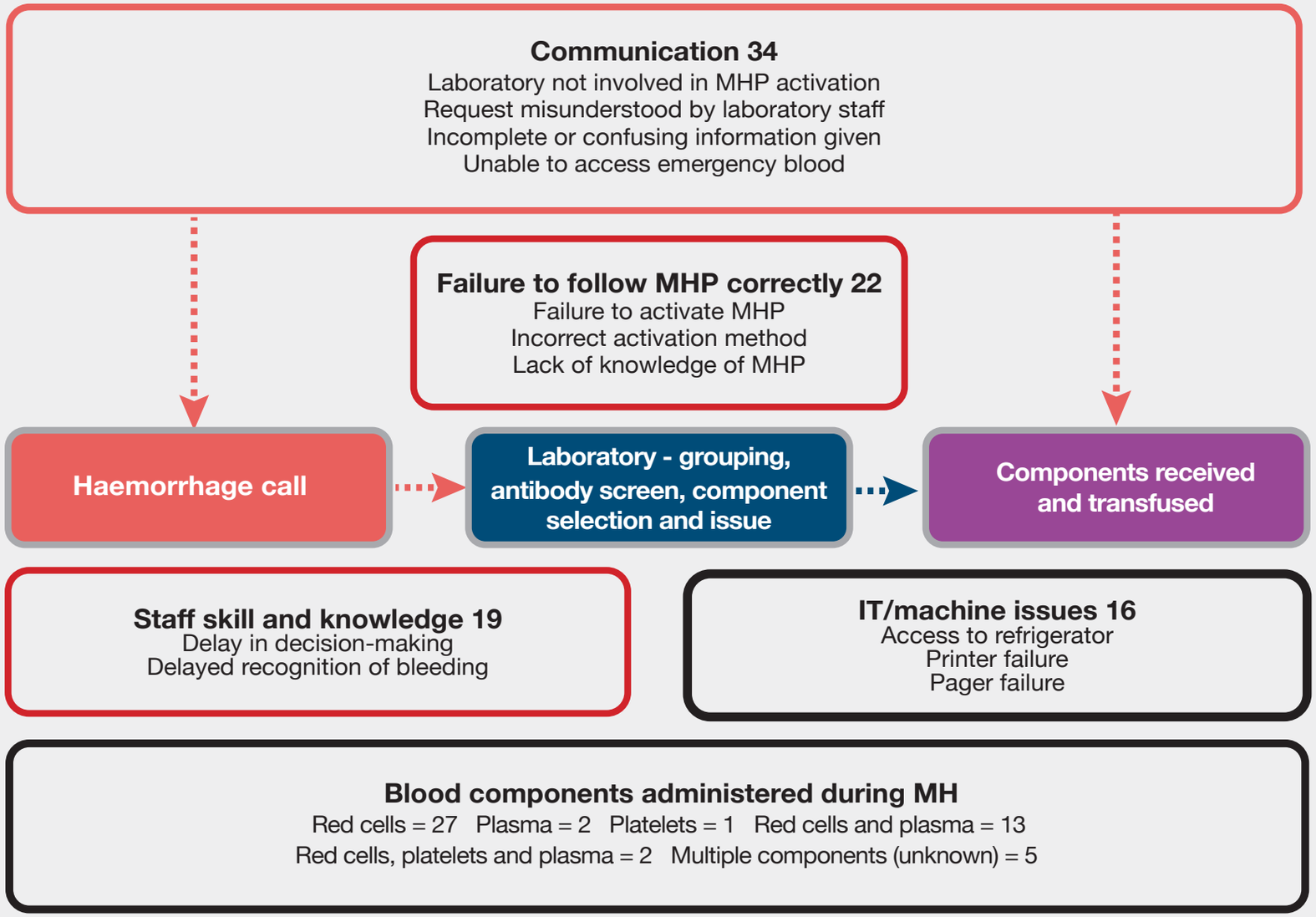


Figure 11a.1: Delayed transfusions by year 2011 to 2022

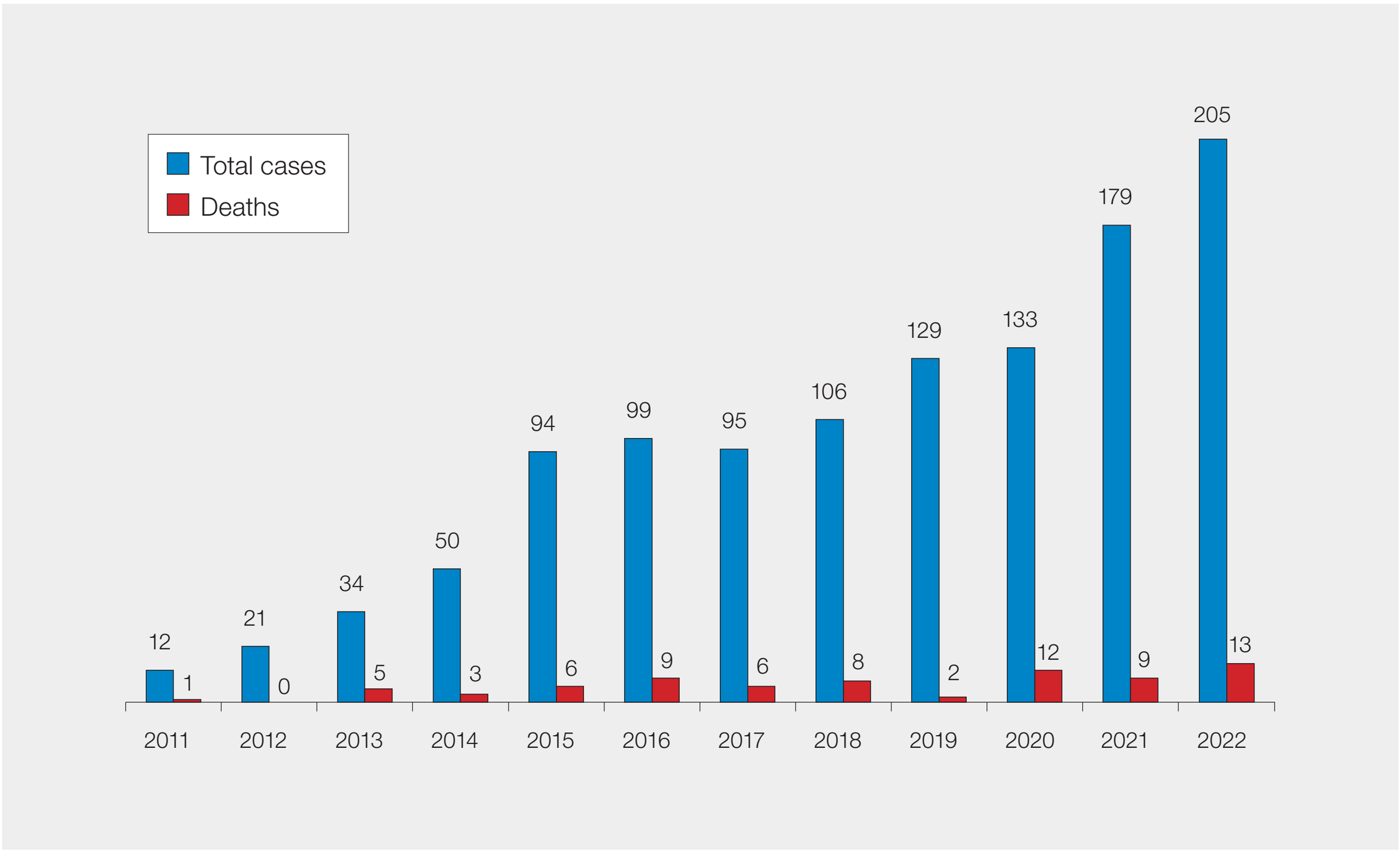


Figure 11a.2: Primary causes of delayed transfusions in 2022 (n=205)

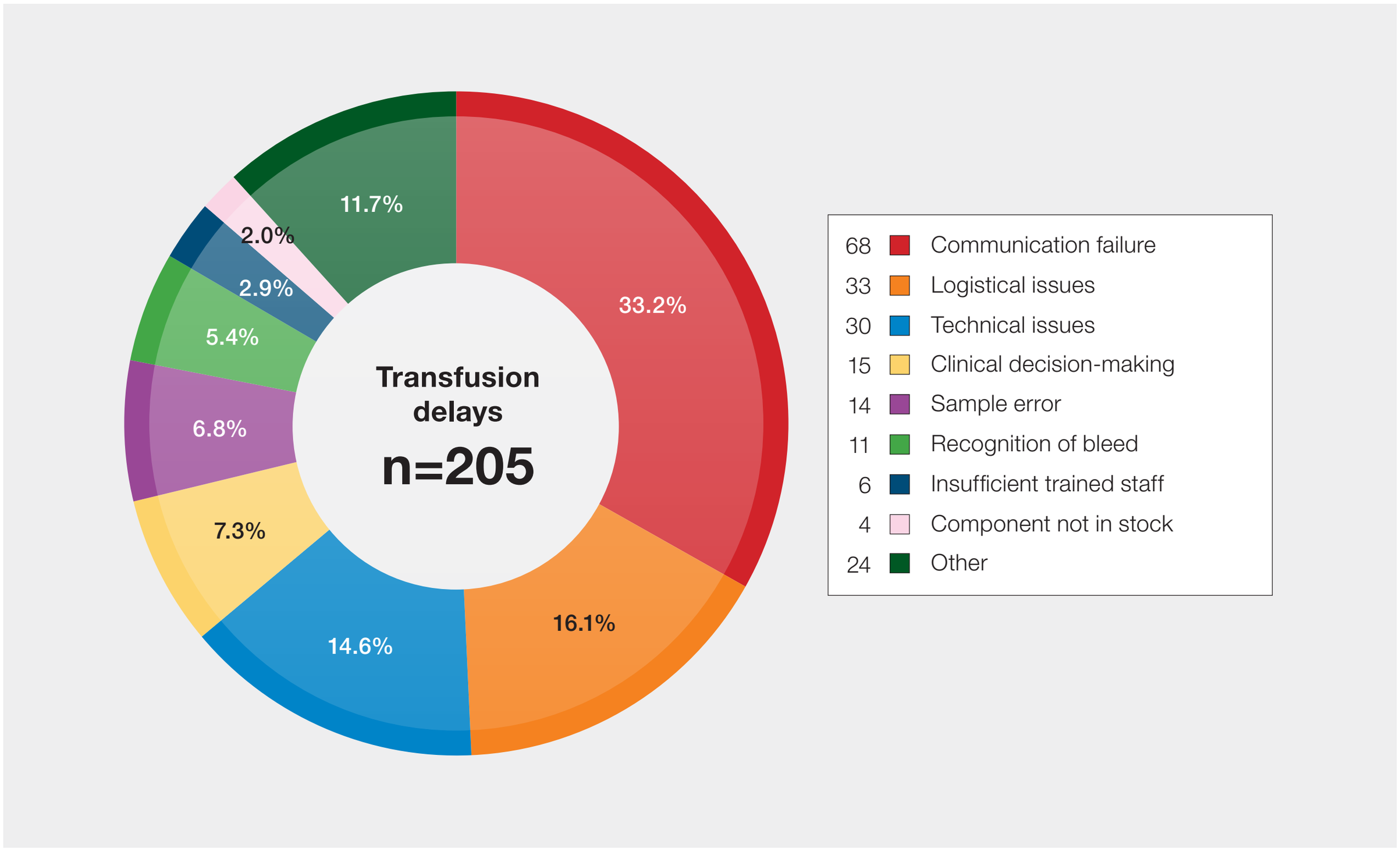
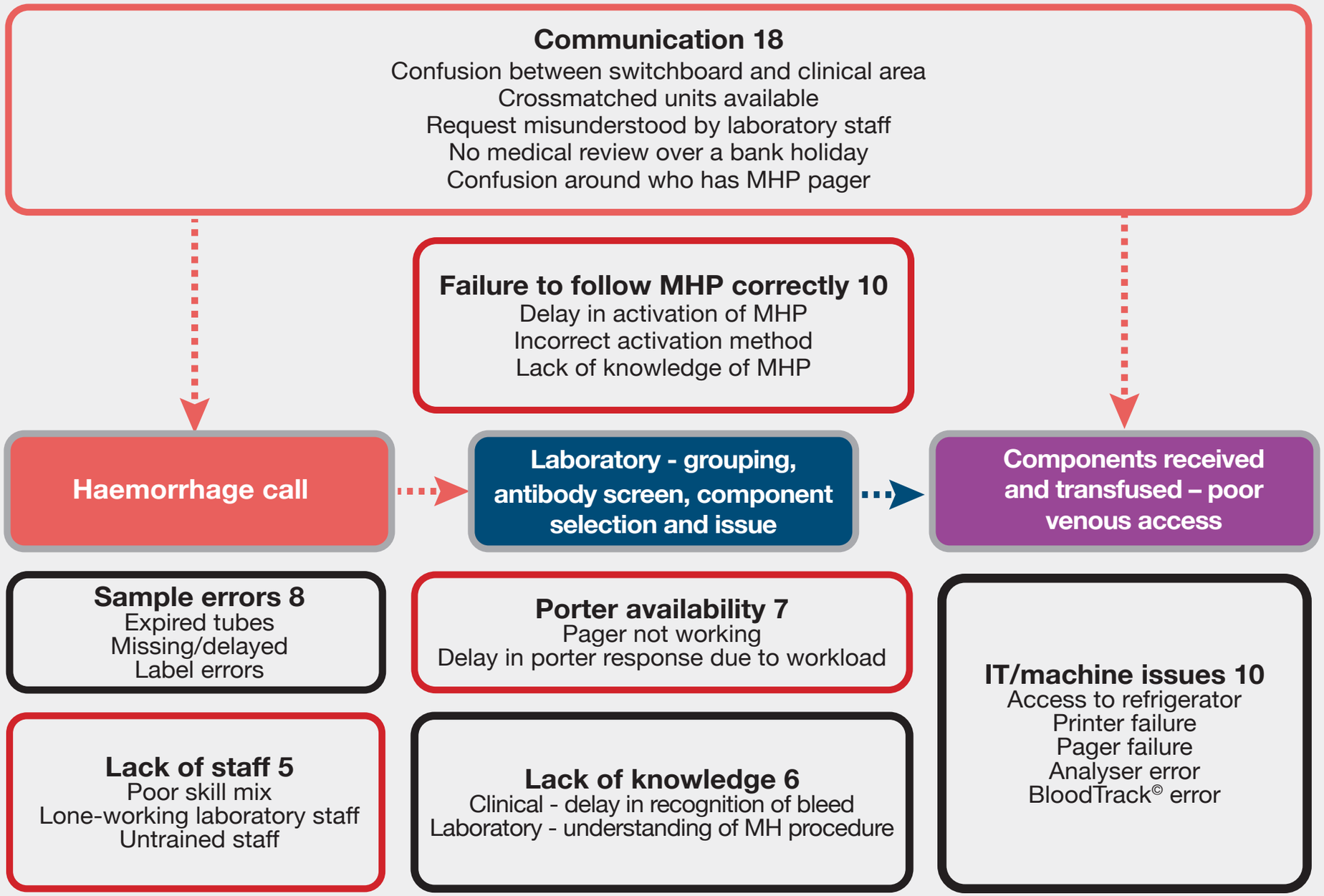


Figure 11a.3: Key factors contributing to delayed transfusions in 41 cases of major haemorrhage



MHP=major haemorrhage protocol; IT=information technology

Figure 11b.1: Step in transfusion process with associated errors

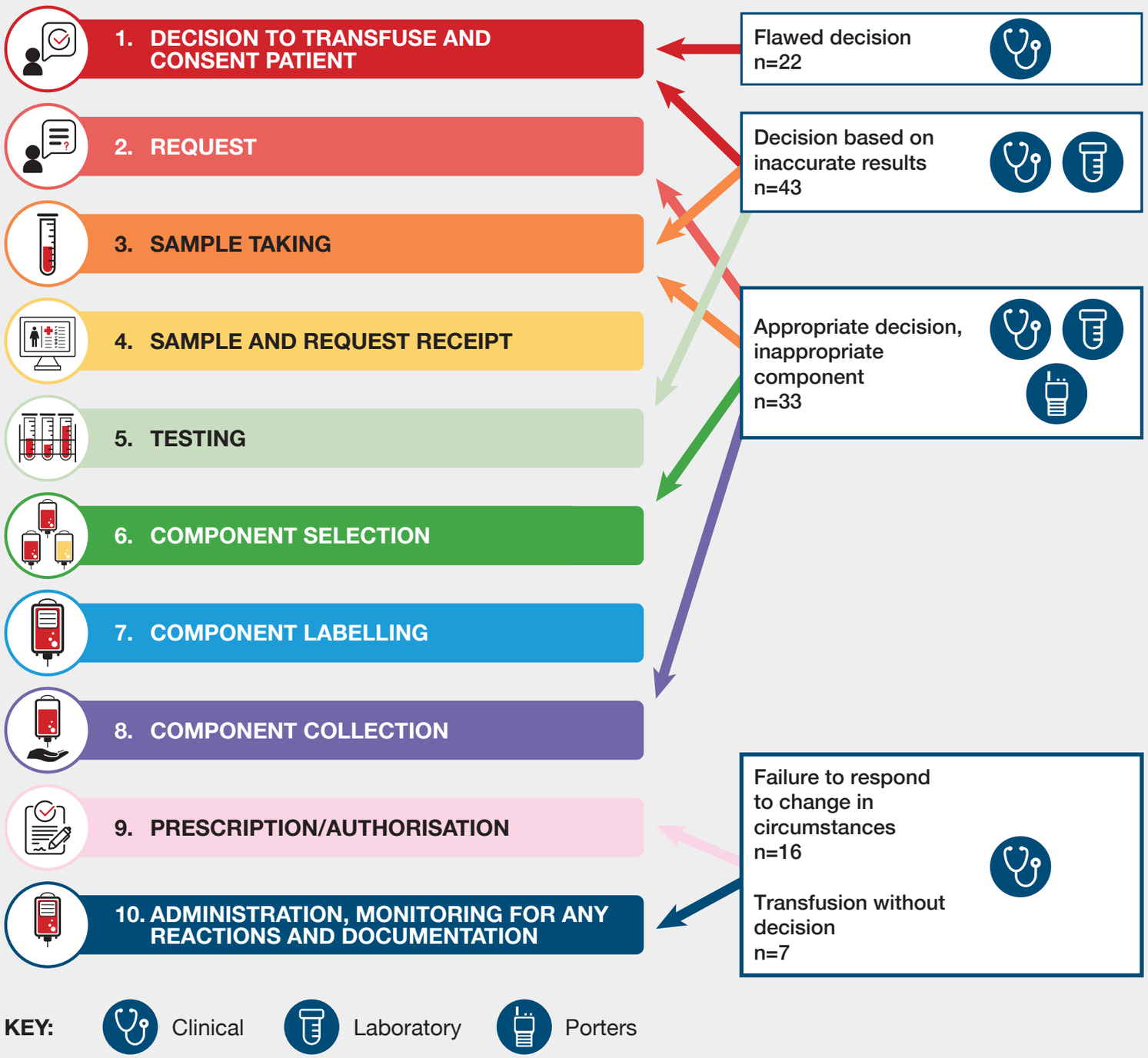


Figure 11a.1: Delayed transfusion reports and deaths by year 2011 to 2021 (n=952, deaths n=61)

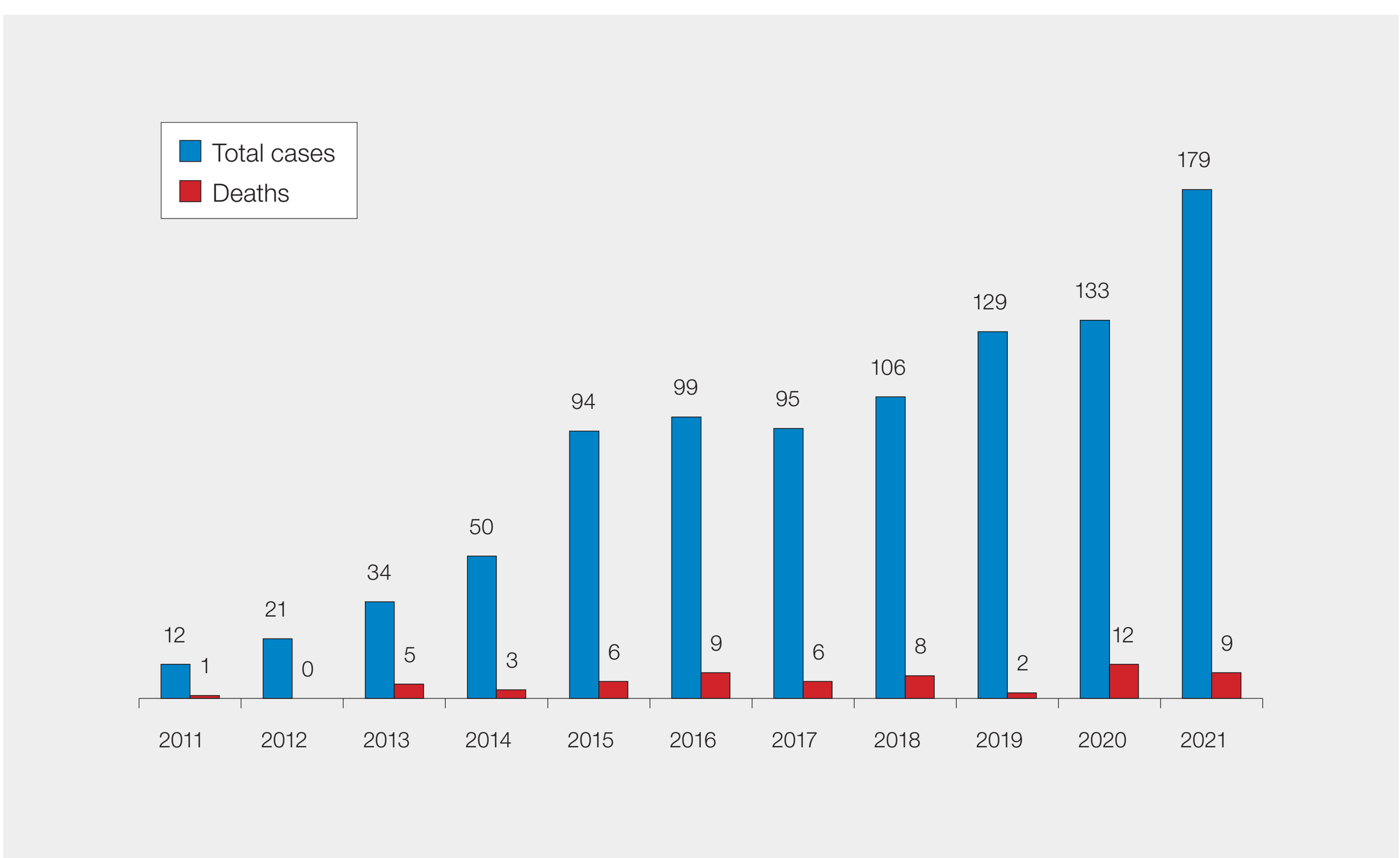


Figure 12a.1: Delayed transfusion reports and deaths by year 2011 to 2020 (n=775, deaths n=53)

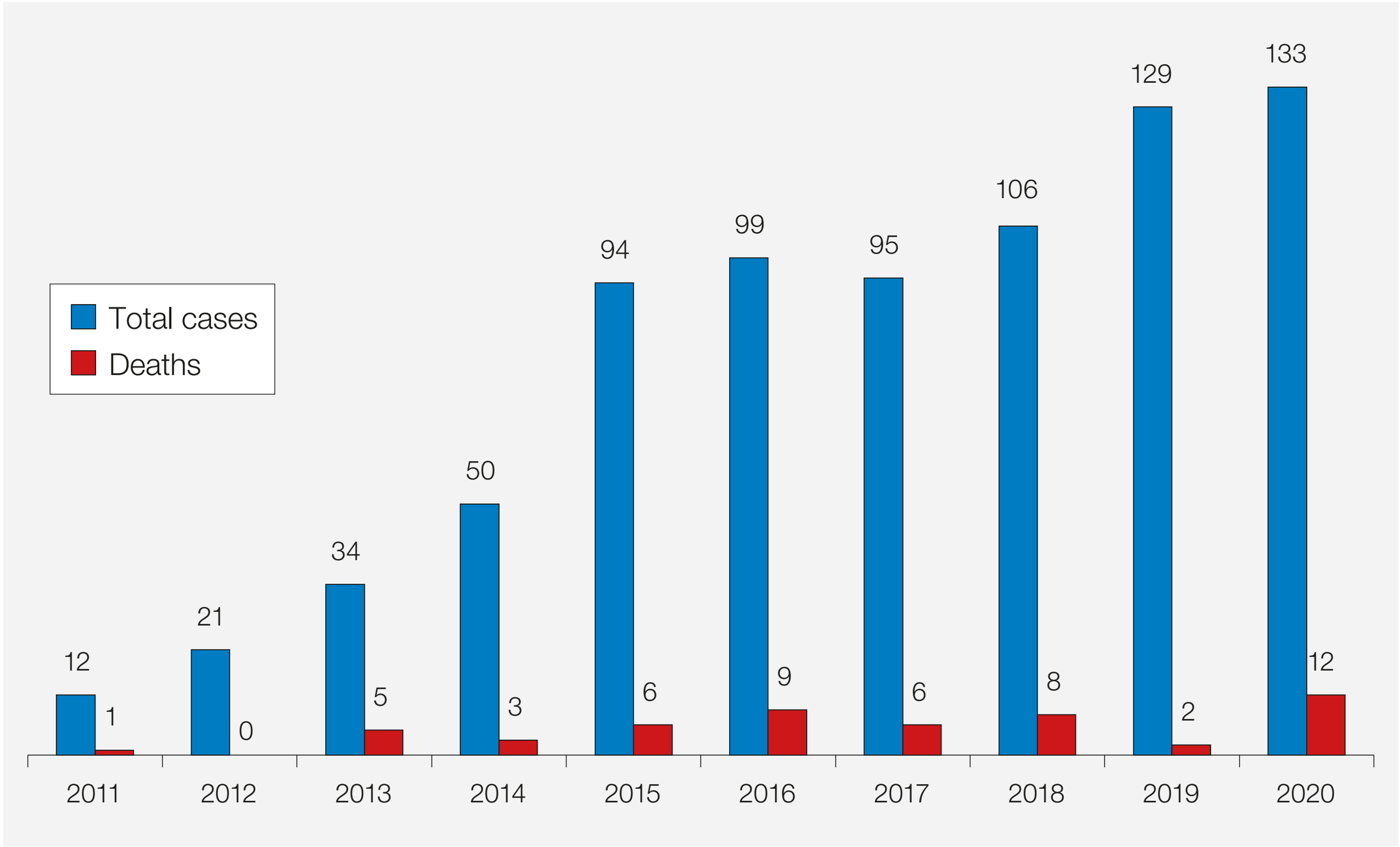


Figure 12a.2: Errors contributing to delayed transfusion

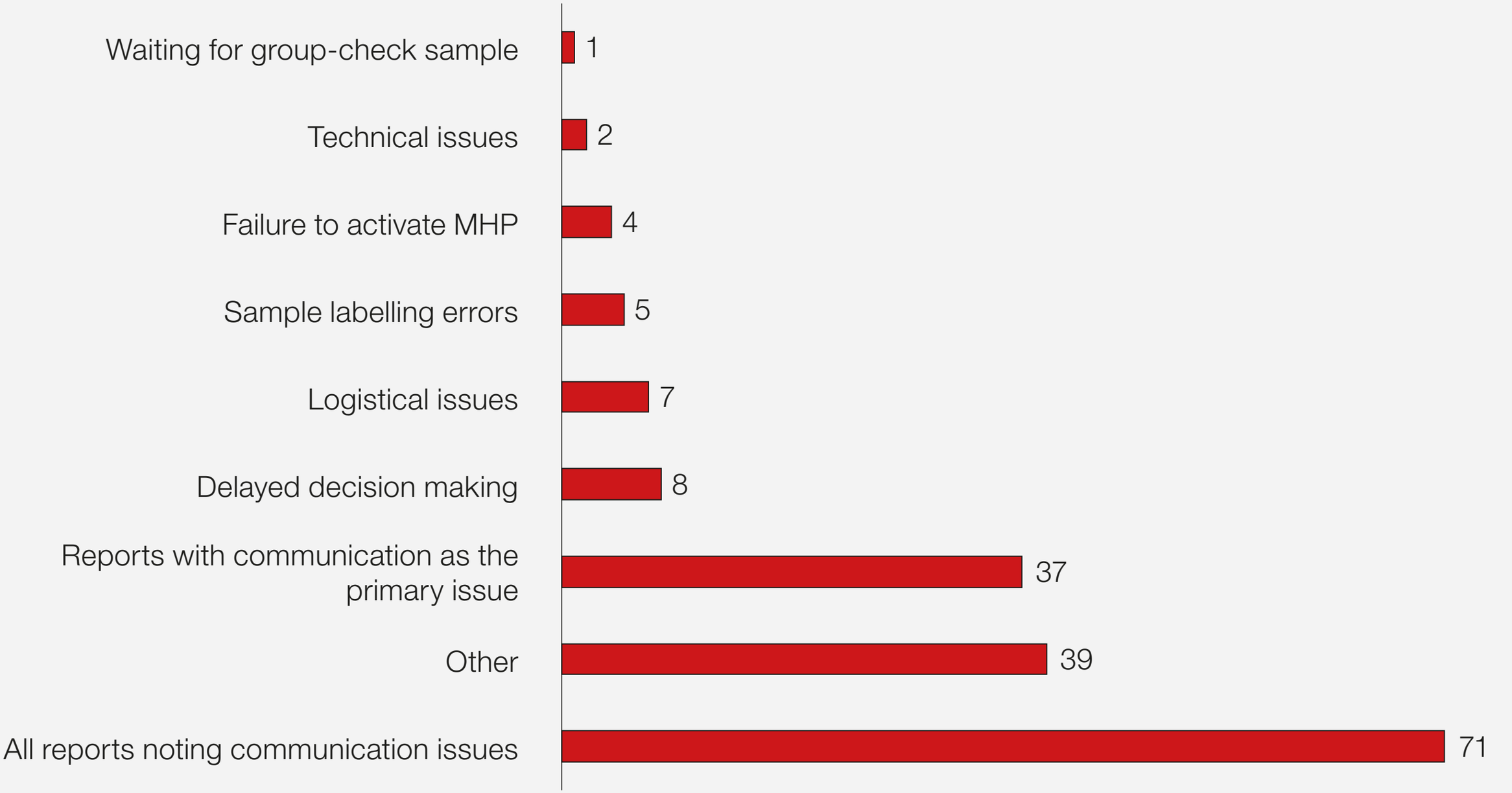


Figure 11a.1: Delayed transfusion reports by year 2010 to 2019

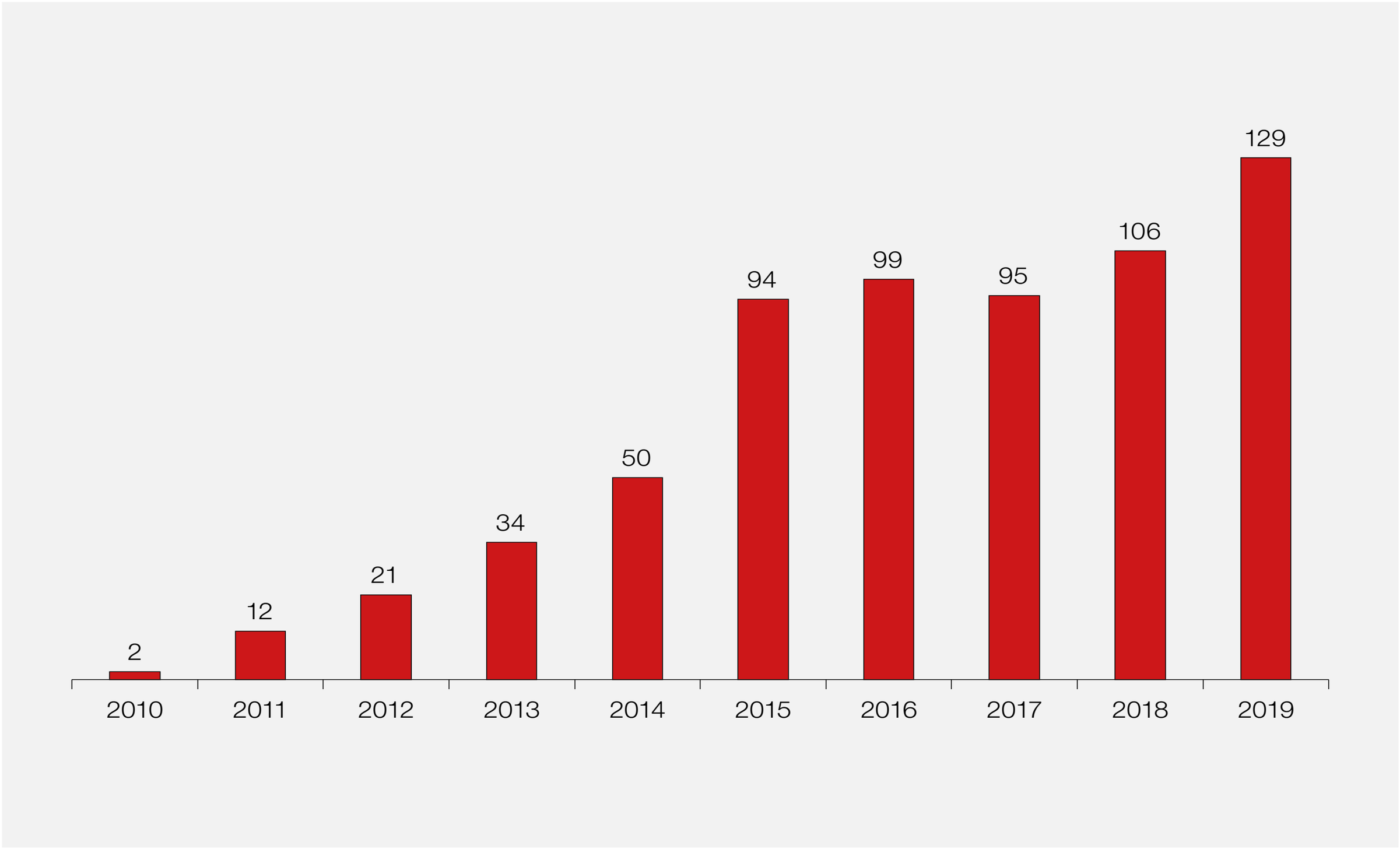
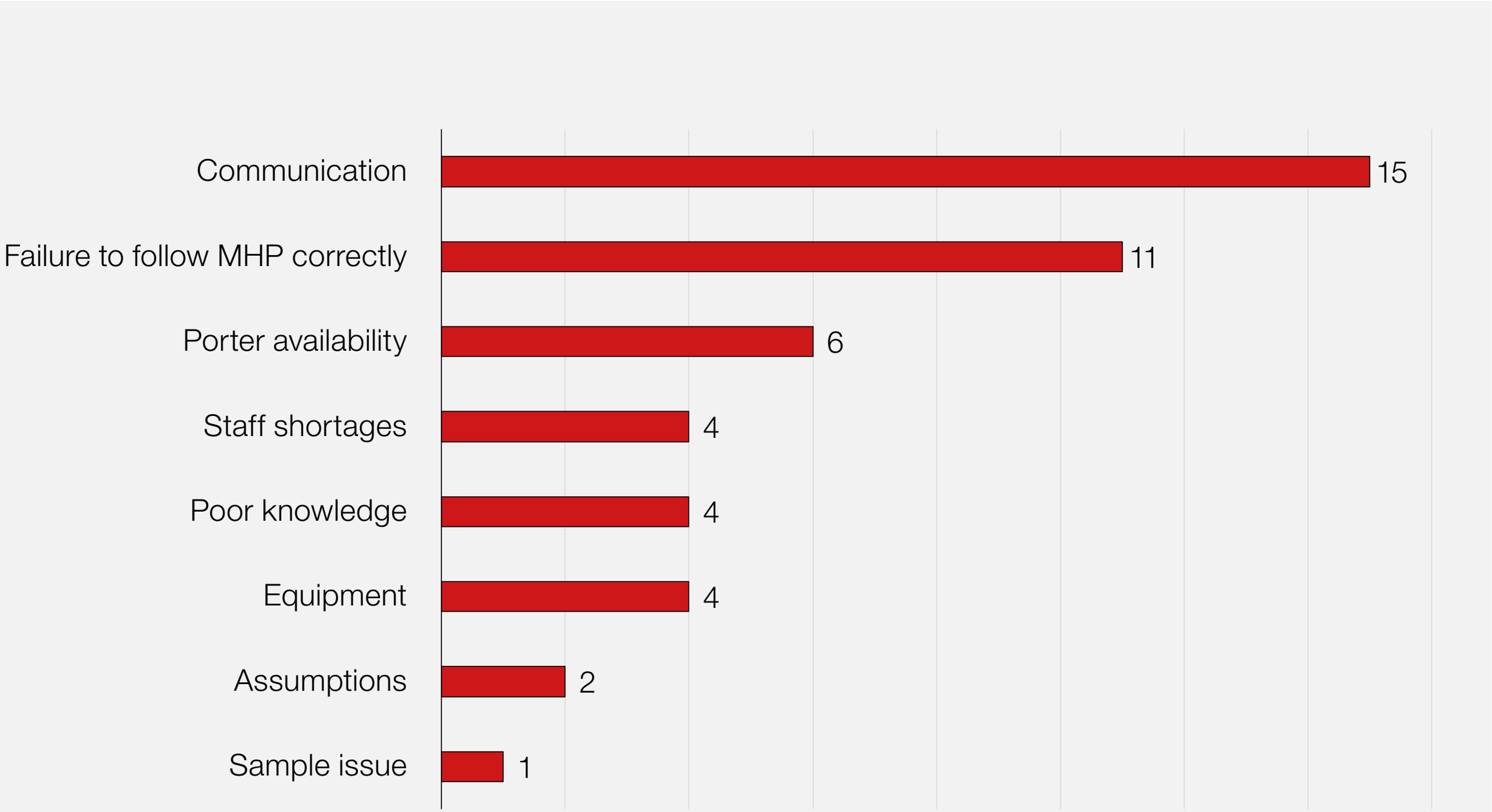
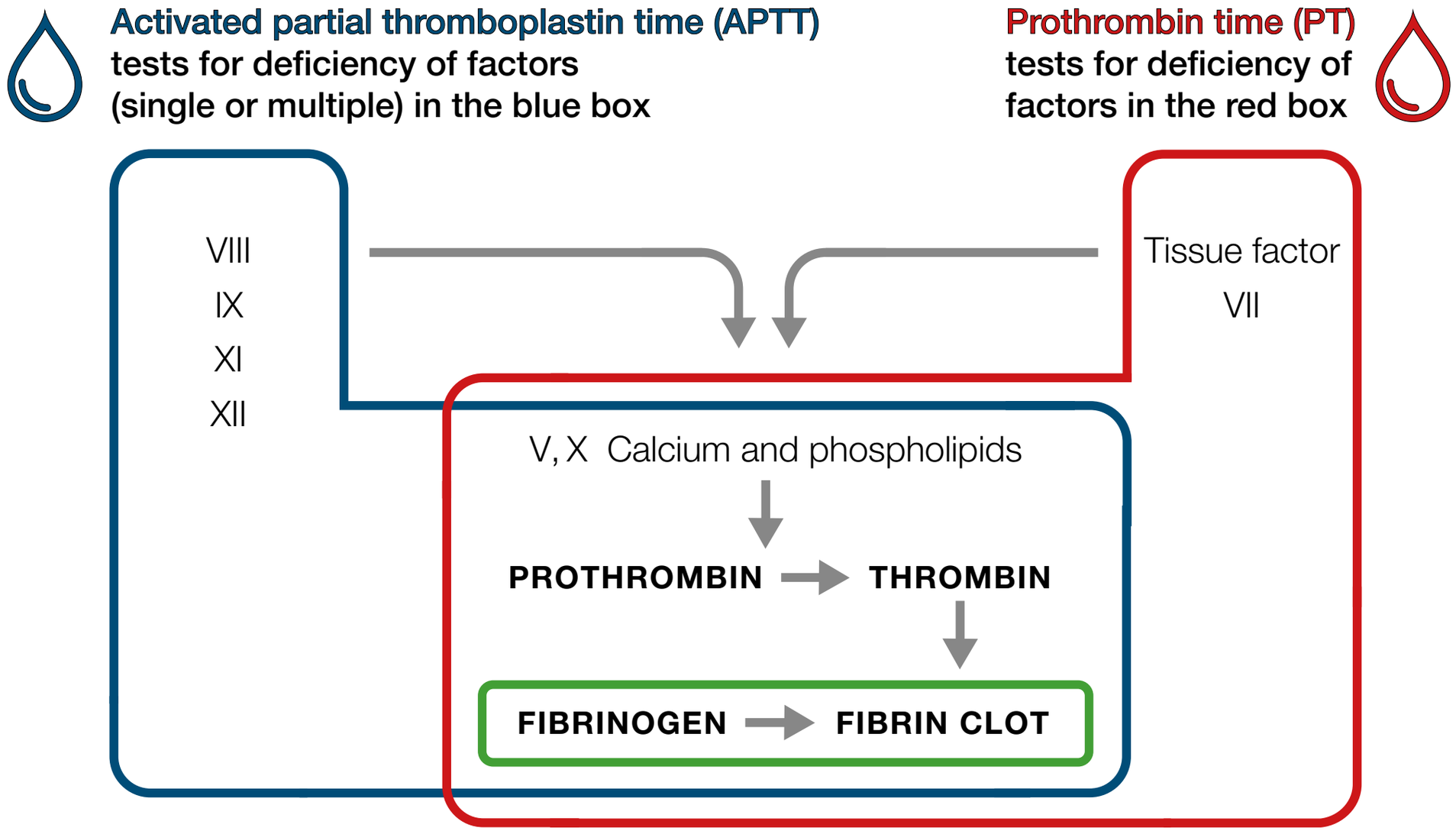


Figure 11a.2: Factors contributing to delayed transfusion in 16 cases



MHP=major haemorrhage protocol

Figure 11a.3: Mechanisms of the coagulation screen to show which coagulation factors affect the standard tests



Thrombin time only looks at this final conversion and depends on adequate amount of fibrinogen

Figure 11a.4: Interpretation of the coagulation screen

Prothrombin time	Activated partial thromboplastin time	Thrombin time	Interpretation
Abnormal	Normal	Normal	Factor VII deficiency
Normal	**Abnormal**	Normal	Deficiency of FXII, XI, **IX, VIII** (single or multiple)
Abnormal	Abnormal	Normal	Deficiency in the common pathway, isolated V or X deficiency. Multiple factors e.g. liver disease, warfarin therapy

Notes: many sick patients have disturbances of coagulation tests that **do not predict bleeding (and in some cases are associated with a thrombotic risk)**. These tests were introduced in the 1960s to screen for congenital factor deficiencies. The PT is very sensitive to FVII deficiency and is used for warfarin monitoring but note that the APTT will also be prolonged (because FIX is reduced) but to a lesser extent. The sample must be taken carefully (good venepuncture, free flow) to avoid activation and in the correct volume (as it is taken into a specific volume of anticoagulant citrate) to avoid erroneous and misleading results.

Isolated prolongation of the APTT can be due to haemophilia A (FVIII deficiency) or B (FIX deficiency,) where the need for diagnosis and treatment is urgent. It is also prolonged in FXII deficiency (common but of no clinical significance) and factor XI deficiency (uncommon and usually not associated with serious bleeding). The thrombin time does not depend on other coagulation factors as thrombin is added to the test system. Many laboratories measure the amount of fibrinogen rather than the thrombin time. (Prolongation of standard coagulation tests can also be caused by inhibitors).

Vitamin K results in increased synthesis of factors II, VII, IX and X so will correct the PT but not FVIII, FXI, V or X deficiency. Normal ranges are different in childhood and any hospital with paediatric patients must use an age-appropriate normal range to avoid unnecessary investigation and treatment.

Figure 11b.1: Example of a blood gas result illustrating the difference between total Hb (A) and O2Hb (B) (not the actual case described above)

Results

				Crit. Low	Reference Low	Reference High	Crit. High
Measured (37.0°C)							
pH		**7.37**		[7.20	7.35	7.45	7.60]
pCO_2	↑	**6.8**	kPa	[2.6	4.3	6.4	9.3]
pO_2	↓	**9.0**	kPa	[6.0	11.0	14.4	--]
Na^+	↓	**135**	mmol/L	[120	136	145	160]
K^+		**4.2**	mmol/L	[2.8	3.5	5.1	6.5]
Cl^-		**99**	mmol/L	[80	98	107	120]
Ca^{++}		**1.19**	mmol/L	[0.75	1.15	1.33	1.60]
Hct	↓	**35**	%	[18	37	50	60]
Glu	↑	**14.4**	mmol/L	[2.5	3.6	5.3	25.0]
Lac	↑	**2.3**	mmol/L	[--	0.3	2.0	4.0]
CO-Oximetry							
A tHb	↓	**110**	g/L	[70	117	174	200]
B O_2Hb		**92.5**	%	[--	90.0	95.0	--]
COHb		**1.3**	%	[--	0.0	3.0	10.0]
MetHb		**0.8**	%	[--	0.0	1.5	--]
HHb	↑	**5.4**	%	[--	1.0	5.0	--]
sO_2		**94.5**	%	[--	94.0	98.0	--]
Derived							
BE(B)	↑	**3.1**	mmol/L	[--	-2.0	3.0	--]
HCO_3^-std		**27.3**	mmol/L	[10.0	21.0	28.0	40.0]

↑↓ Outside Reference Range