AI with a Clinician's Lens

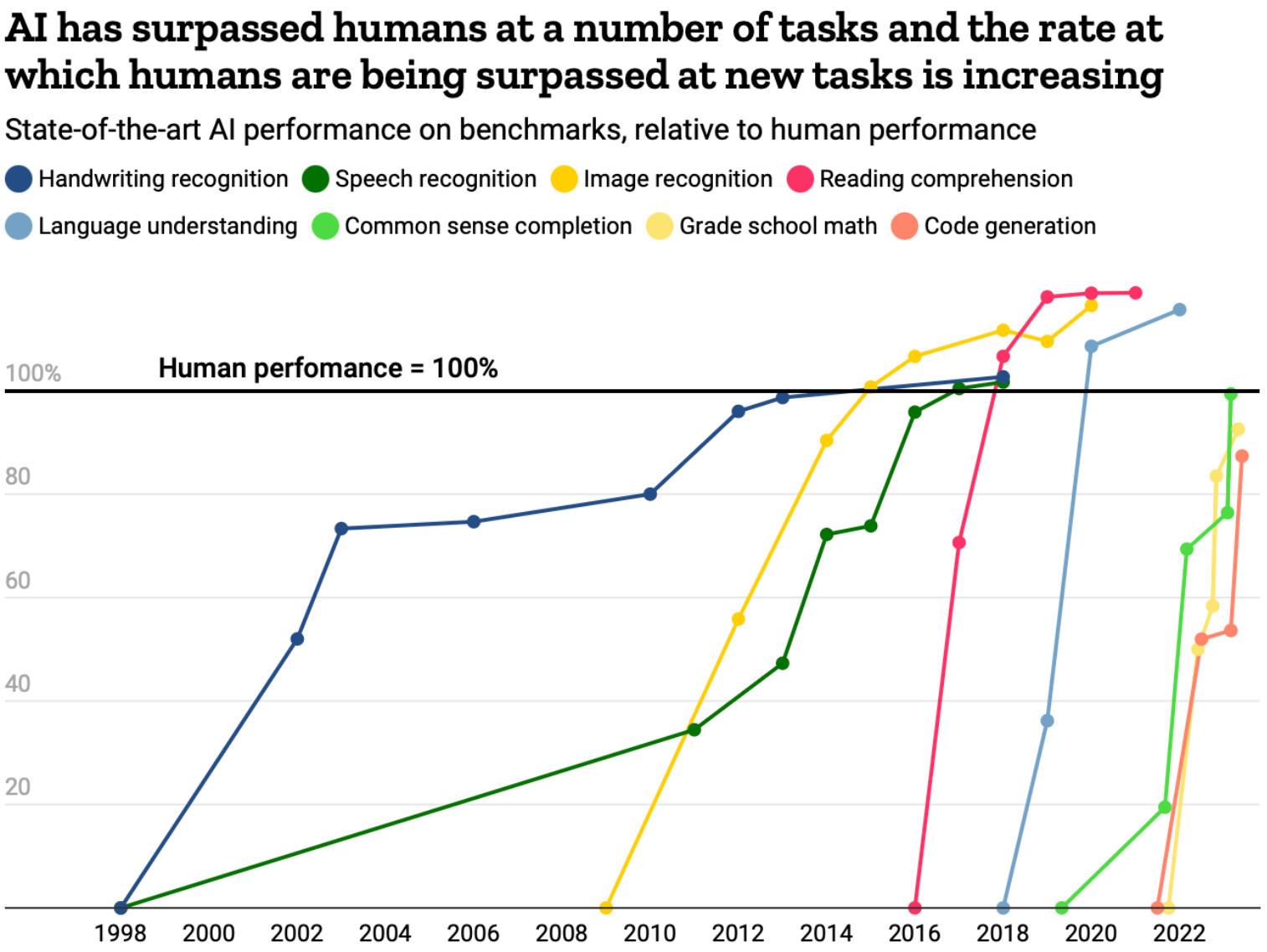
Lynden Roberts
Chief Medical Information Officer
Rheumatologist
Monash Health

Human intelligence is amazing, but not uniquely so

- Squirrels' spatial memory: hide by burying 1000’s of nuts and retrieve them month’s later using memory
- Chimpanzee’s outperform humans in short term memory number recall

Intelligence has many domains

AI has surpassed humans in a number of these



	Artificial Intelligence	Human Intelligence
Thinking speed	AI processes data using algorithms and mathematical models. It can process vast amounts of data much faster than humans.	Humans process data using cognitive processes within biological structures. Human cognition is slower in processing large amounts of data, but can make complex decisions quickly even with limited data.
Learning & adaptability	AI learns based on data and feedback loops. AI can rapidly adapt and learn when given new data.	Humans learns based on experience, intuition, and creativity. Humans adapt to new situations and make decisions based on context.
Accuracy	AI accuracy depends on volume and quality of training data. Unable to perform common sense thinking. Hallucinations noted with deep neural nets.	Human decision making is affected by cognitive bias. Humans can display common sense thinking.
Emotions	AI lacks emotions and empathy.	Humans feel emotions and empathy.
Creativity	AI has limited ability to be creative or think outside of the box.	Humans are creative, have imagination, and innovation.
Ethics	AI does not have a moral code or conscience.	Humans have a moral code and conscience that guides decision-making.
Physical Limitations	AI can operate 24/7	Humans is limited by physical capabilities and requires rest and maintenance.
	AI is a human creation, developed through complex	Humans are a natural phenomenon, evolved over millions of years. Humans are capable of changing

Does Healthcare need AI?

- Over next 10-15 years we will have:
  - 2x Demand
  - 1.1x workforce
  - 1.1x money
  - AND current system is failing on targets and expectations

So… we are completely stuffed … or are we?

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITA

Case 8-2025: A 72-Year-
Mental Status and Acid

Authors: Petra Simic, M.D., Ph.D., David M. Dudzinsk
M.D., Ph.D. Author Info & Affiliations

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What's the differential diagnosis?

Figure 24.3 *An early CPC in the Allen Street Amphitheatre: Hugh Cabot is standing, and Oscar Richardson is seated at the table*

What can Gen AI already do clinically?

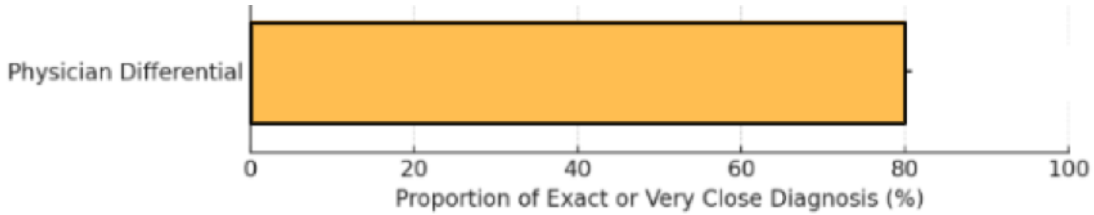

Figure 1: Barplot showing the accuracy of including the correct diagnosis in the differential for MD-PIE, the LLMs and physicians on the NEJM CPCs

Yasma Esteitieh[1], Shaurjya Mandal[2], and George Laliotis[3] https://doi.org/10.1101/2025.01.28.25321282

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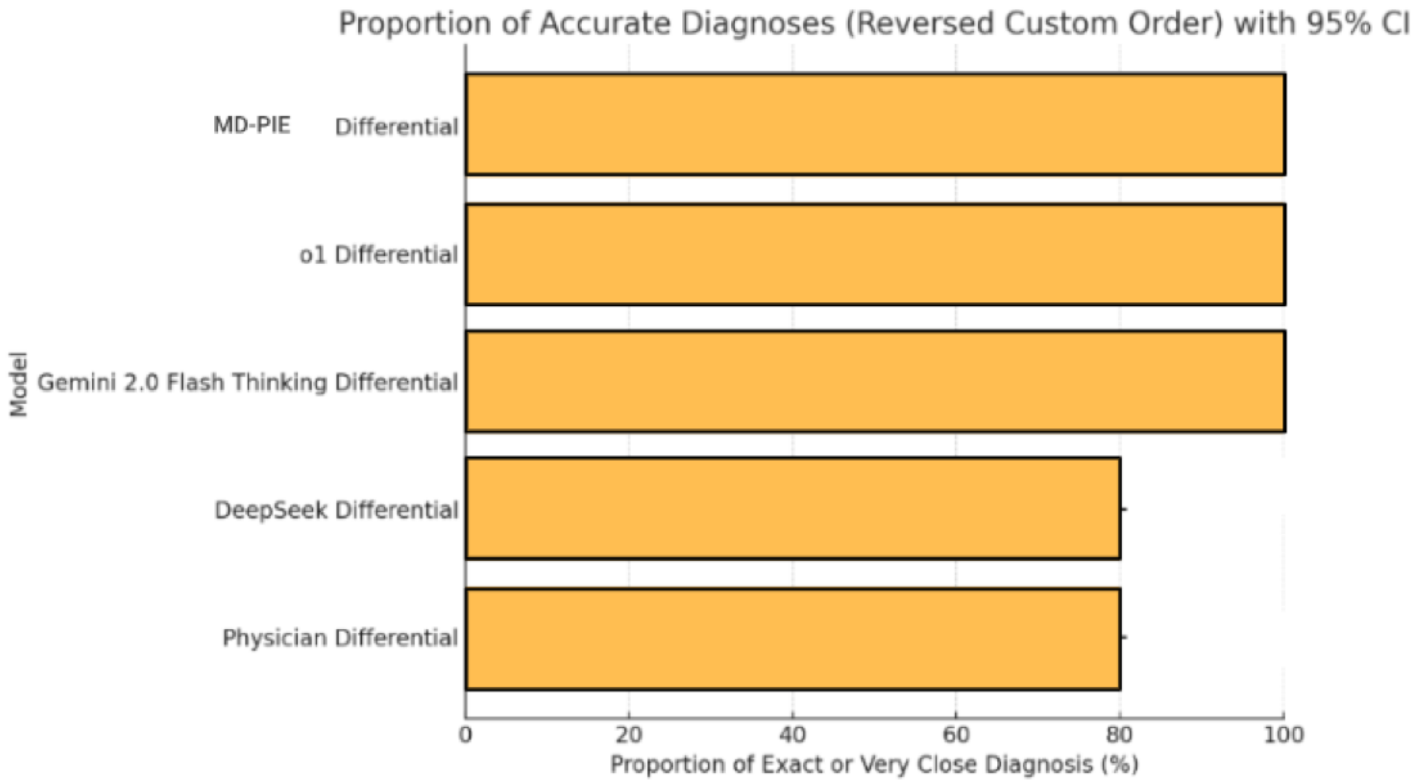

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Australian Rheumatology published Grand Round case

70 year old man presents with 1 month history of fevers, night sweats, weight loss (27kg over 4 months) and anaemia on the background of 7 year history of panniculitis Diagnosed in 2010 on skin biopsy – recurrent painful skin lumps Previous biopsies have shown septal panniculitis, ie erythema nodosum pattern, no vasculitis or granulomas Originally saw immunologist and multiple dermatologists. No associated autoimmune, infectious or haematological disease identified. Self managed with prednisone – up to 75mg daily Previously trialed Hydroxychloroquine, Saturated Solution of Potassium Iodide Trialed on Dapsone in late 2016 (increased to 100mg) – ICU admission with Methaemoglobinaemia, Neutrophillic Dermatosis with skin necrosis, Acute Kidney injury, Anterior Uveitis, Hospital Acquired Pneumonia and prolonged delirium. Readmitted in April due to Pulmonary embolism, had flair of panniculitis treated with Prednisone 50mg Rheumatology involved: Commenced on Methotrexate 20mg April of 2017. Prednisone weaned Folliculitis over posterior neck and scalp in July Incidental painless thigh lesion biopsied showed vasculitis, ?cutaneous PAN Past medical history Diabetes mellitus Type 2 Prostate cancer 2011 with radical prostatectomy Hep B Core Ab positive OA of knees (L TKJR), previous cervical fusion Possible history of Gout, previously on Allopurinol but no flares for a long time Depression and Obsessive compulsive disorder Encephalitis age 19 – history unclear Genital herpes Medications Rivaroxaban 10mg daily Pantoprazole 40mg daily Irbesartan 150mg daily Ezetimibe 10mg nocte Metformin 1g tds Gliclazide 60mg bd Frusemide 20mg mane Paracetamol 1g tds Quetiapine 25mg nocte Escitalopram 30mg mane Mirtazepine 7.5mg nocte Methotrexate 15mg ceased Anaphylaxis to penicillin Social History Independent, lives with his wife, daughter and son-in-law Non-smoker, ex heavy alcohol use 70 year old man presents with 1 month history of fevers, night sweats and weight loss on the background of 7 year history of recurrent panniculitis On prednisone 7.5mg Daily and Methotrexate 20mg weekly. Associated Macrocytic anaemia and oral ulcers Examination Febrile T38 – 39 Cushingoid, no active panniculitis No synovitis No lymphadenopathy. Mild splenomegaly Proximal wasting and weakness of ULL and LL. Tremulous right arm, Clonus on right side. Reflexes were present in the upper limbs and brisk, normal in the lower limbs. Mild wasting of the thenar eminences. Unremarkable cardiovascular examination – no peripheral stigmata of infective endocarditis Respiratory examination revealed reduced breath sounds on the right base. Initial Investigations: CRP 155, ESR 108 Hb 70 MCV 99 WCC 3 (lymphocytes 0.7) PLT 216. Normal retics Na 133 Cr 60 (eGFR >90) Albumin 26, ALP 160 GGT 191 AST/ALT normal Iron, transferrin and transferrin saturation all low, Ferritin raised to 1049 CXR – pre-existing right basal atelectasis

further information: CT Chest, Abdomen and Pelvis Pre-existing – splenomegaly 17.5cm A few subcentimetre mediastinal LNs. Pre-existing right basal bands of atelectasis. No effusions Uncomplicated sigmoid diverticulosis TTE – normal LV and RV, mod dilated LA, mildly dilated ascending aorta, normal valves. RVSP 37 Bone marrow biopsy – Hypercellular marrow with normal erythroid cellularity Granulopoiesis markedly increased (may be reactive or related to hypersplenism) Small T lymphocyte population (<1% of total nucleated cells with dual CD4/8 expression) Haemophagocytosis present and increased in activity No evidence of a lymphoproliferative disorder No AFBs. Negative culture PET No avid LNs except for mildly avid right upper mediastinal LNs (?inflammatory), diffusely increased uptake in spleen and bone marrow of vertebral bodies HBV viral load negative. HCV serology and cryoglobulins negative. Negative HIV ANA 1:80 cytoplasmic, Negative DsDNA, ANCA, ENA, CCP, Rheumatoid factor. Previous ACE negative PSA negative Anaemia became normocytic following transfusion. No haemolysis. Haptogobin 3.9 but normal LDH, no RBC antibodies. Normal reticulocytes CMV negative IgM and IgG. EBV IgG positive. No paraprotein – band of low concentration detected in gamma globulin region. FLC ration 2.06 Quanteferon gold in 2010 negative. Repeated in 2017 and again this admission, negative. Q fever, Brucella, syphilis, Legionella, Strongyloides serology negative Ongoing blood cultures negative Swinging fevers up to T39, profuse sweats, anorexia and general decline

further information: Increased Prednisone to 25mg - 50mg for 2 weeks. ID recommended Meropenem for atypical infection

CT angio of abdomen did not show medium vessel vasculitis

TOE – very small mobile echodensity on mitral valve left atrial side ?degenerative, confirmed by cardiology.

Spleen biopsy showed scattered necrotising epithelioid granulomas including Langhan's type giant cells. ?mTB ?sarcoid.

Negative AFB, mTB PCR (MAC, TB and Intracellulare), Bacterial 16S

Repeat bone marrow biopsy

toxic changes with increased granulocytic hyperplasia and green body inclusions

increased haemophagocytic activity

Small granulomas on the trephine

No AFBs and negative cultures

Soluble IL-2 receptor 4800, (2000 is level for controls). Repeat ferritin 1800, normal triglycerides. Gastroscopy, Colonoscopy, Bronchoscopy and washings - Viral cytopathic effects suggestive of HSV or CMV. Immunohistochemisty show HSV is positive. Perianal pain with intermittent PR bleeding: abscess drained – neutrophillic infiltrate consistent with abscess on histopathology.

further information: Commenced quadruple therapy for tuberculosis based on granulomas and likely exposure. Added moxifloxacin for pneumonia and eventually added meropenem and vancomycin. Respiratory failure in context of NG feeding and aspiration. Right basal pneumonia. CTPA – evidence of new pulmonary embolism, new bulky mediastinal lymphadenopathy. Underwent core biopsy of hilar LN under radiological guidance – complicated by pneumothorax. Histopathology showed necrotising lymphadenopathy similar to what can be seen in lupus, kikuchi, viral processes, no granulomas. (PCR, 16S no infection). Rash - neutrophilic dermatosis. ?autoimmune ?drug hypersensitivity. In ICU – on NIV, floridly delirious, febrile and suffering then commenced Tocilizumab which coincided with dramatic improvement including resolution of fevers, rash and delirium. Treatment for tuberculosis ceased after 1 month as no improvement and had widespread neutrophilic rash (?hypersensitivity). No growth on any cultures. Stepped down IV antibiotics as was clinically improving

Further information: Fevers recurred with reduction in dose frequency of Tocilizumab, trialed Anakinra. Continued low dose of prednisone.

New problems developed

Headaches – LP showed elevated protein, negative cultures.

Episode of orbital cellulitis responded to Vancomycin

Further decline in December/January with swinging fevers, cachexia and new delirium and rigidity – NMDA receptor antibodies positive on serum

Progressive pancytopenia with thrombocytopenia and recurrent PR bleeding, rising ferritin 5000 - 10,000. Repeat LP and BMAT deemed unsafe

Re-increased dose of Prednisone followed by pulse Methyl Prednisolone with deterioration despite this

Deterioration in conscious state:

MRI Brain showed progressive frontal atrophy (previous finding), slight asymmetry of hippocampal area

EEG suggestive of both NCSE and encephalopathy

Passed away in January following significant decline, hypotension requiring inotropes and eventually bradycardic arrest

Differential d...
Claude 3.7 So...
Switch assistant

Select assistant

Search assistants...

Create new assistant

Differential diagnosis for clinician
Expert doctor for differential diagnosis

convert ics calendar to spreadsheet
Efficient calendar-to-spreadsheet converter

compare documents
Document comparison and difference identifier

Type2 Diabetes Referral Triage
Expert diabetologist for referral triage

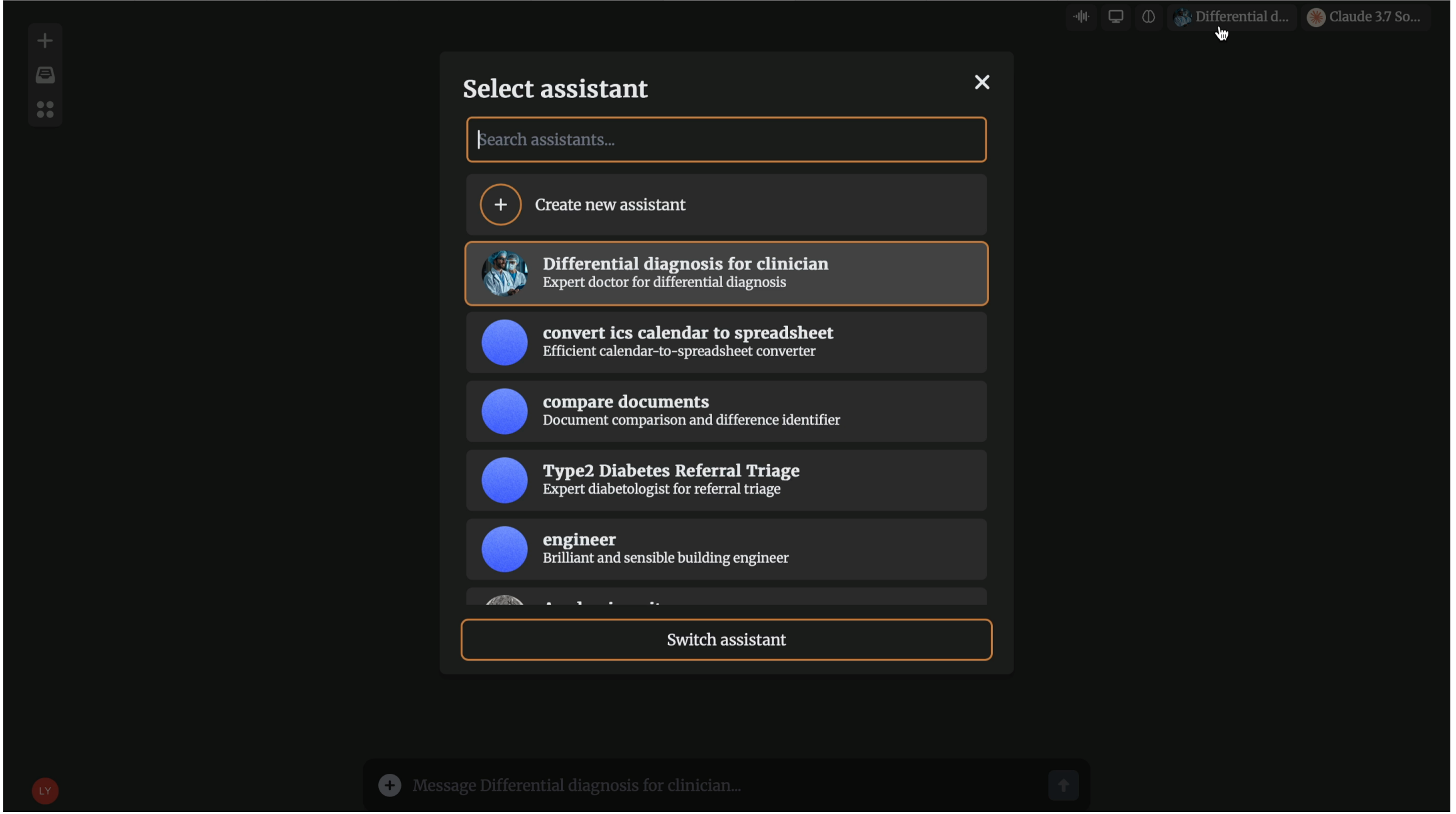
engineer
Brilliant and sensible building engineer

Switch assistant

Message Differential diagnosis for clinician...

Outpatient Referral Triage from GP requesting specialist appointment

- 26 yo male with a painful right knee for a few days. He wondered if he'd sprained it at footy on Saturday. Woke with pain and swelling in the knee on Sunday and pain has been increasing since then. He's having difficulty walking on the knee, using crutches. He feels feverish and has lost his appetite. No rigors. Examination shows a very swollen red knee which is very painful to move through a very limited range. The knee feels hot. PHx: nil. I've sent him for blood tests but don't have the results yet. I've started colchicine and naproxen. His uncle has gout so I thought this would be gout.


Differential d...
Claude 3.7 So...
Select assistant
Search assistants...
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Differential diagnosis for clinician
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E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	
Melissa	Lynden	O3-mini	Gemini 2.0 Flash Experimental	01	Claude 3.5 Sonnet	Claude 3.5 Haiku	Claude Opus	ChatGPT-4o	GPT-4o	01-preview	01-mini	GPT-4o mini	Gemini 1.5 pro	Gemini Experimental 1206	Amazon nova pro	Amazon nova lite	Grok beta	Groq Llama 3.3 versatile	Groq mixtral	Nvidia nemotron 70B	Hermes 3	Meta llama 3.1 405B	Meta llama 3.3 70B	Mistral large 2	Qwen 2.5 72B	
S	S or R	SSR	SSS	SSS	SSS	UUU	UUS	SSS	SSS	SSS	SSS	SSS	SUH	SSS	SSS	SSS	SSS	SSS	SHS	SSS	SSS	SSS	SSS	SSS	SSS	5
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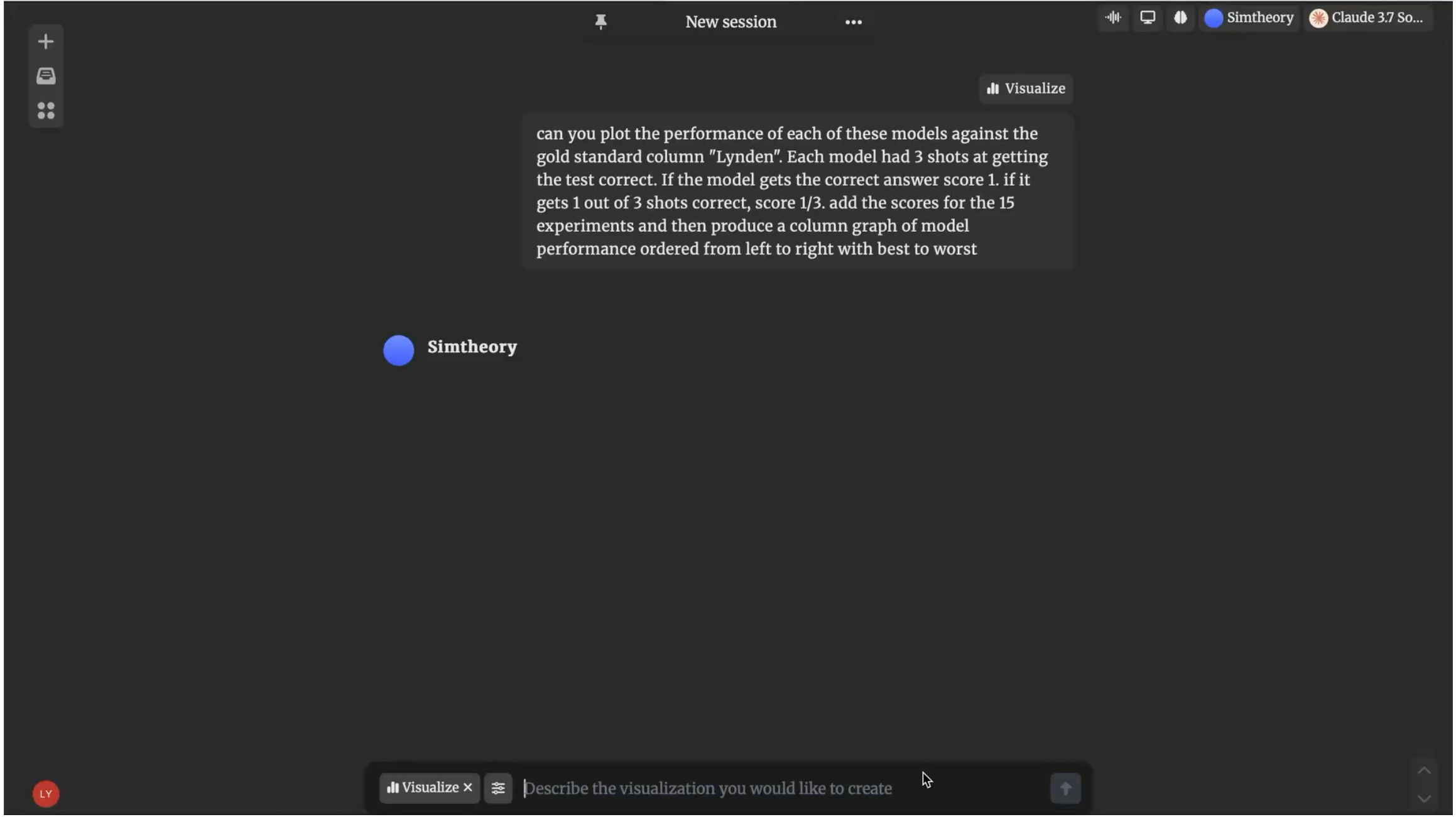
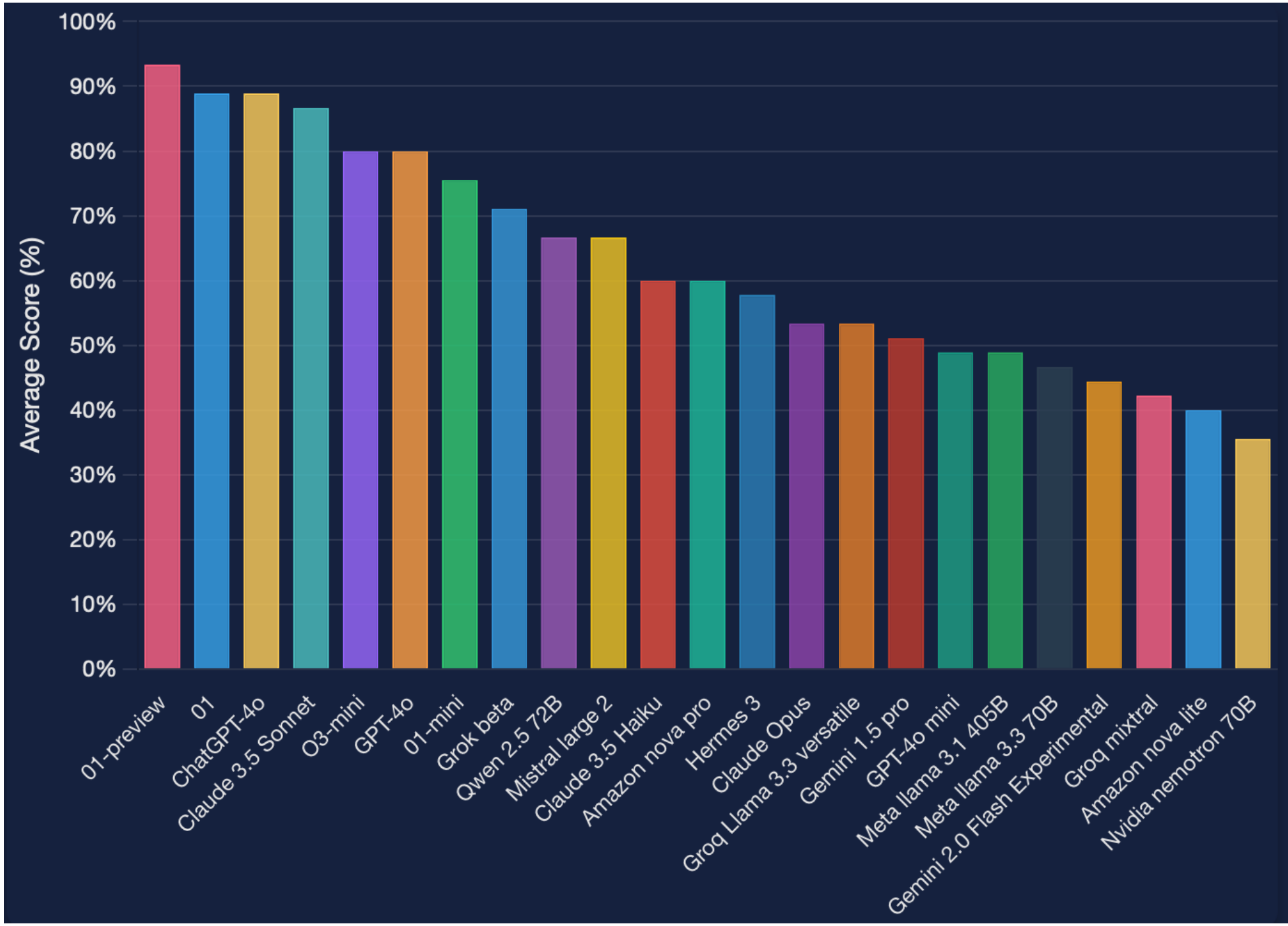
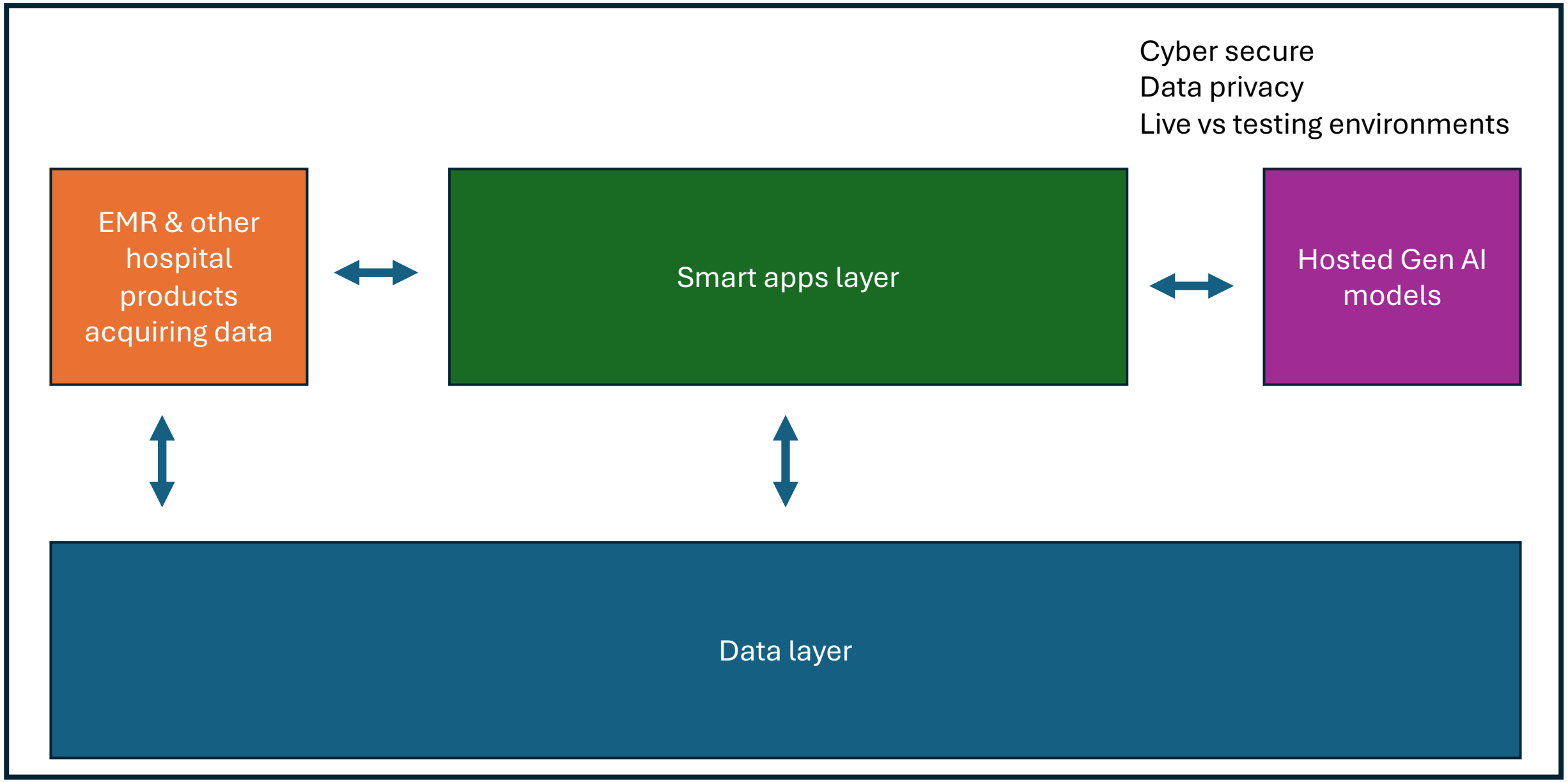

New session
Simtheory
Claude 3.7 So...
Visualize
can you plot the performance of each of these models against the gold standard column "Lynden". Each model had 3 shots at getting the test correct. If the model gets the correct answer score 1. if it gets 1 out of 3 shots correct, score 1/3. add the scores for the 15 experiments and then produce a column graph of model performance ordered from left to right with best to worst
Simtheory
Visualize ×
Describe the visualization you would like to create
LY

LLMs are really good at this complex clinical triage categorization task

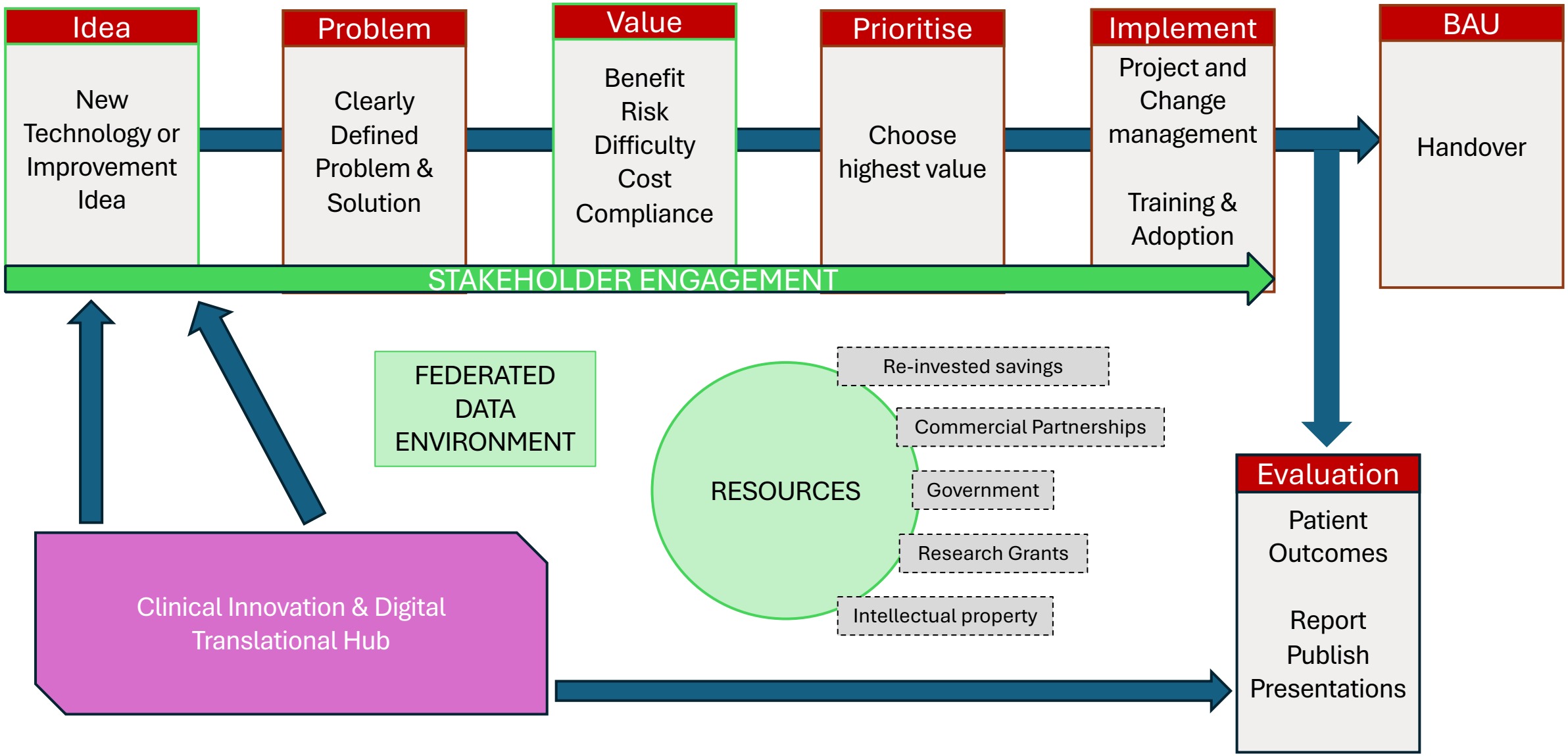
much better than the current human triage system



The digital ecosystem to support Gen AI development/testing/deployment



Learning Health System – innovation process



INTERNAL MEDICINE JOURNAL

RACP Specialists. Together

REVIEW

Challenges for implementing generative artificial intelligence (GenAI) into clinical healthcare

Lynden J. Roberts,[1] Rajiv Jayasena,[2] Sankalp Khanna,[3] Leslie Arnott,[4] Paul Lane[5] and Chris Bain[6]

[1]Monash Health, [2]Australian E-Health Research Centre, CSIRO, [4]Centre for Digital Transformation of Health, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, and [6]Digital Health, Faculty of IT, Monash University, Melbourne, Victoria, and [3]Australian E-Health Research Centre, CSIRO, and [5]The Prince Charles Hospital, Brisbane, Queensland, Australia

What unique abilities of GenAI signal opportunities for healthcare?
Challenges of clinical GenAI implementation
The challenge of inexact outputs
Drifting and shifting performance
Existing challenges of traditional AI are more complex with GenAI
Regulatory and explanatory considerations
GenAI 'credentialing' as a conceptual way forward for regulation
Medico-legal considerations
Workforce dynamics and cultural resistance
Patient, carer, consumer and community perspectives