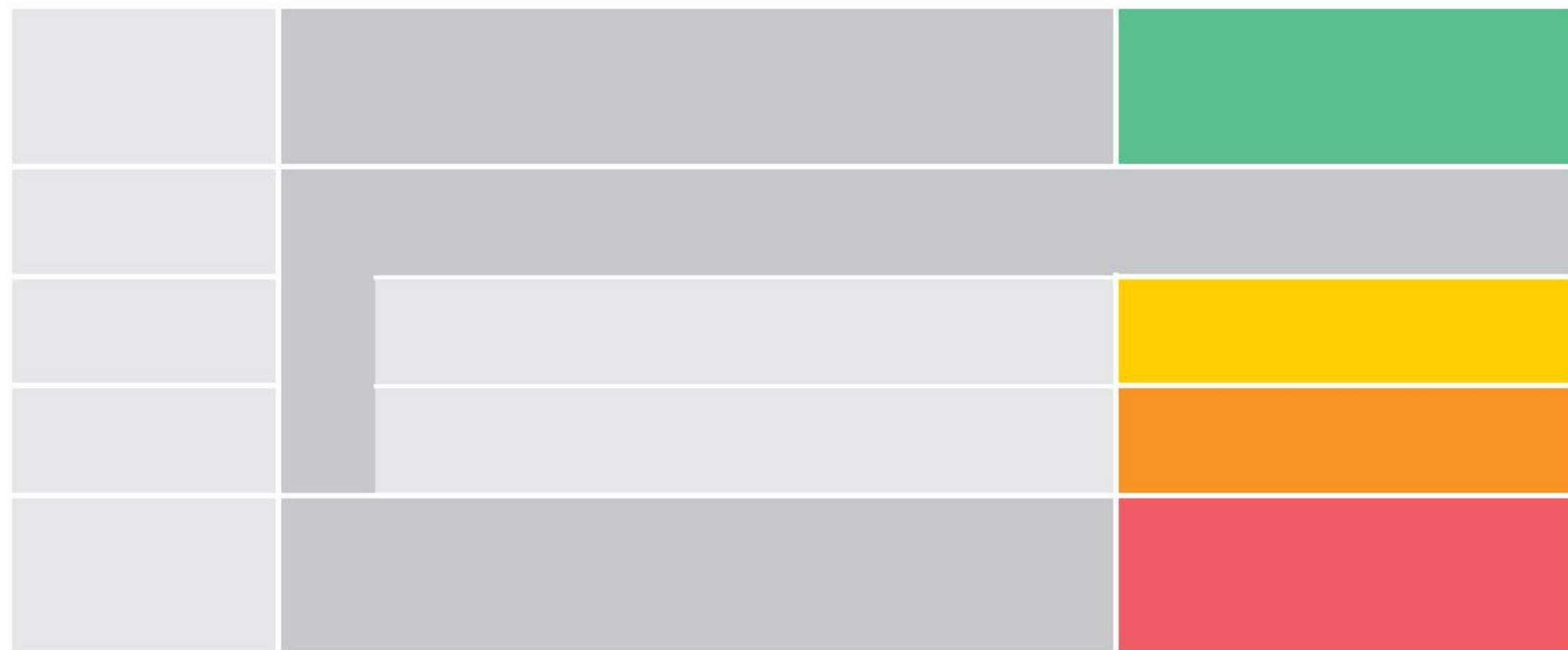
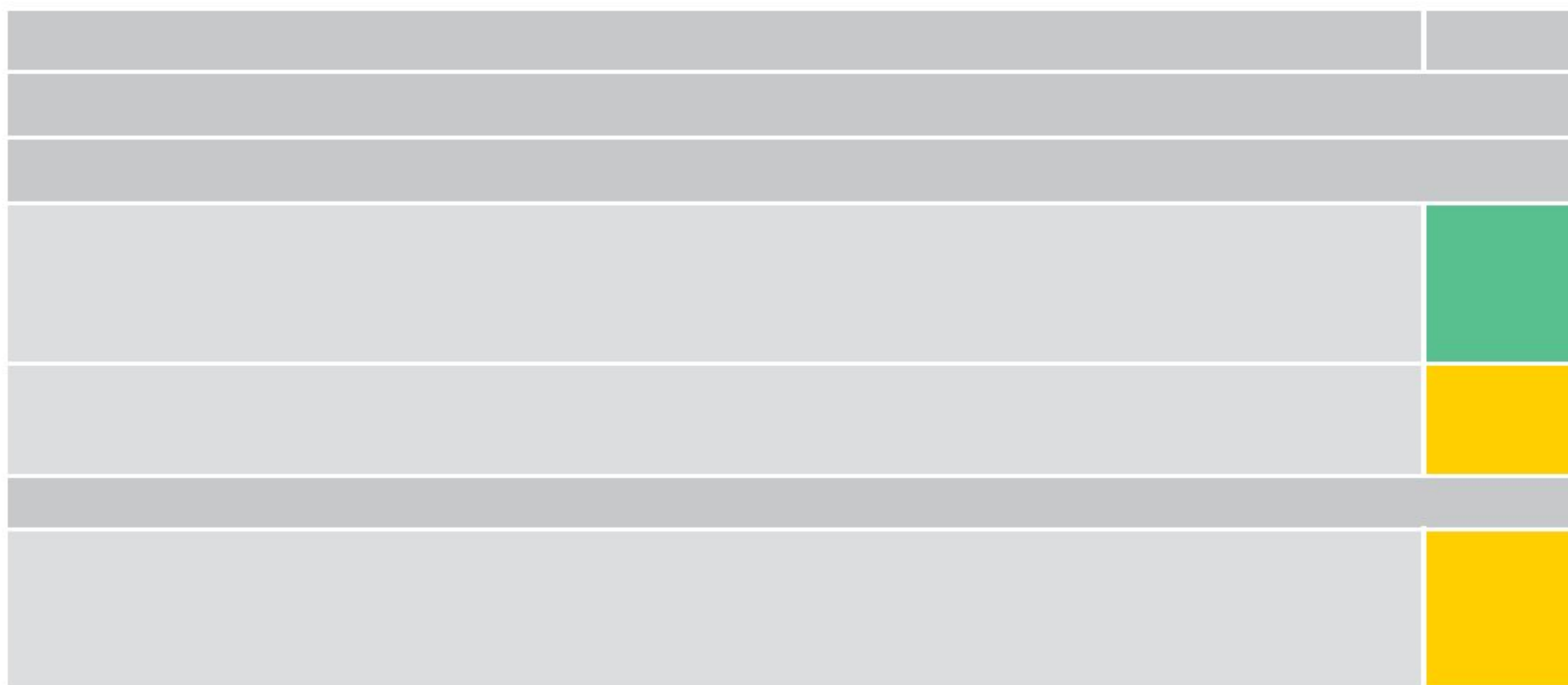
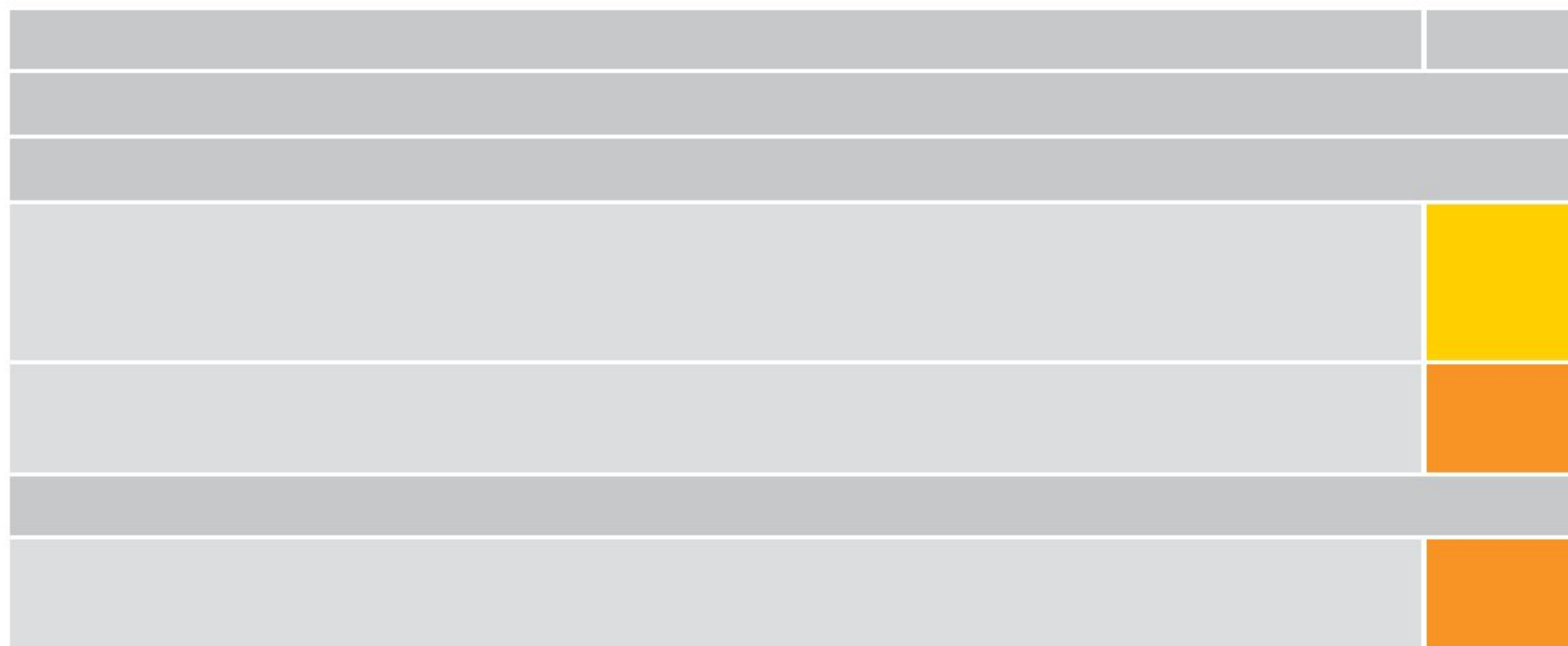
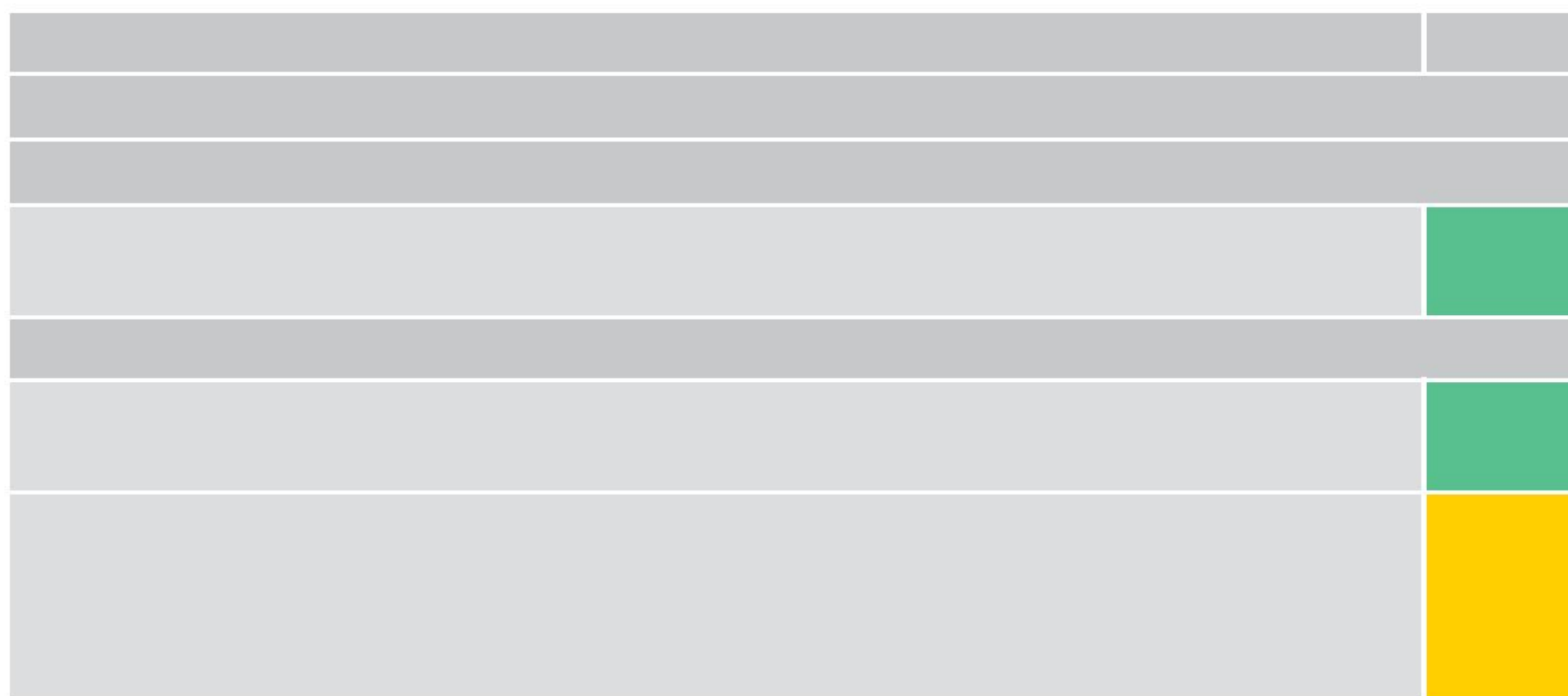




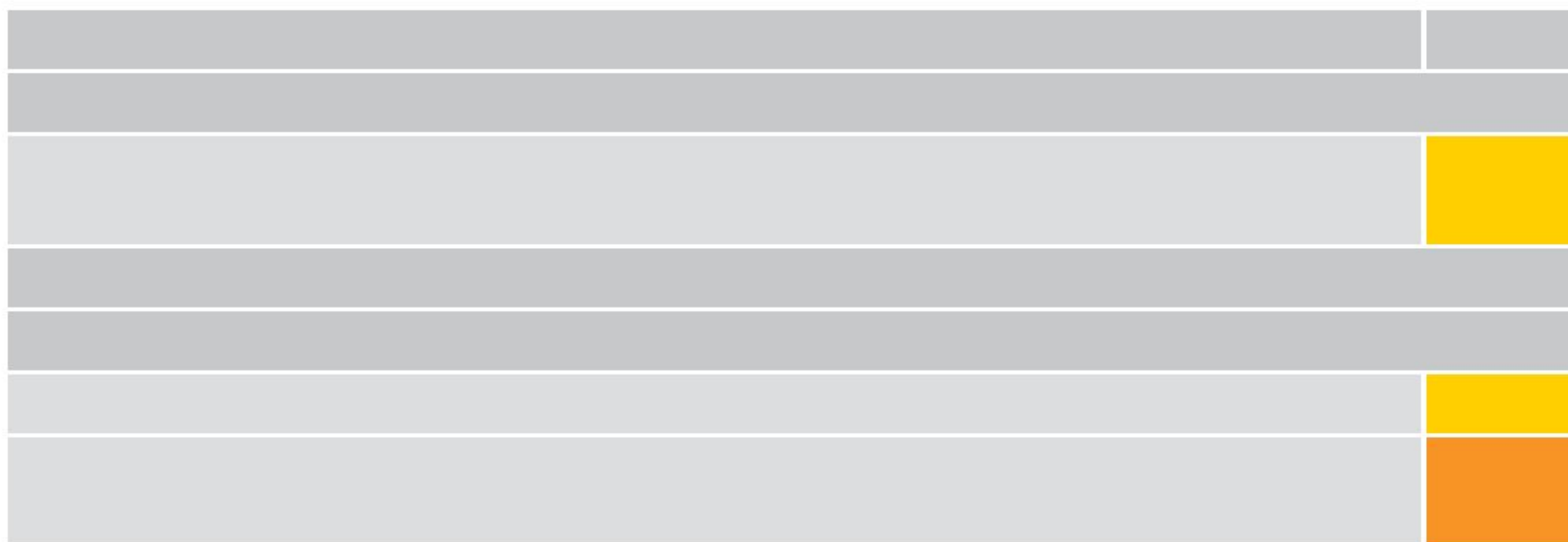


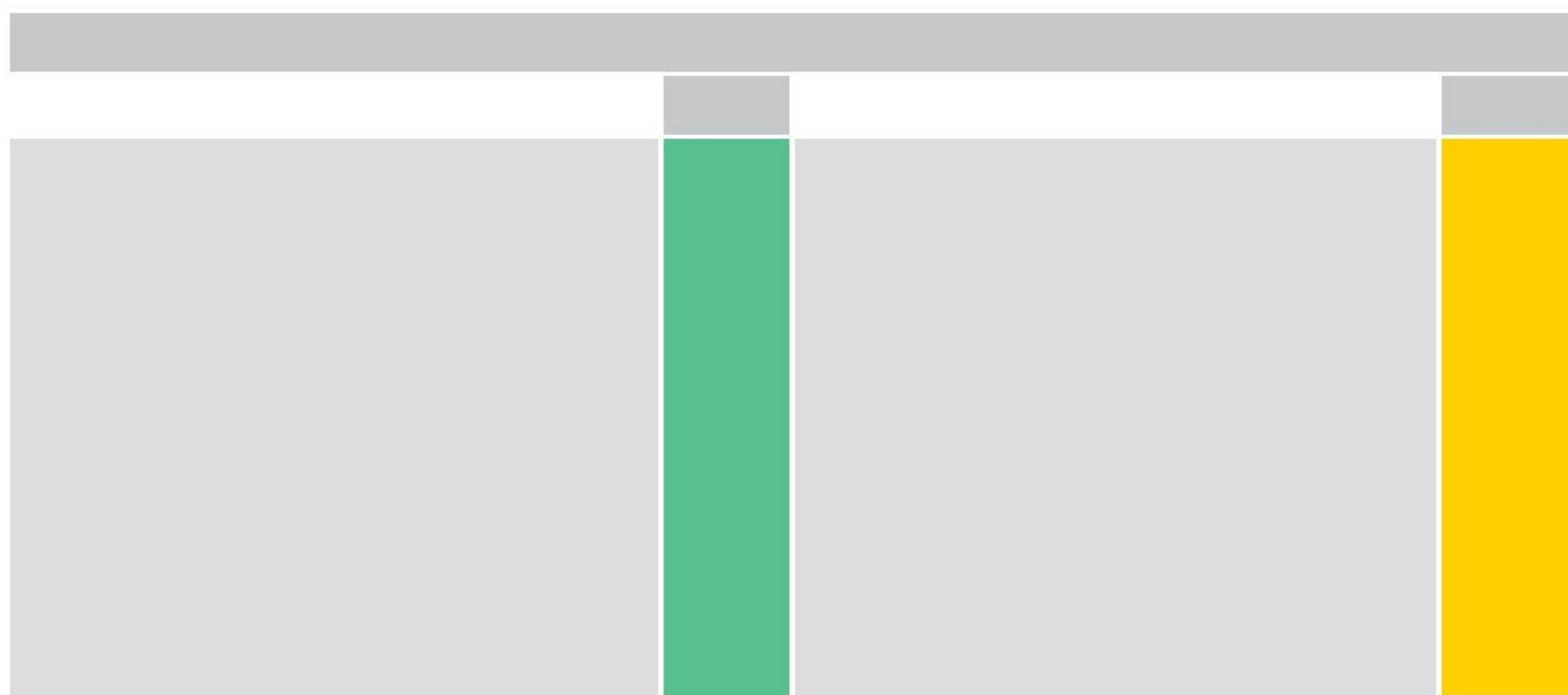


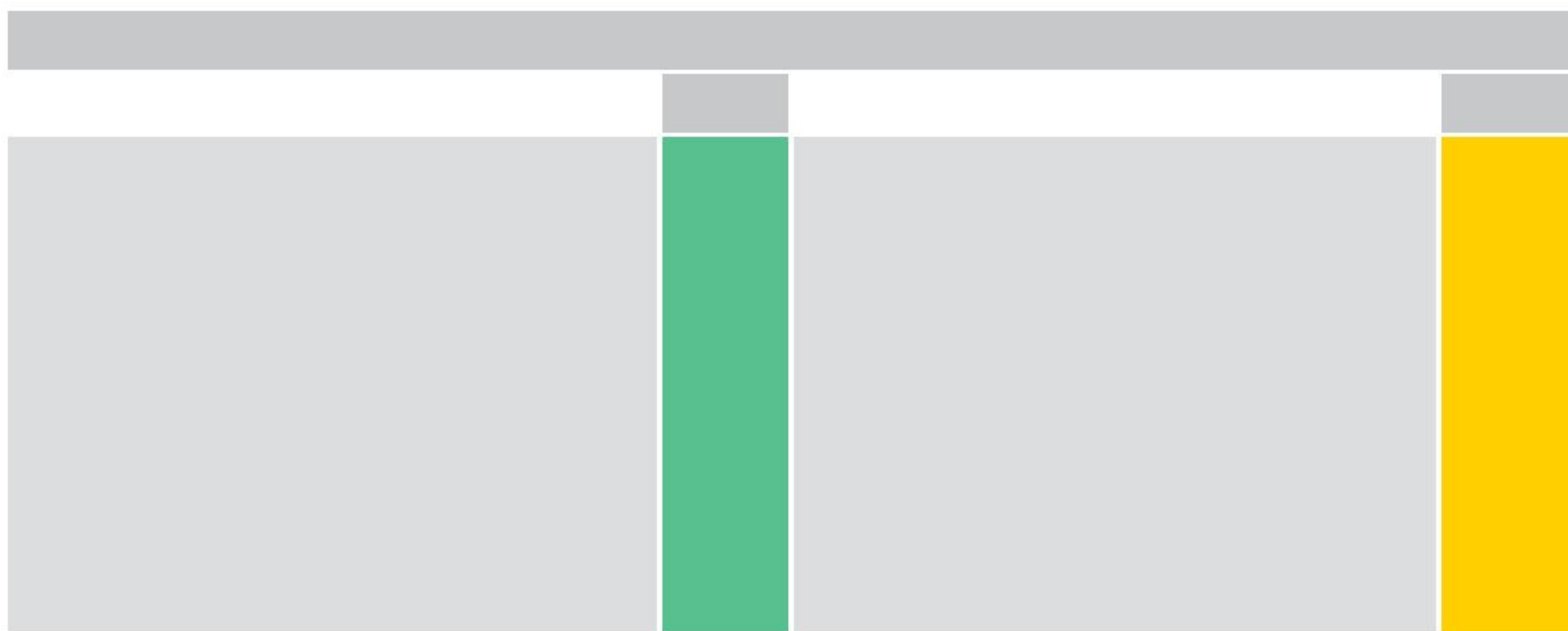


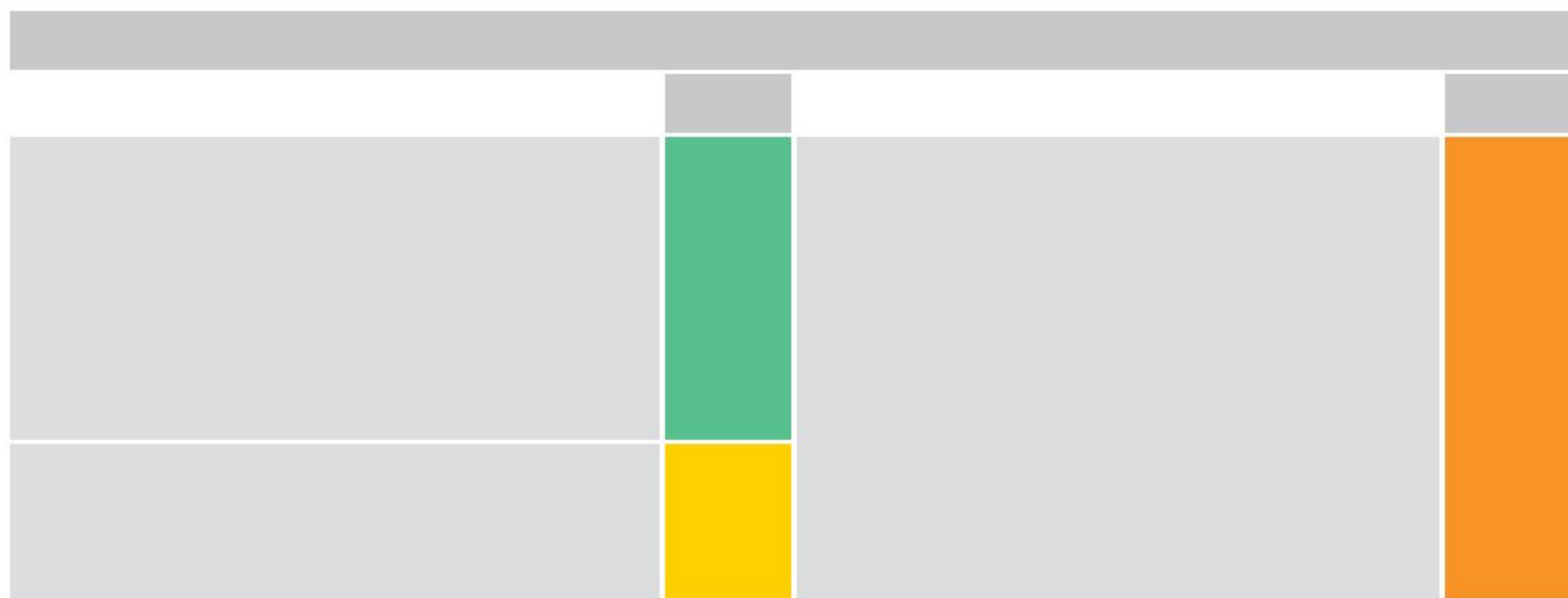


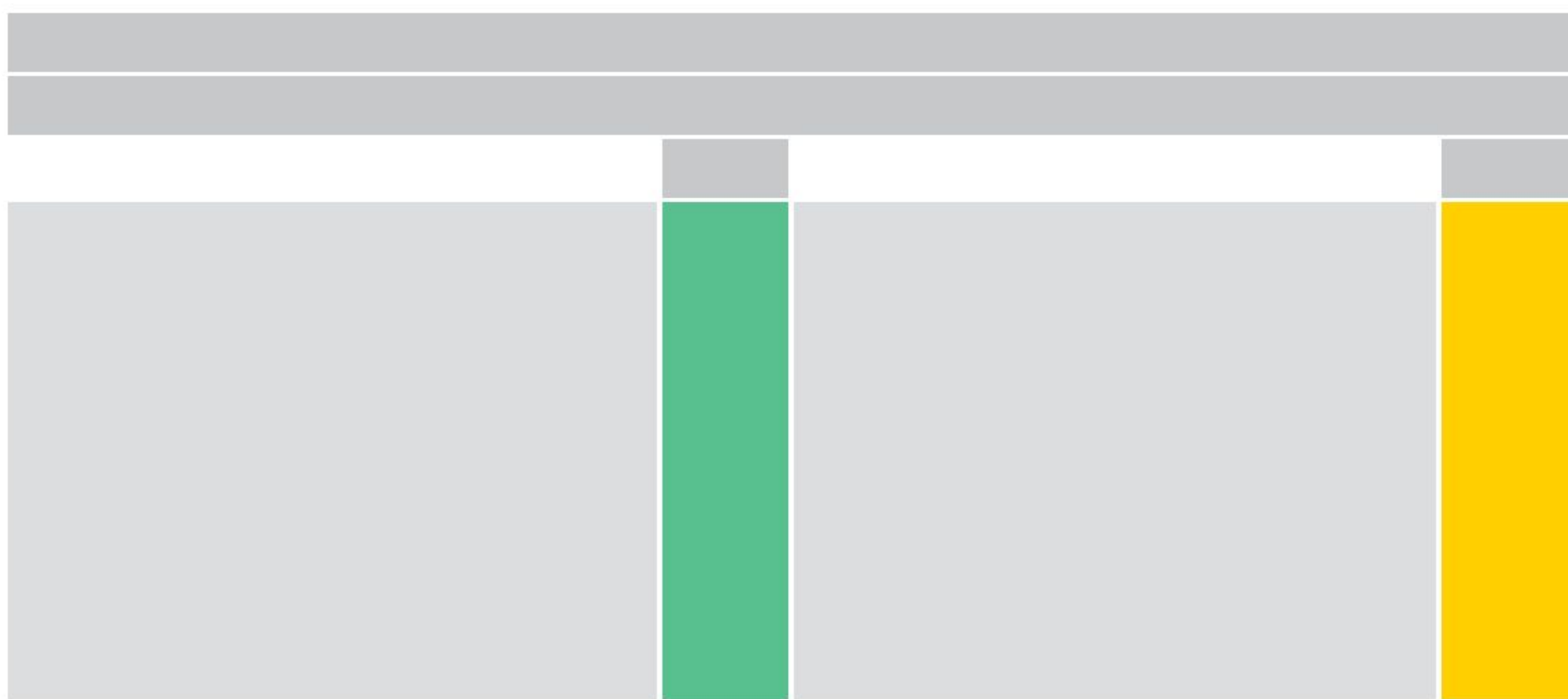


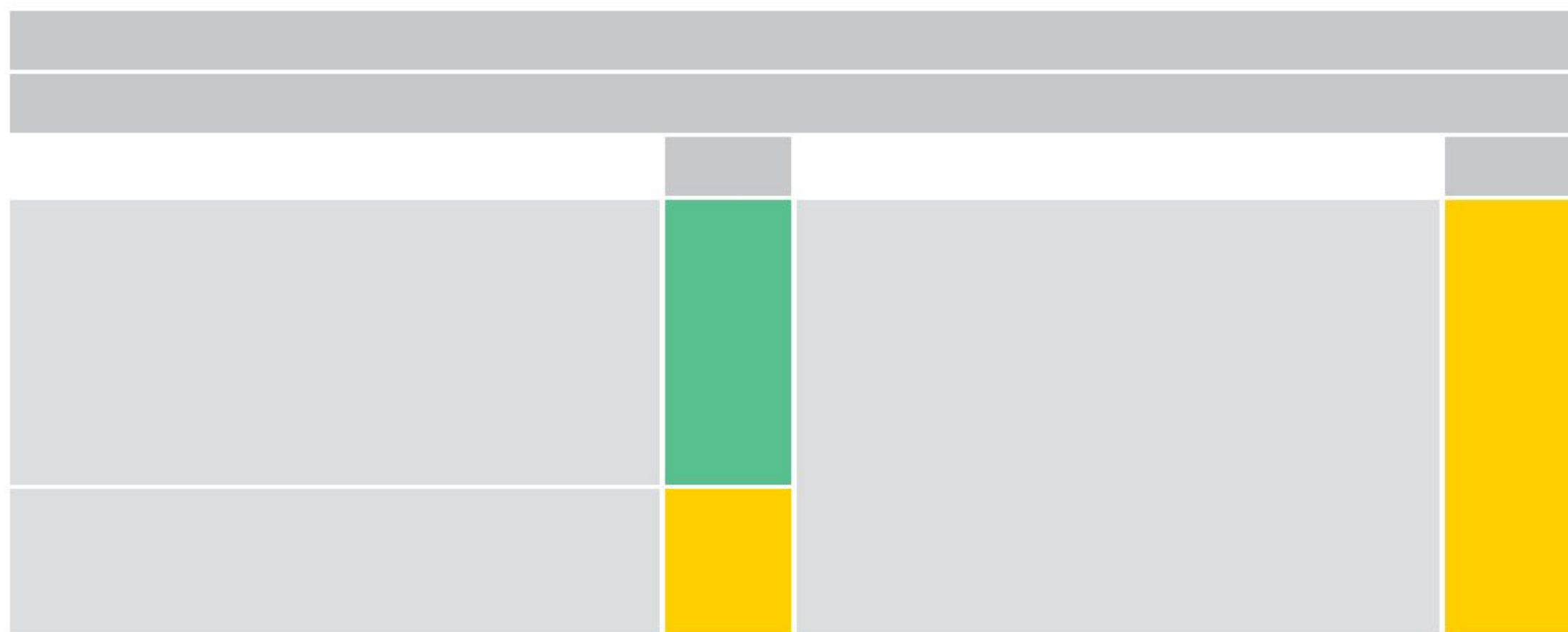




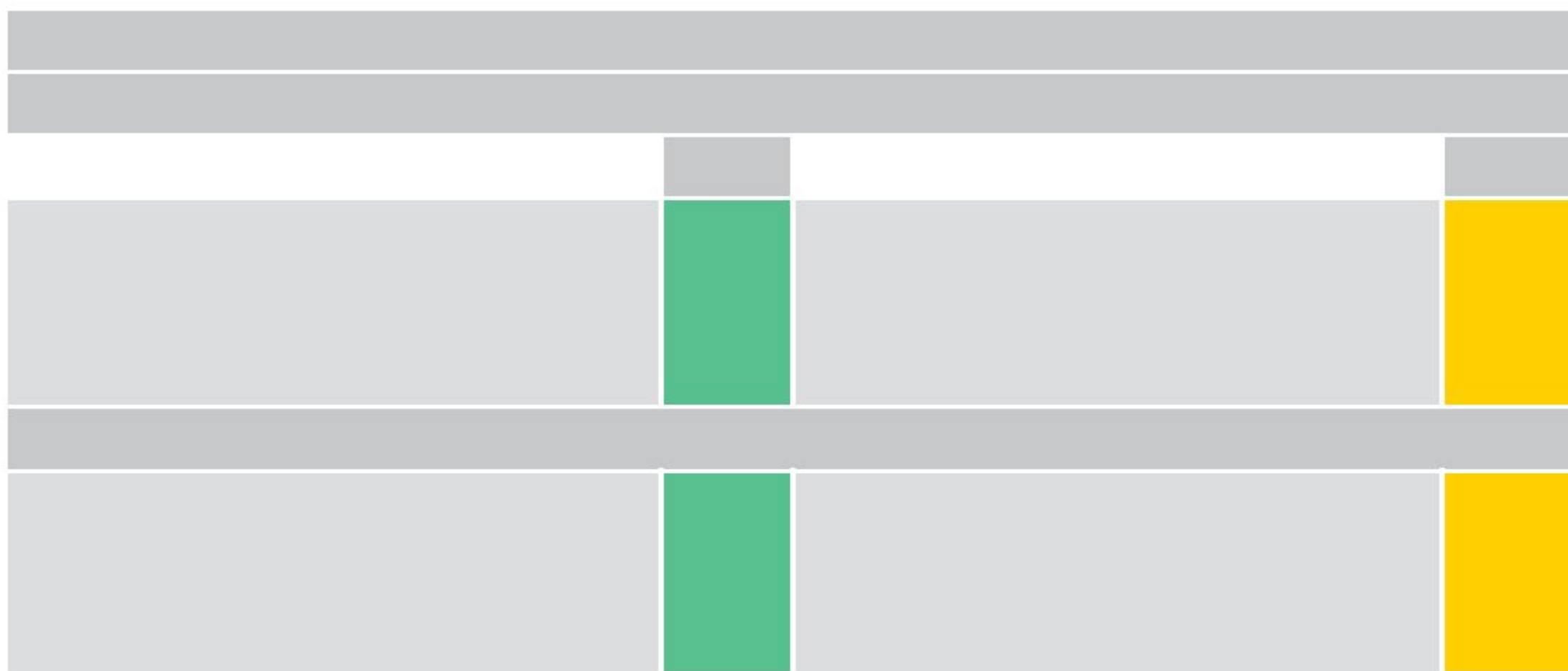


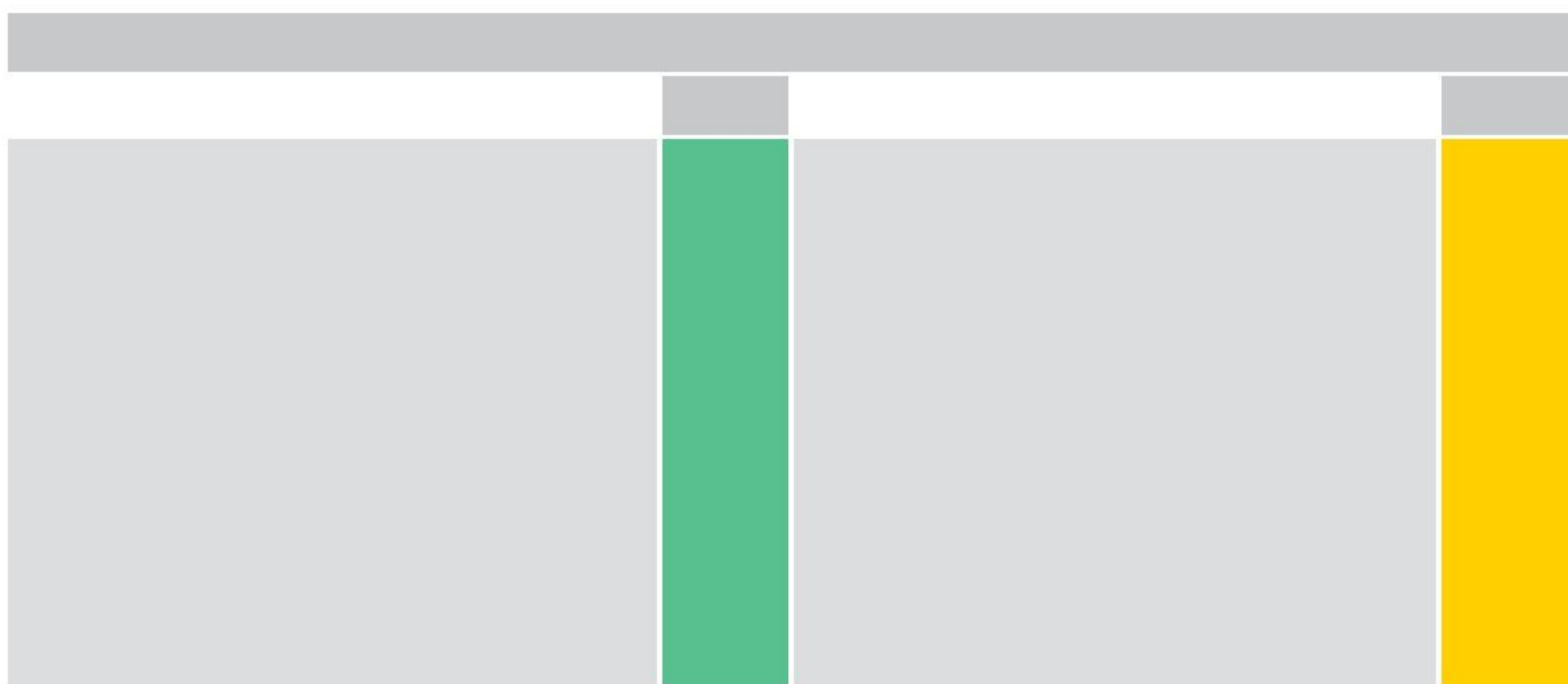


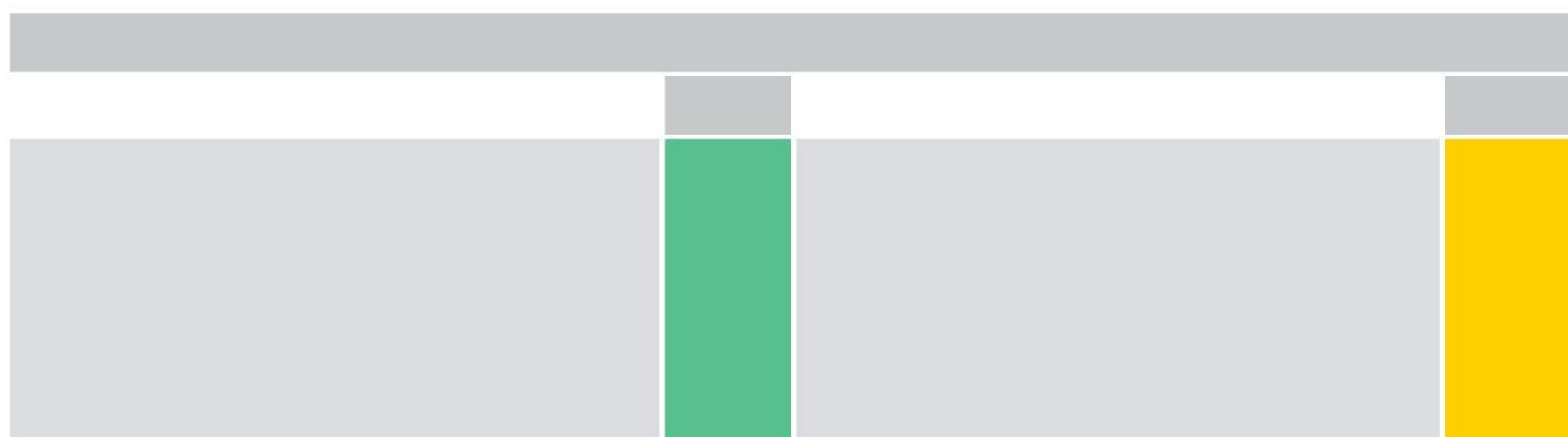




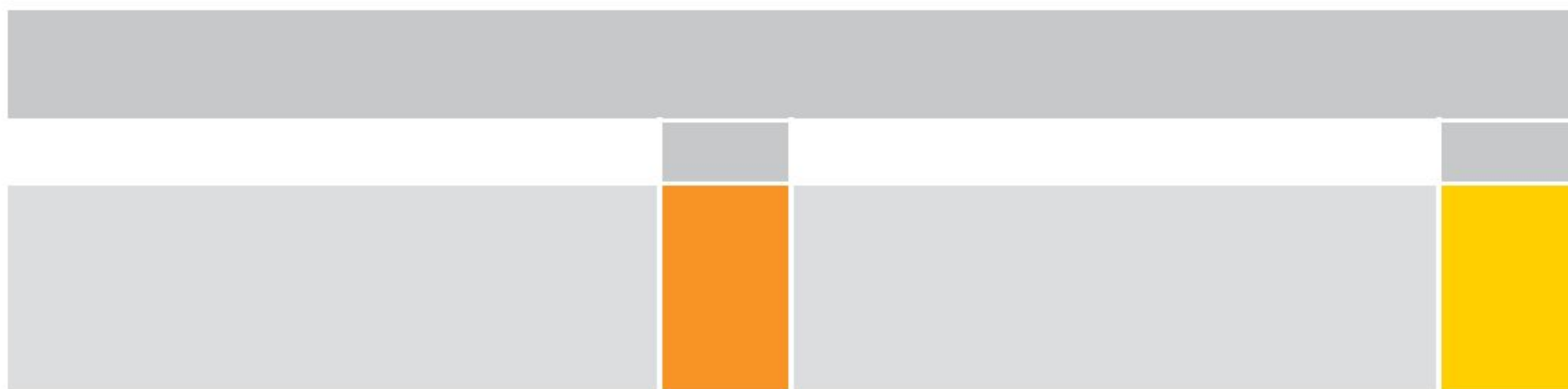
The diagram illustrates a grid of colored rectangles. The top row consists of two gray rectangles. Below this, there are two rows of three rectangles each. The first row has a gray rectangle, a green rectangle, a gray rectangle, and a yellow rectangle. The second row has a gray rectangle, an orange rectangle, a gray rectangle, and a yellow rectangle.

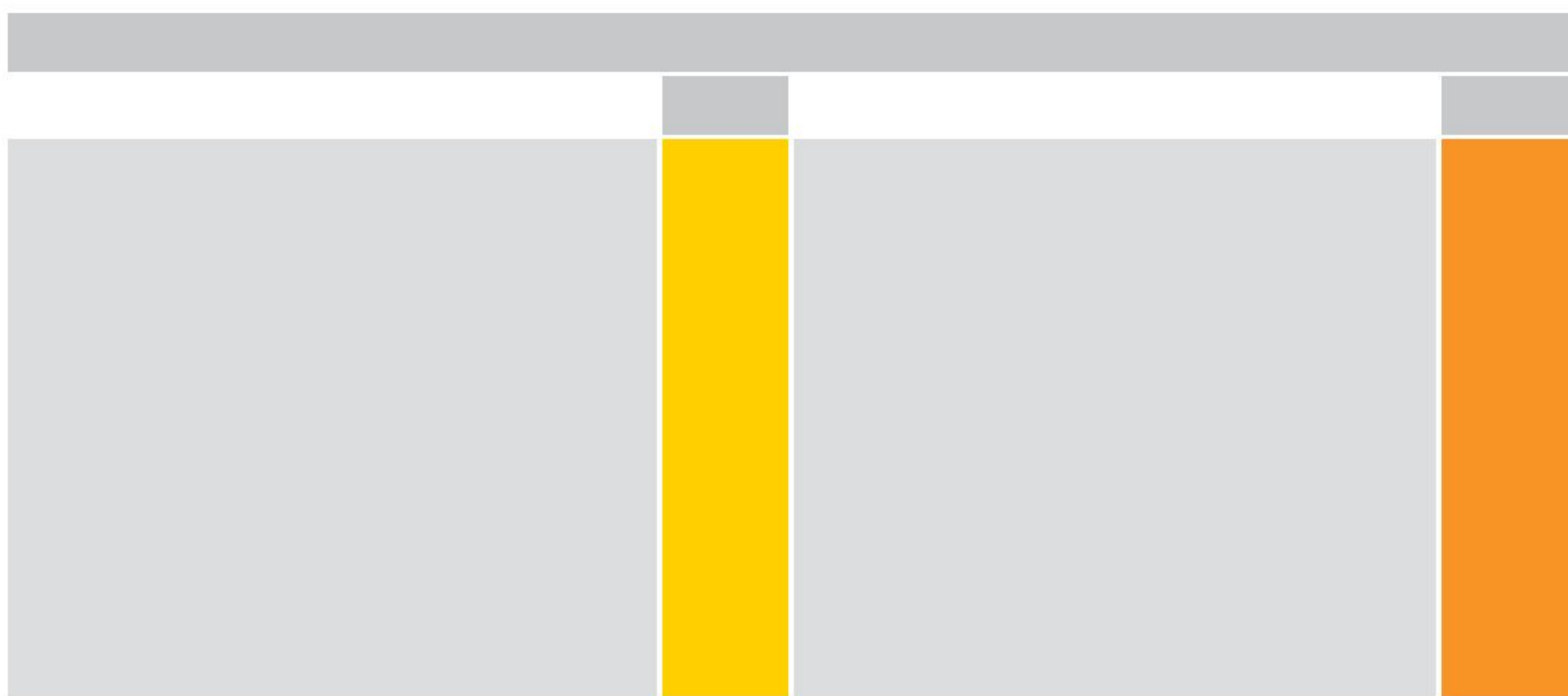


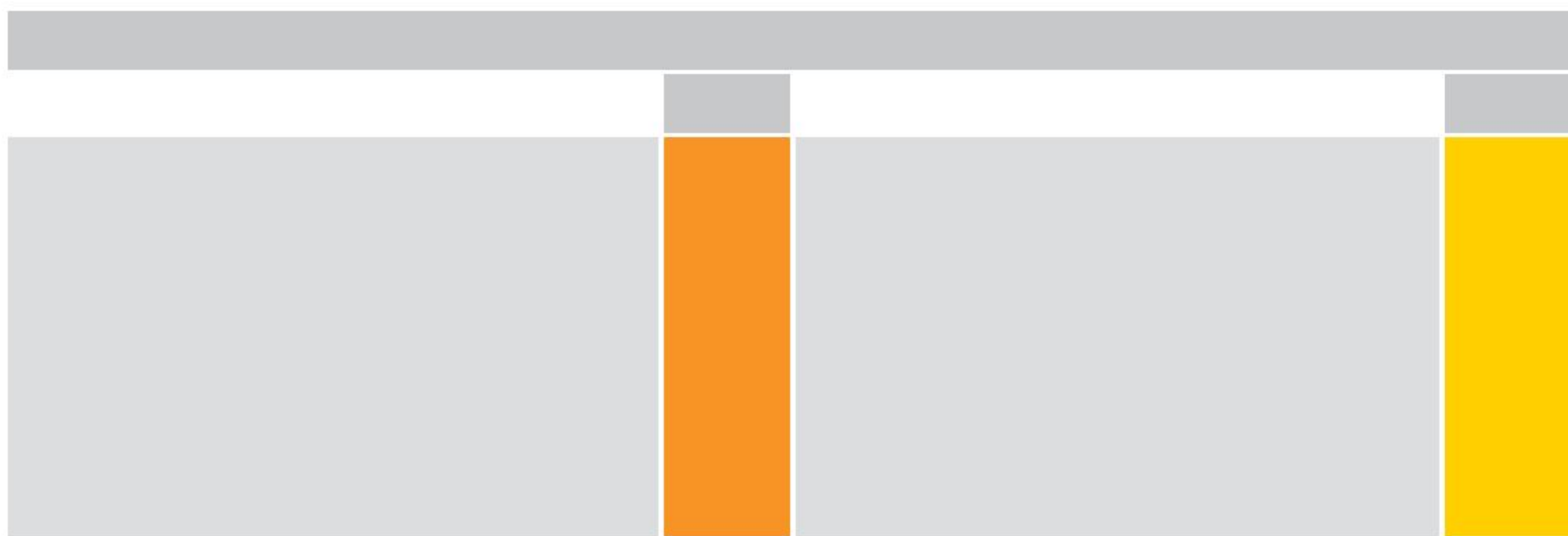


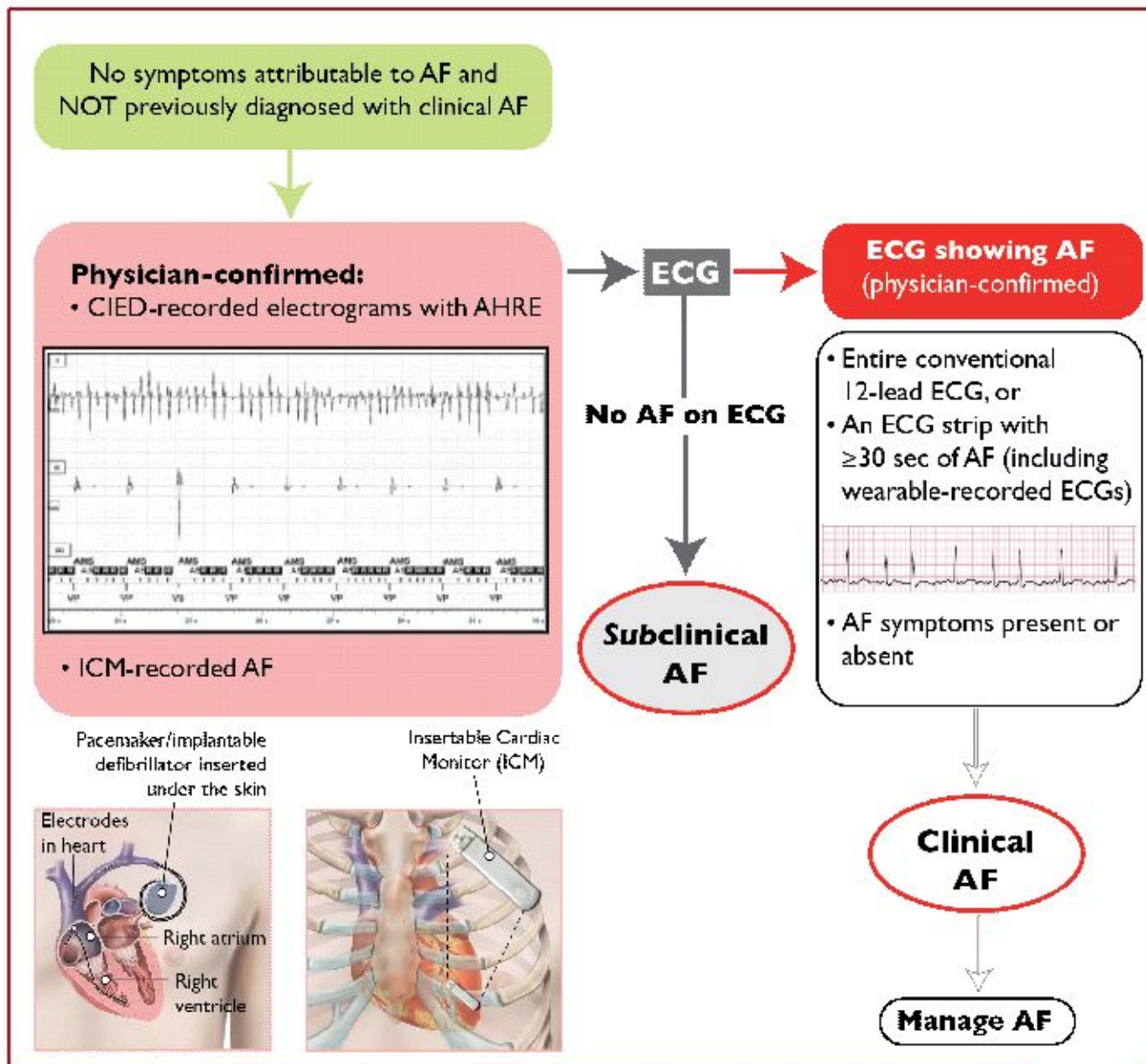


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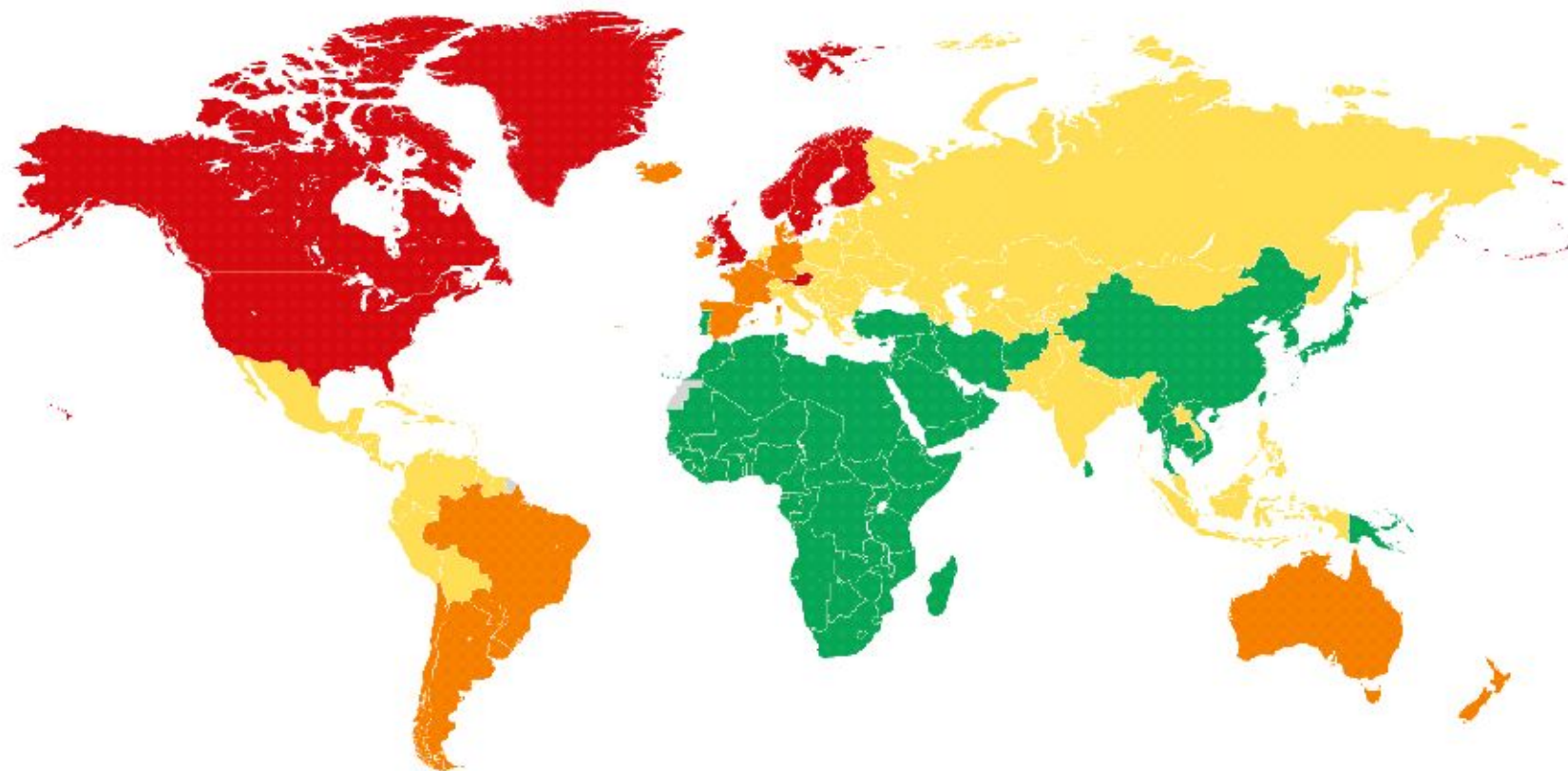






GLOBAL PREVALENCE OF AF

(globally, 43.6 million individuals had prevalent AF/AFL in 2016)



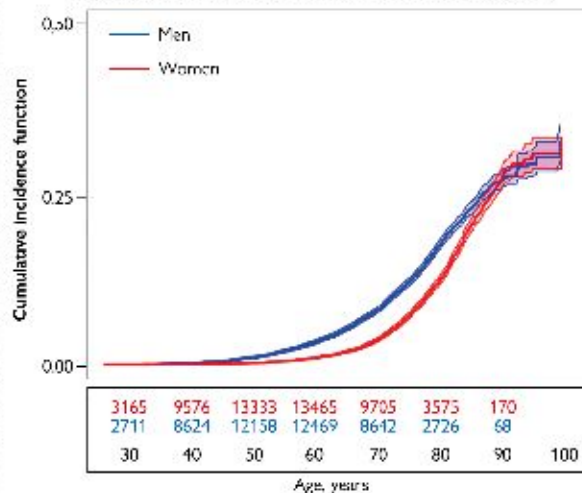
LIFETIME RISK for AF 1 in 3 individuals



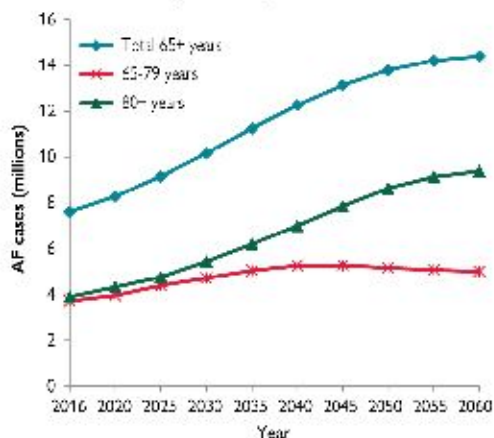
of European ancestry
at index age of 55 years
37.0% (34.3% to 39.6%)

AF is more common in males

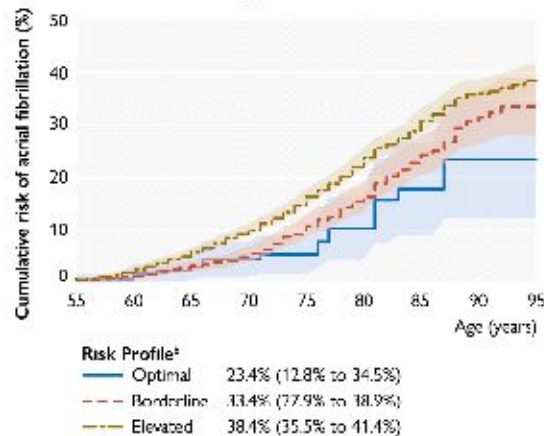
Cumulative incidence curves and 95% CIs
for AF in women and men with death as a competing risk

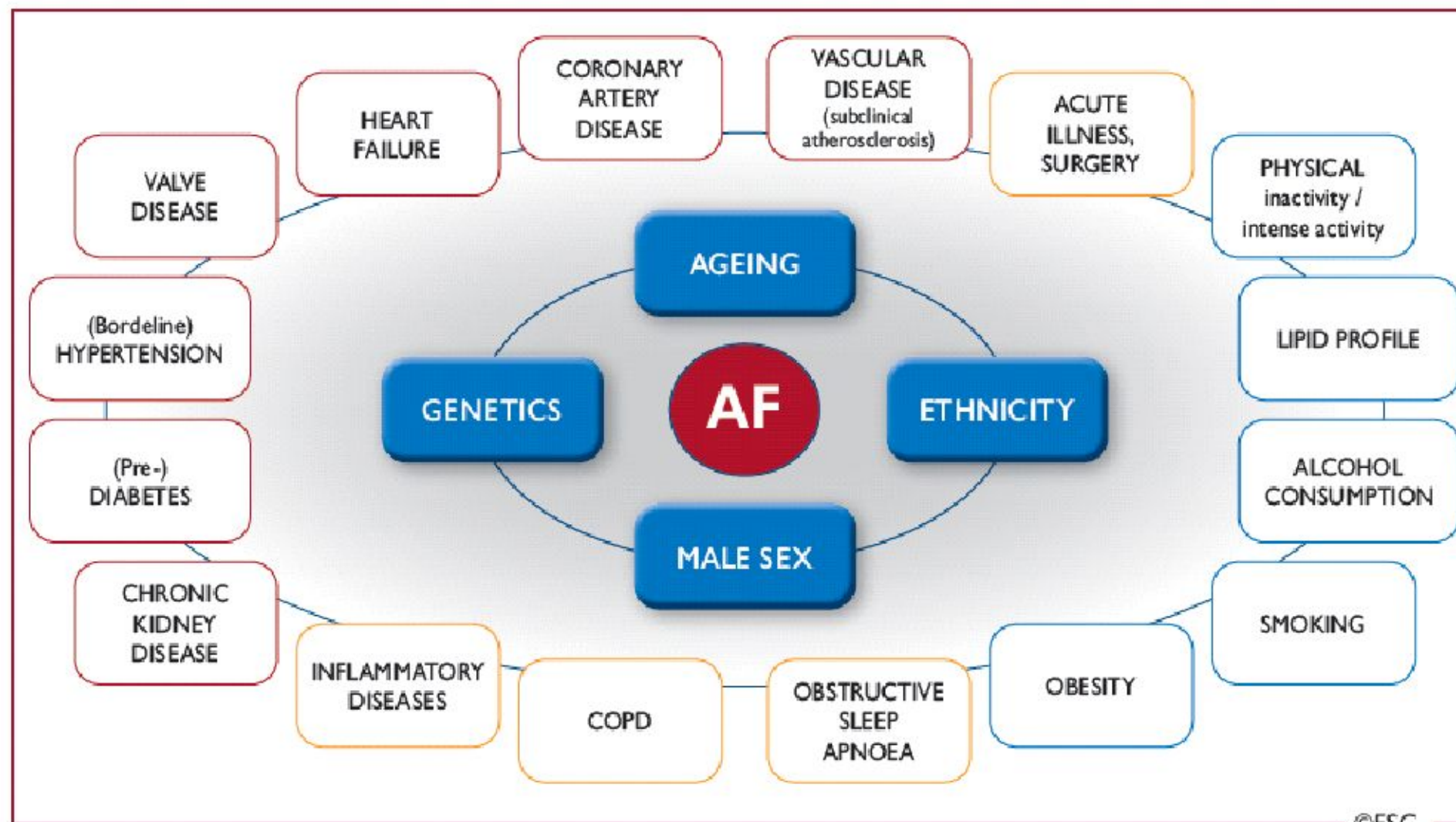


Projected increase in AF prevalence among elderly in EU 2016-2060



Lifetime risk of AF increases with increasing risk factor burden*





Clinical Presentation



Asymptomatic or
Silent (!)



Symptomatic

Palpitations, dyspnoea,
fatigue,

Chest tightness/pain,
poor effort tolerance,
dizziness, syncope,
disordered sleep, etc

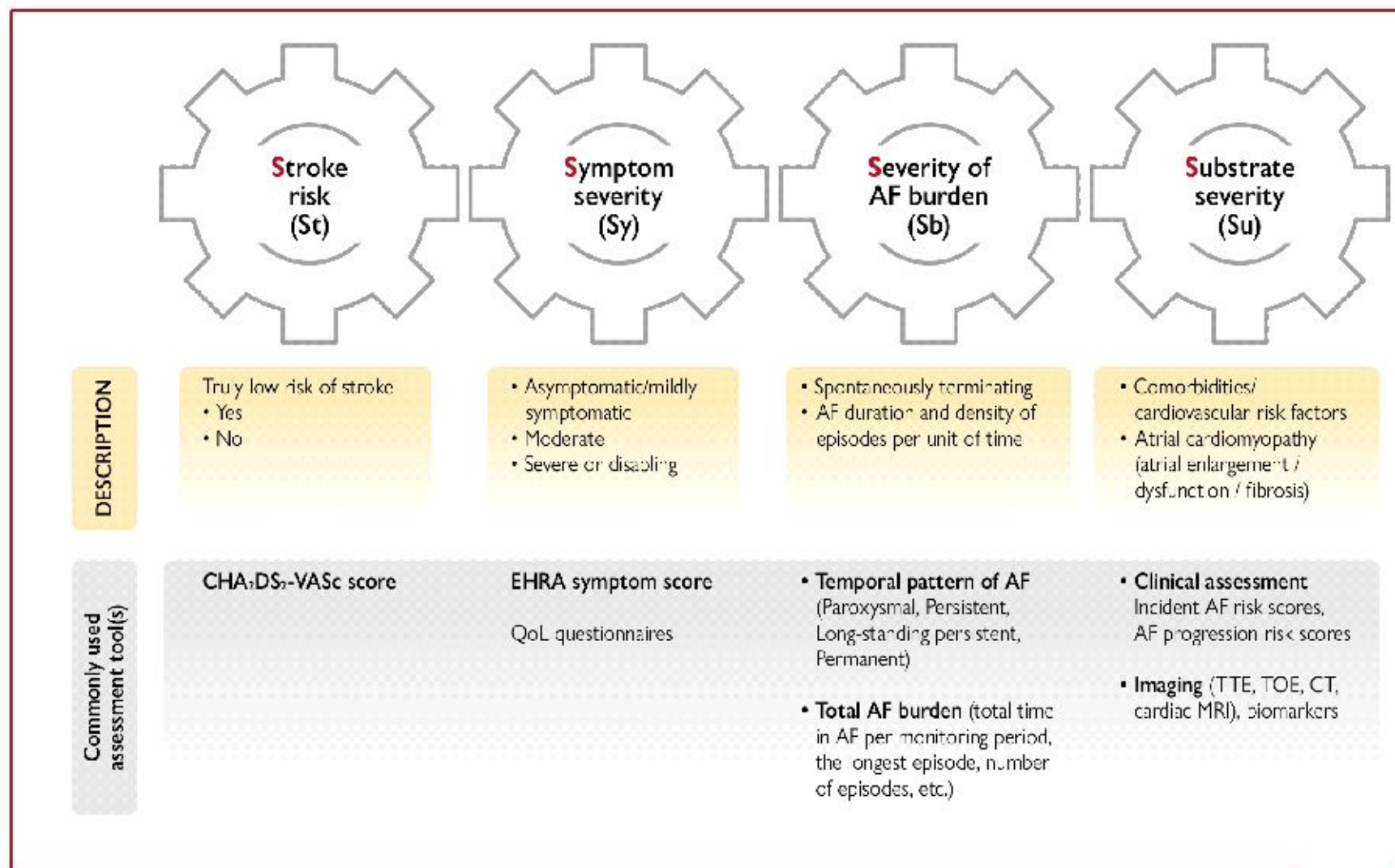
**Haemodynamically
unstable**

- Syncope
- Symptomatic hypotension
- Acute HF, pulmonary oedema
- Ongoing myocardial ischaemia
- Cardiogenic shock

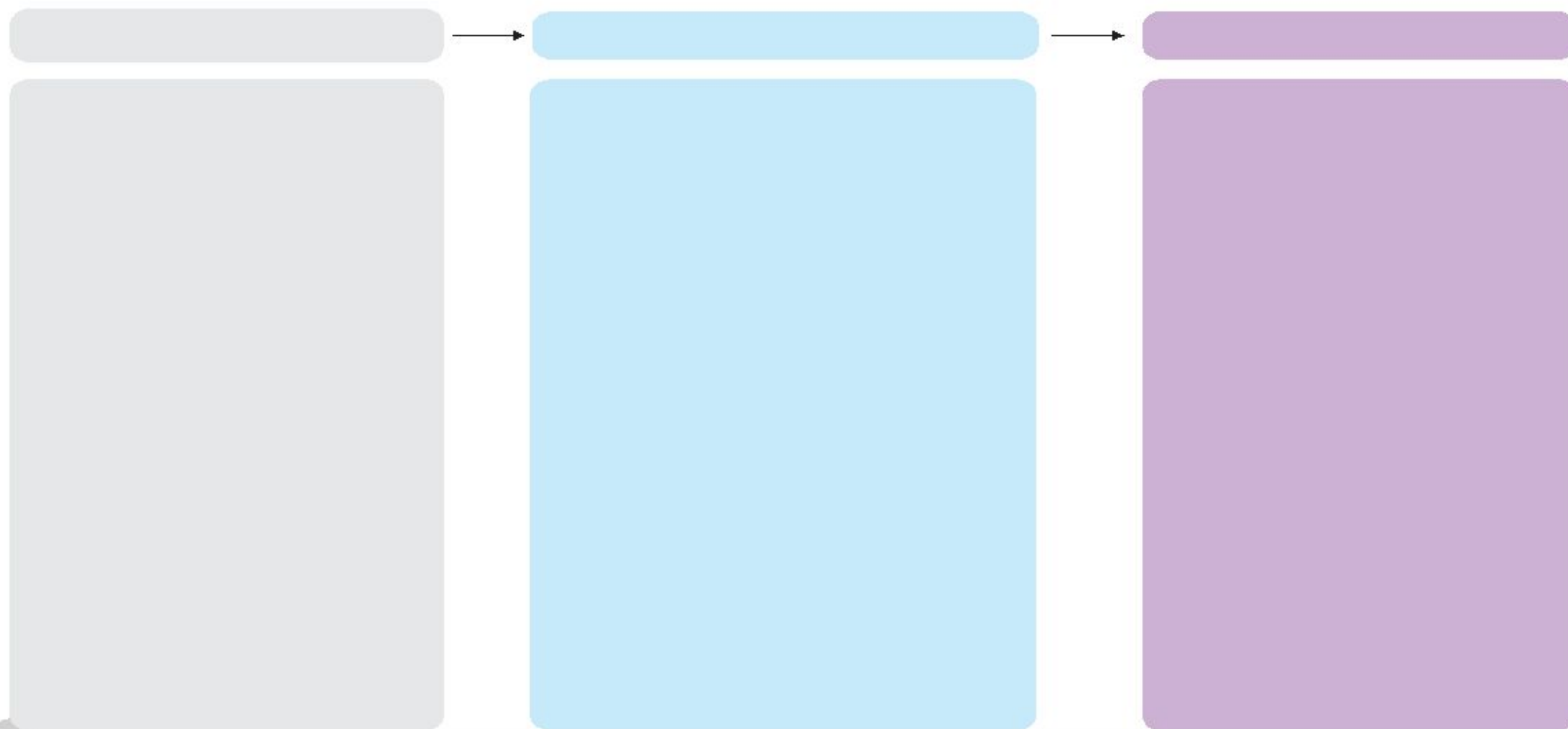
Haemodynamically stable

AF-related OUTCOMES

AF-Related Outcome	Frequency in AF	Mechanism(s)
 Death	1.5-3.5 fold increase	Excess mortality related to: • HF, comorbidities • Stroke
 Stroke	20-30% of all ischaemic strokes, 10% of cryptogenic strokes	• Cardioembolic, or • Related to comorbid vascular atheroma
 LV dysfunction / Heart failure	In 20-30% of AF patients	• Excessive ventricular rate • Irregular ventricular contractions • A primary underlying cause of AF
 Cognitive decline / Vascular dementia	HR 1.4 / 1.6 (irrespective of stroke history)	• Brain white matter lesions, inflammation, • Hypoperfusion, • Micro-embolism
 Depression	Depression in 16-20% (even suicidal ideation)	• Severe symptoms and decreased QoL • Drug side effects
 Impaired quality of life	>60% of patients	• Related to AF burden, comorbidities, psychological functioning and medication • Distressed personality type
 Hospitalizations	10-40% annual hospitalization rate	• AF management, related to HF, MI or AF related symptoms • Treatment-associated complications







Left atrial remodelling
associated with AF

Anatomy

Dilatation and change in geometry

Structure

Fibrosis

Function

Altered electrophysiology,
LA reservoir, conduit and booster
pump function

LA/LAA thrombus detection

Value of LA imaging
techniques in AF

+

Value of imaging
techniques in AF

TEE and TOE



Cardiac CT



Cardiac MRI



EP mapping



LV size, geometry and
function assessment

Heart valves morphology
and function

Right-heart chambers and
pericardium imaging

Advanced/Investigation imaging:

- Echocardiographic TDI and LA strain, etc.
- MRI delayed enhancement or T1 imaging
- CT imaging of substrate, etc.

INTEGRATED AF MANAGEMENT



Patient-centred

Optimised stroke prevention

Symptom control with rate or rhythm control

Management of cardiovascular risk factors/comorbidities

Patient education/self-management
(including personal goals and/or action plan,
exacerbation management)

Healthcare professional education

Lifestyle modification
(i.e., smoking cessation, dietary intervention
to lose weight, exercise)

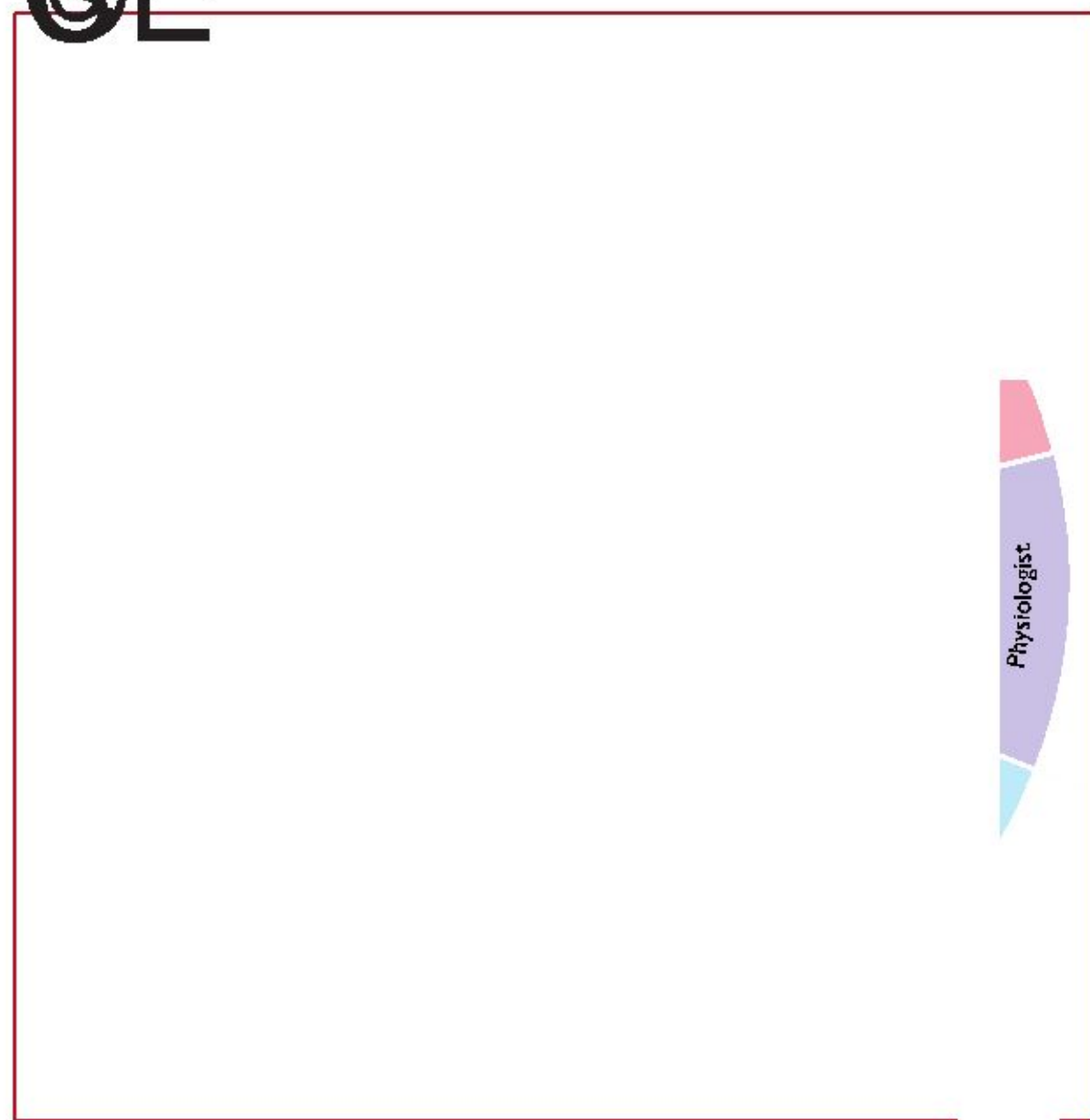
Psychosocial management
(cognitive behavioural therapy, stress management,
other psychological assessment and/or treatment)

Strategies to promote medication adherence

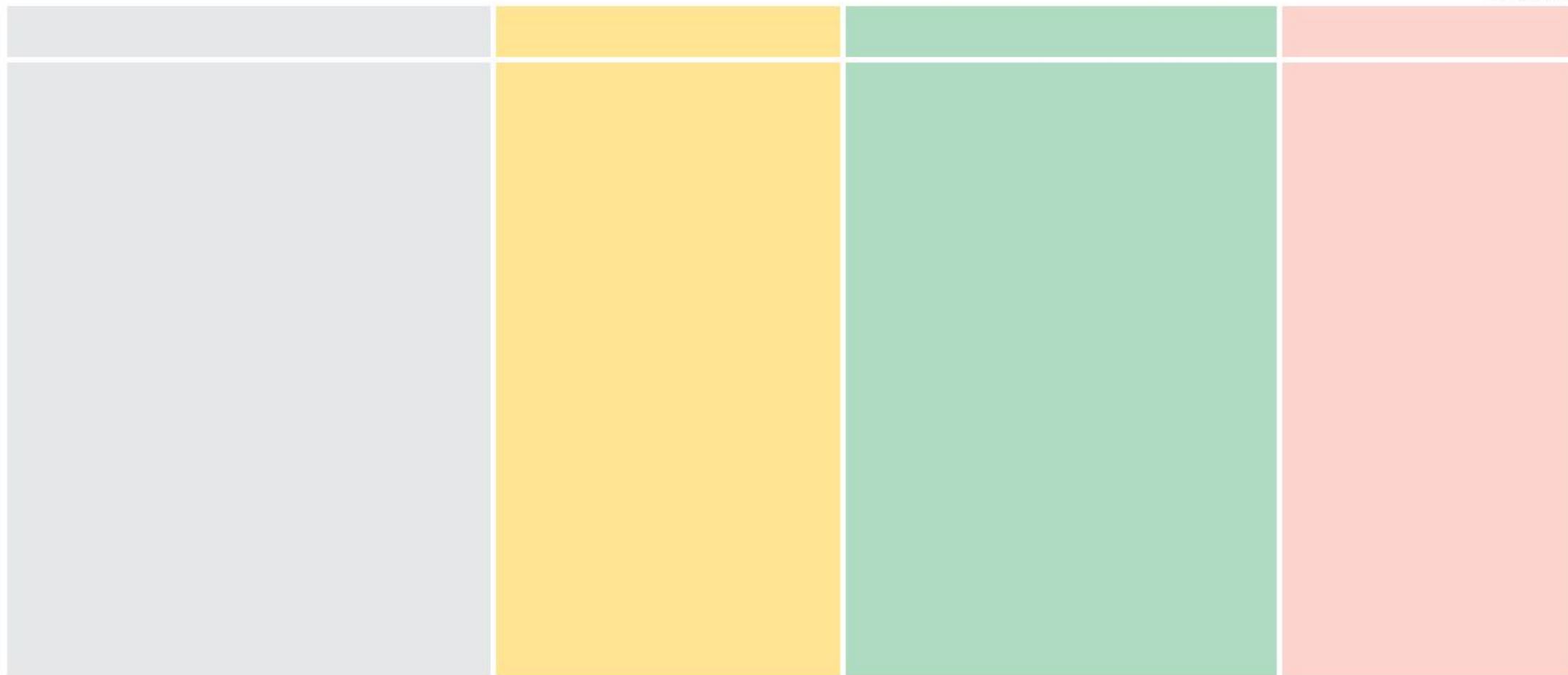
Multidisciplinary team approach

Active participation and formation of teams of HCPs from different disciplines; integration of services. MDT meeting (as needed)

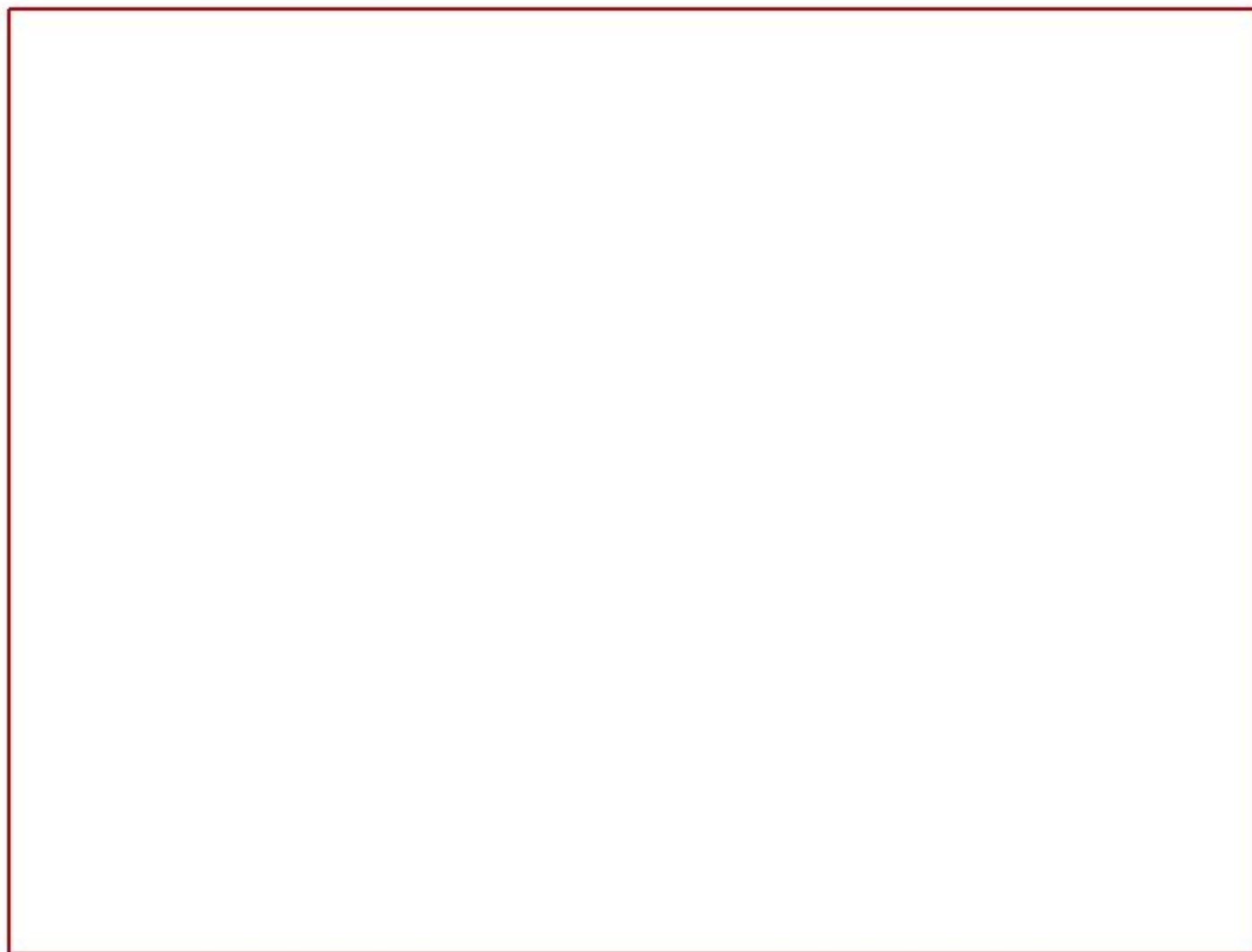
Structured follow-up and clear communication between primary and secondary care

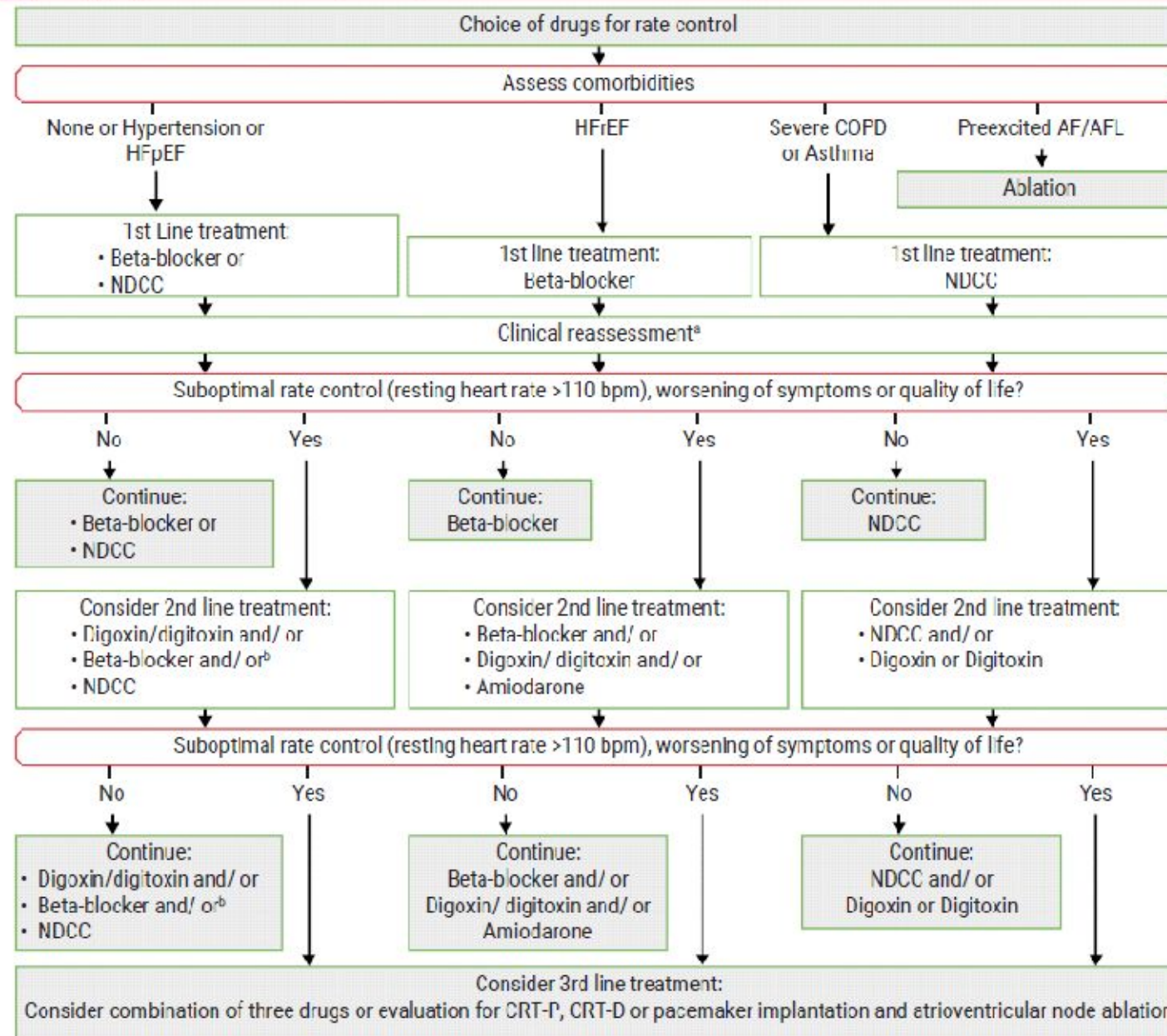


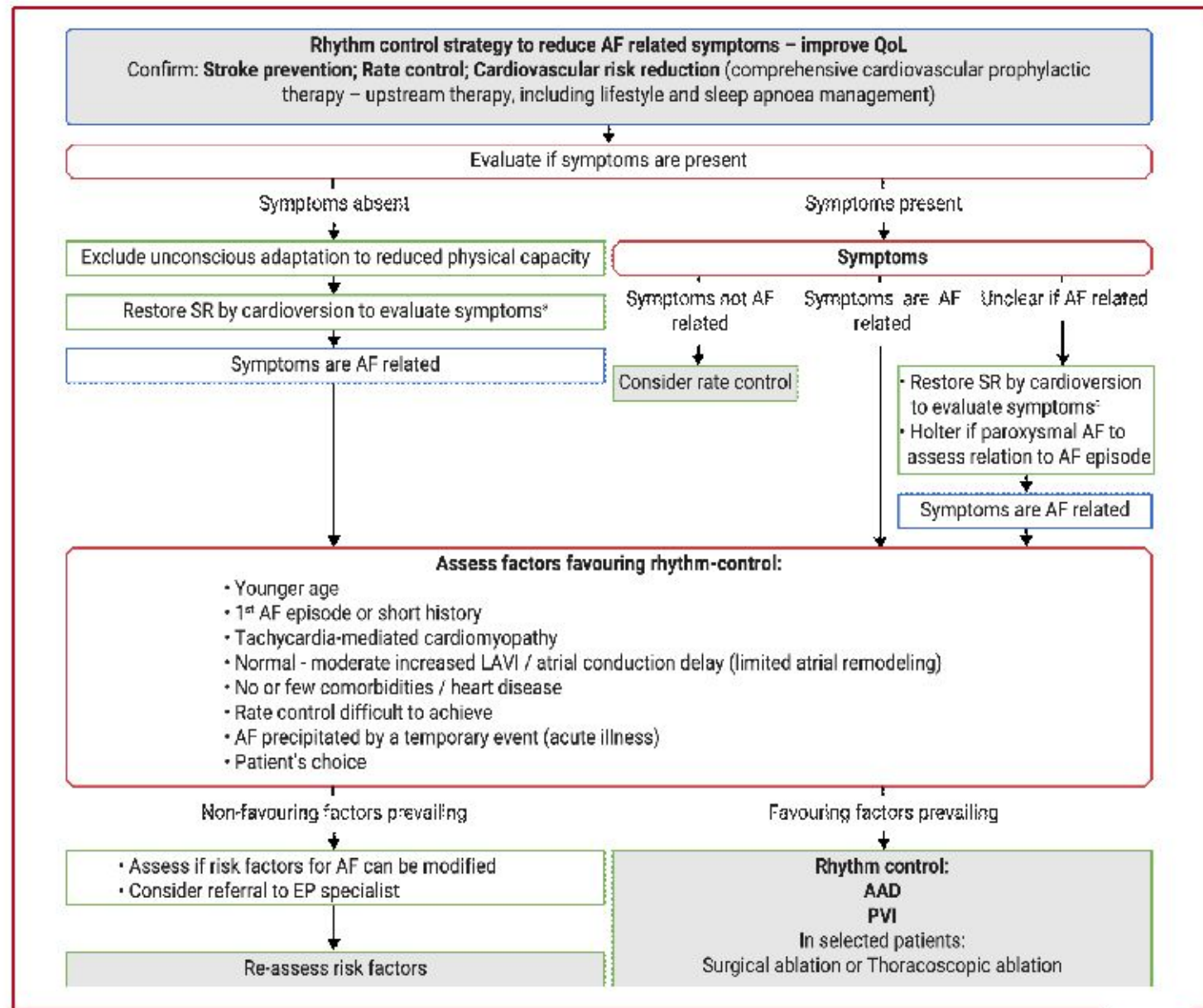
Physiologist

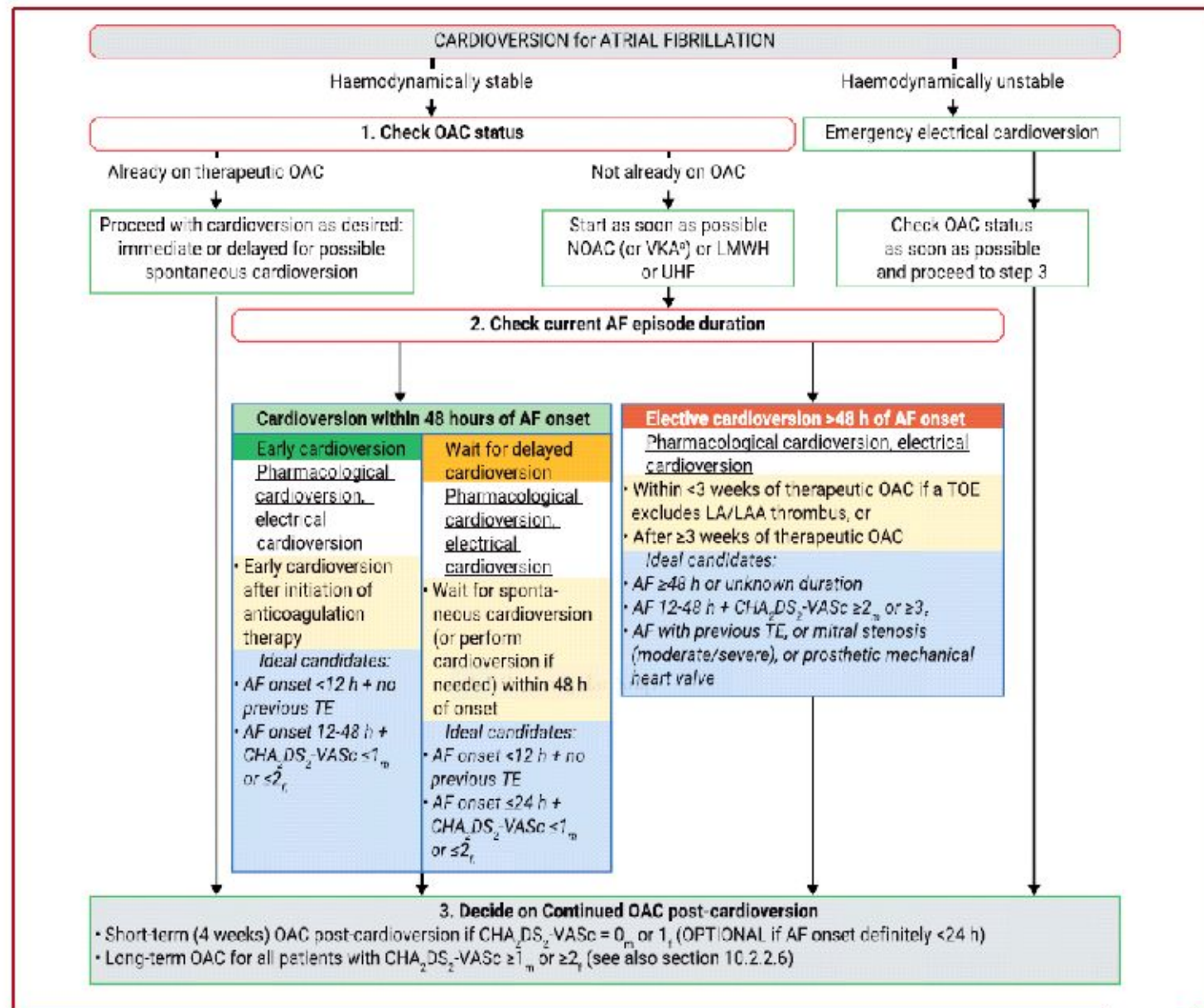


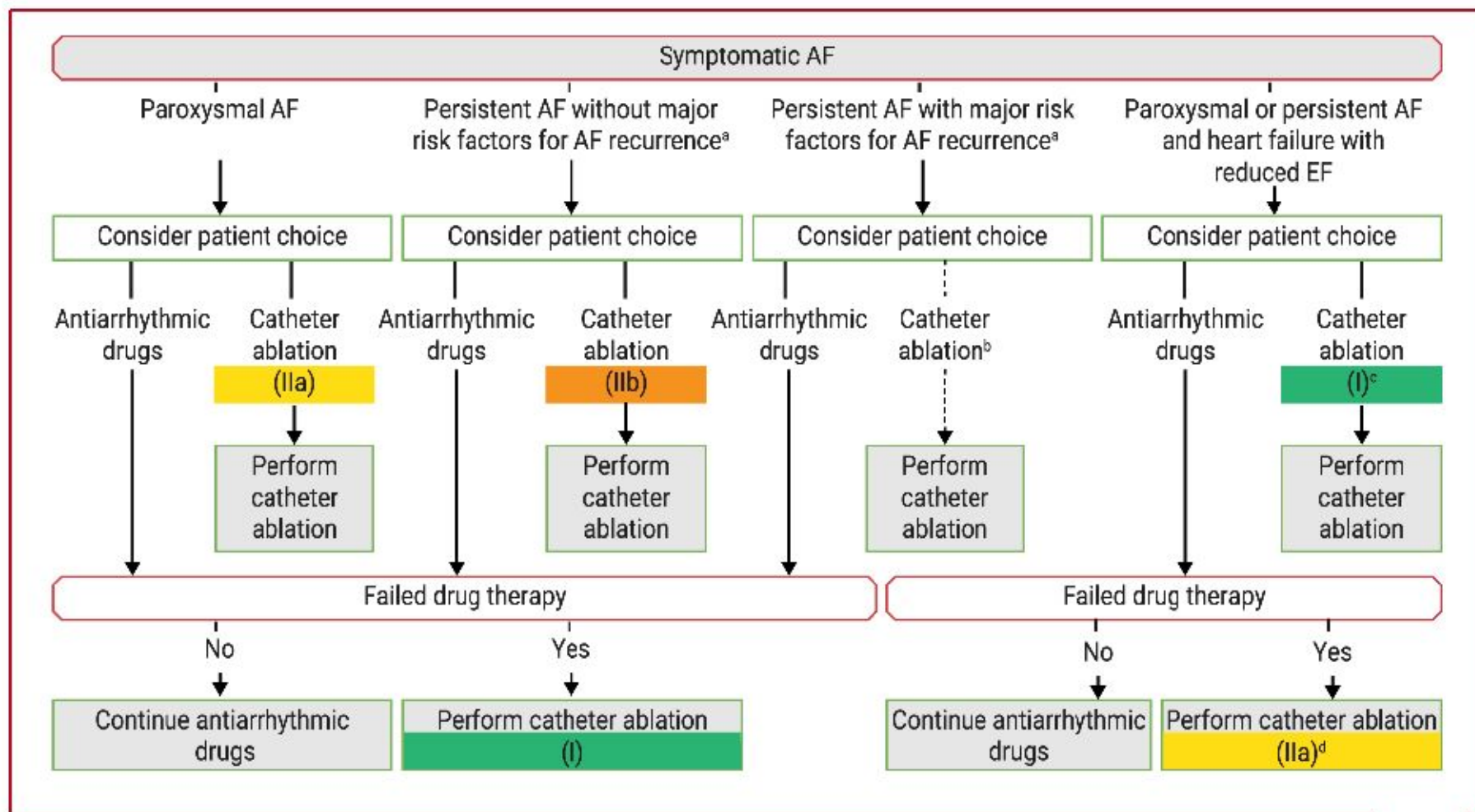
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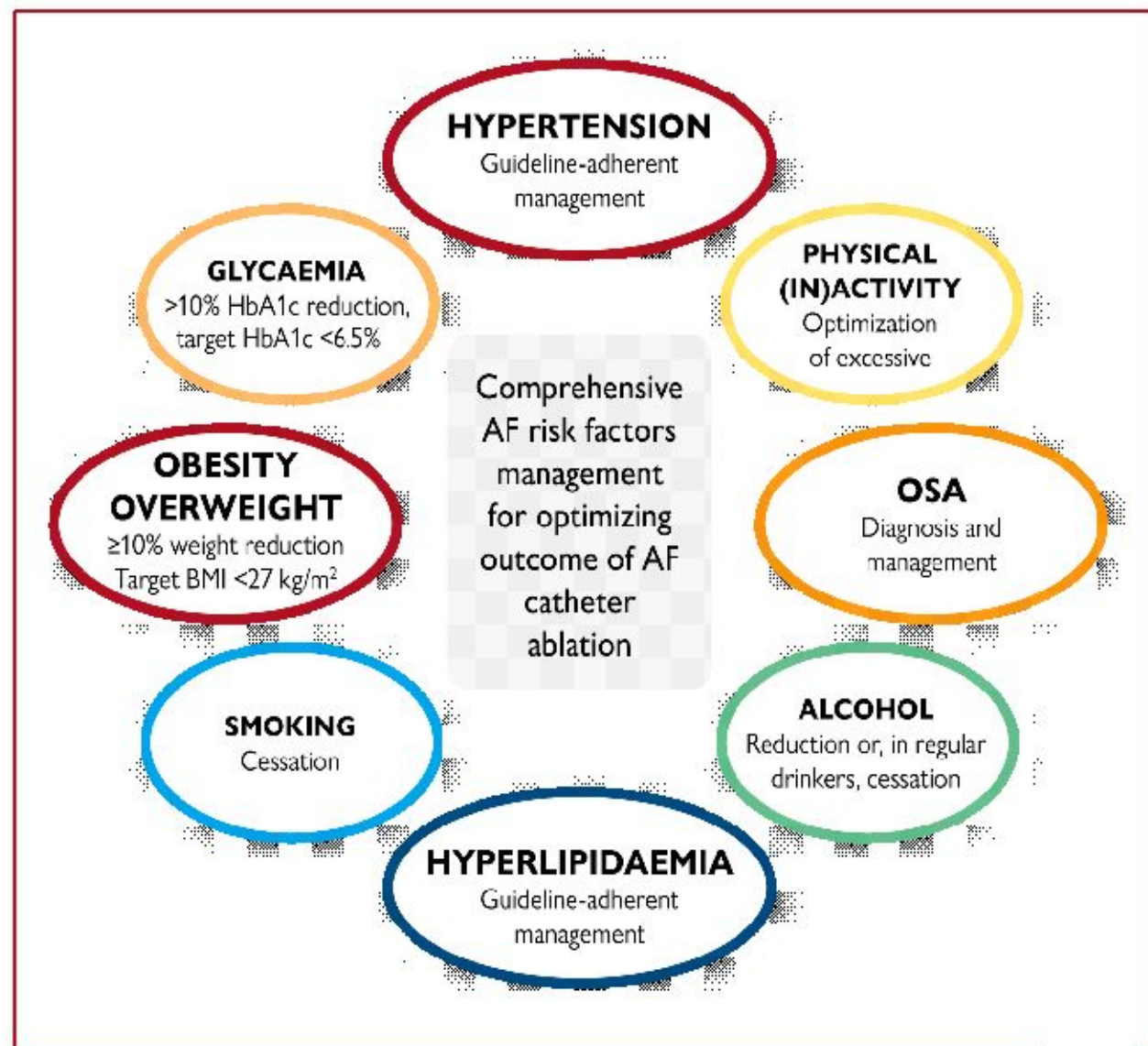


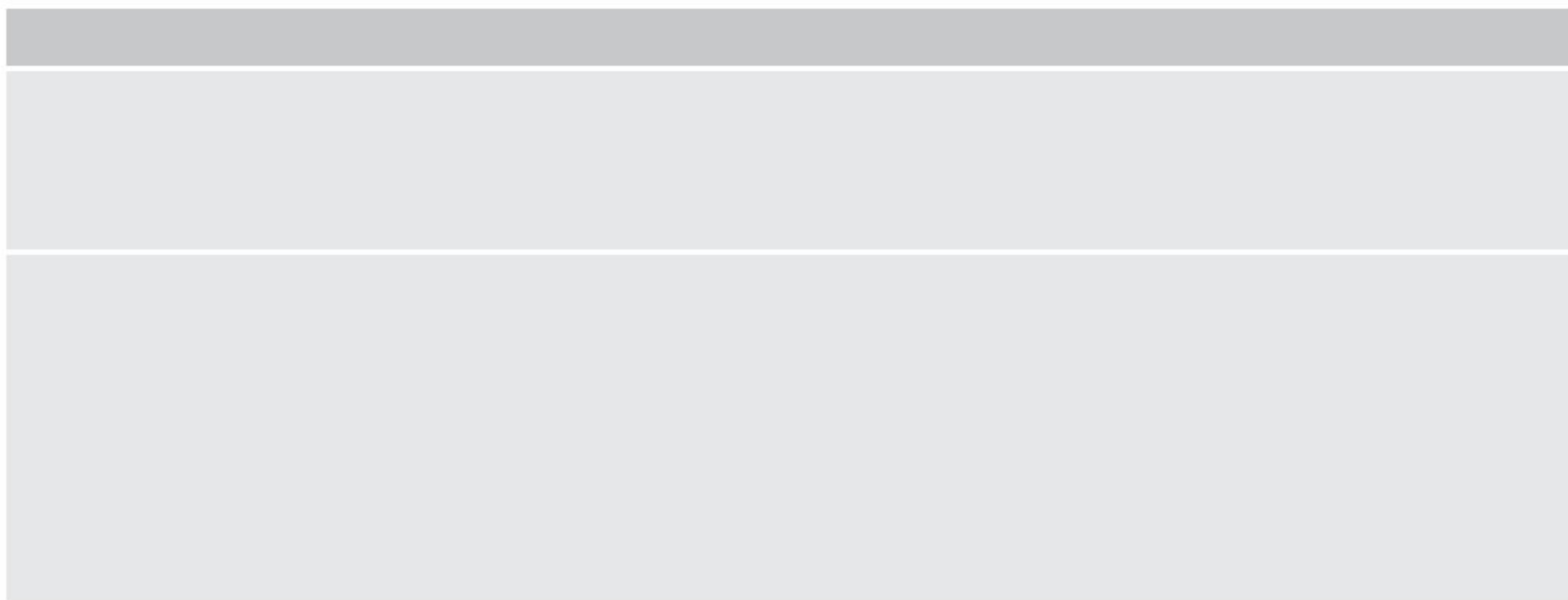


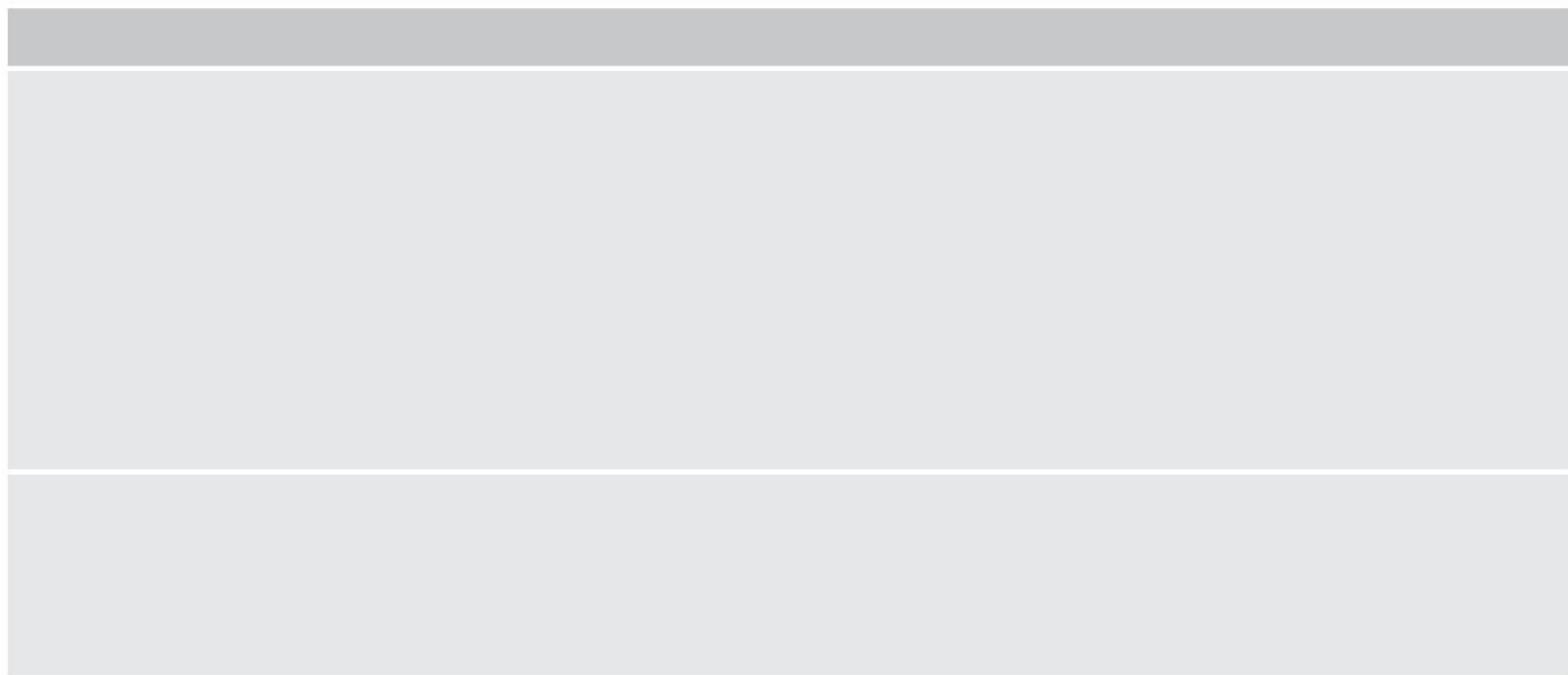








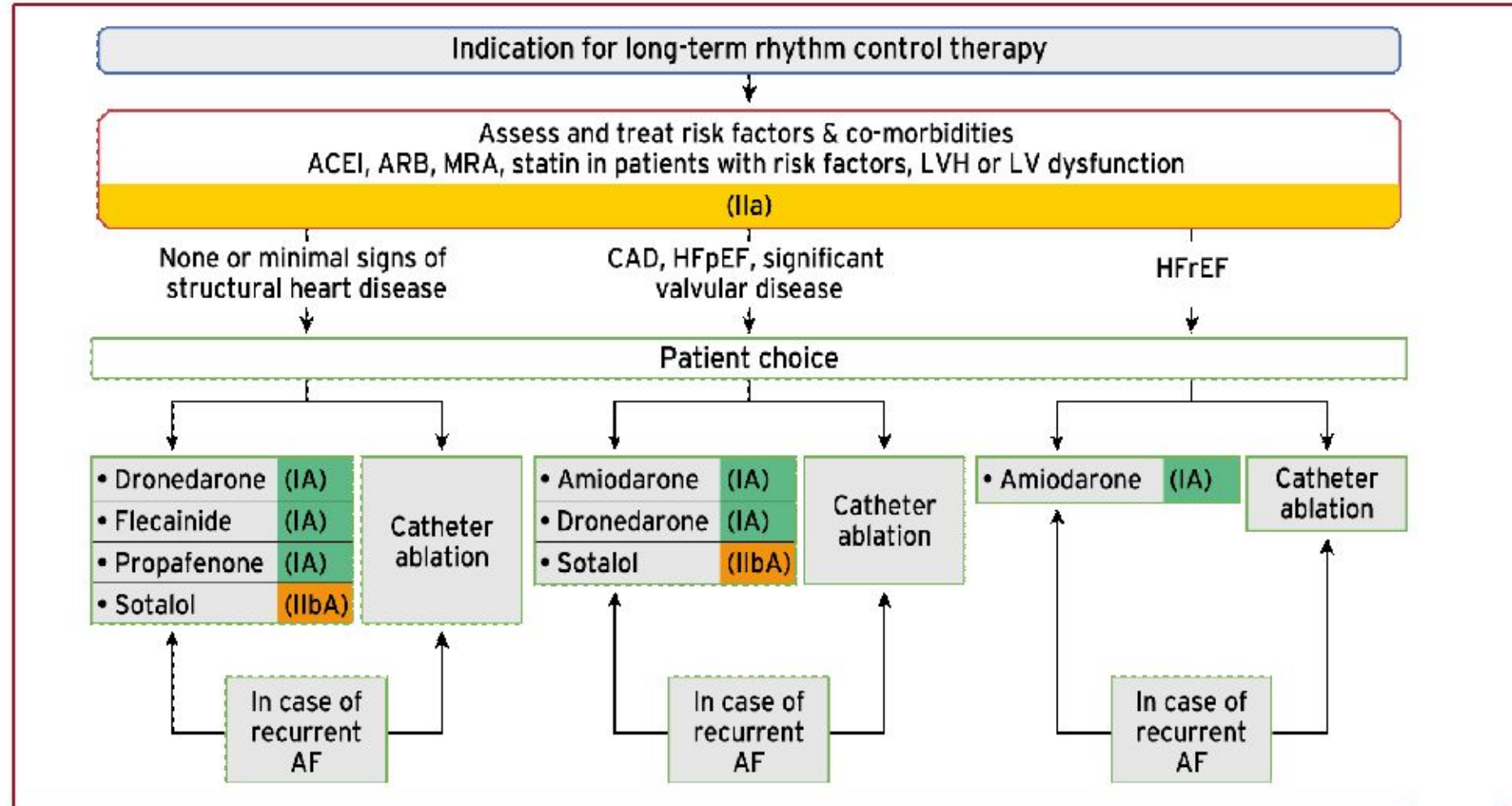


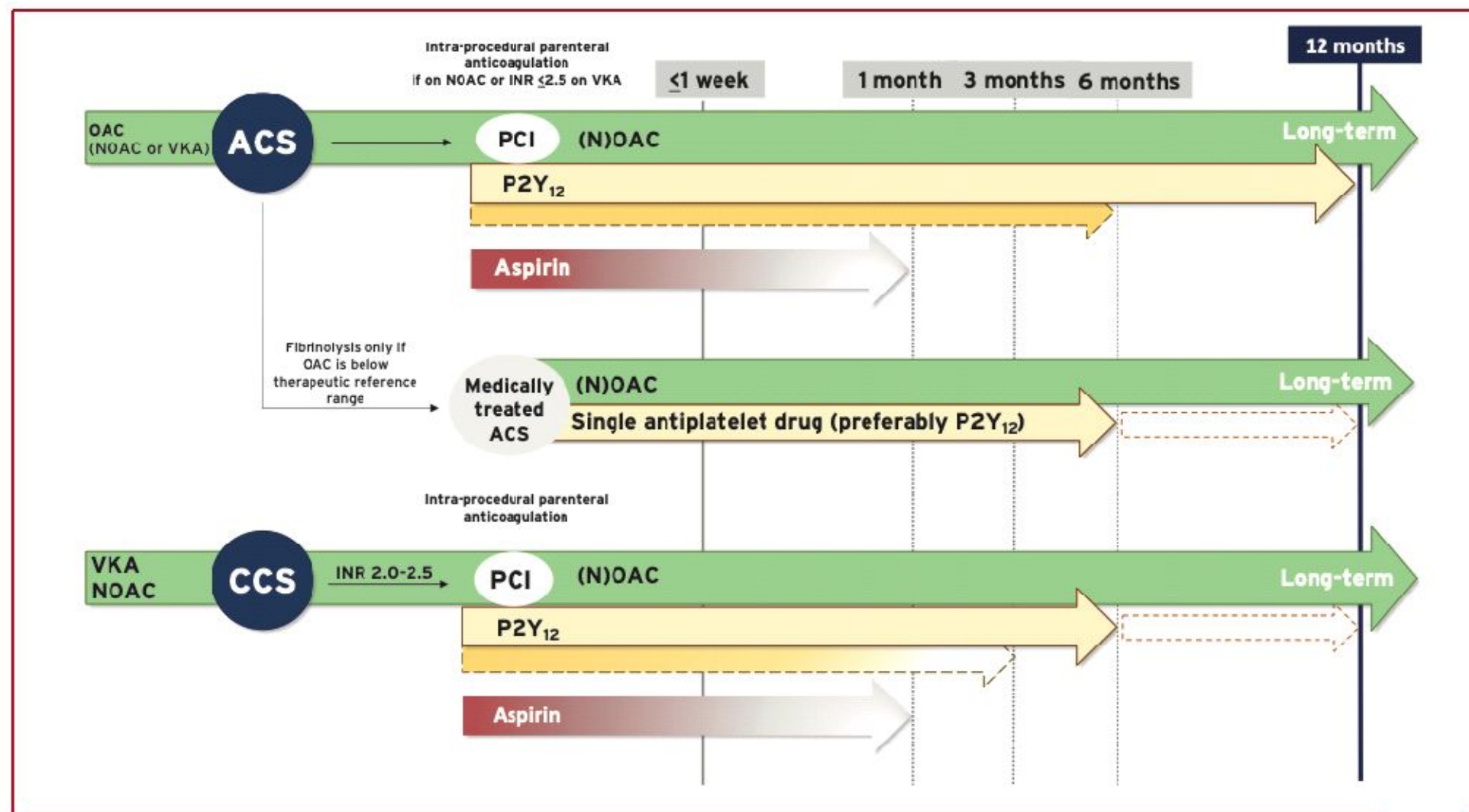


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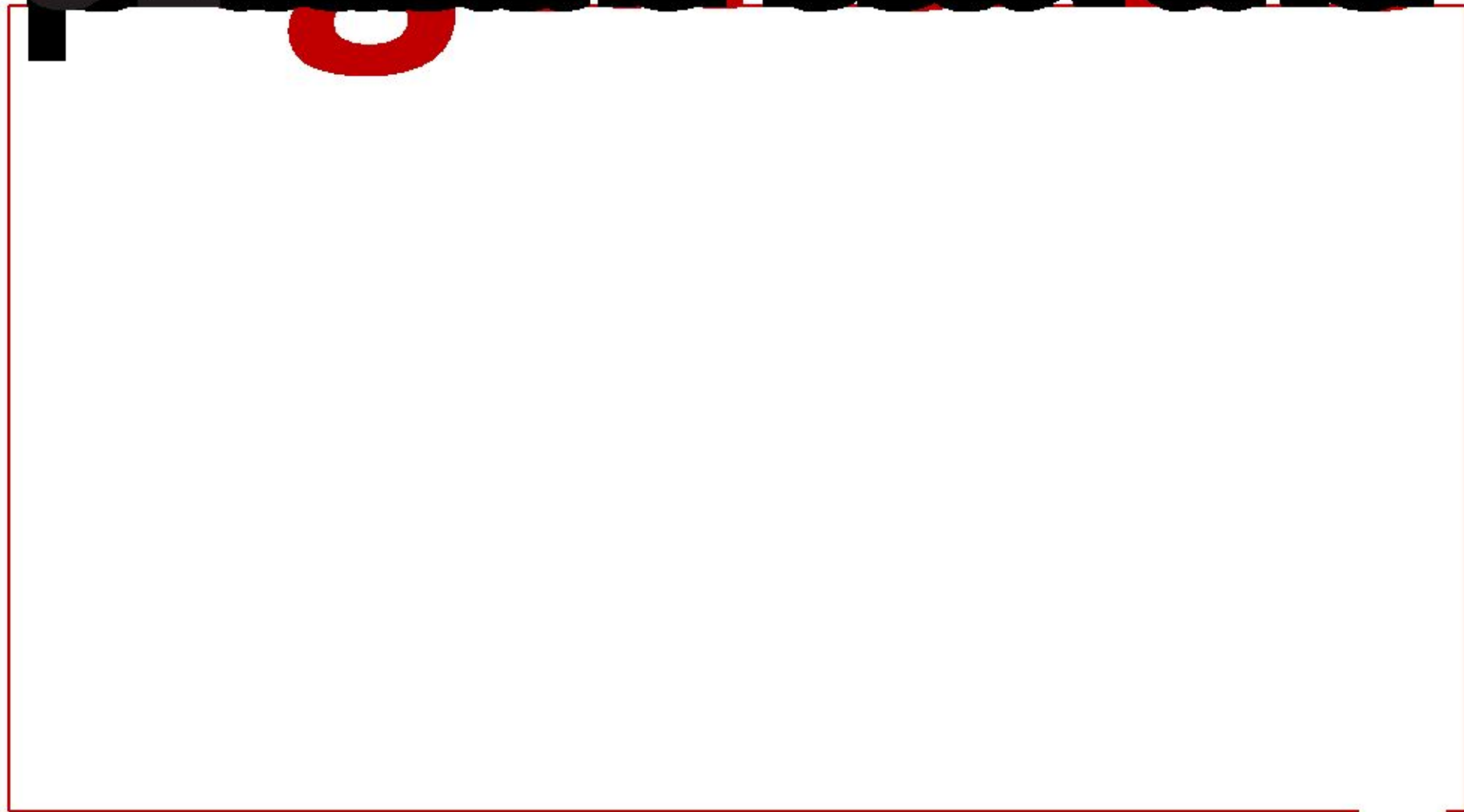
Recommendations for stroke risk management peri catheter ablation (1)

Recommendations	Class	Level
In AF patients with stroke risk factors not taking OAC before ablation, it is recommended that preprocedural management of stroke risk includes initiation of anticoagulation and: • Preferably, therapeutic OAC for at least 3 weeks before ablation, or • Alternatively, the use of TOE to exclude LA thrombus before ablation.	I	C
	IIa	C
For patients undergoing AF catheter ablation who have been therapeutically anticoagulated with warfarin, dabigatran, rivaroxaban, apixaban, or edoxaban, performance of the ablation procedure without OAC interruption is recommended.	I	A

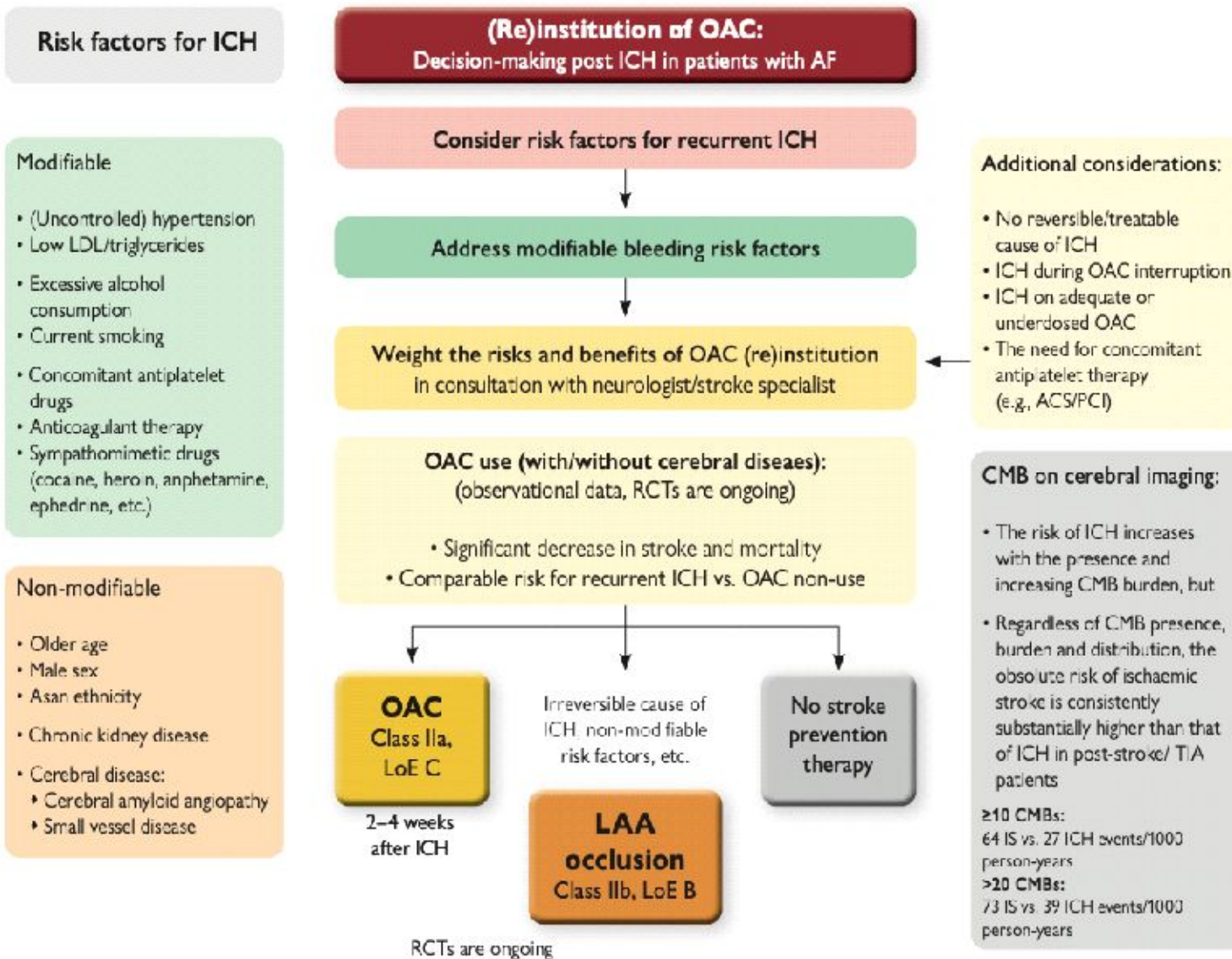


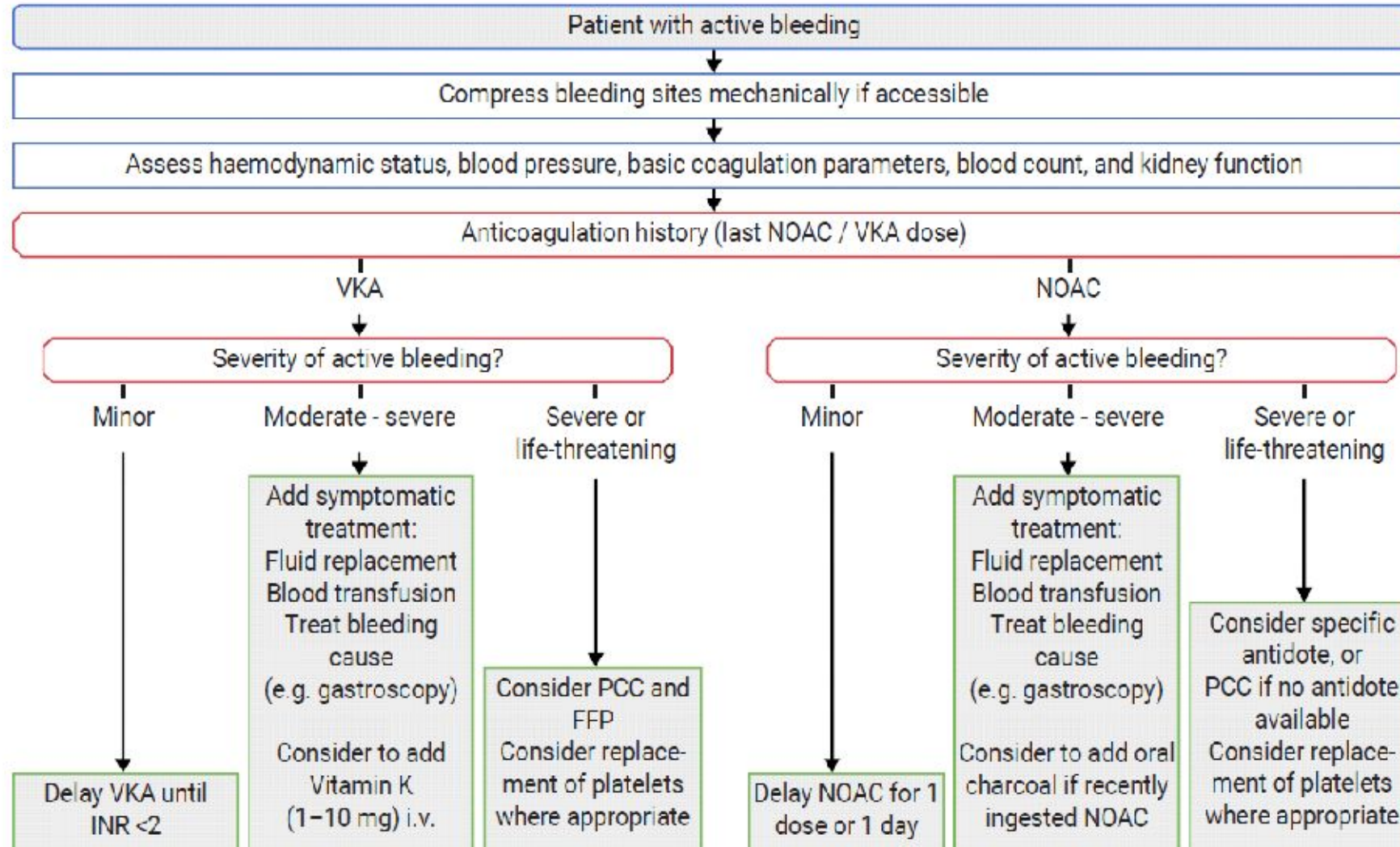


SECRET



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ESC

European Society
of Cardiology



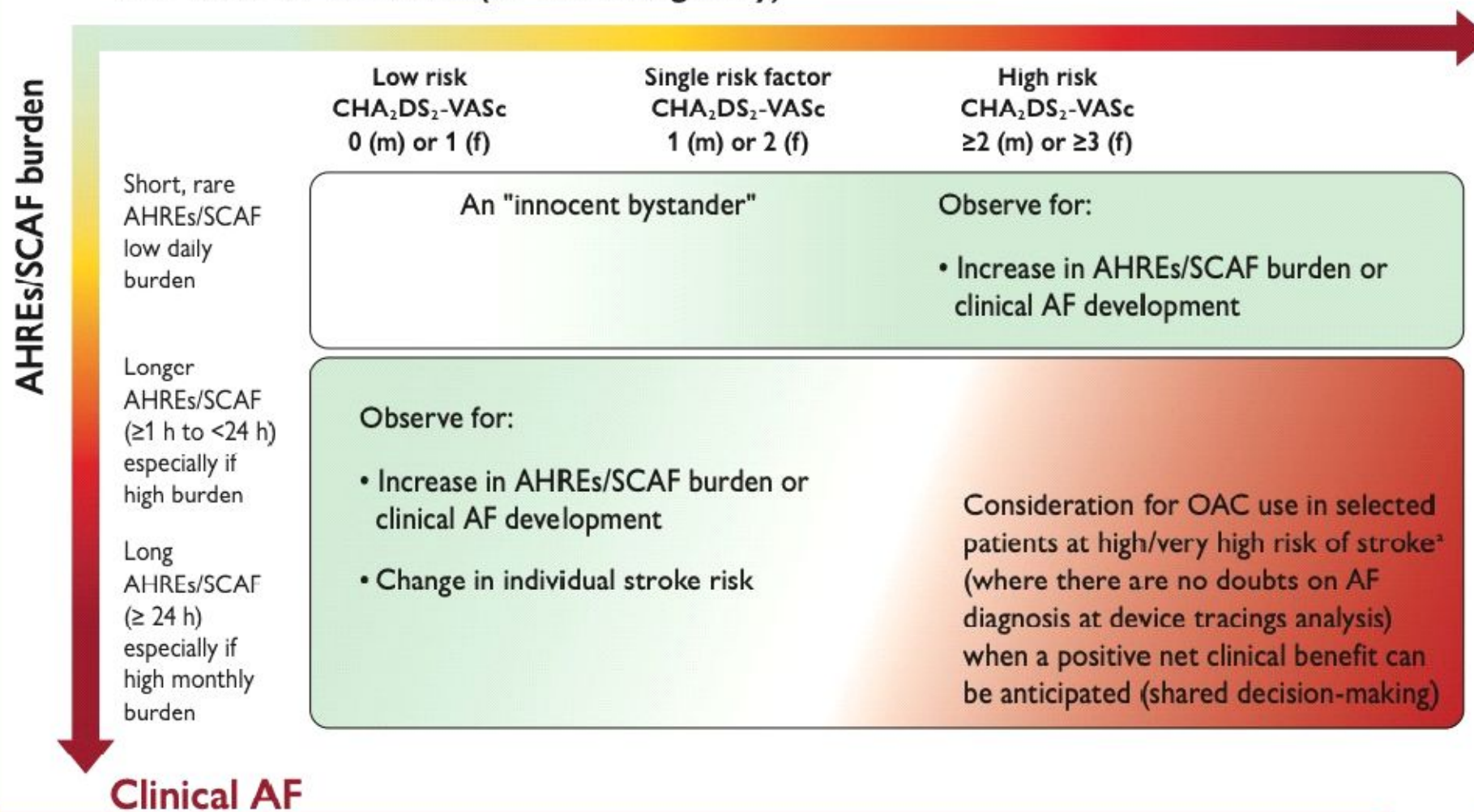
Six-month incidence of transition to higher AHRE burden^a
 (n = 6580, pooled from three prospective studies)

	<i>Baseline burden</i>			
6-month progression	5 min to <1 h	1 h to <6 h	6 h to <12 h	12 h to <23 h
Transition to ≥1 h	33.5%			
Transition to ≥6 h	15.3%	42.2%		
Transition to ≥12 h	8.9%	27.5%	55.8%	
Transition to ≥23 h	5.1%	16.0%	40.6%	63.1%

Stroke rates^b per AHRE burden and CHA₂DS₂-VASc category
 (n = 21 768 device patients not taking OAC)

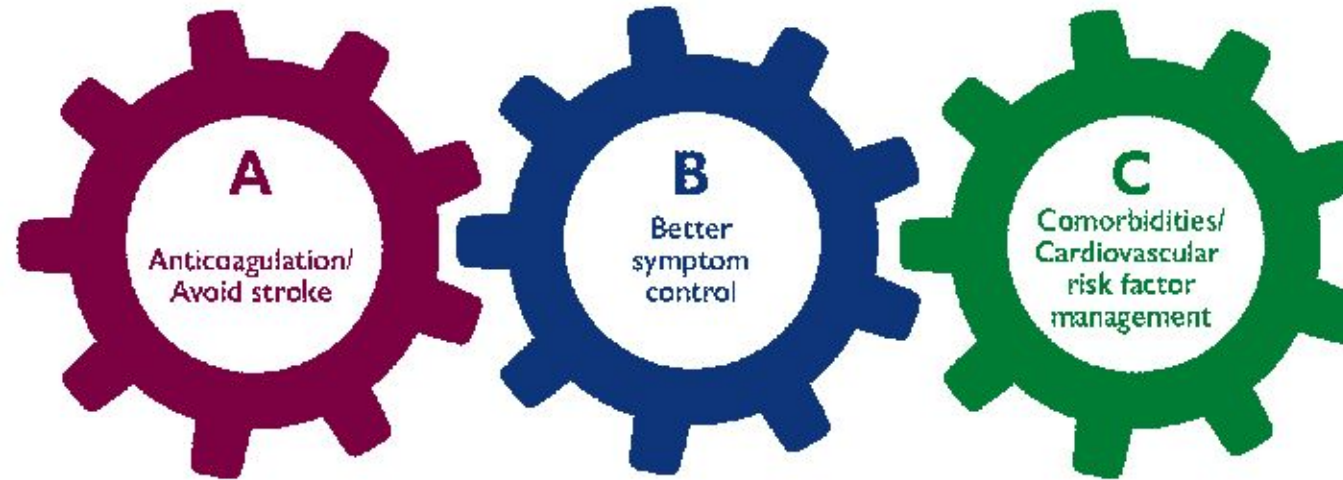
	<i>Baseline maximum daily burden</i>		
CHA ₂ DS ₂ -VASc score	No AF	AF 6 min–23.5 h	AF >23.5 h
0	0.33%	0.52%	0.86%
1	0.62%	0.32%	0.50%
2	0.70%	0.62%	1.52%
3-4	0.83%	1.28%	1.77%
≥5	1.79%	2.21%	1.68%

THE RISK OF STROKE *(re-assess regularly)*





Treat AF: The ABC pathway



1. Identify low-risk patients
CHA₂DS₂-VASc 0(m), 1(f)
2. Offer stroke prevention if
CHA₂DS₂-VASc ≥1(m), 2(f)
Assess bleeding risk, address
modifiable bleeding risk factors
3. Choose OAC (NOAC or VKA
with well-managed TTR)

Assess symptoms,
QoL and patient's
preferences

Optimize rate
control

Consider a rhythm
control strategy
(CV, AADs, ablation)

Comorbidities and
cardiovascular risk
factors

Lifestyle changes
(obesity reduction,
regular exercise,
reduction of alcohol use,
etc.)



