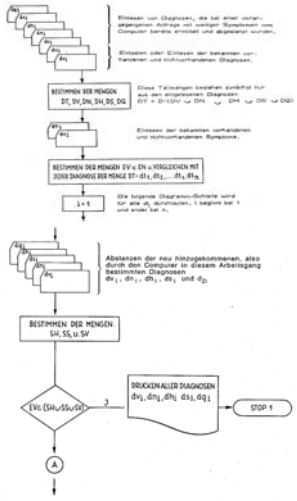
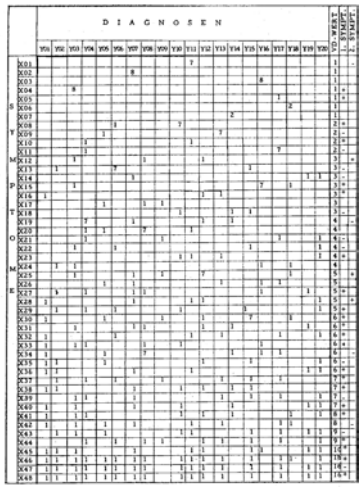
Computer Assisted Diagnostic System 1968





Computer Assisted Diagnostic System 1968

- Negation
- Konjunktion
- Disjunktion

Außerdem:

- Symptom S ist obligat und beweisend für Krankheit K: $S \leftrightarrow K$
- Symptom S ist fakultativ und beweisend für Krankheit K $S \rightarrow K$
- Symptom S ist obligat und nicht beweisend für Krankheit K: $S \leftarrow K$
- Symptom S schließt Krankheit K aus: $S \rightarrow \neg K$

Computer Assisted Diagnostic System 1968

Relationen zwischen Symptomen (S_i) und Diagnosen (D_j)

S_i OC D_j: obligatory occurrence and confirmation, i.e., the symptom has to be present in the patient in order to establish the diagnosis, and, if it is present, it confirms the diagnosis. If the symptom is definitely absent, the diagnosis is excluded.

$$\text{OC} \triangleq \textbf{if } S_i \textbf{ then } D_j \text{ or } \textbf{if not } S_i \textbf{ then not } D_j . \qquad [2.1]$$

EXAMPLE 1: *if* endoprothesis of the knee in X-ray *then* arthroplasty of the knee or *if not* endoprothesis of the knee in X-ray *then not* arthroplasty of the knee.

S_i FC D_j: facultative occurrence and confirmation, i.e., the symptom does not have to be present in order to establish the diagnosis, but if the symptom occurs, the diagnosis is thus confirmed.

$$\text{FC} \triangleq \textbf{if } S_i \textbf{ then } D_j . \qquad [2.2]$$

EXAMPLE 2: *if* intracellular uric acid crystals in joint effusion *then* gout.

S_i ON D_j: obligatory occurrence and nonconfirmation, i.e., the symptom has to be present in order to establish the diagnosis. Therefore, if the symptom is absent, the diagnosis is excluded.

$$\text{ON} \triangleq \textbf{if not } S_i \textbf{ then not } D_j . \qquad [2.3]$$

EXAMPLE 3: *if not* onset of disease prior to 16th year of age *then not* juvenile rheumatoid arthritis.

Computer Assisted Diagnostic System 1968

Relationen zwischen Symptomen (S_i) und Diagnosen (D_j)

S_i EX D_j: exclusion, i.e., if the symptom is present, the diagnosis is excluded.

$$\text{EX} \triangleq \textbf{if } S_i \textbf{ then not } D_j . \qquad [2.4]$$

EXAMPLE 4: *if* Waaler Rose titer $\geq 1:64$ *then not* seronegative rheumatoid arthritis.

S_i FN D_j: facultative occurrence and nonconfirmation, i.e., the symptom does not have to be present in order to establish the diagnosis, and, if it occurs, it does not confirm the diagnosis. The symptom certainly provides evidence for the diagnosis, but it only expresses the existence of an association between the exhibited symptom and the underlying disease.

$$\text{FN} \triangleq \textbf{if } S_i \textbf{ then may be } D_j . \qquad [2.5]$$

EXAMPLE 5: *if* elevated amylase in serum *then may be* acute pancreatitis.

Jan Łukasiewicz
(1878-1956)

Mehrwertige Logik von Łukasiewicz

	¬
1	0
½	½
0	1

∧	1	½	0
1	1	½	0
½	½	½	0
0	0	0	0

∨	1	½	0
1	1	1	1
½	1	½	½
0	1	½	0

→	1	½	0
1	1	½	0
½	1	1	½
0	1	1	1

↔	1	½	0
1	1	½	0
½	½	1	½
0	0	½	1

Computer Assisted Diagnostic System 1968

A diagnosis (D) can be absent (0), present (1) or possible (2).
The truth tables for the relationships are as follows:

1) obligatory and proving (OP): S ≡ D

S/D	0	1	2
0	1	0	2
1	0	1	2
2	2	2	2

2) obligatory excluding (E): S → ¬D or ¬(S ∧ D)

S/D	0	1	2
0	1	1	1
1	1	0	2
2	1	2	2

3) facultative and proving (FP): S → D

S/D	0	1	2
0	1	1	1
1	0	1	2
2	2	1	2

4) obligatory and not proving (ON): S ← D or S ∨ ¬D

S/D	0	1	2
0	1	0	2
1	1	1	1
2	1	2	2

5) facultative and not proving (FN): The FN-relationship is a tautology (permanent true) and consequently unspecific.

S/D	0	1	2
0	1	1	1
1	1	1	1
2	1	1	1

Computer Assisted Diagnostic System 1968

Wissensrepräsentation

$$S_i \text{ OC } D_j \triangleq \forall p(S_i(p) \rightarrow D_j(p)) \wedge \forall p(D_j(p) \rightarrow S_i(p)) \wedge \exists p(S_i(p) \wedge D_j(p)) \quad [2.6]$$

$$S_i \text{ FC } D_j \triangleq \forall p(S_i(p) \rightarrow D_j(p)) \wedge \neg \forall p(D_j(p) \rightarrow S_i(p)) \wedge \exists p(S_i(p) \wedge D_j(p)) \quad [2.7]$$

$$S_i \text{ ON } D_j \triangleq \forall p(D_j(p) \rightarrow S_i(p)) \wedge \neg \forall p(S_i(p) \rightarrow D_j(p)) \wedge \exists p(S_i(p) \wedge D_j(p)) \quad [2.8]$$

$$S_i \text{ EX } D_j \triangleq \forall p(S_i(p) \rightarrow \neg D_j(p)) \wedge \exists p(S_i(p) \wedge \neg D_j(p)) \wedge \exists p(D_j(p) \wedge \neg S_i(p)) \quad [2.9]$$

$$S_i \text{ FN } D_j \triangleq \neg \forall p(S_i(p) \rightarrow D_j(p)) \wedge \neg \forall p(D_i(p) \rightarrow S_i(p)) \wedge \exists p(S_j(p) \wedge D_j(p)) \quad [2.10]$$

Medizinisches Wissen aus einem Lehrbuch

7.2.11 Maligne Tumoren

Neben dem Kaposi-Sarkom kommen auch andere maligne Tumoren gehäuft bei HIV-Patienten vor.

Non-Hodgkin-Lymphome

Non-Hodgkin-Lymphome (NHL) treten bei [redacted] 5–10% aller AIDS-Patienten auf. Histologisch handelt es sich [redacted] um hochmaligne B-Zell-Lymphome. Ein disseminierter und extranodaler Befall liegen [redacted] vor.

Die **Symptome** richten sich nach dem Befallsmuster: Lymphknotenschwellungen und Allgemeinbeschwerden (Fieber, Nachtschweiß) sind [redacted] vorhanden; bei Knochenmarkbefall kommt es zur Panzytopenie, bei Befall des Magen-Darm-Traktes zu Bauchschmerzen und Gewichtsabnahme. Im Labor findet sich [redacted] eine Erhöhung der LDH.

Für die **Prognose** spielt neben der Tumorausbreitung das Stadium der HIV-Infektion eine ganz entscheidende Rolle. Patienten mit kompensiertem Immunstatus können einer Standard-Chemotherapie (CHOP-Schema) unterzogen werden und damit prinzipiell in eine komplette Remission gebracht werden. Dagegen ist die Prognose bei Patienten mit manifestem AIDS, schlechtem Allgemeinzustand oder ausgeprägtem Immundefekt extrem schlecht. Der Nutzen einer aggressiven Chemotherapie ist hier sehr fraglich, da die Therapie-induzierte weitere Verschlechterung des Immunstatus zu meist nicht beherrschbaren infektiösen Komplikationen führt.

Andere Tumoren

Maligne Tumoren, die durch Papillomaviren induziert werden, sind bei HIV-Patienten gehäuft beobachtet worden. Hierzu zählen das Zervixkarzinom der Frau und Plattenepithelkarzinome der Analregion. Außerdem wurde über ein vermehrtes Auftreten von Hodgkin-Lymphomen berichtet.

Literatur

- DeVita, V. T., S. M. D. Hellman, S. Rosenberg (eds.): AIDS: etiology, diagnosis, treatment and prevention. Lippincott-Raven, New York 1996.
- Fields, B. N. (ed.): Virology. Raven, New York 1996.
- Mandell, G. L., J. E. Bennett, R. Dolin (eds.): Principles and Practice of Infectious Diseases. Churchill Livingstone, New York 1995.
- Richman, D. D., R. J. Whitley, F. G. Hayden (eds.): Clinical Virology. Churchill Livingstone, New York 1997.
- Wormser, G. (ed.): AIDS and other manifestations of HIV infection. Lippincott-Raven, New York 1997.

Classen/Diehl/Kochsiek: Innere Medizin, München, Wien Baltimore: Urban & Schwarzenberg, 4. Auflage 1998, S. 433.

Hepatitis Viruses

Hepatitis A Virus

Though hepatitis A virus is classified in the family Picornaviridae and most closely resembles the enteroviruses, it presents diagnostic features that are unique. At the present time, there is no system widely available (either tissue culture or antigen detection) for detecting the virus particle itself, although it is known to be shed in the feces acutely before and during the hepatitis, and there are tissue culture systems that will support its growth. Laboratory diagnosis is therefore best accomplished by serology. Fortunately, the presence of IgM antibody to hepatitis A virus is quite specific for acute infection, and methods for its measurement are widely available.

The appropriate specimen is a serum sample obtained during the acute hepatitis, or shortly thereafter. In essentially all cases of hepatitis A, IgM antibody will be present as measured by the commercially available RIA or ELISA kit, which employs the IgM capture technique. This antibody lasts, on average, 3 months; therefore, while false positives are possible, they are unlikely (33). The detection of IgG antibody provides evidence of prior infection and is also evidence of immunity.

Hepatitis B Virus

The subject of laboratory tests in the diagnosis of hepatitis B infection is treated more fully in Chapter 77, but the principles will be reviewed here. The various antibodies to different antigens in the virus particle (or associated with this particle) develop at different stages of infection and recovery, and the clinician can take advantage of this to time and characterize the individual's infection.

Acute HBV infection is best revealed by the presence of surface antigen (HbsAg); this antigen is present immediately before, as well as for the first few days of illness. If a subject is to become a chronic carrier, this antigenemia is prolonged. As (or after) antigen disappears from the blood, the first antibody to appear is that to the viral core, in the form of IgM and then IgG antibody (anti-HBc). Like HbsAg, IgM anti-HB_c is transient when recovery occurs normally, and it persists for a variable period of time in those with more prolonged infections (its persistence probably depends on continuing active viral replication) (88); anti-HB_c IgG is long-lasting. Quite late after infection, usually several weeks or even months after acute onset of illness, IgG antibody to the surface antigen (anti-HBs) appears and is a marker of past infection. Both e antigen and antibody are important prognostic indicators but rarely aid diagnosis.

Hepatitis C Virus

Because of its importance in the field of blood banking, progress in diagnosis of hepatitis C virus infection has been rapid. Shortly after segments of the RNA genome of the virus were expanded and cloned from chimpanzee plasma by reverse transcriptase PCR (21), the first generation of antibody tests was ready for distribution to blood banks (89). This test, based on the C100 protein which was first cloned, was, unfortunately, neither very sensitive nor specific. It was, however, widely used to screen blood donors, first as the only serologic test but in conjunction with liver enzyme measurement, then with a confirmatory recombinant immunoblot assay, the RIBA test, which is still used (162). A second generation ELISA was distributed which contained not only C100 (which represents the NS4 protein of the virus) but fusion proteins representing the NS3 and core proteins. This was a considerable improvement in both its sensitivity and specificity over the previous test (169). A third generation EIA is due for distribution soon.

Reverse transcriptase PCR (RT-PCR) and branched-chain DNA solid phase systems both clearly represent tests with clinical utility in the diagnosis of hepatitis C infection. Both of these correlate more closely than antibody tests with hepatic dysfunction and probably also correlate with infectivity (91,44).

Fields, B.N. et al. (1996) *Fields virology*. Lippincott-Raven Publishers, Philadelphia.

CANCER OF THE PANCREAS

GENERAL CONSIDERATIONS Carcinoma of the pancreas is now the fourth commonest cancer causing death in the United States; only cancer of the lung, colon, and breast occur more frequently. It accounts for 10 percent of all tumors of digestive organs and over 20,000 deaths per year. The incidence has increased 300 percent since 1930 to approximately 11 per 100,000 population. [illegible] (1.5:1), and the peak incidence is between the ages of 60 to 70. Although [illegible], incidence of carcinoma of the pancreas is 2.0 to 2.5 times greater in *smokers* than in nonsmokers, and about 2 times greater in patients with *diabetes mellitus*. Patients with *calcific pancreatitis* also have an increased incidence of pancreatic carcinoma. Some reports have suggested an association between heavy coffee intake and increased risk of pancreatic cancer, but whether a true causal relationship exists is uncertain. The tumors are usually adenocarcinomas arising from ductal epithelium. The head of the pancreas is involved in about 65 percent, the body and tail in 30 percent, and the tail alone in 5 percent. At the time of diagnosis the tumor is confined to the pancreas in only 15 percent of patients; 25 percent demonstrate local invasion or regional lymph node spread, and the remaining 60 percent exhibit distinct metastases.

CLINICAL FEATURES [illegible] The weight loss in carcinoma is extensive (average total loss about 25 lb) and is not fully explained by anorexia and maldigestion. The weight loss in patients with lesions in the body and tail, in whom malabsorption should be minimal, is often as pronounced as when the carcinoma is in the head of the pancreas.

Pain occurs at some time in the course of the disease in 75 to 90 percent of patients. With tumors of the head of the pancreas, the pain is likely to be in the epigastrium and right upper quadrant; with lesions in the body of the pancreas, pain often localizes in the midline, whereas with lesions in the tail, pain may be referred to the left upper quadrant. Abdominal pain may be vague or be a steady dull, aching, or boring pain often radiating through to the back. Severe and unrelenting pain suggests extension into the retroperitoneal area with invasion of the neural plexus around the celiac axis ganglion.

[illegible] When it occurs it is progressive and accompanied by pruritus. Both constipation and diarrhea have been cited as the predominant alteration of bowel habits. Emotional disturbances frequently occur in cancer of the pancreas and may take the form of insomnia, restlessness, rage, anxiety, depression, suicidal tendencies, and a sense of impending doom.

128 Thomssen, Bredt, Germer: 6. Infektionskrankheiten

III. Medizinische Parasitologie

Von **Wolfdietrich Germer**

A. Protozoen-Infektionen

1. Toxoplasmose

Erreger: Toxoplasma gondii, eine weltweit vorkommende Kokzidie. Endwirt für T. gondii sind Katzen, in deren Darmepithel eine geschlechtliche Vermehrung (Gamogonie) unter Ausbildung von Oozysten stattfindet. Letztere sind – ausgeschieden – gegen äußere Umwelteinflüsse sehr widerstandsfähig. Sie können wieder Katzen, aber auch zahlreiche Zwischenwirte und Menschen infizieren. Nach oraler Aufnahme werden in deren Darm Sporozoiten frei, und es kommt – unter ungeschlechtlicher Vermehrung – zu einer raschen Ausbreitung der Parasiten im ganzen Körper, bevorzugt in den Zellen des RES. Bei wachsender Immunabwehr bilden sich bevorzugt in Gehirn und Muskulatur (Herz, Zwerchfell, Extremitäten) des Wirtes Zysten. Diese Gewebezysten sind für Fleischfresser infektiös.

Übertragung: 1. Diaplazentar, besonders im letzten Schwangerschaftsdrittel. 2. Verzehr von rohem oder ungenügend gekochtem Fleisch, insbesondere Schweinefleisch. 3. Kontakt mit Katzenkot. 4. Organtransplantation und Transfusion leukozytenreicher Blutprodukte. Die Infektionshäufigkeit nimmt mit dem Alter zu und erreicht schließlich 70–80%. Die überstandene Infektion hinterläßt – bei latenter Anwesenheit der Parasiten in den Zysten – eine solide Immunität.

Klinik: Die meisten Infektionen bleiben asymptomatisch. Nur bei 10–20% kommt es zu Krankheitserscheinungen, insbesondere zu Lymphdrüsenschwellungen (bevorzugt zervikal, jedoch auch submental, mediastinal, abdominal u. a.), die mit langanhaltenden Temperaturen, Myalgien, Kopfschmerzen, gelegentlich Hepatosplenomegalie und Auftreten atypischer Lymphozyten einhergehen können. Der Verlauf ist gutartig und selbstbegrenzt. Nur selten kommt es zu Myokarditis, Pneumonitis, Myositis und besonders bei Immundefekten (M. Hodgkin, myeloische Leukämie, AIDS) zu Enzephalo-Meningitis mit meist letalem Ausgang.

Die kongenitale Toxoplasmose, die nur vorkommt, wenn sich die Mutter während der Schwangerschaft erstmalig infiziert, kann zu Abort, Tot- oder Frühgeburt führen. Klinische Zeichen beim Neugeborenen sind Enzephalitis, Pneumonitis, Hydrozephalus, Verkalkungen im Gehirn, Strabismus, Mikrophthalmie, Thrombopenie, Hepatosplenomegalie u. a. Eine Chorioretinitis macht oft erst Monate bzw. Jahre nach der Geburt Erscheinungen.

Diagnose: Der Erregernachweis aus Blut, Knochenmark, Lymphknoten, Liquor, Plazenta gelingt nur in Ausnahmefällen. [illegible]

Differentialdiagnose: Mononukleose, Tbc, Sarkoidose, M. Hodgkin, Listeriose, Virusmeningitis. Die kongenitale T. ist abzugrenzen gegen Zytomegalie, Röteln, Lues.

Therapie: Die Lymphadenopathie bei Toxoplasmose bedarf in der Regel keiner Behandlung. Behandelt werden müssen: akute Infektionen bei immundefekten Patienten, aktive Chorioretinitis, frisch infizierte Schwangere und kongenital infizierte Neugeborene.

1. Pyrimethamin: 100–200 mg/tgl. beim Erwachsenen, 1 mg/kg beim Neugeborenen. In Kombination mit
2. Sulfadiazin: 50–100 mg/kg/tgl. für 4–6 Wochen oder länger. Im ersten Schwangerschaftstrimester ist Pyrimethamin kontraindiziert.
3. Spiramycin: 100 mg/kg/tgl. für 30–45 Tage. Bei T.-Meningitis und -Chorioretinitis zusätzlich
4. Prednison: 1–2 mg/kg/tgl. Zusätzlich bei Pyrimethaminbehandlung.
5. Folsäure: 5 mg 2 × pro Woche.

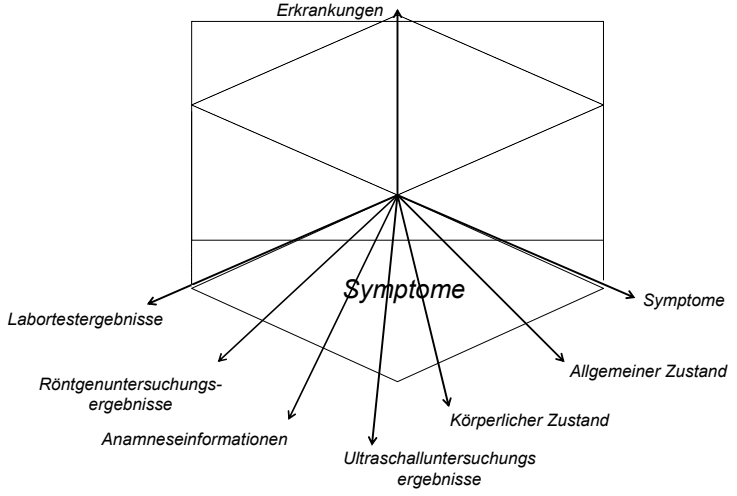
Prophylaxe: Serologisch T.-negative Schwangere sollten kein rohes Fleisch essen und engen Kontakt mit Katzen meiden. Toxoplasmose ist meldepflichtig.

L. A. Zadeh, 1962: From Cercuit Theory to System Theory

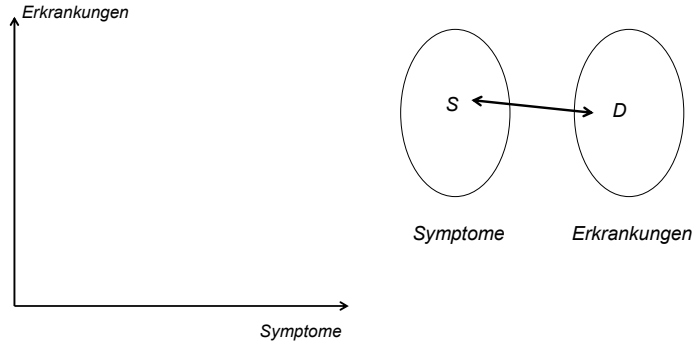
In fact, there is a fairly wide gap between what might be regarded as „animate“ system theorists and „inanimate“ system theorists at the present time, and it is not at all certain that this gap will be narrowed, much less closed, in the near future.

There are some who feel that this gap reflects the fundamental inadequacy of the conventional mathematics – the mathematics of precisely-defined points, functions, sets, probability measures, etc. - for coping with the analysis of biological systems, and that to deal effectively with such systems, which are generally orders of magnitude more complex than man-made systems, **we need a radically different kind of mathematics, the mathematics of fuzzy or cloudy quantities which are not describable in terms of probability distributions**.

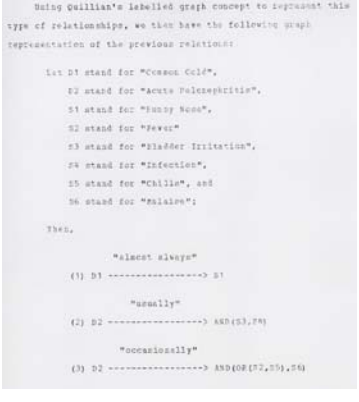
Indeed, the need for such mathematics is becoming increasingly apparent even in the realm of inanimate systems, for in most practical cases the *a priori* data as well as the criteria by which the performance of a man-made system is judged are far from being precisely specified or having accurately-known probability distributions.



Symptome-Diagnosen-Relation



Alonso Perez-Ojeda, 1976



Using Quillian's labelled graph concept to represent this type of relationships, we then have the following graph representation of the previous relations:

Let D1 stand for "Common Cold",
D2 stand for "Acute Pulenephritis",
S1 stand for "Runny Nose",
S2 stand for "Fever"
S3 stand for "Bladder Irritation",
S4 stand for "Infection",
S5 stand for "Chills", and
S6 stand for "Malaise";

Then,

"almost always"
(1) D1 ----------> S1

"usually"
(2) D2 ----------> AND(S3,S4)

"occasionally"
(3) D2 ----------> AND(OR(S2,S5),S6)

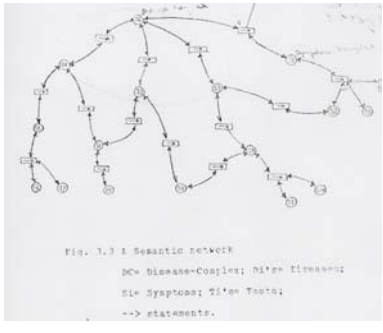


Fig. 3.3 A Semantic network

DC= Disease-Complex; Di's= Diseases;
Si= Symptoms; Ti's= Tests;
--> statements.

A Fuzzy Logical Model of Computer-Assisted Medical Diagnosis

(From the Department of Medical Computer Science (Director: Prof. Dr. G. Grabner), University of Vienna, Austria)

K.-P. Adlassnig

A model of a computer-assisted diagnostic system using fuzzy subsets has been developed. The physician documents symptom—diagnosis presence relationships and symptom—diagnosis conclusiveness relationships by means of labels of the fuzzy subsets *never, almost never, very very seldom, very seldom, seldom, more or less seldom, not known, more or less often, often, very often, very very often, almost always, always*.

Symptoms are regarded as fuzzy subsets of reference sets. The reference set includes all values the symptom may assume. The degree of membership of a value in the fuzzy subset of a symptom is calculated when the patient's symptom pattern is available. By means of compositions of fuzzy relations, four different diagnostic indications are determined for every diagnosis under consideration: *presence indication, conclusiveness indication, non-presence indication* and *non-symptom presence indication*.

By performing the diagnostic process, the system provides the physician with *proven diagnoses, excluded diagnoses* and *diagnostic hints*, including *reasons* for the diagnoses displayed. *Proposals* for further investigations may also be requested.

Key-Words: Computer-assisted Diagnosis, Fuzzy Subsets, Medical Documentation, Diagnostic Hints, Symptom—Diagnosis Relationships, Proven and Excluded Diagnoses

EIN MODELL ZUR COMPUTERUNTERSTÜTZTEN DIAGNOSE MIT FUZZY LOGIK

Es wurde ein computerunterstütztes medizinisches Diagnosemodell unter Verwendung von Fuzzy-Teilmengen entwickelt. Die medizinische Vorarbeit besteht in der Dokumentation des Vorhandenseins eines Symptoms bei einer Diagnose und der Beweiskraft eines Symptoms für eine Diagnose. Zur Dokumentation verwendet der Mediziner die Bezeichnung der Fuzzy-Teilmengen *nie, fast nie, sehr sehr selten, sehr selten, selten, mehr oder weniger selten, unbekannt, mehr oder weniger oft, oft, sehr oft, sehr sehr oft, fast immer, immer*.

Computer Assisted Diagnostic System II, *CADIAG-II*

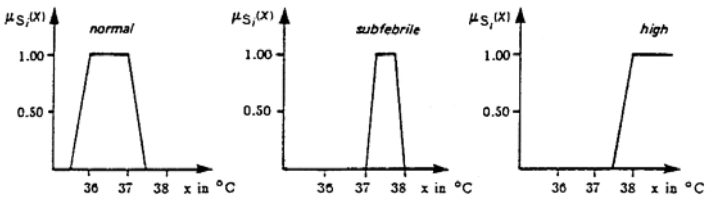


FIG. 1. Some fuzzy subsets for body temperature.

Ludwik Fleck (1896 - 1961)

„Während der Naturwissenschaftler typische, normale Phänomene sucht, studiert der Arzt gerade die nicht typischen, nicht normalen, krankhaften Phänomene. Und dabei trifft er auf diesem Weg sofort auf einen gewaltigen Reichtum und Individualität dieser Phänomene, die die Vielheit ohne klare, abgegrenzte Einheiten begleiten, voller Übergangs- und Grenzzustände. Es gibt keine genaue Grenze zwischen dem, was gesund ist, und dem, was krank ist, und nirgends trifft man wirklich ein zweites Mal auf dasselbe Krankheitsbild. Aber diese unerhört reiche Vielheit immerfort anderer und anderer Varianten muss gedanklich bezwungen werden, denn dies ist die Erkenntnisaufgabe der Medizin."

Alonso Perez-Ojeda, 1976

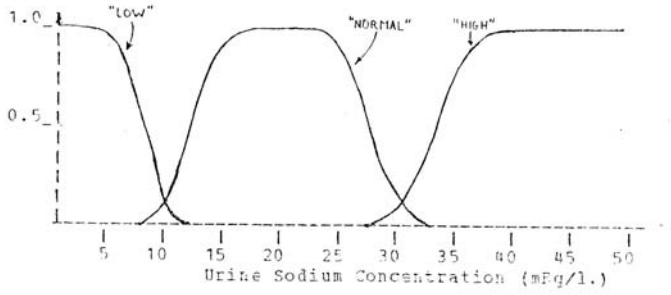


Fig. 3.1 The interpretation of "Low", "Normal" and "High" with respect to "urine sodium concentration".

CADIAG II

TABLE I
AN EXAMPLE OF THE REPRESENTATION OF MEDICAL INFORMATION ON THE PATIENT

Quantitative Value		Symptom	Fuzzy Value
		Potassium, greatly reduced	$\mu_{S_1} = 0.00$
		Potassium, reduced	$\mu_{S_2} = 0.00$
Measured potassium level of 5.3 mmol/l	Fuzzy interpreter	Potassium, normal	$\mu_{S_3} = 0.40$
		Potassium, elevated	$\mu_{S_4} = 0.60$
		Potassium, greatly elevated	$\mu_{S_5} = 0.00$

TABLE II
LINGUISTIC FUZZY VALUES, NUMERICAL INTERVALS, AND REPRESENTATIVE NUMERICAL VALUES DESCRIBING FREQUENCY OF OCCURRENCE AND STRENGTH OF CONFIRMATION

Frequency of Occurrence			Strength of Confirmation		
Value λ_o	Interval	Representative Value μ_o	Value λ_c	Interval	Representative Value μ_c
Always	[1.00, 1.00]	1.00	Always	[1.00, 1.00]	1.00
Almost always	[0.98, 0.99]	0.99	Almost always	[0.98, 0.99]	0.99
Very often	[0.83, 0.97]	0.90	Very strong	[0.83, 0.97]	0.90
Often	[0.68, 0.82]	0.75	Strong	[0.68, 0.82]	0.75
Medium	[0.33, 0.67]	0.50	Medium	[0.33, 0.67]	0.50
Seldom	[0.18, 0.32]	0.25	Weak	[0.18, 0.32]	0.25
Very seldom	[0.03, 0.17]	0.10	Very weak	[0.03, 0.17]	0.10
Almost never	[0.01, 0.02]	0.01	Almost never	[0.01, 0.02]	0.01
Never	[0.00, 0.00]	0.00	Never	[0.00, 0.00]	0.00
Unknown		*	Unknown		*

if antecedent **then** consequent **with** (O,C) [2.11]

where the relationship tuple (O,C) contains linguistic and numerical values λ_O and μ_O, and/or λ_C and μ_C.

EXAMPLE 6 (cf. Example 2): *if* intracellular uric acid crystals in joint effusion *then* gout *with* (λ_O = *seldom* [μ_O = 0.25], λ_C = *always* [μ_C = 1.00]).

EXAMPLE 7 (cf. Example 5): *if* elevated amylase in serum *then may be* acute pancreatitis *with* (λ_O = very often [μ_O = 0.90], λ_C = strong [μ_C = 0.70]).

SC_iD_j, S_iS_j, and D_iD_j relationships are treated in an analogous way. An example for an SC_iD_j rule is:

EXAMPLE 8: *if* low back pain, and limitation of motion of the lumbar spine, and diminished chest expansion, and the patient is male, and between 20 and 40 years of age *then may be* ankylosing spondylitis *with* (λ_O = very often [μ_O = 0.90], λ_C = strong [μ_C = 0.80]).

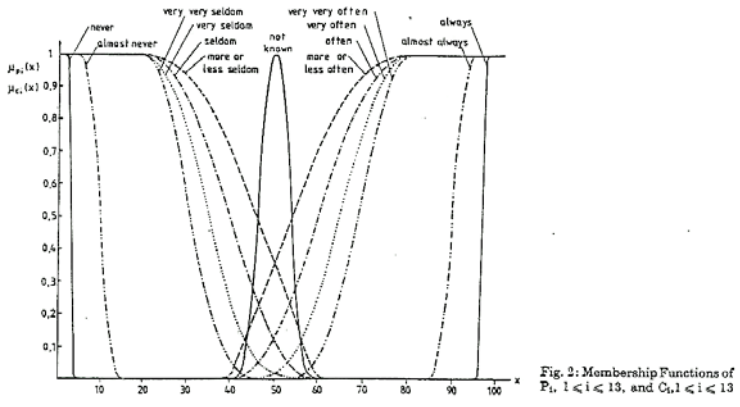


Fig. 2: Membership Functions of P_i, $1 \leq i \leq 13$, and C_i, $1 \leq i \leq 13$

Beispiele

- Example 1 (indicating):

  IF *elevated amylase level in serum*
  THEN *acute pancreatitis*
  WITH (λ_O = *very often* [μ_O = 0.90], λ_B = *strong* [μ_C = 0.70]).

- Example 2 (necessary and sufficient):

  IF *rheumatoid arthritis* and
  splenomegaly and
  leukopenia less than 4 giga/l
  THEN *Felty's Syndrom*
  WITH (λ_O = *always* [μ_O = 1.00], λ_C = *confirming* [μ_C = 1.00]).