## *REGULAR ARTICLES*

# Using Bloom's Taxonomy of the Cognitive Domain to Teach a Required Course in Pharmacology and Toxicology

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This manuscript constitutes a testimonial paper prepared at the time of retirement. The basic concepts presented here are not original with the author, but the comments and the documents assembled as addenda will have some appeal to those interested in educational goals and objectives related to teaching in the cognitive domain, especially in the areas of pharmacology and toxicology.

In 1979, at the request of the administration, the author prepared a statement (Addendum A) on competency-based education. This statement was subsequently adopted as policy and printed in the *University of the Pacific School of Pharmacy Handbook* (1-10). This document was an attempt to communicate certain basics to peer faculty to secure a common terminology and to encourage ex-

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perimentation. It seems that individual interest in competency-based teaching and testing naturally waxes and wanes, depending on the level of interest of the administration. Since it imposes a level of difficulty beyond that of intuitive or "spontaneous" teaching and testing and implies accountability, certain faculty will never attempt it. In consequence, a sustained application of the principles in Addendum A appears to be rather rare.

Earlier, the author had become convinced that testing procedures *do* dictate the way that students will study the content of a course and that there was an increasing need for pharmacy students to acquire cognitive skills beyond rote memorization in preparation for real-life practice. Since 1974, he has been teaching and testing the second semester of the required pharmacology and toxicology course at the University of the Pacific following the precepts of Bloom's *Taxonomy of Educational Objectives: The Classification of Educational Goals. 1: The Cognitive Domain* (7).

On the first day of class, samples of used test questions (sorted according to Bloom's six levels of cognitive difficulty) and the competency-defined syllabus of the course are handed out. Addendum B represents the most recent version of this document. The theory behind testing difficulty and problem-solving in real life is then discussed in class in light of present physiological and psychological knowledge regarding exposure and reinforcement as related to short- and long-term memory. Students generally perceive this lecture as a natural extension of previous study on the physiology of the central nervous system. The reader should note that the definitions of the six levels of the cognitive domain are worded quite differently in Addendum A and Addendum B. Addendum B carries definitions that the students find intelligible and meaningful.

The students are then told that all test questions for the course will be constructed presuming that the pharmacist is to make only decisions beneficial to the health of the patient; i.e., the pharmacist is to act as the patient's advocate in the health care system in regard to drug therapy. The full implications of this concept are not always comprehended by the students, so they are told to answer every question presuming that someone in their family (mother/father, husband/wife, or child) is going to take the medicine to be dispensed. This simple statement (reinforced throughout the semester)

helps to establish the correct ethical perspective. It is very well accepted and represents teaching in the affective domain (Addendum A). Once accepted, this attitudinal principle is as important professionally as mastery of the course's cognitive material.

So that they may become problem-solving clinical pharmacists, the students are told that there are eight pharmacological/toxicological properties of a systemically-acting drug that must be correlated in practice with that drug's generic/trade name. These properties are the ones that must be studied and understood intellectually. Specifically, these are:

1. Pharmacological/toxicological mechanism(s) of action as correlated with drug concentration(s) in body fluids
2. Potency (related to a prototype drug) as correlated with a specific (desired) action and route of administration
3. Capacity (intrinsic activity) to accomplish the specific (desired) action (e.g., aspirin vs. propoxyphene vs. meperidine vs. morphine regarding the blockade of sharp/bright pain)
4. Onset of effect (drug uptake) as associated with a specific route of administration
5. Duration of effect as correlated with a specific route of administration
6. Binding/detoxification/elimination mechanisms for the active form(s) of the drug
7. Expected side effects (unavoidable adverse reactions experienced at therapeutic levels in the body)
8. Expected toxic effects (avoidable adverse reactions experienced at greater than therapeutic levels in the body or those adverse reactions experienced in certain patients on chronic dosage).

It is emphasized that *all* test questions will involve the use of the above-named properties. This list provides a specific focus for studying the course.

There follows an announcement that tests for the past three semesters will be placed on file in the School of Pharmacy Library (no answers provided) and that students are encouraged to copy and study these tests. They are told that thinking through these old tests

is an effective way to study and will increase their problem-solving skills. It is emphasized that the only way to increase cognitive skill beyond the level of simple recall (Level 1, Addendum B) is by practice. As an enticement, they are also told that a certain number of these questions may appear on the tests that they will take for their course grade. This number is unspecified but, in reality, never exceeds 25%. This means that at least 75% of each test must represent new questions. This ratio appears to encourage students to look at the questions in the library and also to discourage them from organizing an archive of old tests.

When cognitive skills beyond simple recall (Level 1) are to be tested (e.g., comprehension, application, analysis, synthesis, and evaluation), there is no single textbook that provides the range of pharmacological/toxicological information needed; therefore, one must improvise. The specified textbooks are *Goodman and Gilman's The Pharmacologic Basis of Therapeutics* and *U.S.P. Dispensing Information, Volumes 1A/B: Drug Information for the Health Care Provider*, and these are supplemented by data handouts using a variety of sources such as comprehensive, well-designed, large-scale clinical studies (when available), drug company monographs prepared for hospital formulary committee members, *Facts and Comparisons*, the American Pharmaceutical Association's now discontinued *Pharmacological and Biochemical Properties of Drug Substances*, etc. (11-14).

Each student receives a 60-page package of supplemental information. This information ranges from a one-page comparison of auranofin and gold sodium thiomalate (Figure 1) to a five-page comparison of the nonsteroidal anti-inflammatory drugs (NSAIDs) (Figure 2). The handouts are photocopied rather than commercially printed because there must be additions, corrections, and deletions made each year. This method of duplicating provides considerable flexibility, since pages sometimes have to be cut and inserts pasted on the master copy the day before a lecture when new information has been secured.

In lecture, the incidence of a specific adverse reaction may be expressed one of three ways: probability, percentage, or ratio of incidence (e.g., 0.0028, 0.28%, 1:350, respectively). However, none of the handouts uses probability, while the small handouts,

such as Figure 1, frequently use percentage. All handouts involving more than two pages of data are prepared using ratio of incidence, since the students find this easier to handle while studying—the larger the ratio, the better the drug.

The author's lectures are set up to focus on the pharmacological/toxicological problems found within each therapeutic drug class. In the lectures, the author has always tended to spend considerable time on one or two chemically dissimilar prototype drugs for each therapeutic class and then much more briefly review all the other drugs noting specifically how they differ (better or worse) from the prototypes(s) (15, 16). The prototype drugs in Figure 2 are aspirin and ibuprofen.

Students need to know when a drug can be considered to have less (or more) toxic potential than the prototype. The criterion that has been adopted is at least a twofold increase (>100%) in the ratio of incidence (i.e., 1:150 vs. 1:200 is an insufficient difference, while 1:150 vs. 1:400 is a difference worth remembering). In a like manner, a twofold decrease (>50%) indicates that the comparison drug can be considered worse than the prototype. While this twofold factor is arbitrary, it does allow the students to study efficiently.

Addendum C and Addendum D provide questions that correspond to Figure 1 and Figure 2, respectively. Such test questions require the students to make decisions *before* the test as to why certain agents are better or worse than others in particular patients. The focus of study takes the form of "Which would I prefer to take myself?" or "Which drug should be recommended for my mother/father/son/daughter?" Some students are hesitant to study in this very pragmatic fashion and attempt to memorize the tables and then develop the answers during the test time, clearly an impossible task in the limited time available. The debated personal decisions made *before* the test tend to be remembered rather easily and provide one with a true sense of the worth of each drug.

It is interesting to note that the most difficult task for the students (by their own admission) involves the memorization of the generic and trade names for each drug. The tests intentionally shift back and forth between generic and trade names from question to question. The handout sheets specify the names to be mastered (usually one

FIGURE 1. Handout: Comparison of Antiarthritic Gold Compounds and Placebo Medication.

| Parameter | Auranofin (Ridaura) | Gold Sodium Thiomalate (Myochrysine) | Placebo |
|---|---|---|---|
| Form | Capsules | Solution | Variable |
| Dose | 3 mg bid or 6 mg 1xday | Weekly IM injection per schedule | Variable |
| Gold Content | 29% | 50% | 0% |
| First-year Cost(MD Cost) | $938($195) | $1,840($876) | — |
| Second-year Cost | $938($195) | $644($195) | — |
| 6-Month Withdrawal for Adverse Patient Reactions | 14% | 26% | 3% |
| 3-Year Withdrawal for Lack of Effectiveness | 22% | 32% | NA |
| 3-Year Withdrawal for All Causes | 55% | 75% | NA |
| Oral Absorption | 25% | Less Than (LT) 1% | — |
| Fate of Absorbed Gold (Route Administered) | Feces: 10% Urine: 15% (PO) | Feces: 30% Urine: 70% (IM) | NA |
| Steady State | 10-12 weeks | 10-12 weeks | NA |
| 12-Week Blood Level: Mean ± SD | 0.71 mcg/ml ± 0.27 | 2.37 mcg/ml ± 0.72 | NA |

| | | | | |
|---|---|---|---|---|
| Synovial Fluid Level | 1/2 | | 1/2 | NA |
| Protein-binding | Serum Protein = 60%; Cellular Protein = 40% | | Serum Protein = 95% (Albumin) | NA |
| Tissue Half-life: Single Dose//Chronic Dose | 17//80 days | | 6//? days | NA |
| Single-dose Retention After 6 Months | 0.4 - 1.3% | | 25 - 42% | NA |
| Vasomotor Reactions: | | 3,475 Patients, with up to 40 months of therapy. Values in parentheses indicate patients forced to drop therapy for that reason. | | |
| Nitritoid | 0% | | LT 1% | 0% |
| Gastrointestinal: | | | | |
| Incr. Bowel Frequency | 41%(2.6%) | | 13%(0.2%) | 11%(0%) |
| Other | 16%(1.9%) | | 11%(0.2%) | 12%(0.2%) |
| Mucocutaneous: | | | | |
| Stomatitis | 11%(0.7%) | | 15%(2.3%) | 6%(0%) |
| Conjunctivitis | 4%(0.1%) | | 4%(0%) | 0.6%(0%) |
| Skin Rash | 19%(2.5%) | | 35%(3.9%) | 9%(0%) |
| Pruritus | 10%(0.7%) | | 21%(1.7%) | 1%(0%) |
| Kidney/Proteinuria: | 3%(0.9%) | | 4%(0.8%) | 2%(0%) |
| Altered Liver Enzymes: | 0.6%(0.2%) | | 0.8%(0.6%) | 0%(0%) |
| Blood: | | | | |
| Thrombocytopenia | 0.7%(0.3%) | | 2%(0.2%) | 0%(0%) |
| Leukopenia | 0.6%(0.1%) | | 2%(0.2%) | 2%(0.2%) |
| Anemia | 0.8%(0.1%) | | 1%(0%) | 0.4%(0%) |

FIGURE 2. Handout: Comparison of Orally effective Nonsteroidal Anti-Inflammatory Drugs.

| I. | Aspirin (ASA/Empirin) | Phenylbutazone (Azolid/Butazolidin) | Meclofenamate (Meclomen) |
|---|---|---|---|
| Dosage, PO (Max/day) | 600-1000 mg tid qid (5.5 g) | 100-200 mg tid (300 mg) | 50-100 mg tid qid (400 mg) |
| % Absorbed | 100% | 100% | ? |
| Max. Blood Level | 1-2 h | 2.5 h | 30-60 min |
| Half-life (Multiple Dose) | ASA: 15 min, SA: 2.4 h (SA: 16-19 h) | 60-90 h (?) | 2 h (3.3 h) |
| Antiinfl. Onset (Peak Effect) | 2-4 d (2-3 w) | 3-7 d (7 d) | 3-14 d (2-3 w) |
| Analgesic | Good | Good | Good |
| Antigout | Uricosuric at doses GT 4 g/d | Uricosuric | Symptomatic only |
| Food Effect on Absorption | Slows absorption | Recommended with food | Decr. absorption |
| Metabolism | Hepatic | Hepatic | Hepatic |
| Excretion | Renal: 100% (85% SA in alk. urine, 5% SA in acid urine) | Renal: 61%<br>Fecal: 27% | Renal: 66%<br>Fecal: 33% |
| Peptic Ulcer | 1:150 | ? | 1:80 |
| GI Bleeding | 1964-71: 64 deaths | 1:600 | 1:100 |
| GI Distress | 1:25 | 1:10 | 1:4 |

| | | | |
|---|---|---|---|
| Headache | Rare | LT 1:100 | 1:20 |
| Drowsiness | Rare | LT 1:100 (Euphoric response more common) | Rare |
| Tinnitus | 1:90 (Monitor marker) | LT 1:100 | 1:50 |
| Hepatotoxic | Rare | ? (Hepatic necrosis) | Rare |
| Nephrotoxic | 1:350 (Produces acidic urine) | ? (Lack of data) | Rare |
| Edema | Rare | 1:10 (1:3 for those GT 60 y of age) | 1:15 |
| Granulocytopenia | Rare | LT 1:100 | Rare |
| Other | Decr. platelet adhesion (useful) | Aplastic anemia potential | — |
| Skin Reactions | In normals: rare; Allergy-prone: 1:50 | 1:50 | 1:8 |
| Photosensitivity | Rare | Rare | Rare |
| Binding to Blood Proteins | SA: GT 90% | 98% | GT 90% |
| Miscellaneous | Contraindicated for ASA "sensitive" individuals. Poor shelf life if poorly formulated & moisture is present (note vinegar odor) causing GI distress by SA. Enteric tabs & suppository forms available. | Contraindicated for children. Avoid in those GT 60 y of age. Can cause hypertension & block $I_2$ uptake by thyroid. 1-Week therapy max. recommended. Side effects mimic old age symptoms. | 1:3 Diarrhea and abdominal pain. Contraindicated for ASA "sensitive" patients, in impaired renal function & in pregnancy. Never use as initial therapy. |

FIGURE 2 (continued)

| II. | Indomethacin (Indocin) | Sulindac (Clinoril) | Tolmetin (Tolectin) |
|---|---|---|---|
| Dosage, PO (Max./day) | 25 mg tid qid (200 mg) | 150-200 mg bid (400 mg) | 200-400 mg tid qid (1800 mg) |
| % Absorbed | GT 90% | 90% | 100% |
| Max. Blood Level | 1-2 h | 24 h | 30-60 min |
| Half-life (Multiple Dose) | 7 h (?) | Sulindac: 7.8 h (?) sulfide = 18 h (?) | 56 min (?) |
| Antiinfl. Onset (Peak Effect) | 2-14 d (2-4 w) | 4-6 d (2-3 w) | 3-7 d (1-2 w) |
| Analgesic | Poor(good antipyretic) | Poor | Poor |
| Antigout | Symptomatic only | Symptomatic only | Not recommended |
| Food Effect on Absorption | Recommended with food | Decr. absorption | Decr. absorption |
| Metabolism | Hepatic | Hepatic | Hepatic |
| Excretion | Renal: 60% Fecal: 33% | Renal: 50% Fecal: 25% (Entero-hepatic recirculat.) | Renal: 90% with 17% unchanged |
| Peptic Ulcer | 1:250 | 1:250 | 1:50 |
| GI Bleeding | 1:600 | 1:600 | 1:250 |
| GI Distress | 1:7 | 1:6 | 1:3 (Nausea: 1:25) |

| | | | |
|---|---|---|---|
| Headache | 1:4 (frontal) | 1:25 | 1:3 |
| Drowsiness | 1:50 | 1:20 | 1:50 |
| Tinnitus | 1:50 | 1:25 | 1:25 |
| Hepatotoxic | Rare | Rare | Rare |
| Nephrotoxic | Rare | Rare (Active form not found in kidney) | Rare |
| Edema | Rare | LT 1:100 | 1:15 |
| Granulocytopenia | Rare | Rare | Rare |
| Other | 1964-73: 25 fatal blood dyscrasias | Diarrhea: 1:14<br>Pancreatitis reptd. | Constipation: 1:50<br>Dyspnea: 1:100 |
| Skin Reactions | Rare | 1:20 | 1:20 |
| Photosensitivity | Rare | Rare | Rare |
| Binding to Blood Proteins | 90-95% | 100% | 99% |
| Miscellaneous | Contraindicated for ASA "sensitive" individuals & children (hepatitis). Decr. lithium excretion. Blocks chemotaxis. Suppository form (bid) available. Not a "first-choice" agent. Avoid in infections. | Contraindicated for ASA "sensitive" individuals, children & in pregnancy. True prodrug: sulfide is active form. | Contraindicated for ASA "sensitive individuals & in pregnancy. Hypertension: 1:50 |

FIGURE 2 (continued)

| III. | Ibuprofen (Motrin/Rufen) | Fenoprofen (Nalfon) | Ketoprofen (Orudis) |
|---|---|---|---|
| Dosage, PO (Max./day) | 300, 400, 600 mg tid qid (2.4 g) | 200, 300, 600 mg tid qid (3.2 g) | 50, 75 mg tid qid (300 mg) |
| % Absorbed | 100% | 80% | 100% |
| Max. Blood Level | 2 h | 2 h | 30 - 120 min |
| Half-life (Multiple Dose) | 1-3 h (?) | 2.5 h (?) | 3 - 5 h (?) |
| Antiinfl. Onset (Peak Effect) | 6 d (1-2 w) | 2-3 d (2-3 w) | 5-10 d (1-2 w) |
| Analgesic | Good | Good | Good |
| Antigout | Symptomatic only | Symptomatic only | Not recommended |
| Food Effect on Absorption | Decr. absorption | Decr. absorption | Recommended with food; decr. absorp. |
| Metabolism | Hepatic | Hepatic | Hepatic |
| Excretion | Renal: 60% Fecal: 30% | Renal: 90% Fecal: 2% | Renal: 60% Fecal: 40% |
| Peptic Ulcer | 1:50 | 1:500 | 1:50 |
| GI Bleeding | 1:250 | 1:500 | 1:100 |
| GI Distress | 1:5 | 1:4 | 1:9 |

| | | | |
|---|---|---|---|
| Headache | 1:20 | 1:5 | 1:30 |
| Drowsiness | 1:50 | 1:7 | 1:100 |
| Tinnitis | 1:35 | 1:5 | 1:9 |
| Hepatotoxic | Rare | Rare | 1:6 |
| Nephrotoxic | 1:160 | Rare | Rare |
| Edema | 1:50 | 1:3 | 1:50 |
| Granulocytopenia | Rare | Rare | Rare |
| Other | Amblyopia: rare | — | — |
| Skin Reactions | 1:20 | 1:10 | 1:100 |
| Photosensitivity | Rare | Rare | Rare |
| Binding to Blood Proteins | 99% | 99% | 99% |
| Miscellaneous | Contraindicated for ASA "sensitive" individuals, children & in pregnancy. Available OTC (200 mg) as Advil, Nuprin, etc. Excellent OTC analgesic/antipyretic. Good shelf life. No potential for acetaminophen-like poisoning. | Contraindicated for ASA "sensitive" individuals, children & in pregnancy. Avoid in patients with genitourinary problems. | Antibradykinin capacity. Contraindicated for ASA "sensitive" individuals & children. |

FIGURE 2 (continued)

| IV. | Suprofen (Suprol) | Flurbiprofen (Ansaid) | Benoxaprofen (Oroflex) |
|---|---|---|---|
| Dosage, PO (Max./day) | 200 mg tid qid (800 mg) | 50, 100 mg bid tid qid (300 mg) | 400-600 mg once daily (1 g) |
| % Absorbed | 90% | GT 90% | ? |
| Max. Blood Level | 1 h | 0.5-4 h | 4 h |
| Half-life (Multiple Dose) | 2-4 h (?) | 5.7 h (6-12 h) | 25-32 h (?) |
| Antiinfl. Onset (Peak Effect) | 5-10 d (1-2 w) | 5-10 d (2-3 w) | 7-14 d (2 w) |
| Analgesic | Good | Good | Good |
| Antigout | Not recommended | Not recommended | Not recommended |
| Food Effect on Absorption | Decr. absorption | Slows absorption | Slows absorption |
| Metabolism | Hepatic | Hepatic | Hepatic |
| Excretion | Renal: 85-95% Fecal: 5-10% | Renal: 70% Fecal: 30% | Renal: 60% Fecal: 30% |
| Peptic Ulcer | 1:60 | 1:1000 | LT 1:100 |
| GI Bleeding | 1:60 | 1:35 | LT 1:100 |
| GI Distress | 1:7 | 1:14 | 1:10 |

| | | | |
|---|---|---|---|
| Headache | 1:30 | 1:20 | LT 1:100 |
| Drowsiness | 1:30 | ? | LT 1:100 |
| Tinnitus | 1:200 | Reported | 1:16 |
| Hepatotoxic | 1:60 | 1:7 | 1:600 (Cholestatic jaundice) |
| Nephrotoxic | 1:00 (Flank pain) | Rare | Rare |
| Edema | 1:20 | 1:7 | Rare |
| Granulocytopenia | Rare | ? | Rare |
| Other | Diarrhea: 1:10 | Vision disturbances reported | — |
| Skin Reactions | 1:50 | 1:150 | 1:5 (Serious) |
| Photosensitivity | Rare | Rare | 1:5 (Phototoxic) |
| Binding to Blood Proteins | 99% | GT 90% | 98% |
| Miscellaneous | 1:5 Discontinue due to side effects. Never initial therapy. Contraindicated for ASA "sensitive" patients. 1:14 have decr. visual acuity. Antibradykinin. Decr. platelet adhesion. | 1:10 Discontinue due to side effects. Never initial therapy. Contraindicated for ASA "sensitive" patients. | 1:150 Serious toxic response; now off market. Lipooxygenase inhibitor. Inc. proteoglycan synthesis. Chemotaxis inhibitor. Here only as reference. Good ad campaigns do not assure good drugs. |

FIGURE 2 (continued)

| V. | Naproxen (Naprosyn) | Diclofenac (Voltaren) | Piroxicam (Feldene) |
|---|---|---|---|
| Dosage, PO (Max./day) | 250,375, 500 mg bid (1 g) | 25, 50, 75 mg bid tid (200 mg) | 10, 20 mg once daily (20 mg) |
| % Absorbed | 100% | GT 90% | ? |
| Max. Blood Level | 2-4 h | 30 min | 3-5 h |
| Half-life (Multiple Dose) | 12-15 h (?) | 1-2 h (?) | 30 h (?) |
| Antiinfl. Onset (Peak Effect) | 14 d (2-4 w) | 2-4 d (2-3 w) | 7-14 d (2-3 w) |
| Analgesic | Good | Good | Good |
| Antigout | Not recommended | Not recommended | Symptomatic only |
| Food Effect on Absorption | Decr. absorption | Slows absorption (60% first-pass metabolism) | ? |
| Metabolism | Hepatic | Hepatic | Hepatic |
| Excretion | Renal: 95% Fecal: 2% | Renal: 50% Fecal: 50% | Renal: 60% Fecal: 30% |
| Peptic Ulcer | 1:60 | 1:500 | 1:130 |
| GI Bleeding | 1:100 | 1:70 | 1:100 |
| GI Distress | 1:6 | 1:5 | 1:5 |

| | | | |
|---|---|---|---|
| Headache<br>Drowsiness<br>Tinnitus | 1:10<br>1:12<br>1:10 | 1:15<br>?<br>Rare | 1:50<br>Rare<br>Rare |
| Hepatotoxic<br>Nephrotoxic<br>Edema<br>Granulocytopenia<br>Other | 1:100<br>Rare<br>1:10<br>Rare<br>— | 1:50<br>Rare<br>Reported<br>Rare<br>Dizzyness: 1:30 | 1:6<br>Nephritis reptd.<br>1:50<br>LT 1:100<br>— |
| Skin Reactions<br>Photosensitivity<br>Binding to Blood Proteins | 1:10<br>Rare<br>99% | LT 1:100<br>Rare<br>100% | 1:50<br>Rare<br>90% |
| Miscellaneous | Contraindicated for ASA "sensitive" individuals & any with impaired renal function. Hepatitis potential. | 1:30 Discontinue due to side effects. Contraindicated for ASA "sensitive" patients. Will incr. lithium levels. Enteric capsules available. | Contraindicated for ASA "sensitive" individuals & any with liver/renal dysfunction, especially patients GT 60 y of age. Never initial therapy. Patients must be carefully monitored. |

trade name and never more than two trade names per drug). The students are told a helpful "trick" to use whenever they are studying: the generic/trade names of each drug should always be thought of and used as one word; i.e., one compares ibuprofen/Motrin® with indomethacin/Indocin®. Ibuprofen/Motrin is used as one word, with indomethacin/Indocin being another single word. This constant pairing truly binds both generic and trade names at a single storage spot in the central nervous system memory banks. If one can remember ibuprofen, then the trade name Motrin automatically follows. To check out that the professor was not expecting more of them than of himself, it was common practice for several years for certain students to call out "ibuprofen?" as we passed in the halls to get back a reply of "Motrin! and what's indomethacin?" This is a good example of mutual reinforcement of Level 1 data.

It should be noted that the author's teacher/course evaluations (normally high) plummeted when Bloom-based testing was instituted in 1974. Initially, the students believed themselves to be under attack and unfairly persecuted—the equivalent of professorial sadism. In spite of the evaluation scores and their protests, this teaching approach was persistently continued. To provide insight, Addendum B was developed with its accompanying lecture. With time, the students realized that studying for Therapeutics I and Therapeutics II had become much easier. Then reports came back that the California State Board *did* use a similar style of testing.

Finally, positive feedback of the best kind began to percolate down to the student body from University of the Pacific graduates who were operating professionally in situations involving critical monitoring of patient drug records and active decision making. Drug-related problems *were* being solved in practice in a manner beneficial to a specific patient's health. It *did* feel good to contribute in a meaningful way! It *was* good for the professional ego to be recognized by others on the health care team as "productive" rather than "passive."

These appreciations from practicing pharmacists slowly led the students to an apprehensive but resigned acceptance of the course and its method of teaching. Some wry humor even surfaced when the 1989 graduating class presented the professor with its "official" Photographic Memory Award (a suitably engraved certificate

suitable for framing). Time after time in lecture, it had been emphasized that a photographic memory will not guarantee success on a Bloom-designed test: it is the learned ability to recognize what is important and the ability to use facts in a productive manner. Therefore, the award recognized the author's ability to excel at the lowest level of cognitive skill—a gentle insult, which is, of course, just what pregraduation "awards" to faculty are all about. The award is certainly evidence that teaching/testing in the Bloom manner had become part of that class's collective consciousness. The author received a more respectable honor when he was given the 1984 All-University Distinguished Faculty Award, as decided by an all-university committee of peers. While not privy to all the factors considered for this award, the first words of the citation (again, suitably engraved and suitable for framing) recognize the quality of his undergraduate and graduate teaching.

In summary, the author still remains convinced at the time of his retirement that testing procedures do dictate the way that students study a course and acquire cognitive skills. He is also convinced that testing for higher levels of cognitive competence does affect in a positive way students' abilities and their willingness to problem solve after graduation. For success, Bloom-type teaching/testing must be done in a consistent manner that has been well defined for the students. This requires both patience and persistence on the part of the professor.

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## ADDENDUM A

Section on competency-based education from the *University of the Pacific School of Pharmacy Handbook*. Brackets are used here to indicate updating of the original 1979 document or to record a note by the author.

### 5:5 *Competency-based teaching and testing*

A definition of professional competency in pharmacy was prepared January 30, 1970 by the California State Board of Pharmacy *Ad Hoc* Committee on the Licensure Examination (1). Subsequently, this definition was written into State of California law and the State Board of Pharmacy examination is constructed question-by-question to test these specific competencies. [The original statement of competency was updated by the Board in March 1983.]

The most efficient method for a professional school to ensure

competency for its graduates is the careful formulation of educational goals and behavioral objectives. The following readings are recommended for faculty who are unfamiliar with the concepts and the methodology (2-4). Most of the arguments against competency-based curricula have been answered effectively by Dobbert (5).

In 1972, the faculty of the University of the Pacific School of Pharmacy voted to adopt a competency-based curriculum; however, it was not until 1976 that the School developed and adopted a guiding list of terminal behavioral objectives for the Pharm.D. program (6). [Note: The original list was updated by the school in 1987.] At the present time, all required Pharm.D. courses must specifically contribute to these terminal objectives. To ensure that this is done, all courses must list their individualized goals and behavioral objectives as part of the course syllabus. Competencies fall into four educational domains:

(*i*) *Cognitive*: The acquisition and use of facts.
(*ii*) *Affective*: The acquisition of attitudinal concepts and the development of personal/professional values.
(*iii*) *Psychomotor*: The acquisition of manipulative skills involving motor co-ordination.
(*iv*) *Experiential*: The integration of all previous domains in real-life situations leading to the development of new personal/professional life styles.

Therefore, testing for competency must always reflect the special demands imposed by the domain within which the competency is located. An examination in physiology properly may lie wholly within the cognitive domain for a pharmacy student, while an examination in pharmaceutics may involve both the cognitive and psychomotor domains. A meaningful exam for competency in clinical pharmacy undoubtedly must involve the experiential domain—a cognitive examination will not suffice to prove competency even though the examination is skillfully prepared.

Most of the basic science courses in the School of Pharmacy strive to teach skills involving the cognitive domain. Teaching within the cognitive domain can be conducted at several levels:

(*i*) *Knowledge*: Simple recall of facts.
(*ii*) *Comprehension*: The ability to interpolate, extrapolate and translate facts.
(*iii*) *Application*: Problem-solving using facts.
(*iv*) *Analysis*: Developing relationships from "random" facts.
(*v*) *Synthesis*: Extrapolating from perceived relationships.
(*vi*) *Evaluation*: Ability to integrate all previous levels and come up with a summative judgement of validity or nonvalidity.

All cognitive levels can be tested using multiple-choice questions. If a course is to develop a problem-solving attitude, then most of the questions on a test should be written for levels (*iii*) to (*vi*). Each level requires the skills of the previous levels, so it is destructive of a student's potential to test only at levels (*i*) through (*iii*).

School of Pharmacy faculty who are unfamiliar with the concepts of testing in the cognitive domain should consult Bloom (7). This book is used by the California State Board of Pharmacy to prepare the licensure examination. They are restricted by law to a machine-scored test. [Note: There is now a written section designed to document written language communication ability.]

Definitive publications on the other domains have been written and should be consulted as they apply to the testing of terminal competencies (8-10). It would be very difficult for a student to challenge legally the validity of a test if it has been prepared in accord with the cited references (7-10).

## ADDENDUM B

Examples of test question style as attached to the course syllabus. [Answers are indicated for this paper.]

*Levels of Cognitive Evaluation*

1. *Knowledge*: Regurgitation of a specific fact
   [a] A physician wants to prescribe a generic form of Benadryl and asks you for the generic name. You correctly reply:

a. diphenhydramine
b. tripelennamine
c. pyrilamine
d. brompheniramine
e. carbinoxamine

2. *Comprehension*: Remembering and using relationships
In regard to the italicized parameter, use the following KEY to compare Drug X relative to Drug Y.

KEY: a. Drug X > Drug Y
b. Drug X < Drug Y
c. Drug X = Drug Y
d. Neither Drug X nor Drug Y is active

[b] *Capacity to relax gastrointestinal smooth muscle*: Drug X: meperidine; Drug Y: diphenoxylate
[b] *Intrinsic activity relative to analgesia*: Drug X: naloxone; Drug Y: nalorphine
[a] *Affinity relative to central analgesic receptors*: Drug X: methadone; Drug Y: codeine
[b] *Capacity to completely block the cough reflex*: Drug X: carbetapentane; Drug Y: codeine
[a] *Capacity for respiratory depression*: Drug X: morphine; Drug Y: meperidine

3. *Application*: Bringing to bear a generalization or fundamental principle
[c] The patient has a sharp repetitive cough of allergic origin. The source of the allergy has been defined and removed from the patient's environment. The cough has caused enough tissue irritation to be self-perpetuating. The cough is nonproductive since the sputum is quite thick. Perhaps the best therapeutic choice would be:
a. codeine + guaifenesin
b. diphenhydramine + guaifenesin
c. noscapine + guaifenesin
d. ephedrine + guaifenesin
e. corticosteroid + guaifenesin

4. *Analysis*: Breaking down a problem into the relevant parts
Keeping in mind that the specified three adverse reactions in each instance can characterize only one drug, associate the individual drugs in the KEY with their respective adverse reaction profile.

KEY: a. Zyloprim
b. Aspirin
c. Arlef
d. Butazolidin
e. Acetanilid

[d] *Chronic therapy*: gastric ulceration, hypertension, sodium-retention edema

[b] *Chronic therapy*: gastric ulceration, tinnitus, prolongation of prothrombin time

[e] *Chronic therapy*: hemolytic anemia, papillary necrosis, hepatotoxicity

[a] *Chronic therapy*: renal calculi, cataract formation, hepatotoxicity

[c] *Chronic therapy*: diarrhea, bone marrow depression, exacerbation of asthma

[d] *Chronic therapy*: euphoria, poor wound healing, ankle edema

5. *Synthesis*: Putting individual facts together to form an understandable whole.

Ideal properties for each class can be defined considering the therapeutic intent and fundamental physiological and pathological principles. For each of the following "ideal" criteria for an antihypertensive agent (essential hypertension, chronic therapy), compare the relative merits of Apresoline and Capoten using the KEY specified.

KEY: a. Only Apresoline satisfies the specified criterion.
b. Only Capoten satisfies the specified criterion.
c. Both Apresoline and Capoten satisfy the specified criterion.
d. Neither Apresoline nor Capoten satisfies the specified criterion.

[b] Have a sustained effect so that a patient can miss at least two days of medication without a return of hypertension

[a] Be predictably well absorbed from the gastrointestinal tract

[c] Maintain the minute volume of the heart within normal limits

[b] Not produce either postural or orthostatic hypotension

[c] Not develop tolerance to the antihypertensive effect upon chronic administration

[b] Not cause rebound hypertension and tachycardia if the dosage is stopped

[c] Not produce hypersensitive *alpha* adrenergic receptors or hypersensitive *beta* adrenergic receptors

[c] After start of therapy, the onset of the antihypertensive effect should be rapid (i.e., within two days).

[c] A patient has had a death in the family (stress) and predictably this has increased his susceptibility to allergens that he would normally tolerate (house dust). He is now experiencing episodes of acute asthma that are frightening. He has hypertension so his physician is very reluctant to consider any sympathomimetic—even those selective for bronchial muscle. The individual also has urinary hesitancy which is aggravated when he takes Benadryl. The physician insists on prescribing an effective antihistamine, so perhaps the best recommendation would be:

a. carbinoxamine
b. cyproheptadine
c. hydroxyzine
d. meclizine
e. antazoline

6. *Evaluation*: Making a holistic judgment
Carefully read the article by Leonards and Levy which appeared in *Clinical Pharmacology and Therapeutics*, 10: 571-5, 1969. Then answer the following question:

[d] Which of the following conclusions is specifically supported by this paper?

a. Formulations containing aspirin, acetaminophen, and caffeine are more effective as analgesics than formulations containing only aspirin as the active ingredient.
b. Highly buffered aspirin formulations are more effective as analgesics than formulations containing only unbuffered aspirin.
c. Aspirin when administered as a buffered solution is more rapidly effective than aspirin given as a buffered tablet.
d. Aspirin given orally as a buffered liquid yields higher plasma levels of salicylic acid than a commercially available tablet formulation of aspirin with an unspecified disintegration time.
e. Some gastrointestinal blood loss must be expected whenever one consumes a solid or a liquid formulation containing aspirin.

## ADDENDUM C

Examples of actual test questions based on the supplemental data sheet illustrated in Figure 1.

November 3, 1988

*Comparative Pharmacology/Toxicology*: Use the specified KEY.

KEY: a. Greater with auranofin than with gold sodium thiomalate
b. Greater with gold sodium thiomalate than with auranofin
c. About equivalent for both auranofin and gold sodium thiomalate

[a] 26. Percentage absorption from the gastrointestinal tract
[b] 27. Incidence of leukopenia on chronic therapy
[b] 28. Incidence of vasomotor collapse at start of therapy
[c] 29. Incidence of proteinuria
[a] 30. Incidence of a laxative effect
[b] 31. Incidence of a decreased platelet count
[c] 32. Time for steady-state blood levels to be reached
[b] 33. Percentage urinary excretion as gold
[c] 34. Incidence of stomatitis

March 10, 1989

*Comparative Toxicology*: Compare the two drugs in the KEY as to which would be preferred from the patient's point of view considering the potential for inducing the italicized adverse drug reaction (ADR):

KEY: a. Ridaura is clearly better than Myochrysine.
b. Myochrysine is clearly better than Ridaura.
c. Ridaura and Myochrysine are generally considered to be equivalent in this regard.
d. Neither Ridaura nor Myochrysine has ever been reported to cause the specified ADR.

[c] 11. *Stomatitis*
[a] 12. *Nitritoid reaction*
[a] 13. *Thrombocytopenia*
[b] 14. *Increased bowel frequency*
[c] 15. *Proteinuria*

## ADDENDUM D

Examples of actual test questions based on the supplemental data sheets illustrated in Figure 2.

October 5, 1988

*Comparative Pharmacology/Toxicology*: Note the best or worst product (as requested) based on respective pharmacologic/toxicologic data provided in the lectures. Use the KEY specified.

KEY: a. ibuprofen
b. fenoprofen
c. naproxen
d. meclofenamate
e. indomethacin

[c] 73. The product preferred for a patient with a history of poor drug compliance

[e] 74. The product preferred because of its high potency as an anti-inflammatory drug when given orally

[e] 75. The product recommended for a patient with a long history of allergy and drug-related skin reactions

[c] 76. The product considered least desirable for a patient with a history of liver disease

[d] 77. The product indicated for the patient who must always remain mentally alert at his job

[b] 78. The product with the greatest potential for cranial nerve toxicity

[a] 79. The product with the most rapid peak anti-inflammatory effect

[a] 80. The product that should be avoided in a patient with compromised kidney function

*Comparative Pharmacology/Toxicology*: As above but use the new KEY below:

KEY: a. tolmetin
b. piroxicam
c. sulindac
d. fenoprofen
e. naproxen

[d] 81. The product recommended for a patient with a past history of gastric and duodenal ulcers
[b] 82. The product with the least incidence of tinnitus
[b] 83. The product preferred for a patient with a history of drug-induced skin reactions including urticaria
[e] 84. The product to be avoided in a patient with compromised liver function
[b] 85. The most potent NSAID of the compounds listed
[b] 86. Useful in patients who are not compliant in taking multiple doses of a drug per day
[a] 87. The product with the highest incidence of the so-called "frontal headache"
[b] 88. The product with the least incidence of drug-induced headache
[b] 89. Recommended for the patient who must remain both alert and noneuphoric while on the job
[b] 90. Not recommended for elderly patients with even the slightest suggestion of kidney impairment
[c] 91. Generally agreed to be the agent with the least potential for reduced kidney function since the drug is not found in the kidney in an active form

February 17, 1989

*Comparative Toxicology*: Compare the adverse drug reaction incidence for the specified NSAID drugs using the following KEY. Presume that all drugs are being given chronically for rheumatoid arthritis at their recommended oral doses.

KEY:
a. Drugs A and B both have the potential to cause the ADR but Drug A is clearly preferable from the patient's point of view.
b. Drugs A and B both have the potential to cause the ADR but Drug B is clearly preferable from the patient's point of view.
c. Drugs A and B both have the potential to cause the ADR and from the patient's point of view they are roughly equivalent.
d. Drug A cannot cause the specified ADR so it is clearly the superior drug for the patient.

e. Drug B cannot cause the specified ADR so it is clearly the superior drug for the patient.

Peptic ulcer:
[a] 70. Drug A: aspirin — Drug B: ketoprofen
[b] 71. Drug A: Motrin — Drug B: Ansaid

Diarrhea:
[b] 72. Drug A: meclofenamic acid — Drug B: sulindac
[b] 73. Drug A: Suprol — Drug B: Motrin

Headache:
[b] 74. Drug A: indomethacin — Drug B: piroxicam
[c] 75. Drug A: Tolectin — Drug B: Nalfon

Leg and ankle edema:
[a] 76. Drug A: sulindac — Drug B: phenylbutazone
[b] 77. Drug A: Naprosyn — Drug B: Clinoril

Cranial nerve toxicity:
[b] 78. Drug A: ketoprofen — Drug B: suprofen
[a] 79. Drug A: Voltaren — Drug B: Naprosyn

Abnormal liver function tests:
[c] 80. Drug A: aspirin — Drug B: piroxicam
[b] 81. Drug A: Orudis — Drug B: Motrin

Decreased visual acuity:
[a] 82. Drug A: aspirin — Drug B: suprofen
[a] 83. Indocin — Drug B: Ansaid

Aplastic anemia:
[b] 84. Drug A: phenylbutazone — Drug B: indomethacin
[c] 85. Drug A: Suprol — Drug B: Orudis

Gastrointestinal distress:
[b] 86. Drug A: tolmetin — Drug B: flurbiprofen
[c] 87. Drug A: Suprol — Drug B: Naprosyn

[a] 88. Which of the following drugs can cause a chronic toxicity syndrome that tends to mimic the "natural" symptoms of aging?
a. phenylbutazone
b. indomethacin
c. piroxicam
d. meclofenamate
e. ibuprofen

[a] 89. Theoretically, the dose must be decreased by 28% for which of the following drugs in order to get an effect equivalent to 650 mg of aspirin orally?

a. salsalate
b. acetylsalicylic acid
c. choline salicylate
d. magnesium salicylate
e. choline magnesium trisalicylate